*Received:December 26, 2025*
*Accepted: January 10, 2026*

*Acad Med J 2026;6(1):30-40*
UDC: 616.132.2:616.12-073.432.19
DOI:
*Original article*

# COMPARATIVE VALUE OF DIASTOLIC AND SYSTOLIC RESPONSES DURING PHARMACOLOGIC STRESS ECHOCARDIOGRAPHY FOR THE DETECTION OF OBSTRUCTIVE CORONARY ARTERY DISEASE

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**Abstract**

**Introduction:** Stress echocardiography (SE) is an established method for detecting coronary artery disease (CAD), primarily based on systolic parameters. Although diastolic dysfunction may occur early during ischemia, the diagnostic value of diastolic assessment during pharmacologic SE remains uncertain.

**Aim:** To evaluate the frequency of diastolic dysfunction during pharmacologic SE and its association with angiographically confirmed obstructive CAD, in comparison with conventional systolic parameters.

**Matrial and methods:** We prospectively evaluated 61 patients who underwent dipyridamole or dobutamine SE followed by coronary angiography. Systolic parameters included wall motion score index (WMSI), left ventricular ejection fraction, systolic tissue Doppler velocity (s′TDI), and global longitudinal strain (GLS%). Diastolic assessment included transmitral inflow velocities, deceleration time (DT), isovolumic relaxation time (IVRT), tissue Doppler e′ velocities, E/e′ ratio, and left atrial volume. Parameters were measured at rest and peak stress, and delta values were calculated.

**Results:** Obstructive CAD was present in 20 patients (32.8%). Systolic parameters, particularly WMSI, significantly differentiated patients with and without CAD. ΔWMSI and Δs′TDI were significantly greater in patients with CAD, whereas GLS% showed non-significant, opposite trends between groups. Diastolic parameters demonstrated expected physiological stress responses, including shortening of DT and IVRT and increased e′ velocities, but neither peak values nor stress-induced changes differed significantly between patients with and without CAD. No diastolic delta parameter provided incremental diagnostic value for CAD detection.

**Conclusion:** During pharmacologic SE, conventional systolic parameters remain superior for identifying obstructive CAD. Diastolic indices appear largely influenced by heart rate and loading conditions and do not improve diagnostic discrimination beyond standard systolic assessment.

***Keywords:*** ctress echocardiography, diastolic function, coronary artery disease, wall motion score index, global longitudinal strain, pharmacologic stress

## Introduction

Coronary artery disease (CAD) accounts for more than nine million deaths worldwide and remains the leading cause of premature mortality[1]. Patients with CAD typically present with exertional chest pain or dyspnea; however, a substantial proportion of symptomatic individuals ultimately do not have obstructive CAD, necessitating further diagnostic evaluation to refine risk stratification[2,3].

Stress echocardiography (SE) is a widely used, cost-effective technique for detecting CAD[4-7]. Despite its broad clinical application, systematic assessment of diastolic function during SE, particularly its diagnostic relevance for CAD, has not been fully integrated into routine practice, which traditionally focuses on systolic parameters and wall-motion abnormalities (WMA) [5,8]. Given that left ventricular (LV) diastolic dysfunction occurs earlier than systolic impairment during myocardial ischemia, and that stress can unmask abnormalities not evident at rest, several studies have reported associations between stress-induced changes in diastolic indices and presence and severity of CAD[9-13]. While diastolic assessment is most often performed during exercise SE, its feasibility and diagnostic value during pharmacologic SE remain less well defined[14].

The aim of this study was to determine the frequency of diastolic dysfunction during pharmacologic SE and to evaluate its association with angiographically confirmed obstructive CAD, in comparison with conventional systolic parameters.

## Material and methods

*Study population*

We prospectively evaluated 105 consecutive patients referred for *dipyridamole* or *dobutamine* SE at the University Clinic for Cardiology in Skopje. Of these, 61 patients who underwent coronary angiography within several weeks after SE were included in the analysis. Indications for SE included chest pain, dyspnea, angina-like symptoms, or inconclusive exercise stress testing. Demographic characteristics, cardiovascular risk factors, comorbidities, symptoms, and medication use were recorded prior to testing.

Exclusion criteria were: previous coronary artery bypass grafting and/or significant valvular disease, elevated troponin during chest pain, severe rhythm and/or conduction abnormalities, significant left ventricular hypertrophy, signs of heart failure and/or LV ejection fraction < 45%, uncontrolled arterial hypertension, comorbid conditions likely to affect test results or limit life expectancy, and a resting wall motion score index (WMSI) >1.2. The study protocol was approved by the Ethics Committee of the Faculty of Medicine, Ss. Cyril and Methodius University in Skopje, and written informed consent was obtained from all participants.

*Stress echocardiography protocol*

Echocardiographic examinations were performed using a GE Vivid 7 system, with offline analysis using EchoPac software (GE Healthcare). All measurements were obtained according to current echocardiographic society recommendations[4-6].

An accelerated high-dose *dipyridamole* protocol was applied (0.84 mg/kg over 6 min), with routine administration of *aminophylline* (up to 240 mg) 5 minutes after test completion or earlier if clinically indicated. *Dobutamine* was administered intravenously starting at 5 μg/kg/min, with dose increments every 3 minutes to 10, 20, 30, and 40 μg/kg/min. Continuous two-dimensional echocardiography and 12-lead ECG monitoring were performed, and blood pressure was measured every 3 minutes.

Echocardiographic images were semi-quantitatively assessed using the 17-segment, 4-point scoring system. The wall motion score index (WMSI) was calculated by dividing the sum of segment scores by the number of interpretable segments. A SE test was considered normal if no new or worsening wall motion abnormality (WMA) was detected, and positive if ≥2 adjacent segments within the same vascular territory demonstrated a ≥1-grade increase in WMA at peak stress.

Left ventricular global longitudinal strain (GLS%) was automatically calculated as the average peak systolic longitudinal strain derived from the three apical views using dedicated software. Images for strain analysis were obtained outside the active SE protocol. All images were recorded at frame rates >50 frames per second, and only segments accepted by both the automated system and the operator were included. All examinations were interpreted by the same experienced cardiologist.

*Assessment of diastolic function during stress*

Diastolic evaluation included left atrial volume (LAV) and indexed LAV (LAVI), mitral early and late diastolic filling velocities (E and A), E/A ratio, deceleration time (DT), and isovolumic relaxation time (IVRT). Tissue Doppler imaging (TDI) was used in apical views to measure early (e') and late (a') diastolic annular velocities at septal and lateral sites, with calculation of averaged values. Pulmonary artery systolic pressure was estimated from tricuspid regurgitation velocity. Diastolic parameters were recorded at rest and at peak stress, in accordance with professional society recommendations[15].

*Coronary angiography*

Coronary angiography was performed in all patients who underwent stress echocardiography with evidence of inducible ischemia, within 4-6 weeks after the echocardiographic examination. The presence of CAD was defined as a luminal coronary stenosis ≥50% in any major epicardial coronary artery. According to coronary angiography findings, 20 patients (32.8%) had obstructive CAD, while 41 patients (67.2%) had no significant coronary stenosis. Among patients with obstructive CAD, single-vessel disease was present in 6 patients (30.0%), whereas 14 patients (70.0%) had multivessel disease. Regarding the distribution of affected coronary arteries, the left anterior descending artery was involved in 32 cases (52.5%), the right coronary artery in 21 cases (34.4%), and the circumflex artery in 13 cases (21.3%). In the group without obstructive CAD, coronary atherosclerotic plaque without significant stenosis was detected in 16 patients (39%).

*Statistical analysis*

Categorical variables are presented as percentages, and continuous variables as mean ±SD. Comparisons between patients with and without CAD were performed using the Student's t-test for continuous variables and Pearson's chi-square test for categorical variables. Comparisons between rest and peak stress within the same group were performed using the paired-samples t-test. Correlations between echocardiographic parameters and hemodynamic variables were assessed using Pearson's correlation analysis.

All statistical analysis were performed using SPSS, version 25.0 (IBM SPSS, Inc., Chicago, Illinois) with a p-value ≤ 0.05 considered statistically significant.

## Results

*Clinical characteristics*

The mean age of the study population was 59.93±7.87 years, with a nearly equal distribution of men and women (47.5% and 52.5%, respectively). Hypertension was the most common comorbidity (78.7%), while prior CAD was documented in only one patient. The

majority of patients were referred for SE because of chest pain (78.7%), while others were presented with dyspnea or angina-like symptoms.

Their clinical characteristics are presented in Table 1. Baseline characteristics were largely comparable between groups; however, patients without CAD showed a trend toward higher body mass index, and included a greater proportion of women. *Dobutamine* was used significantly more often in patients with CAD, whereas *dipyridamole* predominated in those without CAD (p=0.0001).

**Table 1.** Clinical characteristics of study population

| Variable | CAD present n=20 | CAD absent n=41 | p |
|---|---|---|---|
| Age (y) | 59.07±7.67 | 61.70±8.17 | 0.224 |
| Men/women (%) | 55.0/45.0 | 43.9/56.1 | 0.294 |
| BMI (kg/m2) | 28.09±3.97 | 30.28±4.55 | 0.059 |
| History of hypertension (n/%) | 18/90.0 | 30/73.2 | 0.118 |
| Diabetes | 13/65.0 | 16/39.0 | 0.122 |
| Dyslipidemia | 17/85.0 | 30/73.2 | 0.244 |
| History of smoking | 6/30.01 | 6/39.0 | 0.346 |
| ***Medication (n%)*** | | | |
| ACE inhibitors/ARBs | 19/95.0 | 30/73.2 | 0.041 |
| Beta blockers | 12/60.0 | 26/63.4 | 0.506 |
| Statins | 19/95.0 | 36/87.8 | 0.351 |
| ***Type of stressor (n/%)*** | | | |
| Dipyridamole | 7/35.0 | 33/80.5 | 0.0001 |
| Dobutamine | 13/65.0 | 8/19.5 | |

ACE=angiotensin-converting enzyme; ARB=angiotensin receptor blocker

*Stress echocardiography*

Systolic blood pressure (BP) did not change significantly from rest to peak stress in either group. Patients without CAD demonstrated a slight, non-significant decrease (137.80± 16.04 to 135.98±18.88, p=0.487), whereas those with CAD showed a non-significant increase (136.50±19.80 to 140.00±21.76, p=0.089). Diastolic BP decreased during stress in both groups, significantly in patients without CAD (p=0.004) and non-significantly in those with CAD (p=0.185).

Heart rate (HR) increased significantly with stress in both groups (without CAD: 74.34 ±13.08, to 95.12±16.81, p=0.0001; with CAD: 76.50±8.75 to 110.00±20.87, p=0.018).

Echocardiographic findings at rest and peak stress are summarized in Table 2. In both groups, ejection fraction (EF), indexed stroke volumes (SVI), and systolic tissue Doppler velocity (s'TDI) increased during stress. Wall motion score index (WMSI) was mildly elevated at rest in both groups. Notably, 22 patients (53.7%) had WMA already present at rest, with no significant difference between patients with and without CAD (p=0.680). WMSI showed a small worsening with borderline statistical significance (p=0.073) at peak stress only in patients with CAD. Global longitudinal strain (GLS%) was below the lower limit of normal at rest in both groups, more pronounced in those with CAD. During stress, GLS% exhibited minimal, non-significant changes, becoming slightly more negative (improved) in patients without CAD and slightly less negative (worsened) in those with CAD.

*Diastolic parameters during stress*

Left atrial volume (LAV) and indexed LAV (LAVi) increased slightly and non-significantly with stress in both groups. Early transmitral flow velocity (E) increased during stress in both groups, significantly in patients without CAD and with borderline significance in those with CAD. The E/A ratio increased slightly in patients without CAD and decreased slightly in patients with CAD, without reaching statistical significance in either group.

Deceleration time (DT) and isovolumetric relaxation time (IVRT) decreased significantly during stress in both groups. Tissue Doppler e' velocity (septal, lateral, and averaged) increased with stress in both groups. The E/e' ratio demonstrated small, non-significant changes at all measured sites in both groups.

**Table 2.** Echocardiographic characteristics of study population

| Parameters | Without CAD n=41 | | | With CAD n=20 | | |
|---|---|---|---|---|---|---|
| | Rest | Stress | p | Rest | Stress | p |
| ***Systolic parameters*** | | | | | | |
| EF% | 61.09±8.78 | 61.63±10.09 | 0.660 | 59.00±8.21 | 60.05±8.23 | 0.584 |
| SVI (ml/m2) | 42.77±10.54 | 49.52±12.15 | 0.0001 | 41.39±13.45 | 48.73±11.33 | 0.106 |
| WMSI | 1.10±0.14 | 1.08±0.11 | 0.294 | 1.12±0.10 | 1.21±0.20 | 0.074 |
| s'TDI (cm/s) | 7.62±1.31 | 8.36±1.68 | 0.001 | 7.62±1.38 | 9.72±2.69 | 0.0001 |
| GLS% | -16.18±3.10 | -16.40±3.79 | 0.571 | -14.42±2.71 | -14.28±3.61 | 0.833 |
| ***Diastolic parameters*** | | | | | | |
| LAV (ml) | 37.05±10.49 | 37.53±13.02 | 0.789 | 39.38±10.00 | 40.76±12.35 | 0.576 |
| LAVi (ml/m2) | 19.32±5.11 | 19.52±6.35 | 0.832 | 20.69±5.23 | 21.25±5.95 | 0.643 |
| E (cm/s) | 83.32±15.52 | 89.93±20.62 | 0.025 | 84.55±18.36 | 96.70±14.65 | 0.052 |
| E/A | 1.02±0.28 | 1.05±0.34 | 0.502 | 1.01±0.21 | 0.96±0.34 | 0.444 |
| DT (ms) | 197.44±49.01 | 151.24±51.23 | 0.0001 | 201.90±53.35 | 158.90±65.59 | 0.012 |
| IVRT (ms) | 85.02±15.98 | 65.78±9.64 | 0.0001 | 81.85±18.96 | 60.50±9.40 | 0.0001 |
| e's (cm/s) | 9.15±2.44 | 10.29±2.61 | 0.004 | 8.25±1.83 | 9.85±2.15 | 0.006 |
| e'l (cm/s) | 10.51±2.85 | 11.41±3.46 | 0.022 | 10.45±2.74 | 11.50±3.31 | 0.078 |
| e'av (cm/s) | 9.82±2.33 | 10.85±2.75 | 0.001 | 9.35±1.90 | 10.67±2.29 | 0.007 |
| E/e's | 9.58±2.57 | 9.21±3.05 | 0.447 | 10.84±4.02 | 10.22±2.46 | 0.534 |
| E/e'l | 8.51±2.89 | 8.54±2.76 | 0.949 | 8.58±2.97 | 8.61±3.62 | 0.976 |
| E/e'av | 8.86±2.47 | 8.88±2.66 | 0.976 | 9.39±2.92 | 9.41±2.65 | 0.980 |

EF=ejection fraction, SVI=indexed systolic volume, WMSI=wall motion score index, s'TDI=systolic tissue velocity, GLS=global longitudinal strain, HR=heart rate, LAV=left atrial volume, LAVi=indexed left atrial volume, E=early diastolic transmitral flow velocity, A=late diastolic transmitral flow velocity, E/A=ratio of early and late transmitral flow velocity, DT=deceleration time, IVRT=isovolumetric relaxation time, e'=early diastolic tissue velocity, E/e'=ratio of early diastolic transmitral velocity to early diastolic tissue velocity.

*Delta values*

Delta values (peak stress minus rest) are summarized in Table 3. Significant differences between groups were observed only for systolic parameters. ΔWMSI increased significantly in CAD group (p=0.017), and Δs'TDI showed a greater rise in patients with CAD (p=0.004), reflecting a hypercontractile response under *dobutamine*. Although the difference in ΔGLS% between groups did not reach statistical significance, patients with CAD exhibited positive (worsened) ΔGLS% values, whereas patients without CAD demonstrated negative (improved) values during stress. No significant differences were observed in any diastolic delta parameters between the groups.

When comparing patients with single-vessel *vs*. multi-vessel CAD, no significant differences were found for ΔWMSI and ΔGLS% (p=0.988 and p=0.930, respectively). Similarly, no significant differences in diastolic delta parameters were observed according to the number of involved vessels.

**Table 3.** Delta values of echocardiographic characteristics in study population

| Parameters | Without CAD n=41 | With CAD n=20 | p |
|---|---|---|---|
| ***Systolic parameters*** | | | |
| ΔEF% | 0.53±7.74 | 1.05±8.43 | 0.814 |
| ΔSVI (ml/m2) | 6.75±8.66 | 7.34±18.27 | 0.871 |
| ΔWMSI | -0.02±0.14 | 0.09±0.21 | **0.017** |
| Δs'TDI (cm/s) | 0.74±1.30 | 2.10±2.19 | **0.004** |
| ΔGLS% | -0.21±2.43 | 0.14±3.03 | 0.617 |
| ***Diastolic parameters*** | | | |
| ΔLAV (ml) | 0.48±11.55 | 1.28±10.15 | 0.794 |
| ΔLAVi (ml/m2) | 0.20±5.99 | 0.55±5.31 | 0.821 |
| ΔE (cm/s) | 6.60±18.17 | 12.15±26.23 | 0.340 |
| ΔE/A | 0.03±0.31 | -0.04±0.27 | 0.331 |
| ΔDT (ms) | -46.19±54.67 | -43.00±68.96 | 0.845 |
| ΔIVRT (ms) | -19.24±20.10 | -21.35±20.56 | 0.704 |
| Δe's (cm/s) | 1.14±2.57 | 1.60±2.32 | 0.508 |
| Δe'l (cm/s) | 0.90±2.60 | 1.05±2.52 | 0.835 |
| Δe'av (cm/s) | 1.02±1.91 | 1.32±1.94 | 0.569 |
| ΔE/e's | -0.36±3.04 | -0.62±4.42 | 0.789 |
| ΔE/e'l | 0.03±3.02 | 0.02±4.11 | 0.998 |
| ΔE/e'av | 0.01±2.57 | 0.02±3.60 | 0.992 |

**Δ=**Delta values (peak stress minus rest)

*Correlation analysis of diastolic parameters with BP and HR*

In patients with CAD, no significant correlations were observed between HR, BP, and delta diastolic parameters. In contrast, among patients without CAD, higher peak-stress HR correlated with: ΔDT (r=-0.370, p=0.006), ΔIVRT (r=-0.425, p=0.022), and Δe'av (r=0.315, p=0.045). No significant differences were observed between *dobutamine* and *dipyridamole* regarding diastolic responses.

## Discussion

### *Systolic indices during stress*

In this prospective study evaluating the additional diagnostic value of diastolic assessment during stress echocardiography, we found that systolic markers, particularly WMSI, remained the strongest discriminators between patients with and without angiographically confirmed CAD. In contrast, none of the evaluated diastolic parameters at peak stress, nor their stress-induced changes, differed significantly between groups. These findings suggest that diastolic indices measured during pharmacologic stress may have limited incremental value for improving the detection of obstructive CAD beyond standard systolic stress-echo evaluation.

As expected, patients with CAD demonstrated a pattern compatible with inducible ischemia, including a significant increase in WMSI. These results are consistent with established literature where regional wall-motion abnormalities remain the cornerstone for CAD detection during stress echocardiography and are essential for prognostic assessment[4-6,16]. Regarding GLS%, only mild and non-significant changes during stress in both groups were observed; however, the direction of change differed between groups, with negative (improved) values in patients without CAD and positive (worsened) values in those with CAD. This finding is in line with heterogeneous and sometimes inconclusive data reported in the literature. While earlier studies expressed some skepticism regarding the incremental diagnostic value of GLS% beyond visual wall-motion analysis for CAD detection, more recent studies have supported its use, particularly when combined with conventional parameters. Such combinations appear to provide synergistic diagnostic value, especially in intermediate-risk patients and in cases where

conventional SE findings are equivocal or uncertain[5,17-23]. Moreover, the study by Haddad *et al.,*[24] demonstrated that incorporated changes in WMSI and, in particular, GLS% during SE improved diagnostic performance for the detection of high-risk CAD, an approach also applied in our study.

However, several limitations may have influenced the lack of more robust GLS% findings and should be considered. GLS% analysis is highly dependent on optimal image quality, and its accuracy may be impaired in patients with suboptimal acoustic windows, particularly at high HR. In addition, vigorous myocardial contraction and altered loading conditions at higher HR may produce artificially more negative strain values, while reduced reliability of myocardial speckle-tracking may occur, highlighting the need for higher than usual frame rates[25]. Aggeli *et al.,*[18] reported superior diagnostic efficiency of two-dimensional strain for evaluation of the anterior coronary circulation compared to the posterior circulation, whereas Illardi *et al*.,[22] found that strain analysis during SE may improve prediction of LAD stenosis, while regional WMSI performs better in the presence of Cx and RCA stenosis, possibly due to limited visualization of myocardial segments supplied by these vessels. Furthermore, the EVAREST observational multicentre study[26], which evaluated real-world performance and accuracy of SE, emphasized that diagnostic accuracy may be affected by pre-existing regional WMA, as observed in our cohort. Such abnormalities may reflect the impact of *dobutamine* on post-systolic shortening, potentially masking impaired segmental contractile function and leading to misinterpretation during visual assessment. It should also be acknowledged that strain analysis lacks definitive and universally accepted cut-off values for diagnosing significant coronary artery stenosis.

With respect to mitral annular peak systolic velocity (s′TDI), several studies have reported reduced values during SE in patients with CAD[5,11,12,27], whereas in our study we observed a significant increase. This seemingly paradoxical finding may be explained by the fact that s′TDI reflects basal longitudinal function, whereas many ischemic lesions are predominantly apical or subendocardial and may not significantly reduce basal annular velocities until more severe ischemia develops. In addition, the global chronotropic and inotropic effects of *dobutamine* increase tissue velocities, and mechanical tethering or compensatory contraction of adjacent non-ischemic segments may preserve annular longitudinal velocities. A further explanation may involve the inotropic effect of low-dose *dobutamine* with heart-rate increase or a biphasic (viability-related) response at low-to-moderate *dobutamine* doses[5,28-30].

**Diastolic indices during stress**

Stress-induced changes in diastolic function were comparable in patients with and without CAD. Expected physiological responses were observed, included shortening of DT and IVRT and an increase in e′ velocities, reflecting enhanced myocardial relaxation during pharmacologic stimulation[9,7,14]. However, the magnitude of these changes did not differ according to the CAD status. Similarly, E/e′, a surrogate of LV filling pressure, remained stable during stress and did not discriminate between patients with or without CAD. Previous studies have shown that impaired early diastolic filling or abnormal relaxation during stress may accompany myocardial ischemia, reflecting delayed relaxation and increased LV stiffness[10,11,31]. However, more recent evidence suggests substantial heterogeneity of diastolic stress responses and significant overlap between patients with and without obstructive coronary lesions[32]. Our findings are consistent with this more recent evidence, indicating that diastolic indices during pharmacologic stress are strongly influenced by loading conditions, chronotropic effects, and heart rate–dependent shortening of diastole, which may obscure subtle ischemia-related abnormalities. This interpretation is supported by the observed correlations between peak heart rate and ΔDT/ΔIVRT, and Δe′ in patients without CAD, reflecting normal physiological

adaptation and reinforcing the concept that diastolic stress responses are predominantly preload- and HR-dependent rather than ischemia-specific under these testing conditions.

*Limitation of the study*

The main limitation of our study is the relatively small sample size, particularly the number of patients with angiographically confirmed CAD, which may have limited statistical power to detect subtle systolic and/or diastolic differences. In addition, a substantial proportion of patients with single-vessel disease, and a considerable number of patients without obstructive CAD had non-obstructive coronary plaque. The use of mixed pharmacologic stressors is another limitation. *Dipyridamole* was used predominantly in patients without CAD, whereas *dobutamine* was more frequently administered in patients with CAD. These agents produce distinct hemodynamic effects that may influence diastolic parameters differently. *Dipyridamole* induces coronary vasodilation rather than yielding a direct inotropic response, which may attenuate diastolic differences between groups. Relatively preserved LV systolic function in our population suggests that more advanced CAD or preexisting baseline diastolic dysfunction may be required to reveal more pronounced diastolic abnormalities during stress. Strain and tissue Doppler measurements were obtained outside the active stress protocol; although consistent with many laboratory practices, this approach may have reduced temporal sensitivity for detecting ischemia-related changes. Microvascular dysfunction was not assessed, despite its potential influence on diastolic responses independent of epicardial CAD. Although a substantial proportion of patients demonstrated positive ischemic responses during SE in the absence of obstructive coronary stenosis (INOCA), further diagnostic testing to investigate underlying mechanism was not performed, as this was beyond the scope of the study. Finally, diagnostic accuracy of SE may be enhanced by incorporating temporal and spatial characteristics of ischemia, including the number of ischemic segments, severity of induced ischemia, time of onset, and pharmacologic dose - parameters that were not systematically analyzed in the present study.

**Conclusion**

In this prospective study, systolic parameters, particularly wall motion score index, remained the most reliable markers for the detection of angiographically confirmed obstructive CAD during pharmacologic stress echocardiography. In contrast, diastolic parameters assessed at peak stress and their stress-induced changes did not differ significantly between patients with and without obstructive CAD and did not provide incremental diagnostic value beyond conventional systolic assessment.

These findings suggest that, under pharmacologic stress conditions, diastolic indices are predominantly influenced by heart rate and loading-dependent physiological responses, which may limit their ability to discriminate ischemia-related abnormalities. While diastolic assessment remains important for comprehensive cardiac evaluation, its routine use for the detection of obstructive CAD during pharmacologic stress echocardiography cannot be supported by our data.

*Conflict of interest statement.* None declared.

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