



REVIEW

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Tissue Doppler imaging for diagnosis of coronary artery disease: a systematic review and meta-analysis

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Abstract

Global and regional left ventricular (LV) systolic dysfunction is a marker of coronary artery disease (CAD), which is conventionally assessed using two-dimensional echocardiography. Tissue Doppler imaging (TDI) has emerged as an adjunct tool in the diagnosis of regional wall motion abnormalities from CAD. We performed a systematic review and meta-analysis to assess the efficacy of TDI indices in the diagnosis of CAD. We searched MEDLINE and the Cochrane Library for controlled studies comparing TDI measurements in those with and without CAD as confirmed by coronary angiography. Meta-analyses of mean differences in TDI velocities between these populations were performed. Screening of titles and abstracts followed by full-text screening identified 8 studies. At rest, TDI was associated with a significant decrease in the pooled maximum systolic velocity among CAD patients compared to those without CAD [mean difference (MD): -0.66; 95% confidence interval (CI): -0.98 to -0.34]. There were no significant differences in maximum early and late diastolic velocities. Post-stress, TDI was associated with a significant decrease in maximum early diastolic velocity (MD: -1.91; 95% CI: -2.74 to -1.09) and maximum late diastolic velocity (MD: -1.57; 95% CI: -2.95 to -0.18) among CAD patients compared to those without CAD. There was no significant difference in maximum systolic velocity post-stress. Our results suggest that TDI may have a role in the evaluation of CAD. Future studies should evaluate the incremental value of TDI velocities over LV ejection fraction and two dimensional wall motion analysis in the detection of CAD and assessment of its severity. (Word Count: 249)

Keywords: Systematic review, Meta-analysis, Tissue Doppler, Echocardiography, Coronary artery disease

Introduction

Coronary artery disease (CAD) is responsible for one out of every six deaths in the United States. Each year, approximately 800,000 Americans have a new coronary event resulting in approximately one death per minute [1]. This extraordinary burden of disease necessitates early diagnosis and treatment.

Non-invasive imaging tests like radionuclide imaging and stress echocardiography are frequently used in clinical practice for detection and evaluation of CAD [2]. Global and regional left ventricular (LV) systolic function is an important marker of CAD in stress echocardiography,

which is conventionally assessed using two-dimensional echocardiography. In 1989, the technique of tissue Doppler imaging (TDI) emerged as a potential modality for assessing systolic and diastolic LV performance [3-5]. TDI visualizes myocardial velocities by measuring low-frequency, high-amplitude signals of myocardial motion. TDI is done using either the spectral pulsed-wave or the color-coded pulsed-wave technique. Spectral Doppler measures the instantaneous velocity in a sample volume of the region of interest, while color-coded Doppler allows simultaneous sampling of the entire ultrasound sector [6-8].

Most of the studies of TDI have employed the technique for measuring LV diastolic function. Other investigators have applied TDI to measure resting or post-stress velocities of various myocardial segments of the LV as an adjunct

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tool in the diagnosis of regional wall motion abnormalities from CAD. However, there has not been a consensus on the value of these techniques in the diagnosis of CAD. In this paper, we perform a systematic review and meta-analysis to assess the efficacy of TDI indices in the diagnosis of CAD.

Methods

Study selection

We searched MEDLINE (inception to June 2012) and the Cochrane Library (inception to June 2012) using keywords and/or medical subject headings (MeSH) for TDI and CAD. The detailed search strategy for MEDLINE is presented in Table 1. Titles and abstracts of the references identified were screened by a single reviewer. This was followed by full-text review by two independent reviewers of those articles meeting inclusion criteria. Disagreements were resolved by consensus. We included in our analysis all controlled trials that: 1) were published in English, 2) compared TDI measurements (spectral pulsed wave or color-coded pulsed wave) in those with and without CAD as confirmed by coronary angiography, and 3) evaluated at least one of the following measurements - maximum systolic velocity, maximum early diastolic velocity or maximum late diastolic velocity.

Data extraction

We extracted data on study design, intervention, patient population, size of the study sample, and our outcome measures. The primary outcomes of interest were the mean differences in the following variables as measured by TDI in those with and without CAD: maximum systolic velocity, maximum early diastolic velocity, and maximum late diastolic velocity. In addition we also extracted data on mean differences in E/E' (the ratio of early diastolic transmitral flow velocity and early

diastolic tissue velocity) and E'/A' (the ratio of early and late diastolic tissue velocity) as measured by TDI in those with and without CAD. For all outcomes, data was extracted as means and standard deviations.

Statistical analyses

We performed meta-analyses of the included controlled studies using the Mantel-Haenszel procedure that assumes a fixed effect size [9]. We measured heterogeneity between study results using the I^2 statistic [10]. We defined significant heterogeneity as an I^2 statistic $\geq 50\%$ which would imply real differences between study results that would not be explained by chance alone. In the presence of significant heterogeneity, we performed additional meta-analyses using the random-effects model that produces more conservative estimates of effect [11]. The estimates used in our meta-analyses were mean differences (MD) and 95% confidence intervals (CI) in TDI measurements for our measures of interest in those with and without CAD. Sensitivity analyses were performed using only those studies that verified both the presence and absence of CAD with angiography.

Results

Study selection

Our literature search initially yielded 330 references. Screening of titles and abstracts followed by full-text screening ultimately identified 8 studies [12-19] that met our pre-defined inclusion criteria (Figure 1).

Study characteristics

Combined, the 8 studies included 568 patients. Details of the study populations, segments in which TDI velocities were studied and the echocardiography machine used are shown in Table 2. No randomized controlled trials were identified; all studies were prospective controlled studies. CAD patients had significant disease confirmed by angiography in all studies. Patients without significant CAD either had no obstructive CAD on angiography or had negative results on non-invasive stress testing.

Table 1 Search strategy for including studies

PHASE 1: SEARCH TERMS FOR TISSUE DOPPLER	
1	tissue adj doppler
2	tissue adj velocity adj imaging
3	(TDI or TVI).mp.
4	or/1-3
PHASE 2: SEARCH TERMS FOR CORONARY ARTERY DISEASE	
5	exp Coronary Artery Disease/
6	exp Atherosclerosis/
7	(coronary adj artery adj disease).mp.
8	CAD.mp.
9	or/5-8
PHASE 3: COMBINING THE SEARCHES AND RESTRICTING TO ENGLISH	
10	4 and 9
11	limit 10 to english language

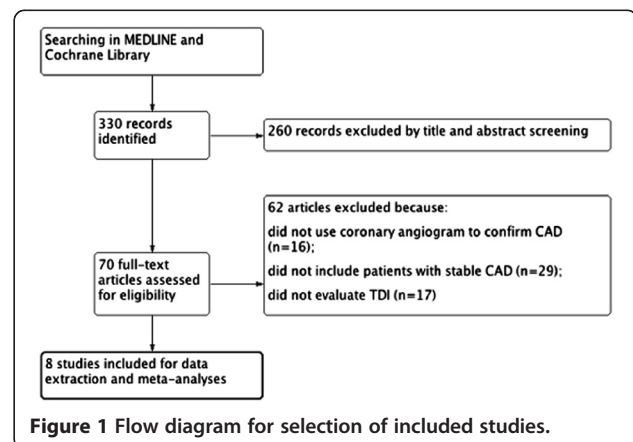


Figure 1 Flow diagram for selection of included studies.

Table 2 Study characteristics

Author, Year (Reference)	Study design	CAD (N)	No CAD (N)	Segments studied	Type of tissue doppler	Machine used
Bolognesi, 2001 [12]	Prospective controlled study	Stable effort angina without previous MI and with normal EF, CAD confirmed with angiography(16)	Negative noninvasive coronary stress tests and normal electrocardiographic and echocardiographic findings in volunteers undergoing cardiac catheterization (6)	Apical four chamber view: septal and anterolateral Apical two chamber view: anterior and inferior	Not reported	Toshiba 380, Rome, Italy
Bruch, 1999 [13]	Prospective controlled study	CAD on angiogram (>70% LAD), no previous MI or cardiac surgery and no echo evidence of regional or global wall motion abnormalities (17)	Normal resting electrocardiogram and a normal regional and global systolic left ventricular function on echocardiogram (20)	Apical four chamber view: midseptal and midlateral	Color-coded pulsed-wave	Toshiba 380, Rome, Italy
Dounis, 2006 [14]	Prospective controlled study	1-2 vessel disease, confirmed by angiogram, normal EF and sinus rhythm (17)	No CAD confirmed by either angiogram or stress testing, normal EF and sinus rhythm(14)	Apical four-, two- and three-chamber views: septal, anteroseptal, anterior, lateral, posterior and inferior	Spectral pulsed-wave	System V, GE Milwaukee, USA
Hoffmann, 2010 [15]	Prospective controlled study	Significant one, two or three vessel disease included. (47)	Suspected angina pectoris and non significant stenosis on angiogram. (35)	Septal, lateral, inferior, anterior, posterior, and anteroseptal	Color-coded pulsed-wave	Vivid 7, GE Healthcare, Horten, Norway
Madler, 2003 [16]	Prospective controlled study	Significant stenosis (>50%) on coronary angiography (90)	Chest pain with no significant stenosis (<50%)on coronary angiography (59)	Apical four and two chamber view: basal septal, basal anterior, basal lateral and basal inferior and mid septal, mid anterior, mid lateral and mid inferior	Color-coded pulsed-wave	System V, GE Vingmed, Horten, Norway
Tsougos, 2008 [17]	Prospective controlled study	Stenosis >70% of at least one coronary artery (72)	No significant stenosis on angiography (42)	Apical four-chamber view: septal and lateral	Spectral pulsed-wave	Vivid 3, GE Vingmed Horten, Norway
Williams, 2005 [18]	Prospective controlled study	Angiography confirmed >50% stenosis in at least one major coronary artery branch; chronic stable symptoms and normal resting global/regional LV function assessed by left ventriculography and echocardiography (16)	Normal coronary angiography; negative, maximum, Bruce protocol exercise testing, and low probability of coronary artery disease (12)	American Society of Echocardiography 16 segment model	Color-coded pulsed-wave	System V, GE Vingmed, Horten, Norway
Zagatina, 2007 [19]	Prospective controlled study	Significant stenosis in the LAD on angiography (88)	Normal angiogram (17)	Long and short-axis parasternal views and the four and two-chamber apical views: basal and mid-septum, basal and midlateral, inferior and anterior	Spectral pulsed-wave	Hewlett Pacard Sonos 2000

Abbreviations: CAD coronary artery disease, EF ejection fraction, LAD left anterior descending, LV left ventricle, MI myocardial infarction.

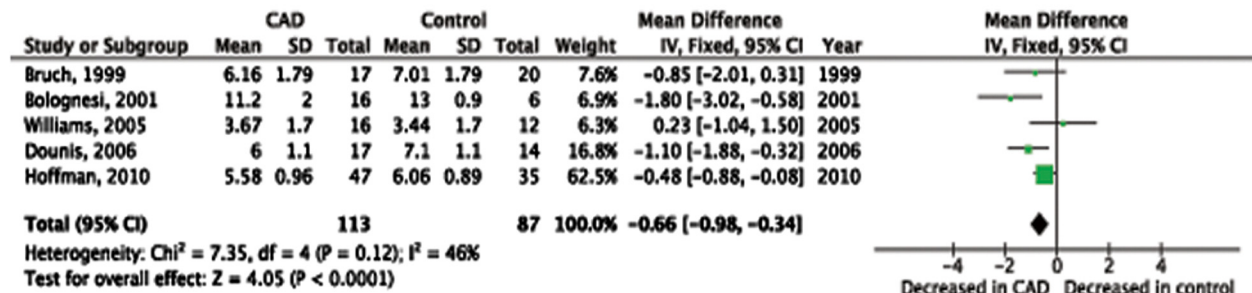
TDI parameters were assessed at baseline in all the studies except one [19] and were assessed after stress testing in 5 studies [14,16-19]. Among the latter, exercise stress testing was performed in 3 studies ([17-19], dipyridamole stress testing was performed in one study [14] and dobutamine stress testing was performed in one study [16]. Three studies reported data on E/E' [12,13,17] and 4 studies on E'/A' [12-14,17].

Primary outcomes (Pre-stress)

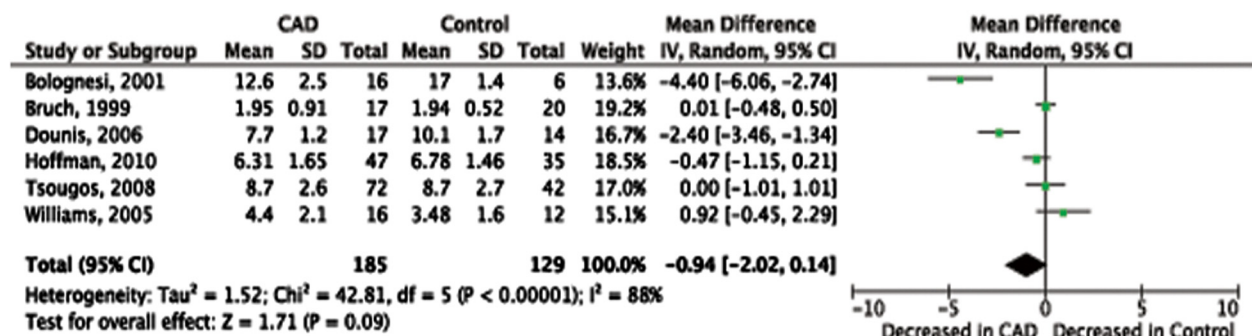
TDI was associated with a significant decrease in maximum systolic velocity among CAD patients when compared to those without CAD (MD: -0.66; 95% CI: -0.98 to -0.34). There was no significant heterogeneity among studies ($I^2 = 46\%$) (Figure 2A). Maximum

early diastolic velocity was significantly decreased among CAD patients when compared to those without CAD (MD: -0.46; 95% CI: -0.79 to -0.13). However, I^2 was significant at 88%. When random-effects meta-analysis was done, the mean difference between those with and without CAD was not significant (MD: -0.94; 95% CI: -2.02 to 0.14) (Figure 2B). TDI was associated with a significant decrease in maximum late diastolic velocity among CAD patients when compared to those without CAD (MD: -0.63; 95% CI: -1.04 to -0.23; $I^2 = 77\%$). Because of the significant heterogeneity, a random-effects meta-analysis was performed which did not demonstrate a significant difference (MD: -0.79; 95% CI: -1.72 to 0.13) (Figure 2C).

[A] Maximum Systolic Velocity



[B] Maximum Early Diastolic Velocity



[C] Maximum Late Diastolic Velocity

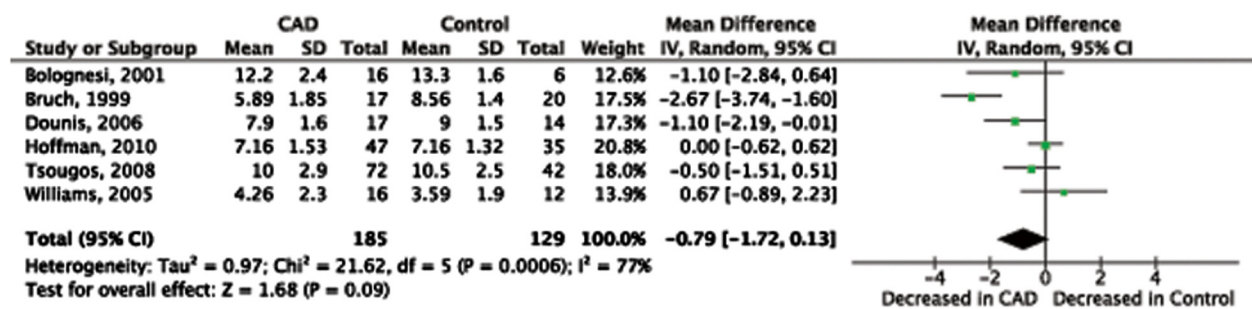


Figure 2 Meta-analysis: Primary outcomes (pre-stress).

Primary outcomes (post-stress)

Meta-analysis demonstrated a significant decrease in maximum systolic velocity among CAD patients when compared to those without CAD (MD: -1.90; 95% CI: -2.69 to -1.11). There was significant heterogeneity ($I^2 = 90\%$). Differences between patients with and without CAD were not statistically significant with a random-effects meta-analysis (MD: -2.64; 95% CI: -5.47 to 0.19) (Figure 3A). TDI was associated with a significant decrease in maximum early diastolic velocity among CAD patients when compared to those without CAD (MD: -1.91; 95% CI: -2.74 to -1.09; $I^2 = 0\%$) (Figure 3B). Maximum late diastolic velocity was also significantly decreased among CAD patients when compared to those without CAD (MD: -1.54; 95% CI: -2.33 to -0.75). A random-effects meta-analysis was performed because of significant heterogeneity ($I^2 = 66\%$), and the mean difference between patients with and without CAD remained

statistically significant (MD: -1.57; 95% CI: -2.95 to -0.18) (Figure 3C).

Sensitivity analyses

Meta-analyses using only those studies that verified both the presence and absence of CAD with angiography showed similar results as the primary analyses. However, there was decreased heterogeneity for the outcome of maximum late diastolic velocity both pre- and post-stress. Additionally, maximum systolic velocity was significantly decreased among CAD patients when compared to those without CAD post-stress, although heterogeneity was still significant (Table 3).

Secondary outcomes

There was not a statistically significant mean difference between E/E' or E'/A' measurements using TDI among

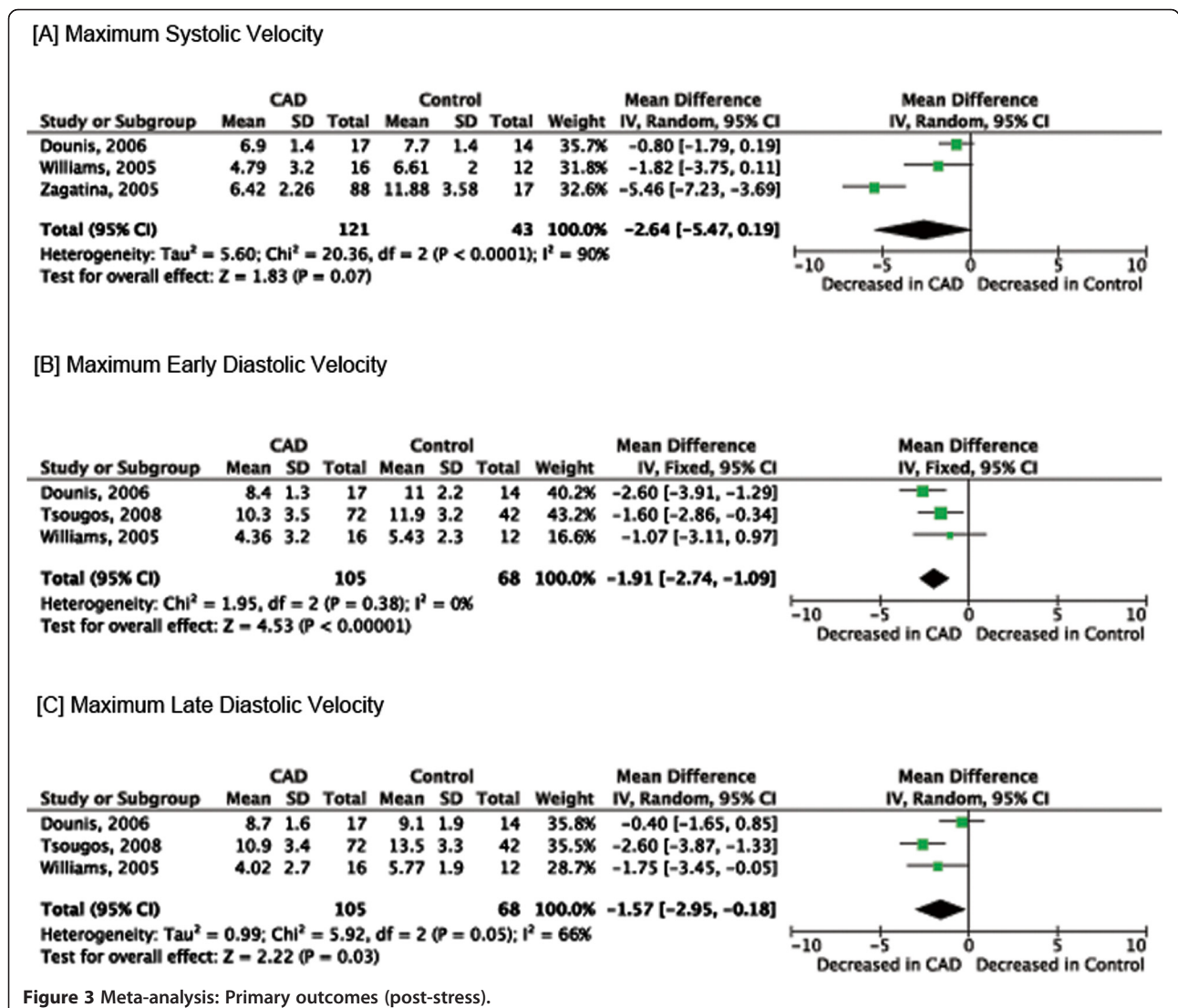


Figure 3 Meta-analysis: Primary outcomes (post-stress).

Table 3 Summary table with pooled results

Outcome	Primary analysis*			Sensitivity analysis of studies using angiography to diagnose or exclude CAD*		
	N of studies	N of patients	Pooled Results – MD (95% CI); I ²	N of studies	N of patients	Pooled Results – MD (95% CI); I ²
Pre-stress						
Maximum systolic velocity	5	200	-0.66 (-0.98 to -0.34); 46%	3	132	-0.54 (-0.91 to -0.17); 44%
Maximum early diastolic velocity	6	314	-0.94 (-2.02 to 0.14); 88%	4	246	-0.89 (-2.52 to 0.74); 89%
Maximum late diastolic velocity	6	314	-0.79 (-1.72 to 0.13); 77%	4	246	-0.13 (-0.61 to 0.35); 0%
Post-stress						
Maximum systolic velocity	3	164	-2.64 (-5.47 to 0.19); 90%	2	133	-3.66 (-7.23 to -0.10); 87%
Maximum early diastolic velocity	3	173	-1.91 (-2.74 to -1.09); 0%	2	142	-1.45 (-2.53 to -0.38); 0%
Maximum late diastolic velocity	3	173	-1.57 (-2.95 to -0.18); 66%	2	142	-2.30 (-3.32 to -1.28); 0%

Abbreviations: *CI* confidence interval, *MD* mean difference.

* Fixed-effects model when I² < 50% and random-effects model when I² ≥ 50%.

CAD patients when compared to those without CAD (Figure 4).

Discussion

To our knowledge, this is the first systematic review and meta-analysis on the use of TDI for the evaluation of CAD. Our results demonstrated significant differences in TDI measurements in those with and without CAD, including significant decreases in the maximum systolic velocity pre-stress, and maximum early diastolic velocity and maximum late diastolic velocity post-stress.

A correlation between decreased systolic velocity and reduced regional myocardial blood flow has also been demonstrated in animal models [20] and a decreased

maximum systolic velocity is highly sensitive and specific for detection of abnormal segmental myocardial motion [21]. In contrast, relatively little data is available specific to maximum early and late diastolic velocities. However, one study demonstrated that reduction of early diastolic velocity was more accurate than maximum systolic velocity for detection of CAD during dobutamine stress echocardiography [22]. A formal evaluation of the test characteristics (sensitivity, specificity, positive and negative predictive value) of TDI was not done because the studies reporting these were done in disparate patient population and used different indices of TDI [23-28].

Our results suggest that decreases in maximal systolic velocity using TDI could be used to detect CAD in

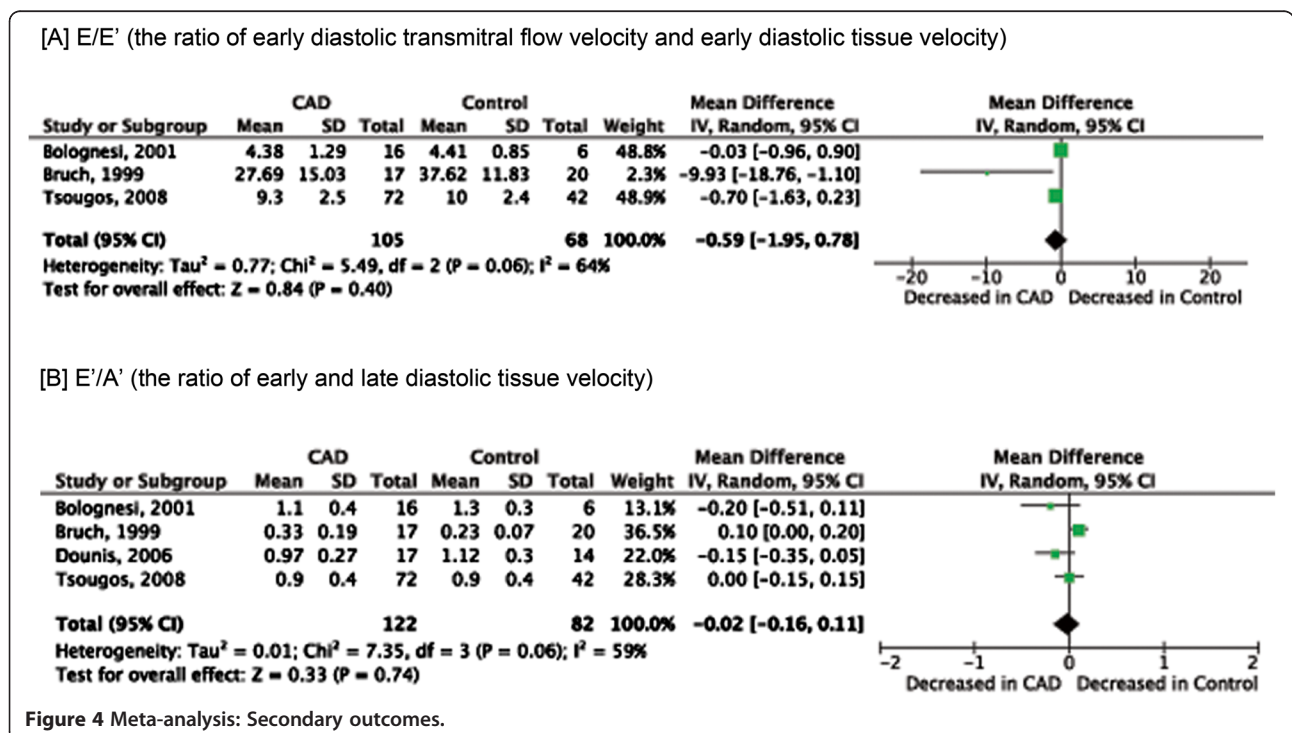


Figure 4 Meta-analysis: Secondary outcomes.

resting studies, especially in patients with poor two-dimensional endocardial definition. It could be incorporated into an echocardiogram based CAD diagnostic score, perhaps combined with wall motion score index, to improve the detection of CAD. Wall motion analysis is qualitative, and the addition of TDI adds a quantitative component in the detection of CAD, theoretically leading to improved intra- and inter-reader variability. Although not addressed by these data, abnormal velocities may also signal a more severe degree of CAD than suggested by wall motion analysis alone [29,30].

Measurement of diastolic velocities could also be incorporated into stress testing algorithms, particularly dobutamine stress echocardiography, in which there is time to measure both two-dimensional and TDI parameters. In the ischemic cascade [31], diastolic dysfunction occurs earlier than systolic dysfunction and thus measurement of diastolic velocities by TDI may prove more sensitive than wall motion analysis alone in the detection of CAD during stress testing.

However, the cutoff values for maximum systolic velocity and diastolic velocities which detect CAD are, as yet, unclear. Furthermore, TDI velocity can only be measured in one dimension and is significantly limited by angle dependence, complicating assessment of multiple wall segments (especially more apical segments). The absence of difference in the maximum systolic velocity post-stress with TDI may be a result of this limitation. Speckle tracking is a new technique that measures myocardial motion in every direction, thus overcoming the limitation of angle dependency and reliably quantifying myocardial ischemia in both infarcted and non-infarcted patients [32,33]. TDI velocities are also influenced by tethering and translation, movement of other adjacent structures and blood flow. Despite these limitations, the American Society of Echocardiography/European Association of Echocardiography guidelines acknowledge that TDI has major advantages like ready availability and quantitative evaluation through online measurement of velocities and time intervals [34].

The conclusions of our meta-analysis are limited by the quality and relatively small number of included studies. Another limitation is the significant heterogeneity observed in the meta-analyses of maximum early and late diastolic velocity (pre-stress); and maximum systolic velocity and late diastolic velocity (post-stress). The heterogeneity could be the result of subtle differences in patient populations, segments in which TDI velocities were measured, models of the echocardiography machines used in the assessments, or type of tissue Doppler used (spectral Doppler, which measures the instantaneous velocity, versus color Doppler which measures the modal velocity). The methods of stress testing used (e.g., dipyridamole vs. dobutamine) to diagnose CAD were different in the included studies and

present an intrinsic limitation in interpreting the analyses. Sensitivity analyses examining only those studies where CAD was evaluated using angiography showed a reduction in heterogeneity in some analyses (Table 3), suggesting that the method used to diagnosis CAD in the studies may have contributed to the observed heterogeneity. A third limitation is that data on head-to-head comparisons between TDI and other non-invasive imaging modalities or wall motion score analysis for diagnosing CAD are not available. It is thus unclear whether TDI has incremental value for diagnosing CAD when compared with resting (or stress) wall motion abnormalities or EKG abnormalities. However, resting wall motion abnormalities and EKG abnormalities are relatively insensitive for detection of CAD, so TDI may be helpful in improving the sensitivity of these tests. Another limitation is that coronary angiography was not performed in all patients across the studies. However, sensitivity analyses that excluded such studies showed similar meta-estimates. There may also be differences in the proportions of patients with variables like hypertension and left ventricular hypertrophy (LVH) across the studies included in this systematic review, and these differences could account for at least some of the heterogeneity observed across studies in the test characteristics of TDI for diagnosing CAD, as well as differences across studies in E/E' or E'/A' measurements. TDI would be low in patients with LVH or hypertension, potentially leading to false positives in patients with hypertension or LVH who do not have CAD. Thus, if the proportion of these patients differed across studies, then the test characteristics of TDI for diagnosing CAD might differ as a result. The proportion of patients with hypertension ranged from 21% to 58% across the included studies which could account for some of the observed heterogeneity in the test characteristics of TDI for diagnosing CAD. The proportion of patients with LVH was not reported in any study. A final limitation is that none of the above studies examined clinical outcomes, like symptomatic CAD.

In conclusion, our results suggest that TDI has a role in the evaluation of CAD. Future studies should evaluate the incremental value of TDI systolic and diastolic velocities over LV ejection fraction and two dimensional wall motion analysis in the detection of CAD and assessment of its severity.

Abbreviations

CAD: Coronary artery disease; CI: Confidence interval; LV: Left ventricle/ventricular; MeSH: Medical Subject Heading; MD: Mean difference; TDI: Tissue Doppler imaging.

Competing interests

None of the authors had any financial or non-financial competing interests

Authors' contributions

RA: Conception and design, analysis and interpretation of the data, drafting of the article, final approval of the article. PG: Collection and assembly of data, final approval of the article. JNK: Critical revision of the article for

important intellectual content, final approval of the article. TA: Critical revision of the article for important intellectual content, final approval of the article. RD: Critical revision of the article for important intellectual content, final approval of the article. GS: Collection and assembly of data, final approval of the article. CAU: Critical revision of the article for important intellectual content, final approval of the article. All authors read and approved the final manuscript.

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References

1. Roger VL, Go AS, Lloyd-Jones DM, Adams RJ, Berry JD, Brown TM, Carnethon MR, Dai S, de Simone G, Ford ES, Fox CS, Fullerton HJ, Gillespie C, Greenlund KJ, Hailpern SM, Heit JA, Ho PM, Howard VJ, Kissela BM, Kittner SJ, Lackland DT, Lichtman JH, Lisabeth LD, Makuc DM, Marcus GM, Marelli A, Matchar DB, McDermott MM, Meigs JB, Moy CS, Mozaffarian D, Mussolino ME, Nichol G, Paynter NP, Rosamond WD, Sorlie PD, Stafford RS, Turan TN, Turner MB, Wong ND, Wylie-Rosett J, American Heart Association Statistics Committee and Stroke Statistics Subcommittee: **Heart disease and stroke statistics—2011 update: a report from the American Heart Association.** *Circulation* 2011, **123**(4):e18–e209.
2. Lee TH, Boucher CA: **Clinical practice. Noninvasive tests in patients with stable coronary artery disease.** *N Engl J Med* 2001, **344**(24):1840–1845.
3. Smiseth OA, Stoylen A, Ihlen H: **Tissue Doppler imaging for the diagnosis of coronary artery disease.** *Curr Opin Cardiol* 2004, **19**(5):421–429.
4. Gorcsan J 3rd, Tanaka H: **Echocardiographic assessment of myocardial strain.** *J Am Coll Cardiol* 2011, **58**(14):1401–1413.
5. Leung DY, Ng AC: **Emerging clinical role of strain imaging in echocardiography.** *Heart Lung Circ* 2010, **19**(3):161–174.
6. Isazak K, Thompson A, Ethevenot G, Cloez JL, Bremilla B, Pernot C: **Doppler echocardiographic measurement of low velocity motion of the left ventricular posterior wall.** *Am J Cardiol* 1989, **64**(1):66–75.
7. McDicken WN, Sutherland GR, Moran CM, Gordon LN: **Colour Doppler velocity imaging of the myocardium.** *Ultrasound Med Biol* 1992, **18**(6–7):651–654.
8. Van de Veire NR, De Sutter J, Bax JJ, Roelandt JR: **Technological advances in tissue Doppler imaging echocardiography.** *Heart* 2008, **94**(8):1065–1074.
9. Collins R, Gray R, Godwin J, Peto R: **Avoidance of large biases and large random errors in the assessment of moderate treatment effects: the need for systematic overviews.** *Stat Med* 1987, **6**(3):245–254.
10. Higgins JP, Thompson SG, Deeks JJ, Altman DG: **Measuring inconsistency in meta-analyses.** *BMJ* 2003, **327**(7414):557–560.
11. DerSimonian R, Kacker R: **Random-effects model for meta-analysis of clinical trials: an update.** *Contemp Clin Trials* 2007, **28**(2):105–114.
12. Bolognesi R, Tsialtas D, Barilli AL, Manca C, Zeppellini R, Javernaro A, Cucchini F: **Detection of early abnormalities of left ventricular function by hemodynamic, echo-tissue Doppler imaging, and mitral Doppler flow techniques in patients with coronary artery disease and normal ejection fraction.** *J Am Soc Echocardiogr* 2001, **14**(8):764–772.
13. Bruch C, Schmermund A, Bartel T, Schaar J, Erbel R: **Tissue Doppler imaging (TDI) for on-line detection of regional early diastolic ventricular asynchrony in patients with coronary artery disease.** *Int J Card Imaging* 1999, **15**(5):379–390.
14. Dounis V, Siegmund T, Hansen A, Jensen J, Schumm-Draeger PM, von Bibra H: **Global myocardial perfusion and diastolic function are impaired to a similar extent in patients with type 2 diabetes mellitus and in patients with coronary artery disease—evaluation by contrast echocardiography and pulsed tissue Doppler.** *Diabetologia* 2006, **49**(11):2729–2740.
15. Hoffmann S, Mogelvang R, Olsen NT, Sogaard P, Fritz-Hansen T, Bech J, Galatius S, Madsen JK, Jensen JS: **Tissue Doppler echocardiography reveals distinct patterns of impaired myocardial velocities in different degrees of coronary artery disease.** *Eur J Echocardiogr* 2010, **11**(6):544–549.
16. Madler CF, Payne N, Wilkenshoff U, Cohen A, Derumeaux GA, Pierard LA, Engvall J, Brodin LA, Sutherland GR, Fraser AG: **Myocardial Doppler in Stress Echocardiography (MYDISE) Study Investigators: Non-invasive diagnosis of coronary artery disease by quantitative stress echocardiography: optimal diagnostic models using off-line tissue Doppler in the MYDISE study.** *Eur Heart J* 2003, **24**(17):1584–1594.
17. Tsougos E, Panou F, Paraskevaidis I, Dagres N, Karatzas D, Kremastinos DT: **Exercise-induced changes in E/E' ratio in patients with suspected coronary artery disease.** *Coron Artery Dis* 2008, **19**(6):405–411.
18. Williams RI, Payne N, Phillips T, D'hooge J, Fraser AG: **Strain rate imaging after dynamic stress provides objective evidence of persistent regional myocardial dysfunction in ischaemic myocardium: regional stunning identified?** *Heart* 2005, **91**(2):152–160.
19. Zagatina A, Zhuravskaya N, Kotelnikova A: **Application of tissue Doppler to interpretation of exercise echocardiography: diagnostics of ischemia localization in patients with ischemic heart disease.** *Eur J Echocardiogr* 2007, **8**(6):463–469.
20. Derumeaux G, Ovize M, Loufoua J, Andre-Fouet X, Minaire Y, Cribier A, Letac B: **Doppler tissue imaging quantitates regional wall motion during myocardial ischemia and reperfusion.** *Circulation* 1998, **97**(19):1970–1977.
21. Katz WE, Gulati VK, Mahler CM, Gorcsan J 3rd: **Quantitative evaluation of the segmental left ventricular response to dobutamine stress by tissue Doppler echocardiography.** *Am J Cardiol* 1997, **79**(8):1036–1042.
22. von Bibra H, Tchnitz A, Klein A, Schneider-Eicke J, Schomig A, Schwaiger M: **Regional diastolic function by pulsed Doppler myocardial mapping for the detection of left ventricular ischemia during pharmacologic stress testing: a comparison with stress echocardiography and perfusion scintigraphy.** *J Am Coll Cardiol* 2000, **36**(2):444–452.
23. Altinmakas S, Dagdeviren B, Turkmen M, Gursurer M, Say B, Tezel T, Ersek B: **Usefulness of pulse-wave Doppler tissue sampling and dobutamine stress echocardiography for identification of false positive inferior wall defects in SPECT.** *Jpn Heart J* 2000, **41**(2):141–152.
24. Badran HM, Elnoamany MF, Seteha M: **Tissue velocity imaging with dobutamine stress echocardiography—a quantitative technique for identification of coronary artery disease in patients with left bundle branch block.** *J Am Soc Echocardiogr* 2007, **20**(7):820–831.
25. Onishi T, Uematsu M, Nanto S, Morozumi T, Watanabe T, Awata M, Iida O, Sera F, Nagata S: **Detection of diastolic abnormality by dyssynchrony imaging: correlation with coronary artery disease in patients presenting with visibly normal wall motion.** *Circ J* 2009, **73**(1):125–131.
26. Peteiro J, Monserrat L, Fabregas R, Manuel Vazquez J, Calvino R, Castro-Beiras A: **Comparison of two-dimensional echocardiography and pulsed Doppler tissue imaging during dobutamine-atropine stress testing to detect coronary artery disease.** *Echocardiogr* 2001, **18**(4):275–284.
27. Rambaldi R, Poldermans D, Bax JJ, Boersma E, Elhendy A, Vletter W, Roelandt JR, Valkema R: **Doppler tissue velocity sampling improves diagnostic accuracy during dobutamine stress echocardiography for the assessment of viable myocardium in patients with severe left ventricular dysfunction.** *Eur Heart J* 2000, **21**(13):1091–1098.
28. Rambaldi R, Poldermans D, Fioretti PM, ten Cate FJ, Vletter WB, Bax JJ, Roelandt JR: **Usefulness of pulse-wave Doppler tissue sampling and dobutamine stress echocardiography for the diagnosis of right coronary artery narrowing.** *Am J Cardiol* 1998, **81**(12):1411–1415.
29. Waggoner AD, Bierig SM: **Tissue Doppler imaging: a useful echocardiographic method for the cardiac sonographer to assess systolic and diastolic ventricular function.** *J Am Soc Echocardiogr* 2001, **14**(12):1143–1152.
30. Trambaiolo P, Tonti G, Salustri A, Fedele F, Sutherland G: **New insights into regional systolic and diastolic left ventricular function with tissue Doppler echocardiography: from qualitative analysis to a quantitative approach.** *J Am Soc Echocardiogr* 2001, **14**(2):85–96.
31. Nesto RW, Kowalchuk GJ: **The ischemic cascade: temporal sequence of hemodynamic, electrocardiographic and symptomatic expressions of ischemia.** *Am J Cardiol* 1987, **59**(7):23C–30C.

32. Sengupta PP, Krishnamoorthy VK, Korinek J, Narula J, Vannan MA, Lester SJ, Tajik JA, Seward JB, Khandheria BK, Belohlavek M: **Left ventricular form and function revisited: applied translational science to cardiovascular ultrasound imaging.** *J Am Soc Echocardiogr* 2007, **20**(5):539–551.
33. Kirkpatrick JN, Lang RM: **Insights into myocardial mechanics in normal and pathologic states using newer echocardiographic techniques.** *Curr Heart Fail Rep* 2008, **5**(3):143–150.
34. Mor-Avi V, Lang RM, Badano LP, Belohlavek M, Cardim NM, Derumeaux G, Galderisi M, Marwick T, Nagueh SF, Sengupta PP, Sicari R, Smiseth OA, Smulevitz B, Takeuchi M, Thomas JD, Vannan M, Voigt JU, Zamorano JL: **Current and evolving echocardiographic techniques for the quantitative evaluation of cardiac mechanics: ASE/EAE consensus statement on methodology and indications endorsed by the Japanese Society of Echocardiography.** *Eur J Echocardiogr* 2011, **12**(3):167–205.

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