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Non-invasive myocardial work index identifies acute coronary occlusion in patients with non-ST-segment elevation-acute coronary syndrome

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Aims Acute coronary artery occlusion (ACO) occurs in ~30% of patients with non-ST-segment elevation-acute coronary syndrome (NSTE-ACS). We investigated the ability of a regional non-invasive myocardial work index (MWI) to identify ACO.

Methods and results Segmental strain analysis was performed before coronary angiography in 126 patients with NSTE-ACS. Left ventricular (LV) pressure was estimated non-invasively using a standard waveform fitted to valvular events and scaled to systolic blood pressure. MWI was calculated as the area of the LV pressure-strain loop. Empirical cut-off values were set to identify segmental systolic dysfunction for MWI (<1700 mmHg %) and strain (more than −14%). The number of dysfunctional segments was used in ROC analysis to identify ACO. The presence of ≥4 adjacent dysfunctional segments assessed by MWI was significantly better than both global strain and ejection fraction at detecting the occurrence of ACO ($P < 0.05$). Regional MWI had a higher sensitivity (81 vs. 78%) and especially specificity (82 vs. 65%) compared with regional strain. Logistic regression demonstrated that elevated systolic blood pressure significantly decreased the probability of actual ACO in a patient with an area of impaired regional strain.

Conclusion The presence of a region of reduced MWI in patients with NSTE-ACS identified patients with ACO and was superior to all other parameters. The regional MWI was able to account for the influence of systolic blood pressure on regional contraction. We therefore propose that MWI may serve as an important clinical tool for selecting patients in need of prompt invasive treatment.

Keywords acute coronary syndrome • echocardiography • haemodynamics • myocardial contraction • hypertension

Introduction

Coronary artery disease presents in a spectrum of ways from silent ischaemia and stable angina pectoris to acute coronary artery occlusion (ACO) and death. The presence of acute ST-segment elevation in the ECG predicts ACO with a high specificity,[1] and mechanical [percutaneous coronary intervention (PCI)] or pharmacological reperfusion is recommended as early as possible.[2] This treatment strategy, in combination with advances in antithrombotic therapy and secondary prevention, has led to a significant reduction in mortality in these patients.[3]

The sensitivity of the ECG in diagnosing ACO is, however, suboptimal, and ~30% of patients with ACO do not develop ST-segment elevation.[4,5] These patients are diagnosed with non-ST-segment elevation-acute coronary syndrome (NSTE-ACS) which is a more heterogeneous group of patients. The optimal timing for invasive therapy in NSTE-ACS patients varies according to different risk cohorts.[6] Previous studies have shown that a delay to PCI >24 h was an independent predictor of short and late mortality in patients with NSTE-ACS.[7] Thus, additional diagnostic tools are needed to identify patients who are in need of prompt reperfusion therapy.

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ACO causes ischaemia and systolic dysfunction in the perfusion territory of the artery. This area is defined as the 'ischaemic risk area' and is an independent predictor of death and a major determinant of final infarct size in patients with ACO.[8–10] Experimental studies have shown a good correlation between the ischaemic risk area and regional systolic dysfunction.[11,12] Regional strain analysis by speckle tracking echocardiography (STE) has previously identified patients with ACO with a good sensitivity. The specificity, however, was low limiting its clinical use.[13] Strain parameters are load dependant, and the low positive predictive value of regional strain may be related to the inability of myocardial deformation indices to account for variations in pre- and afterload, hence not reflecting systolic function accurately. An alternative approach is the estimation of regional myocardial work which is the result of both deformation and opposing force.[14] For example, an increase in afterload may lead to impairment in systolic strain in the presence of preserved or even increased myocardial work. Therefore, when there are changes in afterload, the use of strain could lead to misinterpretations of contractile function. This could secondarily cause false conclusions with regard to myocardial function. In principle, regional myocardial work is superior to myocardial strain in evaluating myocardial function, as it takes into account deformation as well as afterload. This is illustrated in *Figure 1*.

Work assessment has previously been dependent on the use of invasive pressure measurements and has consequently not been feasible in clinical routine. Our group recently introduced a non-invasive method to quantify segmental work using segmental strain and a standardized LV pressure (LVP) curve adjusted to brachial cuff pressure and valvular events.[15] This non-invasive estimation of work, calculated as the myocardial work index (MWI), showed a strong correlation with the equivalent invasive work measurements both experimentally and clinically.

The aim of the present study was to investigate whether the non-invasive MWI could improve selection of NSTE-ACS patients with ACO. MWI was compared with other regional and global echocardiographic parameters.

Methods

The analyses were performed on consecutive NSTE-ACS patients referred for coronary angiography in a single tertiary coronary care centre. The details of the design and study population have been reported previously.[13] Briefly, 150 patients with the following criteria: ≥18 years of age, a clinical diagnosis of NSTE-ACS, and planned coronary angiography within 3 days of admission to the primary hospital, were included. Exclusion criteria were previous myocardial infarction, QRS-width of >120 ms, severe valvular disease, previous heart surgery, extensive comorbidity, or atrial fibrillation. All patients were clinically and haemodynamically stable. The regional ethics committee approved the study, and all patients provided informed consent.

Echocardiography

Echocardiography recordings were performed immediately before coronary angiography. Longitudinal strain values were measured by STE from three apical LV views {mean frame rate 82 ± 13 s^{-1} [mean ± standard deviation (SD)]} and analysed by an 18-segment model. The timing of mitral and aortic valve closure and opening were obtained for work estimation (see below). Twenty-four (16%) patients were excluded due to suboptimal image quality when determining valvular events.

Calculation of non-invasive LVP and MWI

The methods used to calculate the non-invasive LVP analogue and the estimated MWI, which have been validated against the invasive standard in humans, are described in detail in the validation paper.[15] In short, an empiric reference curve for LVP was constructed by collecting invasive LVP data from a number of patients during various conditions. The curves were normalized by stretching or compressing the LVP curves along the time and pressure axes to produce a standard LVP curve. The patient-specific, non-invasive LVP curve was estimated by measuring the time of valvular events by echocardiography and adjusting the

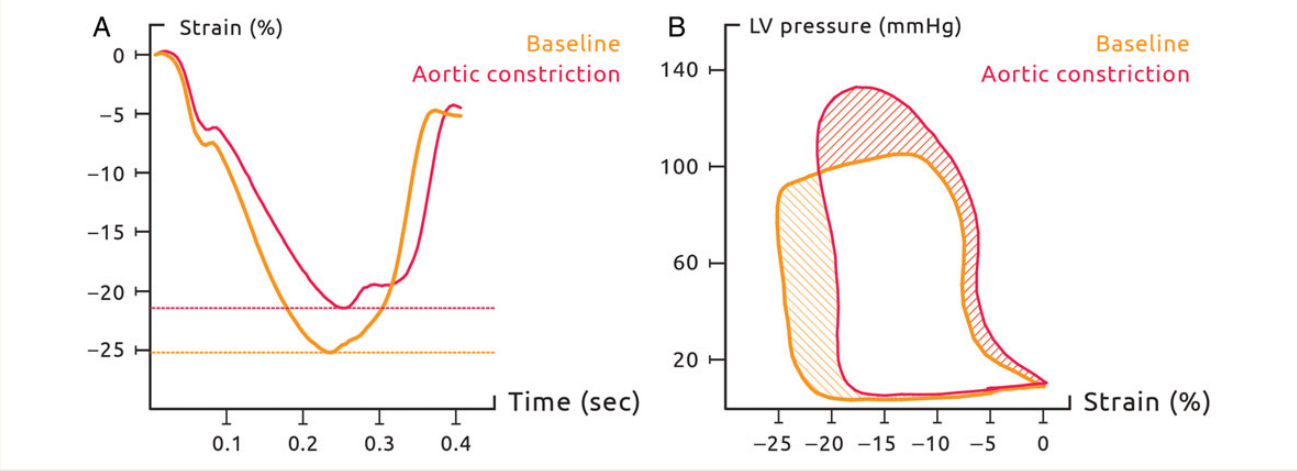

Figure 1 Myocardial strain (*A*) and LV pressure-strain loops (*B*) from a previous unpublished canine experiment in our laboratory before (yellow) and after aortic construction (red). Aortic constriction led to an increase in LV pressure by ~30 mmHg and an impairment in strain by 3–4%. This is illustrated by the horizontal lines showing the peak strain measurement for each condition in *A*. In *B*, LV pressure-strain loop areas for the same experiment revealed no change (<0.5%), and hence equal segmental work, before and after aortic constriction. The shaded areas reflect the variations in work during baseline (yellow) and aortic constriction (red) during the cardiac cycle.

standard LVP curve to the duration of isovolumic contraction, LV ejection, and isovolumic relaxation. To scale the amplitude of the curve, the patient's systolic brachial cuff pressure, measured immediately before the echocardiographic recordings were obtained, was used as a substitute for peak LVP. The patient-specific, non-invasive LVP curve was then used in combination with each of the individuals segmental strain curves to calculate non-invasive LVP-strain loops. The area of the loop served as an index of regional myocardial work (mmHg %). Strain and pressure data were synchronized using the onset R-wave in the ECG. Inter- and intra-observer variabilities were assessed in 16 randomly selected patients.

Regional echocardiographic parameters: the functional risk area

The ischaemic risk area can only be determined by perfusion imaging, contrast echocardiography, or single photon emission computer tomography. These tests are time consuming and are mostly unavailable in the emergency setting. A feasible analogue to the ischaemic risk area is the 'functional risk area (FRA)' (*Figure 2*), introduced by Eek *et al.*,[13] defined as an area of adjacent segments with systolic dysfunction. In their study, a peak strain value of more than −14% was used to dichotomize segments into normal or dysfunctional. In the same manner, a cut-off for regional MWI was determined by using the number of dysfunctional segments as the threshold variable in receiver operator curve (ROC) analysis to identify ACO. A wide range of values were applied to determine the optimal cut-off for dichotomizing segments. A regional MWI value of <1700 mmHg % gave the largest area under the curve (AUC) at identifying patients with ACO and divided segments into functional or dysfunctional. The number of adjacent dysfunctional segments (MWI of <1700 mmHg %) constituted the FRA by MWI (*Figure 2*).

Global echocardiographic parameters

LV ejection fraction (EF) was calculated using the modified Simpson's rule from four- and two-chamber views. The segmental peak strain and MWI values for all segments, from mitral valve closure to mitral valve opening, were averaged to obtain global strain and global work, respectively.

Coronary angiography

ACO on the angiogram was defined as a TIMI flow of 0 (no perfusion) or 1 (penetration without perfusion) in the infarct-related artery. Acute occlusions were differentiated from chronic total occlusions by angiographic appearance and by the ease with which a guide wire could be passed.

Statistics

Values are expressed as mean ± SD or median (inter-quartile range). Differences between groups were analysed by Student's *t*-test or Mann–Whitney *U* test. Categorical data were analysed by χ^2 test or Fisher exact test. Reproducibility was reported as inter-class correlation coefficients, kappa-agreement, and a Bland–Altman plot. ROC analysis was used to find optimal cut-offs for sensitivity and specificity for each parameter. Significance between ROC curves was assessed by the DeLong method[16] through computer software (Analyze-it Software, v. 2 Leeds, UK). The remaining statistical analyses were performed using SPSS v.21 (SPSS Inc., Chicago, IL, USA) and STATA (StataCorp. 2013, Stata Statistical Software: Release 13. College Station, TX, USA). A two-tailed *P*-value of <0.05 was considered significant.

Logistic regression models were constructed to determine potential independent covariates when identifying patients with ACO. FRA by strain and MWI were assessed in separate models. Systolic blood pressure and end-diastolic volume (EDV)/body surface area (BSA) were

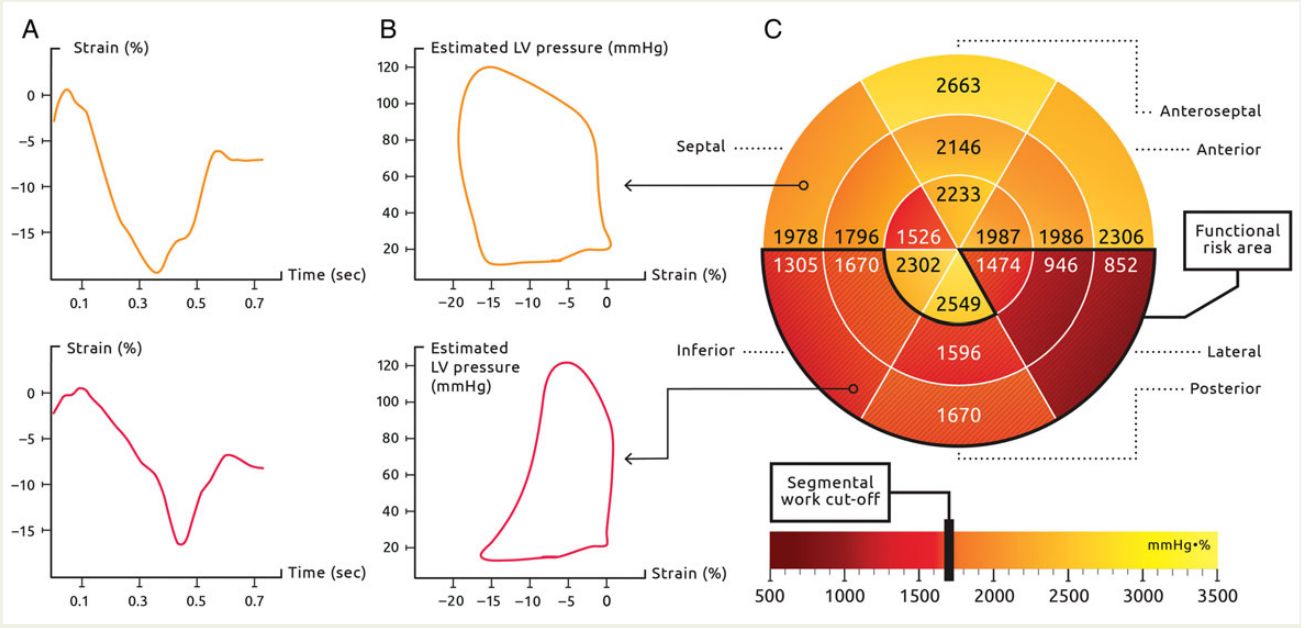

Figure 2 Data from a patient with an acute occlusion in the right coronary artery. (*A*) Two myocardial strain curves from one normal (yellow; top) and one dysfunctional (red; bottom) segment. (*B*) Two estimated LV pressure-strain loops from the same patient. The top left LV pressure-strain loop (yellow) shows a normal segment with a MWI of 1978 mmHg %. The bottom left LV pressure-strain loop (red) shows a dysfunctional segment, within the functional risk area, with a MWI of 1305 mmHg %. (*C*) A bull's eye plot showing segmental work in an 18-segment model. Eight of the segments had impaired systolic function with a MWI of <1700 mmHg % (segmental work cut-off). The functional risk area comprises these segments (shaded) and is marked by the black line. (*D*) Coronary angiogram revealed an occluded right coronary artery (black arrow, top) successfully treated with percutaneous coronary intervention (bottom).

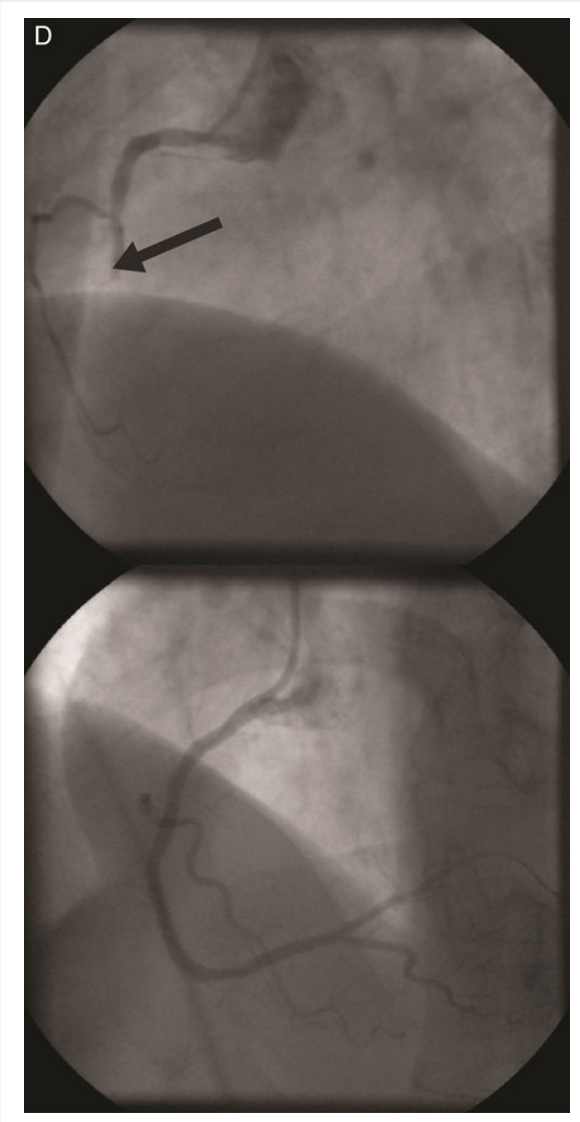

Figure 2 Continued

included as independent covariates as shown below. EF, global strain, and global work were not included as they are not independent of regional strain or work. The following combinations of covariates were included in the models:

(i) ln(odds for ACO) = β_0+β_1*FRA strain + β_2*systolic blood pressure + β_3*EDV/BSA
(ii) ln(odds for ACO) = β_0 + β_1*FRA MWI + β_2*EDV/BSA

Results

Patient characteristics

Twenty-seven (21%) patients had an ACO based on angiographic findings. The territorial distribution of the culprit lesion was 7 (26%) in LAD (2 proximal and 5 distal or branch), 11 (41%) in LCX (6 proximal and 5 distal or branch), and 9 (33%) in RCA (8 proximal and 1 distal or branch). Of these 27 patients, 7 (26%) had collateral perfusion, 11 (41%) had partial collateral perfusion, and 9 (33%) had no collateral perfusion. Patient characteristics, medication, and risk factors are listed in *Table 1*. The time from first hospital admission to coronary angiography was 2.2 ± 0.7 days. Peak Troponin T values were significantly higher in patients with ACO (1088 ± 1110 ng/L) compared with patients without ACO [470 ± 970 ng/L ($P <$ 0.01)]. There were no differences in the signs of ischaemia on ECG between the two groups. There were no significant differences in the blood pressures between patients (*Table 2*).

Table 1 Patient characteristics

	Non-occlusion (n = 99)	Occlusion (n = 27)
Age (years)	58 ± 9	59 ± 8
Body mass index (kg/m^2)	26.7 ± 3.4	27.4 ± 2.9
GRACE score	113 ± 36	131 ± 29*
Normal angiography	28 (28%)	0 (0%)*
Normal ECG	65 (66%)	16 (60%)
Risk factors–no. (%)		
Male gender	70 (71)	25 (93)*
Current smoker	32 (32)	12 (44)
Hypercholesterolaemia	21 (21)	9 (33)
Diabetes mellitus	8 (8)	4 (15)
History of CAD	7 (7)	1 (4)
Hypertension	40 (40)	10 (37)
Medication prior to coronary angiogram–no. (%)		
Aspirin	98 (99)	27 (100)
Clopidogrel	95 (96)	27 (100)
LMWH	95 (96)	27 (100)
β-blocker	85 (86)	16 (59)*
Statin	92 (93)	25 (93)
Warfarin	0 (0)	0 (0)
ACE inhibitor or ARB	28 (28)	5 (19)
Gp IIb/IIIa inhibitor	0 (0)	0 (0)

Continuous data are displayed as mean ± SD.
CAD, coronary artery disease (patients with previous unstable angina or myocardial infarction were excluded from the study); LMWH, low molecular weight heparin; ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blockers; Gp, glycoprotein. GRACE risk score for 6-month mortality or myocardial infarction.
*$P < 0.05$ when compared to patients with no occlusion.

Identification of ACO

Echocardiographic parameters revealed findings consistent with reduced systolic function in patients with ACO (*Table 2*). These patients had a reduced EF, global work, impaired global strain, and an increase in FRA by both strain and MWI.

FRA by MWI was superior to all other parameters at identifying patients with ACO with an AUC of 0.86 (*Table 3*). This regional parameter was significantly better than both global strain and EF (*Figure 3*). Four or more dysfunctional adjacent segments based on MWI assessment resulted in a sensitivity of 81% and specificity of 82% when classifying patients by the presence of ACO. Twenty-two of the 27 patients with ACO were identified using this method. The mean GRACE score for 6-month mortality or myocardial infarction

in the false-negative patients was 103 ± 23. A typical example of a patient with an ACO and an FRA of ≥4 segments assessed by MWI is shown in *Figure 2*. Results from the various parameters including cut-offs, sensitivity, specificity, AUCs, and positive and negative predictive values are summarized in *Table 3*. The intra-class correlation coefficients for intra- and inter-observer variability were 0.92/0.89 and 0.94/0.94 for mitral valve closure/opening and 0.90/0.90 and 0.89/0.94 for aortic valve opening/closure. The corresponding values for MWI were both 0.99 for intra- and inter-observer variability. The Kappa agreement for a dichotomous MWI of <1700 mmHg % was 0.94, and the Bland–Altman plot (*Figure 4*) shows a good agreement between the observers.

Table 2 Clinical and echocardiographic findings

	Non-occlusion ($n = 99$)	Occlusion ($n = 27$)
Haemodynamic variables		
Systolic blood pressure (mmHg)	148 ± 22	145 ± 27
Diastolic blood pressure (mmHg)	83 ± 14	83 ± 14
Heart rate (bpm)	61 ± 10	66 ± 10*
Troponin T (ng/L)	470 ± 970	1088 ± 1100*
Negative Troponin	14 (14%)	0 (0%)
Echocardiography		
LVEF (%)	59.1 ± 7.4	54.3 ± 10.3*
Global strain (%)	−17.4 ± 2.4	−14.9 ± 2.8*
Global work (mmHg %)	2262 ± 449	1781 ± 360*
FRA by work (segments)	1(0–3)	7 (5–11)*
FRA by strain (segments)	2 (0–5)	7 (4–11)*
LV EDV/BSA (mL/m^2)	53.6 ± 9.3	58.3 ± 11.5*

Data are displayed as mean ± SD or median (IQR).
LVEF, left ventricular ejection fraction; FRA, functional risk area; EDV, end-diastolic volume; BSA, body surface area.
*$P < 0.05$ compared with patients with no occlusion.

The impact of alterations in pre- and afterload on the regional myocardial parameters

The systolic blood pressure was a significant covariate [−0.03 per mmHg; 95% CI; −0.05 to 0; ($P < 0.05$)] when using the logistic regression model for regional strain analysis to identify ACO. In other words, an increase in systolic blood pressure decreased the ability of a FRA of ≥4 segments by strain analysis to identify ACO. Based on the first logistic regression model, the predictive probability of ACO was 0.52 for FRA strain in a patient with a systolic blood pressure of 120 mmHg. This fell to 0.28 in a patient with a systolic blood pressure of 160 mmHg. The mean EDV/BSA for the study population was used in these calculations. The effects of variations in afterload on the regional parameters are displayed in *Figure 5* where regional strain and work are compared in a normotensive patient *with* an ACO (5A-B) and a hypertensive patient *without* an ACO (5C-E).

EDV/BSA was a significant covariate in both models but did not confound the ability of the regional parameters to identify ACO.

Discussion

In the present study, we demonstrated the ability of a non-invasive regional MWI to identify acute coronary artery occlusion in patients with NSTE-ACS. Detection of an area with reduced myocardial work was superior to all other echocardiographic parameters at identifying ACO.

The presence of ≥4 adjacent segments with systolic dysfunction based on MWI measurements showed a good sensitivity and specificity at identifying patients with ACO in our study cohort. All echocardiographic parameters identified patients with ACO which were missed by the ECG criteria with a fair sensitivity. There was, however, a high proportion of false-positive patients using all parameters except work indices. The specificities for the non-work parameters were low, emphasizing the need for improved diagnostic tools. From a clinical perspective, most of the parameters included

Table 3 Details of the receiver operator characteristic analyses of echocardiographic parameters to identify acute coronary artery occlusion

	Regional parameters		Global parameters		
	Functional risk area work	Functional risk area strain	Global work	Global strain	Ejection fraction
Cut-off	≥4 segments ≤1700 mmHg %	≥4 segments ≥ −14%	≤2000 mmHg %	≥ −16.5%	≤57%
Sensitivity (%)	81	78	70	70	63
Specificity (%)	82	65	82	70	62
Area under the curve	0.86	0.81	0.80	0.75	0.66
Negative predictive value (%)	94	91	91	90	86
Positive predictive value (%)	55	38	51	39	31
False positives	18	35	18	30	38

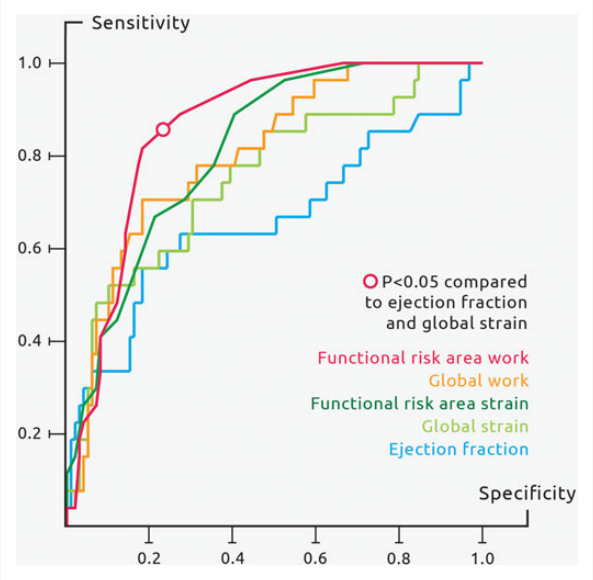

Figure 3 Receiver operator characteristic analyses of echocardiographic parameters to identify coronary artery occlusion in patients with non-ST-elevation–acute coronary syndrome. The myocardial work index was significantly better than ejection fraction and global strain at identifying patients with acute coronary artery occlusion.

could be used to rule out an ACO. The presence of a reduced area of systolic function accentuates how the parameters differ. In a patient with an FRA of ≥4 segments based on MWI, the positive predictive value for an ACO was 55%. The positive predictive value for an ACO in a patient with a FRA of ≥4 segments based on strain was, however, 38%.

Assessment of regional myocardial function; the effect of alterations in load

Acute coronary syndrome leads to a sudden reduction in blood flow to the myocardium, causing an impairment in regional function within minutes.[17] In an ischaemic segment, the reduction in systolic function does not only depend upon the contractile state of the myocardium, but also on variations in preload and afterload. This has been shown experimentally where an elevation in afterload led to a further decrease in systolic shortening of an ischaemic segment.[18]

Regional myocardial work assessment by LVP-segment length loops is a feasible method of quantifying myocardial performance during ischaemia in an experimental setting.[19] In the present study, we demonstrate that this is also possible in humans. Strain is sensitive to alterations in loading conditions, and systolic blood pressure significantly affected strain measurements in their ability to identify patients with ACO. The dependency of strain on systolic pressures was recently confirmed in a clinical study.[20] In this meta-analysis, systolic blood pressure was the only factor that had a significant impact on strain compared with all other covariates including intervendor

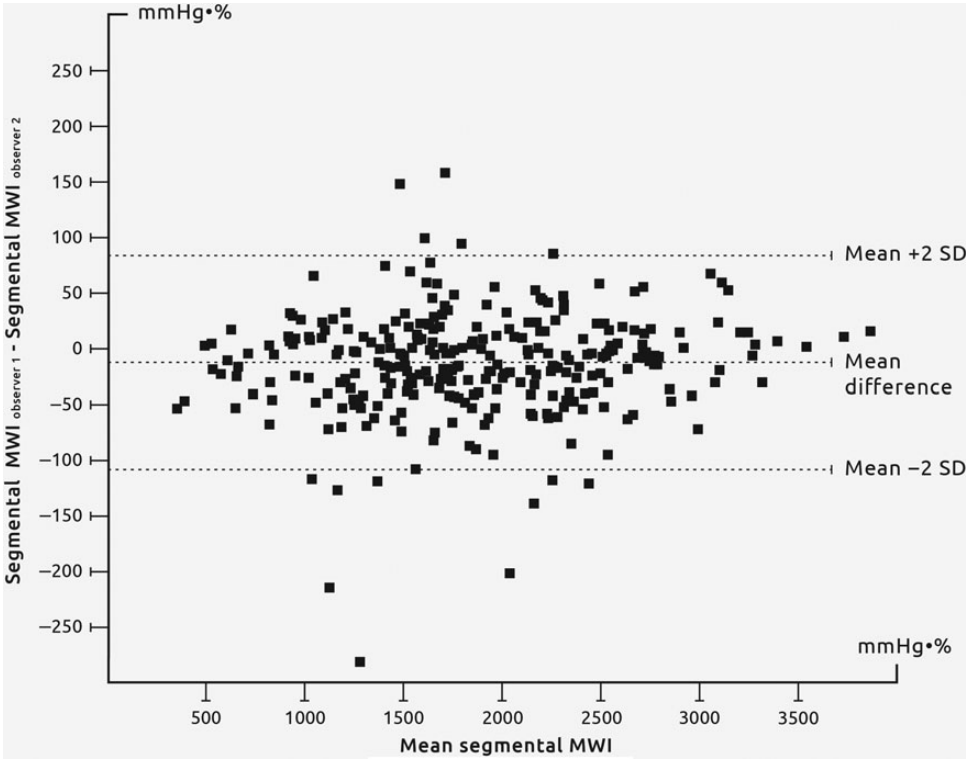

Figure 4 The agreement between two observers for the myocardial work index shown by a Bland–Altman plot. There was practically no systematic difference between the observers and an acceptable disagreement. SD, standard deviation.

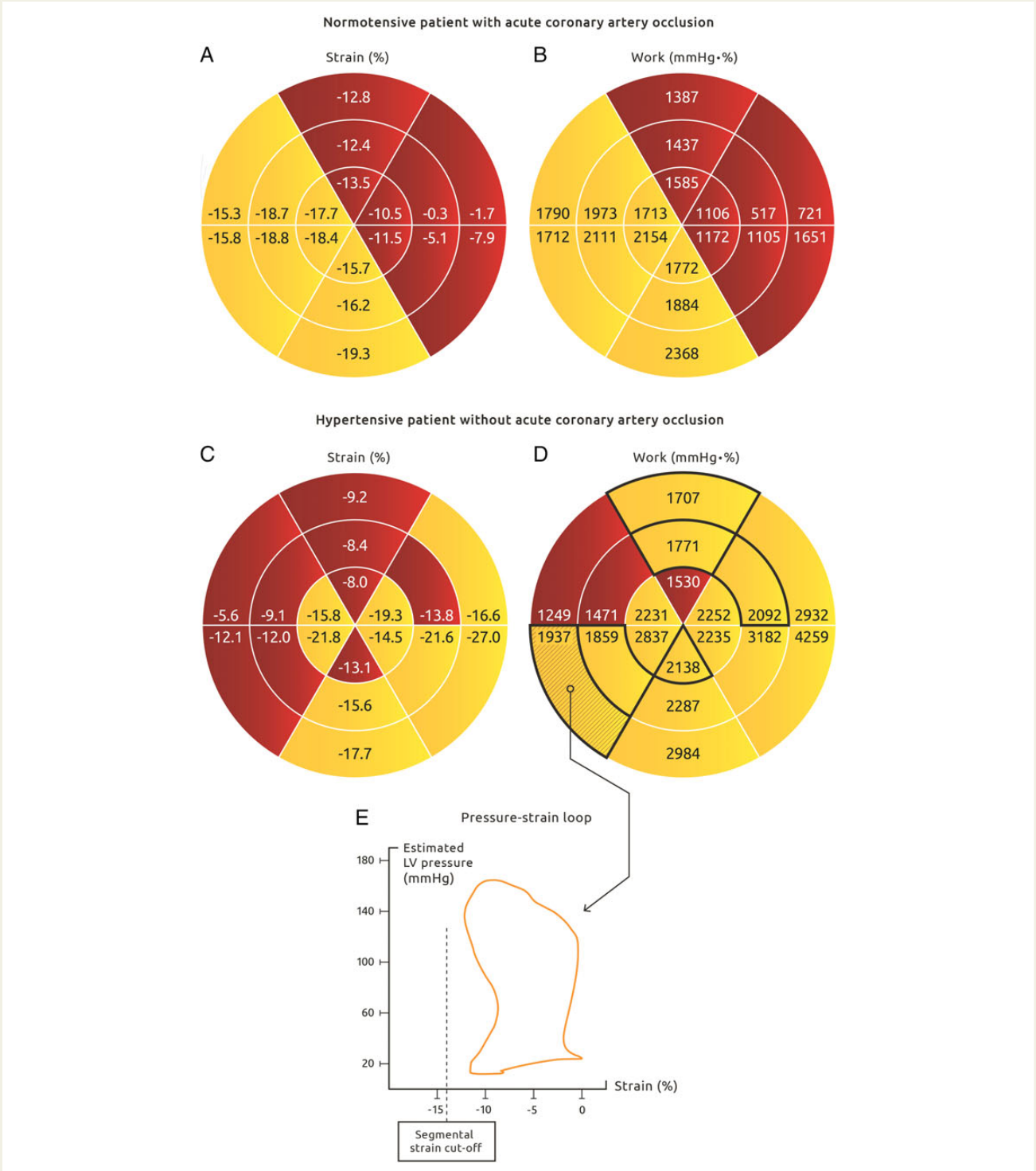

Figure 5 Bull's eye plots of segmental strain and MWI from a normotensive patient with ACO and a hypertensive patient without ACO. Segments with impaired systolic function are colored red and normal segments are colored yellow according to the cut-offs set for strain and MWI, respectively. (*A* and *B*) Data from a patient *with* ACO and normal systolic blood pressure (132 mmHg). The segmental measurements of strain and MWI show an identical functional risk area (FRA) of nine segments. The patient had an occlusion in the posteromedial branch of the circumflex artery. (*C* and *D*) Data from a patient *without* ACO and a high systolic blood pressure (160 mmHg). Strain measurements demonstrated a FRA comprising eight adjacent segments with impaired strain categorizing the patient into the false-positive group (C). The MWI showed that there was normal work (D yellow segments with black perimeter) in several of the segments with hypokinesia based on strain measurements (C). (*E*) An LV pressure-strain loop from a segment that was categorized as dysfunctional using strain showing a normal index of myocardial work. The segment was operating below the cut-off for strain, but at a high systolic blood pressure.

differences. *Figure 5* exemplifies how regional contraction is affected by load and shows that segments can falsely be interpreted as dysfunctional using strain values alone due to a high systolic blood pressure (5C). The MWI deemed several of these segments as normal (5D), classifying the patient as a true negative. *Figure 5E* shows how the LVP-strain loop area for one of these segments is normal, even though the segment's strain value was subnormal. The MWI was therefore able to correct the falsely interpreted reduction in systolic function based on strain measurement in patients with a high afterload and no ACO.

Another explanation for different interpretations between MWI and strain may be related to peak strain values that have a limited ability to reflect myocardial performance. Peak strain depends not only on the contractile properties of the segment, but also on the afterload at the time of contraction. Identical peak segmental strain values occurring in early or mid-systole do not correspond to identical segmental systolic function, as afterload is higher during mid-systole.

Timing of invasive therapy for patients with NSTE-ACS

NSTE-ACS represents a heterogonous group of patients with a highly variable prognosis, and the optimal timing of angiography has not been well defined.[6] The ability to identify patients with ACO is clinically important, as they are amenable to revascularization treatment and an improved long-term prognosis.

Previous studies have supported early angiography and intervention (<24 h) in NSTE-ACS patients. This is particularly true among high-risk patients (GRACE score >140).[21] The use of risk stratification models is helpful, but relies on biochemical markers which are elevated several hours after an ACO.

Immediate intervention, analogue to primary PCI for STEMI patients, has also been evaluated for NSTE-ACS patients without an overall positive effect.[22] Nevertheless, sub-populations within this heterogeneous patient group may benefit from immediate PCI. We believe that our echocardiographic method could be an important tool when considering timing of invasive therapy, both immediate and early, in NSTE-ACS patients. The method may potentially be used in viability assessment, aiding the decision-making process in relation to invasive vs. medical treatment. These issues need to be examined in future prospective studies.

Limitations

The LV pressure-strain loop is an estimation of regional work which is calculated from a non-invasive LVP derivative and STE. The calculation does not take into account local curvature and represents an index of work rather than being a direct measurement of work. The limitations of LV pressure-strain loop calculation are dealt with in the validation study.[15] Despite these limitations, the present study has shown the value of using MWI as a clinical tool, although further validation in other patients, especially in the earlier stages of ACS, is desired.

Twenty-four patients were unfortunately excluded due to insufficient quality of the echocardiographic recordings when determining valvular events. The recordings were originally performed for strain analyses without specific concern for valvular timing. In further clinical studies, this can be addressed more specifically. There was, however, the same proportion of patients with ACO in the group excluded compared with the study population. We therefore believe that this would not have changed the main conclusion of this study.

The cut-offs for MWI and strain were determined by retrospective analysis. These values should be validated prospectively in both patient and healthy populations. Since brachial cuff pressure was used as an estimate for peak LVP in the calculation of MWI, the systematic difference between cuff pressure and peak LVP is inherent in the cut-off value for MWI.

Finally, one can object that differences in heart size may have influenced the regional work index. This is, however, already normalized by using strain (relative deformation) instead of actual shortening distance in the calculation of MWI.

Conclusion

In this study, a region of reduced myocardial work in patients with NSTE-ACS identified patients with ACO with a high sensitivity and specificity. This method was superior to strain analyses by accounting for the effect of systolic blood pressure on myocardial systolic shortening. Although further studies are needed, we propose that this method may be an important tool to identify ACO in patients with NSTE-ACS in the emergency department.

Conflict of interest: K.R., M.E. and O.A.S. are a co-inventors of a patent application WO 2012055498 A4 (Method for myocardial segment work analysis) licensed to GE Healthcare.

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IMAGE FOCUS

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Giant coronary sinus in rheumatic heart disease: a rare case presentation

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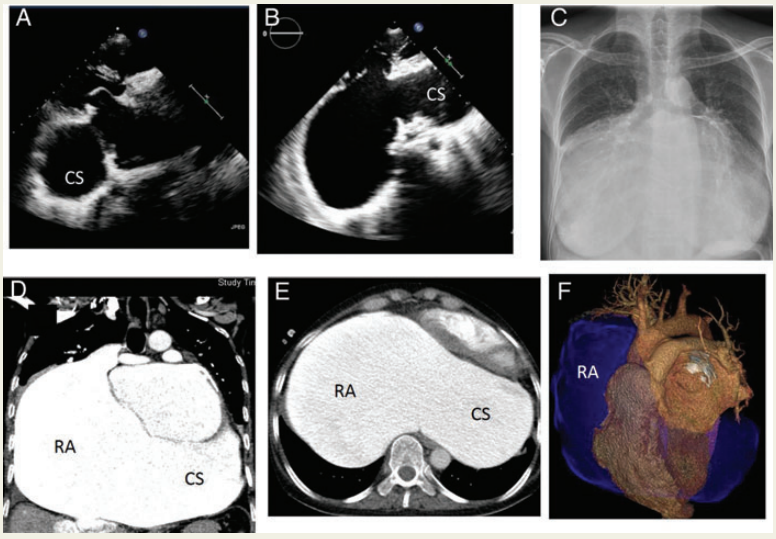

A 41-year-old female, who had undergone balloon mitral valvotomy 4 years back, presented with Class 3 dyspnoea. She had elevated jugular venous pressure up to the angle of mandible with pedal oedema. An echocardiogram showed severe mitral valve disease with severe tricuspid regurgitation with dilated right atrium. The tricuspid regurgitant gradient was 33 mmHg and the estimated PA systolic pressure was 48 mmHg. The coronary sinus was grossly dilated (see *Panels A* and *B*, see Supplementary data online, *Videos S1–S4*). Chest X-ray showed a cardiothoracic ratio of >90% (see *Panel C*). The left atrial upper border showed linear calcification. Contrast-enhanced CT with volume-rendered reconstruction showed a grossly dilated right atrium (see *Panels D–F*). The coronary sinus was dilated measuring 10.1 × 7.3 cm in diameter and 1873 mL in volume, which is the largest size reported in literature till now. A left superior vena cava (LSVC) was noted to drain into the coronary sinus.

Dilatation of the coronary sinus is rarely seen in normal healthy adults. The cause of dilatation in order of decreasing frequency is persistent LSVC, rheumatic heart disease, total anomalous pulmonary venous drainage, coronary cameral fistula, post-operative obstruction, and unroofing of coronary sinus.

In our patient, the giant coronary sinus was due to multiple factors such as persistent LSVC, severe tricuspid regurgitation, and pulmonary hypertension. This is the largest coronary sinus reported to the best of our knowledge.

Supplementary data are available at *European Heart Journal – Cardiovascular Imaging* online.