Diastolic Backward-Traveling Decompression (Suction) Wave Correlates With Simultaneously Acquired Indices of Diastolic Function and Is Reduced in Left Ventricular Stunning

Andrew Ladwiniec, MBBS, MRCP; Paul A. White, PhD; Sukhjinder S. Nijjer, PhD, MRCP; Michael O’Sullivan, PhD, FRCP; Nick E.J. West, MD, FRCP; Justin E. Davies, PhD, FRCP; Stephen P. Hoole, DM, FRCP

Background—Wave intensity analysis can distinguish proximal (propulsion) and distal (suction) influences on coronary blood flow and is purported to reflect myocardial performance and microvascular function. Quantifying the amplitude of the peak, backwards expansion wave (BEW) may have clinical utility. However, simultaneously acquired wave intensity analysis and left ventricular (LV) pressure–volume loop data, confirming the origin and effect of myocardial function on the BEW in humans, have not been previously reported.

Methods and Results—Patients with single-vessel left anterior descending coronary disease and normal ventricular function (n=13) were recruited prospectively. We simultaneously measured LV function with a conductance catheter and derived wave intensity analysis using a pressure–low velocity guidewire at baseline and again 30 minutes after a 1-minute coronary balloon occlusion. The peak BEW correlated with the indices of diastolic LV function: LV dP/dt\textsubscript{min} (r=−0.59; P=0.002) and τ (r=−0.59; P=0.002), but not with systolic function. In 12 patients with paired measurements 30 minutes post balloon occlusion, LV dP/dt\textsubscript{max} decreased from 1437.1±163.9 to 1299.4±152.9 mmHg/s (median difference, −110.4 [−183.3 to −70.4]; P=0.015) and τ increased from 48.3±7.4 to 52.4±7.9 ms (difference, 4.1 [1.3–6.9]; P=0.01), but basal average peak coronary flow velocity was unchanged, indicating LV stunning post balloon occlusion. However, the peak BEW amplitude decreased from −9.95±5.45 W·m⁻²·s⁻²×10⁵ to −7.52±5.00 W·m⁻²·s⁻²×10⁵ (difference 2.43×10⁵ [0.20×10⁵ to 4.67×10⁵; P=0.04]).

Conclusions—Peak BEW assessed by coronary wave intensity analysis correlates with invasive indices of LV diastolic function and mirrors changes in LV diastolic function confirming the origin of the suction wave. This may have implications for physiological lesion assessment after percutaneous coronary intervention.

Clinical Trial Registration—URL: http://www.isrctn.org. Unique identifier: ISRCTN42864201.

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Key Words: diastole ■ hemodynamics ■ myocardial stunning ■ percutaneous coronary intervention ■ systole
WHAT IS KNOWN

• Wave intensity analysis is a technique that enables the calculation of proximal and distal influences on coronary flow from simultaneously acquired coronary pressure and flow velocity.

• The 2 predominant drivers of coronary flow are the early forwards compression wave, caused by left ventricular (LV) ejection, and the backwards expansion (or suction) wave, caused by microcirculatory decompression in early diastole.

• Late myocardial stunning can be induced by coronary occlusion at the time of percutaneous coronary intervention.

WHAT THE STUDY ADDS

• The magnitude of the backwards expansion wave is related to the indices of LV diastolic performance, confirming a close link between LV mechanics and coronary flow.

• Reduction in LV systolic and diastolic performance by the induction of late myocardial stunning results in a parallel reduction in the magnitude of the backwards expansion wave, suggesting a potential role for wave intensity analysis to investigate changes in diastolic LV function from intracoronary measurements.

Methods

Study Population

Seventeen consecutive male patients with an isolated left anterior descending type-A stenosis, awaiting elective PCI who had normal LV function with no regional wall motion abnormality assessed by left ventriculography were prospectively recruited. Patients were excluded if they had diabetes mellitus, had a myocardial infarction in the preceding 3 months, or were not in sinus rhythm. Patients were asked to abstain from caffeine, alcohol, nicotine, and oral/sublingual nitrates and nicorandil for a 24-hour period before their procedure.

Ethics

The study was approved by the Local Research Ethics Committee/Institutional Review Board (LREC06/Q0106/52) and confirmed to the Declaration of Helsinki. The trial number was ISRCTN42864201. The study was approved by our Institutional Review Committee, and all subjects gave written informed consent.

Catheter Laboratory Protocol

Bilateral femoral arterial (7-F and 8-F) and venous (6-F) sheaths were inserted. PCI was performed with 6-F or 7-F guiding catheters. Central venous pressure monitoring (Pv) and Millar catheter calibration were performed via the venous sheath. All patients were anticoagulated with a heparin bolus (70–100 U/kg) after arterial sheath insertion to achieve an activated coagulation time ≥250 s. No hemodynamic altering medication was administered during the procedure. The ambient temperature of the catheter laboratory was kept constant, and the environment was quiet.

To determine LV pressure–volume relations, a 7-F, 8-electrode conductance catheter with an integrated micromanometer tip (Millar Instruments, Houston, TX) was advanced via a femoral sheath into the LV apex. It was placed along the longitudinal axis of the ventricle to minimize motion during the cardiac cycle. The catheter was connected to a signal conditioning and processing unit (CD Leycom, Zoetermeer, the Netherlands). A 20-kHz, 30-pA current was applied to the distal and proximal electrodes, and the remaining 6 electrodes were used to measure time-varying ventricular conductance, G(t), as the sum of the intervening 5 segments. Continuous recordings were taken for simultaneous comparison with coronary hemodynamic data. The relationship between the time-varying volume, V(t), and the time-varying conductance, G(t), is given by the formula: V(t)=1/α(L×r)[G(t)–G(p)]; α is the ratio of the conductance-derived volume to true ventricular volume, L is the interelectrode distance, r is the resistivity of blood in Ω/cm, and G(p) is the parallel conductance because of the conductance of structures outside the ventricular blood pool. Volume correction for G(p) was calculated from the formula: Vc=1/α(L×r)[G(p)] as estimated by the hypertonic saline injection technique described by Baan et al.12 which allows the conductance (and hence volume) outside the heart to be subtracted from the total volume to derive ventricular volume. The slope coefficient, α, calculated to correct for inhomogeneity of the electric field, was determined by an average Fick cardiac output, measured 3x, to further improve ventricular volumetric accuracy.

A Combowire XT 9500 (Volcano Corp, San Diego, CA)10 0.014” guidewire with a zero-offset Doppler flow velocity and pressure sensor at the tip was used to acquire coronary hemodynamic data. After pressure calibration, the tip of the wire was positioned 3 to 5 cm beyond the stenosis, in a segment of vessel that was straight and free from side-branches. Rotation of the wire, to ensure that the tip was in the center of the vessel, optimized the velocity signals. The wire tip position was fluoroscopically stored to ensure that successive measurements were made with the wire tip in the same vessel location. Measurement of intracoronary flow velocity and pressure were acquired initially at baseline for a minimum of 30 s. A low-pressure balloon occlusion was then performed at ≤4 atmospheres for 1 minute, with the intention of inducing myocardial stunning, but not modifying the stenosis. Coronary occlusion was confirmed by contrast injection during balloon inflation. After 30 minutes, coronary flow and pressure measurements were repeated under baseline conditions. After hemodynamic measurements, the PCI procedure was completed as is standard practice.

Measurements were recorded digitally onto a ComboMap (Volcano Corp) at 200 Hz and stored on disk for offline analysis. These
data were then analyzed using dedicated customized software (Study Manager, Academic Medical Center, University of Amsterdam, The Netherlands; and a Matlab (Mathworks Inc., Natick, Massachusetts) environment for wave-intensity analysis (Imperial College London, United Kingdom).

**Coronary Angiographic and Hemodynamic Assessment**

Angiographic stenosis severity was assessed before each low-pressure balloon occlusion by quantitative coronary angiography (Cardiac Viewer CV-1000, version 2.1.0; Liverpool, New York). The pressure gradient ($P_d/P_a$, where $P_d$ is the distal pressure and $P_a$ is the aortic pressure) was assessed before and at +30 minutes after low-pressure coronary balloon occlusion and the collateral flow index ($CF_I = (P_w/P_v)/(P_a–P_v)$, where $P_w$ is the distal wedge pressure measured during balloon occlusion and $P_v$ is the central venous pressure) was measured during first low-pressure coronary balloon occlusion.

**Data Analysis: LV Assessment**

Typical pressure volume loops at baseline and during balloon occlusion are illustrated in Figure 1. Conductance catheter data were analyzed offline with PVAN software (Millar Instruments) using 5 cardiac cycles at baseline, and 30 minutes after balloon occlusion. Mean index of diastolic function (maximum rate of pressure decline: LV $dP/dt_{min}$ and time constant of LV isovolumic relaxation: LV $\tau$); systolic function (maximum rate of pressure generation: LV $dP/dt_{max}$); and LV end-diastolic pressure and volume, stroke volume, and ejection fraction were calculated at these time points.

![Image]

Figure 1. Left ventricular (LV) conductance catheter and simultaneous coronary wave intensity measurements. **Top left**: antero-posterior cranial angiogram of a mid-left anterior descending lesion in a study participant with a Millar conductance catheter in the LV cavity. **Top right**: the same participant undergoing balloon occlusion. **Bottom left**: ensemble averaged coronary pressure (solid line) and flow velocity (dashed line) in a study participant undergoing simultaneous LV assessment. Wave intensity analysis is shown in the top; black waves represent those associated with coronary flow acceleration, and white waves associated with flow deceleration, upward deflections represent waves originating from the aorta and downward deflections represent waves originating from the microvasculature. The early forwards compression wave (eFCW) and backwards expansion wave (BEW) are labeled. **Bottom right**: plotted pressure–volume relations at baseline (blue) and during balloon occlusion (red).
Waves intensity represents the rate of energy per unit area transported by travelling waves in arteries and is derived from phasic changes in local pressure and flow velocity. The blood pressure and Doppler velocity recordings were filtered with a Savitzky–Golay filter and ensemble averaged using the ECG R-wave for timing. Wave intensity was calculated from simultaneous baseline pressure and flow measurements taken in the vessel being treated, over 30 cardiac cycles. Measurements were taken at baseline and 30 minutes post balloon occlusion. The change in pressure was separated into wave components originating from the proximal vessel and from the microvasculature assuming the density of blood to be 1050 kg/m³ and estimating wave speed using the sum of squares method. The peak wave energy (measured in W·m⁻²·s⁻²×10⁵) was calculated for the early forwards compression wave (eFCW) occurring at the onset of systole and the BEW, the first negative wave occurring at the onset of ventricular relaxation, identified by the onset of diastole (Figure 1). Data from baseline and 30 minutes after coronary balloon occlusion were pooled to explore the relationships between coronary wave intensity and LV hemodynamics.

Statistical Analysis
Stata v.12 (StataCorp) was used for statistical analysis. Continuous values are expressed as means±SD or median (25th–75th percentile) as appropriate. Continuous variables were compared using a paired t test or, where appropriate, a Wilcoxon signed-rank test. No adjustments for multiple comparisons were made. Correlations were quantified using Pearson correlation coefficient or Spearman correlation coefficient as appropriate. Probability values were 2-sided, and values of P<0.05 considered significant.

Results
Of 17 patients recruited into the study, 13 had coronary flow velocity traces of sufficient quality to perform WIA; the other 4 patients were excluded. Patient demographic, baseline hemodynamic, and angiographic data for the 13 patients included in analysis are summarized in Table 1. One of the 13 patients underwent coronary and LV assessment before balloon occlusion, but not at 30 minutes and was therefore excluded from any pre/post comparisons. All recruited patients had stable angina (Canadian Cardiovascular Society class ≥2) with evidence of ischemia on functional testing.

Relationship Between Wave Intensity Analysis and LV Hemodynamics
The relationship between peak BEW intensity and indices of diastolic function is illustrated in Figure 2. Including baseline

| Table 1. Baseline Demographic, Hemodynamic, and Angiographic Characteristics |
|-------------------|-------------------|
| Demographics (n=13) |                |
| Age, y             | 55.4±6.8         |
| Left ventricular ejection fraction, % | 61.4±4.7         |
| Body mass index, kg/m² | 29.5±4.2        |
| Treated hypertension, n (%) | 5 (38)          |
| Current smoker, n (%) | 2 (15)           |
| Medications, n (%) |                |
| Aspirin             | 13 (100)         |
| Clopidogrel         | 13 (100)         |
| Statin              | 13 (100)         |
| β-blocker           | 12 (92)          |
| ACE inhibitor or ARB| 10 (77)          |
| Long-acting nitrate | 7 (54)           |
| Hemodynamics       |                |
| Systolic blood pressure, mmHg | 123.8±18.4 |
| Diastolic blood pressure, mmHg | 66.2±9.2       |
| Heart rate, bpm    | 56.7 (7.1)       |
| Angiographic characteristics |             |
| Stenosis severity, % | 79.8±12.6       |
| Reference vessel diameter, mm | 3.2±0.6        |
| Proximal LAD, n (%) | 5 (38)           |
| Mid LAD, n (%)      | 8 (62)           |
| Coronary hemodynamics |            |
| Pd/Pa               | 0.79±0.18        |
| CFIp                | 0.13±0.09        |

ACE indicates angiotensin-converting enzyme; ARB, angiotensin II receptor blocker; CFIp, collateral flow index (by pressure); LAD, left anterior descending artery; Pa, aortic pressure; and Pd, distal pressure.

Figure 2. Association between backwards expansion wave (BEW) intensity and left ventricular (LV) diastolic function. Relationship between peak BEW intensity and dP/dtmin (left) and τ (right). Solid dots represent baseline measures and hollow dots represent measures 30 min post 1-min balloon occlusion.
measurements for all 13 study participants and measurements 30 minutes post balloon occlusion for the 12 study participants with paired measurements (a total of 25 data points). Peak BEW correlated with indices of diastolic LV function: LV dP/dtmin (rs = -0.59; P = 0.002) and τ (rs = -0.59; P = 0.002). This relationship was still present if only baseline measurements were used: LV dP/dtmin (rs = 0.58; P = 0.037) and τ (rs = -0.77; P = 0.002). We did not find a relationship between LV dP/dt max and peak BEW intensity (rs = -0.36; P = 0.08). We also did not identify a statistically significant association between peak eFCW intensity and LV systolic function (LV dP/dt max; rs = 0.08; P = 0.69; Figure 3).

Effect of Myocardial Stunning
LV hemodynamics and coronary wave intensities at baseline and 30 minutes post balloon occlusion are listed in Table 2 and illustrated, along with LV hemodynamics during balloon occlusion in Figure 4. In those patients with paired measurements, low-pressure balloon inflations did not alter coronary stenosis severity measured by quantitative coronary angiography (78.5±12.1% versus 77.4±11.4%; P = 0.79) or Pd/Pa (0.79±0.18 versus 0.81±0.19; P = 0.13). All patients had transient ECG evidence of myocardial ischemia at the time of balloon occlusion; but at 30 minutes post balloon occlusion, there was no evidence of ongoing myocardial ischemia in the form of symptoms or ECG changes in any of the 12 patients who underwent paired measurements. LV dP/dt max decreased from 1437.1±163.9 to 1299.4±152.9 mm Hg∙s⁻¹ (median difference, −110.4 [−183.3 to −70.4]; P = 0.015) and τ increased from 48.3±7.4 to 52.4±7.9 ms (difference, 4.1 [1.3–6.9]; P = 0.01). There was no change in average peak coronary flow velocity, suggesting LV stunning 30 minutes post balloon occlusion rather than ongoing ischemia; the definition of the former stipulates LV dysfunction in the setting of normal epicardial flow, whereas the latter would be expected to have resulted from reduced flow and lower average peak coronary flow velocity, which was not the case. Simultaneously acquired coronary wave intensity analysis assessment revealed a decrease in peak BEW intensity from −9.95±5.45 W∙m⁻²/s²×10⁵ at baseline to −7.52±5.00 W∙m⁻²/s²×10⁵ (difference 2.43×10⁵ [0.20×10⁵ to 4.67×10⁵; P = 0.04) at 30 minutes post balloon occlusion. We also found a numeric, but not statistically significant, reduction in average peak coronary flow velocity, suggesting LV stunning 30 minutes post balloon occlusion rather than ongoing ischemia; the definition of the former stipulates LV dysfunction in the setting of normal epicardial flow, whereas the latter would be expected to have resulted from reduced flow and lower average peak coronary flow velocity, which was not the case. Simultaneously acquired coronary wave intensity analysis assessment revealed a decrease in peak BEW intensity from −9.95±5.45 W∙m⁻²/s²×10⁵ at baseline to −7.52±5.00 W∙m⁻²/s²×10⁵ (difference 2.43×10⁵ [0.20×10⁵ to 4.67×10⁵; P = 0.04) at 30 minutes post balloon occlusion. We also found a numeric, but not statistically significant, reduction

Table 2. Hemodynamic Assessment Pre and 30 Minutes Post Balloon Occlusion

<table>
<thead>
<tr>
<th></th>
<th>Baseline (n=12)*</th>
<th>30 min Post Balloon Occlusion (n=12)</th>
<th>Mean Difference (95% CI)/Median Difference (25th–75th Percentile)*</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left ventricle</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ESV, mL</td>
<td>60.2±17.6</td>
<td>69.1±22.0</td>
<td>8.9 (0.0 to 17.8)</td>
<td>0.053</td>
</tr>
<tr>
<td>EDV, mL</td>
<td>150.6±44.0</td>
<td>144.0±42.4</td>
<td>-6.6 (-13.8 to 0.5)</td>
<td>0.066</td>
</tr>
<tr>
<td>SV, mL</td>
<td>90.4±26.4</td>
<td>74.9±26.5</td>
<td>-15.5 (-22.7 to -8.3)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>EF, %</td>
<td>60.8±4.5</td>
<td>51.5±7.7</td>
<td>-9.3 (-16.6 to -2.0)</td>
<td>0.017</td>
</tr>
<tr>
<td>EDP, mmHg</td>
<td>15.0±4.2</td>
<td>15.2±4.2</td>
<td>0.2 (-1.3 to 1.8)</td>
<td>0.75</td>
</tr>
<tr>
<td>dP/dt max, mmHg∙s⁻¹</td>
<td>1437.1±163.9</td>
<td>1299.4±152.9</td>
<td>-110.4 (-183.3 to -70.4)*</td>
<td>0.015</td>
</tr>
<tr>
<td>dP/dt min, mmHg∙s⁻¹</td>
<td>-1973.9±411.2</td>
<td>-1956.9±393.7</td>
<td>17.0 (9.8 to 24.2)</td>
<td>0.79</td>
</tr>
<tr>
<td>T, ms</td>
<td>48.3±7.4</td>
<td>52.4±7.9</td>
<td>4.1 (1.3 to 6.9)</td>
<td>0.01</td>
</tr>
<tr>
<td>Coronary WIA</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Basal APV, cm∙s⁻¹</td>
<td>15.2±3.9</td>
<td>15.3±4.6</td>
<td>0.08 (0.05 to 0.11)</td>
<td>0.86</td>
</tr>
<tr>
<td>Peak eFCW, W∙m⁻²/s²×10⁵</td>
<td>11.68±8.69</td>
<td>6.81±3.80</td>
<td>-2.81 (-8.69 to 0.98)*</td>
<td>0.08</td>
</tr>
<tr>
<td>Peak BEW, W∙m⁻²/s²×10⁵</td>
<td>-9.95±5.45</td>
<td>-7.52±5.00</td>
<td>2.43 (0.20 to 4.67)</td>
<td>0.04</td>
</tr>
</tbody>
</table>

*Denotes median difference (25th–75th percentile) rather than mean difference (95% CI).
in peak eFCW intensity from 11.68±8.69 W·m⁻²s⁻¹×10⁵ at baseline to 6.81±3.80 W·m⁻²s⁻¹×10⁵ 30 minutes post balloon occlusion (median difference −2.81(−8.69 to 0.98); P=0.08). Figure 5 shows an example of wave intensity analysis for 2 patients before and 30 minutes after a 1-minute balloon occlusion, showing a reduced BEW at 30 minutes.

Discussion

We demonstrate, for the first time in patients with coronary disease, the relationship between coronary wave intensity and simultaneously acquired measurements of LV systolic and diastolic performance. Using the technique of WIA, we have demonstrated a close association between LV diastolic performance and diastolic coronary flow, corroborating our understanding that the BEW is generated by myocardial relaxation. In addition, our finding that induced changes in LV diastolic performance are associated with similar changes in BEW intensity supports the widely held belief that the myocardium and coronary flow are intimately linked.

**BEW and Myocardial Relaxation**

We have demonstrated a direct correlation between indices of diastolic function (LV 𝛿 and LV dP/dtᵁ) and peak BEW. This is consistent with the mechanistic explanation of the origin of the BEW; decompression of the microvasculature in early diastole due to myocardial relaxation results in the suction of blood from the coronary artery into the microvasculature. In such circumstances, the rate of myocardial relaxation, represented by dP/dtᵁ, and the time constant of LV isovolumic relaxation (τ) would be expected to be related to the rate of microvascular decompression, and therefore the amplitude of the BEW. BEW wave energy is territory dependent and has been shown to fall with a reduction in perfused myocardial mass.¹⁵ BEW intensity is likely to be related to a combination of perfused myocardial mass and LV diastolic performance. We intentionally selected left anterior descending stenoses to attempt to control for myocardial mass, and differences in wave intensity analysis attributed to different myocardial territories. We performed a paired assessment using the patient as their own control to assess the influence of LV stunning. Increasing LV hypertrophy is associated with not only diastolic dysfunction¹⁶ but also increased perfused myocardial mass, although the associated BEW is smaller.⁴ Diastolic dysfunction is associated with an adverse prognosis in hypertensive patients, independent of LV mass or blood pressure control¹⁷ and also occurs in those with dilated cardiomyopathy. Our results and those of others⁴ suggest that these patients have a blunted BEW, microvascular dysfunction, and potentially a mechanism for reduced tissue perfusion. However, whether there are any sequelae to a reduced BEW with respect to clinical outcomes in these patients is not yet clear.

**Wave Intensity Analysis and Systolic Function**

We did not demonstrate a statistically significant relationship between LV systolic function and eFCW intensity. This is
somewhat surprising given that we observed changes in systolic function (dP/dt max was reduced at 30 minutes) and dP/dt max is closely related to LV ejection fraction, and LV ejection is the driver of the eFCW. This might simply be the result of our small sample size; indeed, if a small number of outliers were excluded, there is a suggestion of the expected positive correlation between the eFCW and dP/dt max. Measurement of forward waves distal to a coronary stenosis can have the effect of abrogating them. The effect of significant coronary stenoses on the eFCW amplitude may therefore have blunted any observable association.

**Effect of Myocardial Stunning on Wave Intensity**

Transient impairment of myocardial contractility after myocardial ischemia, despite normal coronary flow is termed myocardial stunning and has been demonstrated in humans. We have now confirmed that stunned myocardium correspondingly diminishes BEW intensity and to a lesser extent eFCW. These findings confirm that myocardial performance and coronary flow are intimately linked helping match supply with demand. The more marked reduction in the BEW during stunning compared with the eFCW may be a chance finding as it was expected that both would be similarly reduced. Alternatively, perhaps local stunning affects the distally arising BEW more so than the proximal eFCW. In this model, all myocardium responsible for the generation of BEW is stunned, whereas the eFCW, propelled forward by global LV function, is only partially affected. Nevertheless, microcirculatory dysfunction has been demonstrated remote from the target lesion vessel and this may be mediated by LV stunning affecting adjacent territories.

![Figure 5](http://circinterventions.ahajournals.org/Downloadedfrom) Two examples of wave intensity analysis before and after myocardial stunning. Wave intensity analysis (top), ensemble averaged coronary pressure (bottom, solid line), and flow velocity (bottom, dashed line) measured before (left) and 30 min after (right) a 1-min balloon coronary occlusion to induce myocardial stunning. The starred wave is the backwards expansion wave.
Despite some suggestions for clinical utility of WIA, at present it remains largely a research tool. However, the clear link that we demonstrate between LV mechanics and coronary wave intensity supports a potential role for the technique as a surrogate in investigating serial changes in LV diastolic performance from intracoronary measurements. This could be applied to the investigation of cardio-protection strategies without the need for peri-procedural direct LV assessment.

Clinical Implications

We have demonstrated that BEW intensity is closely related to LV diastolic function and is reduced in myocardial stunning induced during PCI. This may have implications in assessing residual disease and a PCI result by instantaneous wave free ratio, as diminishing the BEW may reflect an increase in microcirculatory resistance that could result in an over-estimation of the true instantaneous wave free ratio. It has been suggested that reduced BEW intensity might have future utility in predicting myocardial viability. Our data confirm that stunned myocardium similarly reduces BEW intensity and caution against deferring revascularization simply on the amplitude of the BEW, particularly in the acute setting.

Limitations

This is a single-center study, with a small number of patients. However, it is the first to compare simultaneous LV assessment and coronary wave intensity analysis in patients with coronary disease, in whom WIA is most likely to find a clinical application. We are unable to confirm that LV function fully recovered, fulfilling the strict definition of stunning, although we think it as highly unlikely that LV dysfunction persisted.

The sum of squares method for the assessment of coronary wave intensity has been validated in subjects with normal coronary arteries, and we make the assumption that coronary disease does not alter wave speed. Assessment of stenosed coronary arteries may alter wave intensity and, given that the hemodynamic severity of those lesions assessed will have varied, this may have confounded our results. This might be particularly relevant for the measurement of eFCW as coronary stenosis diminishes the magnitude of forward waves measured distal to it to a greater extent than backward waves which originate distally. This could have led to a type II error for eFCW, but does not negate the positive result observed for the BEW. The size of the study is such that additional variables such as stenosis severity cannot be controlled for statistically. However, the demonstrated correlation between BEW and indices of diastolic function, with a fall in BEW amplitude in parallel with a reduction in LV diastolic performance, makes a chance association implausible.

The myocardium influences coronary flow through the microvasculature in systole as well as diastole. Myocardial contraction results in microcirculatory compression which can be seen as a compression wave travelling from the microvasculature to the aorta in early systole, known as the early backwards compression wave. The early backwards compression wave has been shown in animal models to be related to LV systolic function, something one might expect based on our findings given that its mechanism is the reverse of BEW. We have found it to be frequently merged and difficult to separate from the late backwards compression wave, which arises because of the reflection of eFCW from stenoses, bifurcations, and microvasculature. We therefore elected not to investigate it.

Finally, there is a possibility that a low-pressure balloon occlusion modified the stenosis in the interrogated vessel. We have demonstrated this not to be the case angiographically and physiologically, but we would expect the effect of such modification to have a positive effect on wave intensity rather than the negative effect we observed with stunning.

Conclusions

The peak BEW assessed by coronary WIA correlates with simultaneous invasively derived indices of LV diastolic function and mirrors changes in LV diastolic function, confirming a link between the coronary BEW and LV diastolic function.

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Disclosures

None.

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