Assessment of 3D motion increases the applicability of accelerometers for monitoring left ventricular function†

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Abstract

OBJECTIVES: Miniaturized accelerometers attached to the epicardium have been shown to provide useful clinical information. However, attachment of such a sensor has been cumbersome due to requirement of aligning the three sensor axes with the cardiac coordinate axes, limiting clinical utility. We propose a new method to process the three-dimensional (3D) accelerometer signal that does not require such alignment.

METHODS: In 20 open-chest pigs, miniaturized 3D accelerometers were fixated on the epicardium in apical and basal regions of left ventricle. Accelerations in circumferential, longitudinal and radial directions were measured and a 3D velocity vector was calculated. Systolic velocity along the 3D vector and velocities in circumferential, longitudinal and radial directions were compared with the positive time derivative of left ventricular pressure during changes in global left ventricular function (epinephrine, esmolol and fluid loading) and to strain echocardiography during left anterior descending artery occlusion.

RESULTS: Distinct changes in all accelerometer velocities were observed during alterations on global and regional left ventricular function. Accelerometer 3D and circumferential systolic velocities in apical region best reflected left ventricular function during interventions on global function by correlating significantly with the positive time derivative of left ventricular pressure, \( r = 0.83 \) and \( r = 0.86 \), respectively. The accelerometer 3D velocity also demonstrated equally good capacity as circumferential velocity in discriminating coronary occlusion from interventions on global left ventricular function with sensitivity/specificity of 0.90/0.83 and 0.90/0.86, respectively.

CONCLUSIONS: Accelerometer 3D systolic velocity showed very good correspondence to changes in global and regional left ventricular function. Our results demonstrate that by the use of the accelerometer 3D motion vector, no alignment of the sensor with the cardiac coordinate axes was required. This increases potential clinical applicability of the accelerometer in cardiac surgery.

Keywords: Left ventricular function • Cardiac monitoring • Motion sensor

INTRODUCTION

Accelerometers are motion sensors, which placed epicardially on the heart during surgery, can provide detailed information of heart wall movement and ventricular function [1–3]. The accelerometer signal can be transformed to estimate heart motion expressed by acceleration, velocity or displacement [4, 5]. By combining electrocardiograms (ECGs) and accelerometer signals, it is possible to measure heart motion within different phases during the cardiac cycle [2, 6], similarly to what is achievable with tissue Doppler imaging and myocardial strain analysis by echocardiography [7]. The use of echocardiography postoperatively is resource demanding and impractical. Analyses of myocardial velocities and deformations are complicated and have to be performed off-line. This limits clinical utility of the method in cardiac surgery. A benefit of the accelerometer technology is that it provides a real-time signal that enables automated beat-to-beat analysis of the heart motion. In addition, by use of a three-axis [three-dimensional (3D)]-accelerometer, heart wall motions can be assessed in three dimensions [8, 9]. The 3D motion of the heart depends on myocardial structure and function and may be different in patients with coronary artery...
disease and patients with severe aortic stenosis and left ventricular (LV) hypertrophy. A 3D method, which enables automatic determination of the main contraction direction of the LV wall, may simplify the monitoring of the patient.

Our previous studies on the technology have focused on single axis analysis where sensor orientation had to be taken into account. Precise alignment of the accelerometer with the LV contraction coordinates may be impractical, thereby limiting potential clinical use of the method. We suggest that this can be solved by the use of 3D data from the sensor in order to automatically determine a 3D motion vector pointing in the direction of maximum systolic motion.

The aims of this study were to investigate epicardial motion patterns by a 3D accelerometer in two different LV regions, and to evaluate an accelerometer 3D motion vector in quantifying changes in global and regional LV function induced by pharmacological interventions or coronary artery occlusion. We hypothesized (i) that accelerometers in two different LV regions had similar capability to monitor changes in global LV function and (ii) that an accelerometer-generated 3D motion vector had similar ability for quantifying changes in LV function as the best-aligned single accelerometer axis.

MATERIALS AND METHODS

Animal preparation

Twenty Norwegian Landrace pigs of either sex (weight: 51 ± 6 kg) were used. The study protocol was approved by the Norwegian Animal Research Authority. Anaesthesia was induced by intravenous pentobarbital 2–3 mg/kg and morphine 0.5–1.0 mg/kg until tracheotomy was completed. General anaesthesia was continued with inhaled isoflurane (1.0–1.5%) and morphine infusion (0.15–0.2 mg/kg). Mechanical ventilation was performed by a Siemens KION 6.0 anaesthesia machine (Solna, Sweden), with inspired oxygen fraction 0.35 and tidal volume/ventilation rate adjusted to keep arterial PCO₂ ≈ 5.5 kPa. Ringer’s acetate was given at the rate of 1000 ml/h to keep a diuresis >1 ml/kg/h. No blood transfusions were given.

The right carotid artery and left atrial appendage were cannulated using 8-French introducers to allow insertion of micromano- metres (MPC-500, Millar Instruments, TX, USA) into the cavities of aorta, LV and left atrium for continuous pressure registrations. A median sternotomy was performed and the pericardium opened. An inflatable occluder (In Vivo Metric, CA, USA) was placed after the second diagonal branch of left anterior descending artery (LAD) and coronary flow measured by a 4-mm ultrasonic flow probe (Medistim, Oslo, Norway) placed distally to the occluder. A 16-mm flow probe was placed on the ascending aorta for measuring cardiac output. One 3D accelerometer (KXM52-1040, Kionix, Inc., NY, USA) with dimension of 5 x 5 x 2 mm was sutured by two stitches (Prolene 6-0) to the LV anterior apical region, supplied by the LAD, and another to the lateral basal region of LV, supplied by the circumflex coronary artery (CX) (Fig. 1A). The accelerometers were randomly attached in three different orientations; one with the sensor pointing to the apex (Fig. 1A), one with the sensor point- ing towards the basis of the LV (180° difference) and one with the sensor oriented 90° to the left in Fig. 1A. This still enabled identification of the circumferential, longitudinal and radial motions. The chest and the pericardium were left open and the pig was placed in dorsal supine position.

From the Millar catheter, LV peak systolic pressure and LV end-diastolic pressure were measured and positive (dp/dt_max) and negative (dp/dt_min) time derivatives calculated. Stroke volume was defined from the start of R-peak on ECG to LV dp/dt_min. Stroke volume (SV) was calculated by dividing cardiac output on heart rate.

Experimental protocol

Global LV function was modified by intravenous boluses of epinephrine (10 μg) to increase contractility, esmolol (100 mg) to decrease contractility and fluid loading [500 ml colloid (Voluvene*) to enhance preload. Regional dysfunction was induced by a 3-min LAD occlusion. Complete occlusion of LAD was verified by measuring zero LAD flow and abnormal regional contraction by echo- cardiography. Blinded drawing of lottery slip randomized sequence of interventions on global LV function. After each intervention on global function, the pig recovered for at least 15 min to regain stable haemodynamic baseline values. LAD occlusion was always performed after the last intervention on global function due to the risk of ventricular fibrillation. The pig recovered in 45 min after the interventions on global function before LAD were occluded. Baseline measurements were undertaken before each intervention.

Echocardiography

The echocardiographic examination was performed by a Vivid 7 scanner with a 2.5 MHz probe (GE Vingmed AS, Norway). Conventional two-dimensional greyscale short-axis images were obtained from the LV basal and apical regions, above and beneath the level of the papillary muscles, respectively. From these images, myocardial circumferential peak systolic strain in apical anterior and basal lateral segments was measured using the speckle tracking method [10] and used as the reference for determining regional dysfunction [11]. Systolic strain represents change in segment length (L) during systolic contraction (ES) relative to end-diastolic length (ED) in percent ([LES − LED]/LED * 100%). Negative strain values signify segmental shortening. A more negative value therefore represents increased contraction, whereas less negative strain represents reduced contraction. Mean strain from three consecutive heartbeats was used for statistical calculation. The frame rate was 62 frames/s. EchoPAC (Version 112, GE Vingmed Ultrasound AS, Norway) was used for off-line echocardiographic analysis.

Accelerometer

The acceleration signals were collected by a NI USB 6009 AD converter (National Instruments, Inc., TX, USA), using the LabVIEW software (National Instruments, Inc.). A high-pass filter was used to remove effects of gravity and breathing motions on the signals. Synchronous and continuous sampling of accelerometer signals, ECG and pressures (500/s) allowed measurement of heart motions in different phases within the cardiac cycle in longitudinal, circumferential and radial directions in LV regions. One of the three sensor orientations is shown in Fig. 1A. A customized Matlab algorithm (The MathWorks, Natick, MA, USA) was used to automatically calculate the accelerometer variables. Acceleration signals in a 10 s interval at all baselines and interventions were analysed ensuring that all heartbeats in at least one ventilation cycle were included. Mathematical time integration of the acceleration signals was performed to obtain the corresponding epicardial...
velocities and displacements (Fig. 1B). The greatest changes in LV function by epicardially attached accelerometers are seen in early systole [12], and therefore for each axis peak systolic velocity within a 150 ms time interval after ECG R-peak ($V_{sys}$) was automatically measured and used as accelerometer indicators for LV systolic function (Fig.1B). All accelerometer analyses were calculated automatically, and intra- and interobserver variability was not an issue.

Calculation of accelerometer three-dimensional $V_{sys}$. The 3D analysis was performed without information on placement of the sensors. A customized algorithm was used to automatically calculate the accelerometer 3D motion vectors in both LV regions:

1. Automatic detection of peak R on ECG [13] and calculation of the 3D motion of the epicardial accelerometer, that is, the 3D displacement loops (Fig. 2), which is based on the displacement in all three directions during one heart cycle (Fig.1B). ECG R-peak was used to determine start and end of the 3D displacement loop (Fig. 2A, left panel).

2. Assessment of 3D reference vector: the vector found from the 3D displacement loop starting at the ECG R-peak to the position furthest away in space from this point during the heart cycle was extracted (Fig. 2A, left panel). This motion vector obtained at baseline (Fig. 2A, bold arrow left panel) represented the 3D reference vector obtained by the accelerometer.

3. Calculation of displacement along the 3D reference vector and thereafter velocity by time differentiation (Fig. 2B, left panel).

4. Automatic calculation of peak systolic velocity within the 150 ms period after peak R on ECG along the 3D reference vector ($3D V_{sys}$). The 3D $V_{sys}$ was evaluated in assessment of LV function in this study.

The 3D motion vector (Fig. 2A, bold arrow) could be influenced by the magnitude of ventricular contraction (increase or decrease in length of 3D motion vector) and main direction of contraction (change in 3D motion vector angle). Subsequent recorded motion during the different interventions was then projected onto the 3D reference vector from baseline (Fig. 2A, right panel).

Statistical analysis

Sample size was calculated on the basis of estimates from pilot experiments with mean difference in response to accelerometer 3D $V_{sys}$ of 4 cm/s ($\delta$), standard deviation (SD) of 5 cm/s ($\sigma$) and $\alpha$ of 0.05. The null hypothesis could be rejected with probability (power) of 0.8 using 14 animals. Parametric statistical methods were used (data showed normal distribution for each intervention), and data are presented as mean ± SD if otherwise not noted. Two-tailed paired-sample t-tests were used for the repeated measurements with the Bonferroni correction of $P$-values. No differences were found between baseline values before each intervention so the first baseline was used for comparison with interventions on LV function (Table 1). Receiver-operating characteristic (ROC) curves were constructed to determine cut-off values for ischaemia detection by accelerometer and to assess sensitivity and specificity to discriminate regional dysfunction from changes in global LV function. Accelerometer measurements were correlated to LV dP/dt and cardiac output by use of Spearman correlation coefficient, as these data did not show normal distribution. $P < 0.05$ was considered significant. Statistical analysis was performed by SPSS (Version 18, SPSS, Inc., IL, USA).
Figure 2: (A) Automatic calculation of accelerometer 3D $V_{sys}$. Accelerometer epicardial 3D displacement loops in LV apical region from 14 heartbeats (10 s measurement interval) during baseline and LAD occlusion. A 3D reference vector (bold arrow, left panel) was extracted at baseline from the sensors position at ECG R-peak to the furthest distance from this point, which corresponded closely to end systole (squares). The baseline 3D reference vector is also shown in the right panel, visualizing that a completely different LV motion occurred during LAD occlusion. Sensor displacement during the first 150 ms after ECG R-peak is marked with blue colour and projected onto the 3D reference vector (dashed arrow). (B) Displacement along the 3D reference vector (upper panel) and the corresponding velocity along the 3D reference vector (mid panel). 3D $V_{sys}$ (arrow) represents peak systolic velocity within the 150 ms (blue segment) along the 3D reference vector and was used as the accelerometer 3D measure of LV systolic function. The effects of LAD occlusion on 3D displacement and 3D velocity are shown in right panels. LV: left ventricular; LAD: left anterior descending artery; ECG: electrocardiogram; 3D: three-dimensional.

Table 1: Baseline characteristics

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<th></th>
<th>First BL</th>
<th>BL epinephrine</th>
<th>BL fluid</th>
<th>BL esmolol</th>
<th>BL LAD occlusion</th>
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<tbody>
<tr>
<td>Heart rate (beats/s)</td>
<td>86 ± 15</td>
<td>86 ± 11</td>
<td>89 ± 13</td>
<td>87 ± 14</td>
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<td>LV peak systolic pressure (mmHg)</td>
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<td>LV end-diastolic pressure (mmHg)</td>
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<td>14 ± 5*</td>
<td>11 ± 3</td>
<td>12 ± 3</td>
<td>13 ± 4*</td>
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<td>LV $dP/dt_{max}$ (mmHg/s)</td>
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<td>1475 ± 335</td>
<td>1401 ± 375</td>
<td>1395 ± 359</td>
<td>1497 ± 358</td>
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<tr>
<td>Cardiac output (l/min)</td>
<td>3.9 ± 1.7</td>
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<td>4.0 ± 1.9</td>
<td>4.1 ± 1.9</td>
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<tr>
<td>Stroke volume (ml)</td>
<td>45.2 ± 14.5</td>
<td>46.6 ± 16.2</td>
<td>41.8 ± 13.9*</td>
<td>46.0 ± 18.6</td>
<td>46.7 ± 20.4</td>
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Data presented as mean ± SD. Paired sample t-test with Bonferroni adjusted P-values for multiple comparisons. Comparison with first baseline: *P < 0.05.
BL: baseline; LV: left ventricular.
RESULTS

Two accelerometers in the LV basal region lost function in one axis and calculation of 3D V_{sys} was therefore precluded for these individuals. In one pig, the pressure catheter in the LV cavity failed. For other variables, complete data were obtained for all pigs during LAD occlusion and in 17 pigs for the interventions with epinephrine and fluid loading and in 15 pigs for the intervention with esmolol.

Haemodynamic variables

The pigs remained circulatory stable during the experiment with no great changes in haemodynamic baseline values (Table 1), compared with haemodynamic changes observed during the interventions on global and regional LV function. No significant changes in haemoglobin and lactate were seen. From the start to the end of the experiment, a minor change in haemoglobin was observed (8.4 ± 0.7–7.3 ± 0.7 g/dl), whereas the serum lactate concentration was unaltered, from 1.1 ± 0.2 to 1.2 ± 0.3 mmol/l. The interventions on global LV function induced significant haemodynamic changes (Table 2). Epinephrine increased heart rate, LVP, LV dp/dt_{max} and cardiac output for significance, whereas the opposite changes were seen for esmolol except for heart rate, which was unaltered. Fluid loading induced increase in LVP, end-diastolic pressure, cardiac output and stroke volume, while heart rate remained unchanged.

LAD occlusion reduced LVP, LV dp/dt_{max} and increased end-diastolic pressure, whereas the increase in heart rate and reduction in cardiac output were not significant. Compared with LAD occlusion, esmolol induced a greater reduction in global LV contractility as assessed by LV dp/dt_{max} (P < 0.05). Heart rate was higher during LAD occlusion than during the intervention with esmolol (P < 0.05).

Strain echocardiography

Representative echocardiographic images were obtained in 18 animals. For the alterations on global LV function, a significant change in myocardial strain was observed for the intervention with epinephrine (Table 2). The greatest change in strain was found in apical anterior region during LAD occlusion (P < 0.01) with a positive strain value observed, that is, systolic myocardial stretching, which is a characteristic of myocardial ischaemia [10]. In this region, strain was significantly more affected during LAD occlusion than during esmolol infusion (P < 0.01). Strain in basal region was unaltered during LAD occlusion.

Accelerometer systolic velocities

Accelerometer velocities at baseline. The accelerometer velocity traces in apical and basal LV regions looked similar to tissue velocity traces achievable by echocardiography (Figs 1B and 4) [7]. Within one heart cycle, it was easy to identify both systolic (V_{sys}) and diastolic velocities (v’ and a’) in all accelerometer traces. In the apical region, the dominating systolic motion by accelerometer was observed in circumferential direction (Figs 1B, 2A and 3A) whereas in the basal region longitudinal motion dominated (Fig. 3B). Epicardial radial motion at baseline was minor in both LV regions.

Interventions on global left ventricular function. Of the accelerometer velocities in the LV apical region, circumferential and 3D V_{sys} changed most when altering global LV function, whereas in the basal region longitudinal, circumferential and 3D V_{sys} most precisely reflected global LV function (Fig. 3). Radial velocities did not change significantly during any intervention on global function, except for the intervention with epinephrine by the accelerometer in apical region.

The correlation between circumferential V_{sys} and 3D V_{sys} in apical region during interventions on global LV function was r = 0.93 (P < 0.001). Similar good correlations were found for these two variables against LV dp/dt_{max} [r = 0.86 (P < 0.001) and r = 0.83 (P < 0.001), respectively] and cardiac output (both r = 0.76, P < 0.001).

In the basal region, longitudinal V_{sys} and 3D V_{sys} correlated significantly (r = 0.93, P < 0.001), but less strong correlations were found between these accelerometer measurements and LV dp/dt_{max} and cardiac output: longitudinal V_{sys} against LV dp/dt_{max} r = 0.71 (P < 0.001) and cardiac output r = 0.61 (P < 0.001), and 3D V_{sys} against LV dp/dt_{max}, r = 0.70 (P < 0.001) and cardiac output, r = 0.58 (P < 0.001). To summarize, the accelerometer measurements in LV apical region most precisely reflected global LV function.

Table 2: Haemodynamic and echocardiographic data during interventions on global and regional LV function

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Epinephrine</th>
<th>Fluid</th>
<th>Esmolol</th>
<th>LAD occlusion</th>
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<tbody>
<tr>
<td>Heart rate (beats/s)</td>
<td>86 ± 15</td>
<td>133 ± 25*</td>
<td>88 ± 11</td>
<td>82 ± 12</td>
<td>93 ± 10*</td>
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<tr>
<td>LV peak systolic pressure (mmHg)</td>
<td>88 ± 14</td>
<td>130 ± 24*</td>
<td>97 ± 12</td>
<td>72 ± 9*</td>
<td>77 ± 14*</td>
</tr>
<tr>
<td>LV end-diastolic pressure (mmHg)</td>
<td>10 ± 4</td>
<td>10 ± 6</td>
<td>15 ± 7*</td>
<td>11 ± 5</td>
<td>13 ± 6*</td>
</tr>
<tr>
<td>LV dp/dt_{max} (mmHg/s)</td>
<td>1442 ± 370</td>
<td>4085 ± 2031*</td>
<td>1546 ± 263</td>
<td>861 ± 223</td>
<td>1167 ± 295*</td>
</tr>
<tr>
<td>Cardiac output (l/min)</td>
<td>3.9 ± 1.7</td>
<td>6.1 ± 2.6*</td>
<td>4.5 ± 1.6*</td>
<td>3.3 ± 1.9*</td>
<td>3.5 ± 2.0</td>
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<tr>
<td>Stroke volume (ml)</td>
<td>45.2 ± 14.9</td>
<td>46.2 ± 13.9</td>
<td>51.8 ± 17.6*</td>
<td>41.5 ± 21.3</td>
<td>38.1 ± 20.1</td>
</tr>
<tr>
<td>LV apical systolic circumferential strain</td>
<td>−23.5 ± 4.8</td>
<td>−31.6 ± 8.2*</td>
<td>−23.3 ± 7.2</td>
<td>−20.4 ± 4.3</td>
<td>9.7 ± 8.4*</td>
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<tr>
<td>LV basal systolic circumferential strain</td>
<td>−24.4 ± 6.5</td>
<td>−29.6 ± 9.1*</td>
<td>−24.1 ± 5.2</td>
<td>−20.9 ± 5.2</td>
<td>−25.9 ± 6.5</td>
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</table>

Data presented as mean ± SD. Paired sample t-test with Bonferroni adjusted P-values for multiple comparisons.

Comparison with baseline: *P < 0.05 and †P < 0.01.

Comparison between esmolol and LAD occlusion: †P < 0.05 and †P < 0.01.

LV: left ventricular; LAD: left anterior descending artery.
values for circumferential \( V_{\text{sys}} \) (6.6 cm/s) and 3D \( V_{\text{sys}} \) (8.7 cm/s) were substantially different from the mean values obtained for these two variables during the interventions on global LV functions (Fig. 5). This implied very good discrimination of regional dysfunction with similar high sensitivity and specificity (90 and 86%, 90 and 83%, respectively).

Except for radial \( V_{\text{sys}} \), LAD occlusion also reduced accelerometer velocities in the basal non-ischaemic CX-supplied region (Fig. 3B). The relative reductions in longitudinal, circumferential and 3D \( V_{\text{sys}} \) were similar. These findings demonstrated that the accelerometer cannot be regarded a strict regional method for ischaemia detection as the regional myocardial strain in the CX-supplied region was unaltered (Table 2).

**DISCUSSION**

The main result in this study was that the accelerometer 3D velocity method performed equally well as the best accelerometer single axis velocity in quantifying changes in LV function. The 3D method was independent of the sensor orientation on the LV surface. This implies that the sensor could be attached to the LV without need for precise alignment to the cardiac contraction coordinates. This increases potential clinical applicability of the accelerometer.

LV motions reflect LV function, and abnormalities in wall motion are early signs of complications [2, 14]. A prerequisite for using wall motions as indicators for LV performance is extensive knowledge of the contraction patterns within different regions of the heart. In the LV apical region, circumferential motion in the counter clockwise direction dominates, whereas in basal regions longitudinal motion predominates [15]. Radial epicardial motion is considered minor [16]. These normal motion patterns were clearly detected by the accelerometer and by combing accelerometer and ECG signals, systolic and diastolic heart phases within the heart cycle were easily identified as seen in Fig. 4. Importantly, the accelerometer provides this information continuously and allows real-time automatic analysis and presentation of signals. The accelerometer technology has been found superior to haemodynamics and ST-segment monitoring in detection of regional ischaemia and has almost similar ability as echocardiographic strain to detect LV regional dysfunction [2, 8]. Echocardiography and magnetic resonance imaging (MRI) provide structural and functional assessment on cardiac function, and are both non-invasive. However, their use is intermittent, and requires skilled examiners and interpretation. MRI is not suitable for perioperative use.

A finding in our study was that optimal site for monitoring global LV function was in the LV apical region. In this region, normal wall motion is dominated by rotation [15], and consistently accelerometer circumferential \( V_{\text{sys}} \) in apical region was highly correlated to both LV \( \frac{dP}{dt_{\text{max}} \text{ and cardiac output. Not surprisingly, the greatest change in accelerometer measurements during LAD occlusion was also found in this region, and circumferential } V_{\text{sys}} \text{ demonstrated high sensitivity and specificity for detecting regional myocardial ischaemia. The accelerometer cannot be regarded as a strictly regional method for detecting ischaemia as also the accelerometer velocities in basal region of LV were reduced during LAD occlusion, despite unchanged myocardial strain in this region. These results imply that epicardial motions by accelerometer are affected by function in other segments as well (tethering effect) similar to tissue velocity measurements by echocardiography [17]. This may not be a disadvantage as one sensor may be

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**Intervention on regional left ventricular function.** LAD occlusion induced significant reductions in accelerometer \( V_{\text{sys}} \) in both LV regions, but the changes were more pronounced in the intervention region (Fig. 3). In the apical region, similar to interventions on global LV function, circumferential and 3D \( V_{\text{sys}} \) performed best and the reductions in these accelerometer variables were significantly greater during LAD occlusion than during esmolol infusion (\( P < 0.01 \)) (Figs 3A and 4). The cut-off values obtained by ROC analyses represent the measured values with maximal sensitivity and specificity for detection of regional dysfunction. The cut-off
sufficient for monitoring several LV regions perfused by different coronary arteries.

Three-dimensional motions by accelerometer

Using only one accelerometer axis for monitoring LV function can be a problem. The motions obtained from the longitudinal, circumferential and radial axes are very different (Fig. 1A). Consequently, if a sensor axis is not perfectly aligned to the cardiac contraction coordinates, the readings will become inaccurate. For example: if the y-axis, which should be parallel to the circumferential direction with the highest velocity (Fig. 1B), was oriented between the circumferential and longitudinal contraction directions, the $V_{sys}$ along the y-axis (supposedly circumferential) would be reduced on expense of increased $V_{sys}$ in the x-axis (supposedly oriented in the longitudinal direction). In worst-case, myocardial ischaemia may not be detected due to such misalignment. A future possibility is that the sensor can be integrated in a temporary pacemaker lead and placed subendocardially. With such a design, a potential problem can be rotation of the sensor and change of axis orientation over time, making monitoring of LV function by measuring only one motion vector uncertain. Precise alignment of the sensor to the cardiac coordinates can be time-consuming and cumbersome. This may limit the probability of using the modality on a routine basis. The main finding in our study was that use of accelerometer 3D data and generation of a 3D motion vector could solve these problems. The dominant LV 3D motion vector and the

![Figure 4: Representative velocity traces in the circumferential direction and along the 3D reference vector over one cardiac cycle in the LV apical region at baseline and during interventions on global and regional LV function. The measurement period of 150 ms from ECG R-peak is marked with blue and $V_{sys}$ indicated. The diastolic early ($e'$) and atrial ($a'$) velocities are also indicated. LV: left ventricular; ECG: electrocardiogram; 3D: three-dimensional.]
successively 3D $V_{sys}$ were automatically determined without knowledge of the sensor placement on the LV surface. In both LV regions, this variable showed similar good capability as the best-aligned single axis $V_{sys}$ in quantifying changes in both global and regional LV function. These results demonstrate that alignment of sensor to the LV contraction coordinates was not necessary.

Our proposed 3D method used maximal total displacement during the cardiac cycle (3D displacement loop) as basis for calculating the reference 3D motion vector shown in Fig. 2A. As seen from this figure, circumferential motion dominated in the apical LV region and hence the calculated 3D motion vector was relatively parallel to the circumferential motion vector. The 3D systolic velocity curves in this region therefore appeared similar to the circumferential systolic velocity traces during all interventions on LV function as demonstrated in Fig. 4, even during regional dysfunction where the greatest quantitative and qualitative changes in accelerometer $V_{sys}$ were observed.

**Clinical perspectives**

The accelerometer and echocardiography techniques are supplemental rather than competing modalities, where the former provides continuous real-time assessment and the latter can provide more precise diagnostics. Miniaturized accelerometers may prove a valuable future method to detect complications and for evaluating effects of medical treatment in cardiac surgery. Recently, a smaller 3D accelerometer with dimension of $1.2 \times 1.5 \times 0.8$ mm (Bosch BMA 355, Germany) has become available. Together with the findings from our study, technological progress and miniaturization increase the probability for accelerometers to be routinely used in patients. Today, placement of temporary pacemaker leads to the heart is standard procedure in cardiac surgery. Incorporation of a miniaturized 3D accelerometer in such a pacemaker lead would allow continuous monitoring of LV function after surgery.

Accelerometers also have potential for improving continuous perioperative monitoring of right ventricular (RV) function, for instance, in the treatment of end-stage heart failure with a LV assist device, either as a stand-alone device or as an integrated part of the device system; however, this needs further investigations.

**Limitations**

Longitudinal contraction in the LV is reduced by pericardiotomy, but is normalized after chest closure [18]. This may explain the observed lesser performance of the accelerometer placed in the basal region of the LV as epicardial longitudinal motion predominates in this region. The size of the accelerometer used in this study did not allow investigating effects of chest closure on accelerometer LV motions. Therefore, our results have to be verified in this condition. Furthermore, for future monitoring of cardiac function, the accelerometer needs to be as small as possible in order to limit the possibility for lacerations in fragile ventricles. This needs to be tested over time in a closed chest condition. It also remains to be determined whether the accelerometer 3D method is sensitive in detecting graft failure and sub-clinical myocardial ischaemia. These issues are currently under investigation by use of a smaller 3D accelerometer.

**CONCLUSIONS**

Epicardially attached 3D accelerometers provided detailed information on LV motions, which could be used to precisely quantify changes in global LV function and to detect regional LV dysfunction induced by coronary artery occlusion. The calculated accelerometer 3D systolic velocity showed equally good performance as the best single axis accelerometer systolic velocity in monitoring LV function. This implied that the sensor could be attached to the LV without need for precise alignment of the sensor to the cardiac contraction coordinates. This makes accelerometers more suitable for clinical use.

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Conflict of interest: Andreas Espinoza, Erik Fosse and Per S. Halvorsen are patent holders of the accelerometer technology for assessment of cardiac function and together with Remme shareholders in Cardiacs A/S.

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