Cardiac hemodynamics in PCI: effects of ischemia, reperfusion and mechanical support
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CHAPTER 5

Acute hemodynamic effects of accelerated idioventricular rhythm in primary percutaneous coronary intervention

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Submitted
Abstract

Objectives. The hemodynamic effects of accelerated idioventricular rhythm (AIVR) in primary percutaneous coronary intervention (PCI) are poorly described, although the transient character of AIVR may provide information on the left ventricle performance in this setting.

Methods. We studied a ST-segment myocardial infarction cohort of 75 consecutive patients (ages 60±11 years) in whom AIVR occurred following reperfusion during primary PCI. Mean systolic blood pressure (SBP), diastolic blood pressure (DBP) and heart rate were determined during periods of AIVR and sinus rhythm. We grouped patients according to the infarct related artery and the site of the coronary occlusion.

Results. AIVR caused an immediate reduction in SBP (124±26 vs. 95±22 mm Hg, p<0.001) and DBP (76±16 vs. 67±15 mm Hg, p<0.001) as compared to sinus rhythm irrespective of infarct-related artery. Reduction in SBP was more pronounced in distal than proximal left coronary artery (LCA) occlusions (35±15 vs. 26±10 mm Hg, p<0.02). SBP during sinus rhythm was lower in proximal LCA occlusions than in distal LCA occlusions (119±17 vs. 144±23 mm Hg, p<0.002) with a concomitant higher heart rate (84±11 vs. 75±10 bpm, p<0.01), whereas these hemodynamic differences between proximal and distal occlusion sites were not observed in the right coronary artery.

Conclusions. AIVR following reperfusion is associated with marked reduction in both SBP and DBP, irrespective of infarct-related artery. Moreover, patients with a culprit lesion in the proximal LCA showed less reduction in systolic blood pressure, probably due to a preexisting more comprised hemodynamic condition as indicated by a lower SBP and increased heart rate.
Introduction

Accelerated idioventricular rhythm (AIVR) is frequently observed during the reperfusion phase in ST-elevated myocardial infarction (STEMI) patients. It is an appearance of a transient and intermittent ventricular ectopic rhythm with a rate commonly between 60 and 110 bpm. The reported incidence of AIVR in patients undergoing thrombolytic therapy varies between 42-88%, partly due to inconsistent or imprecise definitions of reperfusion status. In a more recent study, in patients undergoing percutaneous coronary intervention (PCI) for a first acute myocardial infarction, the incidence of AIVR was 15%. The hemodynamic effects of AIVR have not been systematically studied in the era of mechanical reperfusion. We hypothesized that these effects may be related to infarct location and site of the culprit lesion. Therefore, we studied for the first time the hemodynamic effects of AIVR on systemic blood pressure in STEMI patients treated with primary PCI.

Methods

Source Population

The data analyzed in our observational study were obtained from STEMI patients who underwent primary PCI at the Academic Medical Center – University of Amsterdam. Primary PCI was performed with standard techniques using the femoral approach and in accordance with current guidelines. All studied patients were treated with aspirin, clopidogrel and heparin before primary PCI. The use of Glycoprotein IIb/IIIa inhibitors during the procedure was at the discretion of the operator.

Data Source

Baseline demographic variables, procedural and angiographic data were prospectively collected and entered by attending specialized nurses and interventional cardiologists in a stand-alone dedicated electronic database at the catheterization laboratory. Hemodynamic and Electrocardiographic Data Collection Throughout the procedure, aortic blood pressure as measured via the 7F guiding catheter, heart rate, and surface 12-lead electrocardiograms were continuously monitored.

Patient Selection

Our study population consisted of 75 consecutive STEMI patients who underwent a first primary PCI and showed AIVR following successful reperfusion. AIVR was defined as a run of > 3 consecutive ventricular beats with a rate between 60 and 110 bpm. Exclusion
criteria were a previous myocardial infarction or coronary artery bypass operation, cardiogenic shock, left bundle branch block, pacemaker rhythm, supraventricular arrhythmias, ventricular arrhythmia other than AIVR. The study complied with the Declaration of Helsinki.

Data analysis
During the first period of AIVR, aortic pressure and heart rate were analyzed using hemodynamic data-acquisition software MacLab 7000 (version 5.2, General Electric Co., Milwaukee, Wisconsin). Mean SBP, DBP and heart rate were calculated as the mean of 10 beats during sinus rhythm, and the mean of at least 3 beats during AIVR. It was accounted for that selected registrations were obtained during stable hemodynamic conditions, without interference of pharmaceuticals (e.g. nitroglycerin). SBP, DBP, and heart rate during AIVR were compared to the values during sinus rhythm. We grouped patients according to infarct-related artery and the site of the culprit lesion. The site of the culprit lesion was classified as proximal, i.e. of the right coronary artery (RCA) segment 1, and of the left coronary artery (LCA), segment 6 of the left anterior descending coronary artery and segment 11 of the left circumflex coronary artery. All other locations of the lesions in the main three epicardial arteries were classified as distal lesions.

Statistical Analysis
The 2-tailed paired \( t \) test was used for paired data to evaluate differences in blood pressure and heart rate between sinus rhythm and AIVR. The 2-tailed \( t \) test was used where appropriate, to compare hemodynamic characteristics between different infarct-related arteries and site of the lesions. Statistical analysis was performed using statistical software package for windows (SPSS version 16.0.1, 2008, Chicago, Illinois). We consider \( p < 0.05 \) to be statistically significant.

Results
Table 1 shows the patient characteristics of our 75 studied patients (ages 60±11 years, 33% women). The infarct-related artery was the left anterior descending coronary artery in 44%, left circumflex coronary artery in 7% and right coronary artery in 49%. Table 2 shows hemodynamic data as obtained during sinus rhythm and AIVR. AIVR caused a marked reduction in SBP (124±26 vs. 95±22 mm Hg, \( p<0.001 \)) and DBP (76±16 vs.67±15 mm Hg, \( p<0.001 \)), as compared to sinus rhythm. This effect was irrespective of infarct-related artery. Figure 1, illustrates a typical registration of the decrease in blood pressure during AIVR. This reduction in SBP was more pronounced in distal than proximal left coronary artery (LCA) occlusions (35±15 vs. 26±10mm Hg, \( p<0.01 \)), as shown in Figure 2. This difference
hemodynamic effects of AIVR

Table 1. Baseline Characteristics

<table>
<thead>
<tr>
<th>Study Population (n=75)</th>
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<tbody>
<tr>
<td>Demographics</td>
</tr>
<tr>
<td>Age in years (mean ± SD)</td>
</tr>
<tr>
<td>Male</td>
</tr>
<tr>
<td>BMI in kg/m² (mean ± SD)</td>
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<tr>
<td>Medical history</td>
</tr>
<tr>
<td>Hypertension</td>
</tr>
<tr>
<td>Diabetes</td>
</tr>
<tr>
<td>Hypercholesterolemia</td>
</tr>
<tr>
<td>Smoking</td>
</tr>
<tr>
<td>Procedural features and outcomes</td>
</tr>
<tr>
<td>Ischemic time in minutes (mean ± SD)</td>
</tr>
<tr>
<td>Location culprit lesion LCA</td>
</tr>
<tr>
<td>Single vessel disease</td>
</tr>
<tr>
<td>TIMI 0–1 flow, pre procedure</td>
</tr>
<tr>
<td>TIMI 2–3 flow, post procedure</td>
</tr>
</tbody>
</table>

Values are percentages unless otherwise indicated

BMI, Body Mass Index; LAD, Left Coronary Artery; TIMI, Thrombolysis in Myocardial Infarction.

Table 2. Mean Systolic Blood Pressure, Diastolic Blood Pressure and heart rate during Sinus Rhythm and AIVR

<table>
<thead>
<tr>
<th></th>
<th>Sinus rhythm</th>
<th>AIVR</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Left coronary artery (n=43)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Proximal Occlusions (n=26)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP (mm Hg)</td>
<td>119±18</td>
<td>94±15</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>DBP (mm Hg)</td>
<td>81±13</td>
<td>71±13</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Heart rate (bpm)</td>
<td>84±12</td>
<td>85±9</td>
<td>NS</td>
</tr>
<tr>
<td>Distal Occlusions (n=17)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP (mm Hg)</td>
<td>144±23</td>
<td>110±21</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>DBP (mm Hg)</td>
<td>79±15</td>
<td>73±13</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>Heart rate (bpm)</td>
<td>75±10</td>
<td>80±8</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Right coronary artery (n=32)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Proximal Occlusions (n=10)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP (mm Hg)</td>
<td>122±31</td>
<td>93±25</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>DBP (mm Hg)</td>
<td>69±15</td>
<td>63±12</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>Heart rate (bpm)</td>
<td>79±8</td>
<td>79±10</td>
<td>NS</td>
</tr>
<tr>
<td>Distal Occlusions (n=22)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP (mm Hg)</td>
<td>115±29</td>
<td>87±23</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>DBP (mm Hg)</td>
<td>70±19</td>
<td>59±17</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Heart rate (bpm)</td>
<td>79±14</td>
<td>82±9</td>
<td>NS</td>
</tr>
</tbody>
</table>

Values are mean ± SD, SBP, Systolic Blood Pressure; DBP, Diastolic Blood Pressure.

in proximal and distal occlusions was not observed in the right coronary artery. SBP during sinus rhythm was lower in proximal LCA occlusions than in distal LCA occlusions (119±17 vs. 144±23 mm Hg, p<0.002) with a concomitant higher heart rate (84±11 vs. 75±10 bpm, p<0.01). SBP in proximal RCA occlusions during sinus rhythm was similar to
distal RCA occlusions. (122±30 vs. 115±29 mmHg, p=NS) without a difference in heart rate (79±8 vs. 79±14 bpm, p=NS).

Figure 1. A typical blood pressure registration of accelerated idioventricular rhythm (AIVR) in a ST-segment elevation myocardial infarction patient during primary percutaneous coronary intervention. Note the immediate decrease in blood pressure during an episode of AIVR, as compared to sinus rhythm.

Figure 2. Illustration of the decrease in blood pressure during AIVR, as compared to sinus rhythm, in relation to the site of the coronary lesion. Note that reduction in systolic blood pressure was more pronounced in distal than proximal left coronary artery (LCA) occlusions (35±15 vs. 26±10 mm Hg, p<0.01). This difference in blood pressure drop was not observed in right coronary artery (RCA) occlusions.
Discussion

In this study, we are the first to have systematically investigated acute hemodynamic effects in STEMI patients with reperfusion-induced AIVR following primary PCI. We found that AIVR following reperfusion is associated with marked reduction in both SBP and DBP, irrespective of infarct-related artery. Moreover, patients with a culprit lesion in the proximal LCA showed less reduction in blood pressure, probably due to a preexisting more comprised hemodynamic condition as indicated by a lower SBP and increased heart rate.

Incidence and mechanisms of AIVR

The reported incidence of AIVR in patients undergoing thrombolytic therapy varies between 42-88%, partly due to inconsistent or imprecise definitions of reperfusion status. It has been a frequently observed phenomenon in daily clinical practice in the catheterization laboratory after reperfusion therapy by primary PCI for STEMI. In a more recent study of 125 consecutive patients that underwent PCI for a first acute myocardial infarction, 24-hour Holter monitoring revealed that AIVR appeared in 15% of the patients. Furthermore, AIVR has been proposed as a specific non-invasive marker for successful coronary artery reperfusion in the prethrombolytic and thrombolytic era. However, in the era of direct mechanical reperfusion strategies, the prognostic relevance of AIVR remains controversial. The occurrence of AIVR is generally considered as benign and without prognostic implications, however this topic of interest this topic has not been studied in large scaled clinical studies.

The underlying electrophysiological mechanism of AIVR has been suggested to result from abnormal ventricular automaticity of the subendocardial Purkinje fibers. Kaplinski et al. described a transient increase of the idioventricular rate after coronary reperfusion and suggested that this increase in ventricular automaticity may be the basis for the occurrence of AIVR. Bonnemeier et al., demonstrated that parasympathetic predominance at the sinus node, which was characterized by lower serum norepinephrine levels leading to lower sinus rates, may favor the occurrence of AIVR after reperfusion in acute myocardial infarction patients. Reperfusion injury following thrombolysis or primary PCI secondary to calcium overload (and other contributing factors such as formation of free oxygen radicals or changes in intracellular pH), may lead to triggered activity. Some investigators have suggested that triggered activity, based on delayed afterdepolarizations, may be another explanation of the occurrence of AIVR. Preliminary hemodynamic data from our institution (Remmelink et al., unpublished data, 2009) suggest that diastolic left ventricular dysfunction, assessed by pressure-volume loops, contributes to the occurrence of AIVR.
AIVR and systemic blood pressure

In our study, we observed a marked decrease in systolic and diastolic blood pressure during an episode of AIVR. An explanation for the immediate decrease in blood pressure may be the absence of atrioventricular sequential pacing. Atrioventricular sequential pacing may lead to optimization of the timing of the mechanical atrial and ventricular synchrony. Moreover, optimal diastolic filling and reduction of diastolic mitral regurgitation may contribute to the hemodynamic improvement.16

Another explanation may be an absence of atrial contraction (i.e., atrial kick) to left ventricular filling in AIVR.17 The significance of atrial contraction to maintain cardiac output has been documented in numerous studies18,19 and is supported by clinical observations showing a marked hemodynamic deterioration in patients developing atrial fibrillation in the setting of ischemic heart disease.20 The current study protocol does not allow a differentiation between these two potential mechanisms responsible for the decreased left ventricle performance.

AIVR and proximal versus distal location of culprit lesion

Interestingly, we found that the location of the culprit lesion, i.e. the distal versus the proximal segment of the left coronary artery, was associated with the degree of systolic blood pressure reduction. Patients with a culprit lesion in the distal LCA showed more pronounced reduction in blood pressure than patients with a proximal lesion, in contrast to patients with a proximal or distal culprit lesion in the RCA showing no significant differences of AIVR on blood pressure. The RCA supplies a relative small area at risk of the left ventricle, independent of the site of the coronary occlusion. Blood pressure and heart rate were similar sinus rhythm irrespective of the location of the lesion. This is in contrast with the observations in patients with a culprit lesion in the LCA. Patients with a proximal LCA culprit lesion, and therefore a larger area at risk, had lower blood pressures and concomitant higher heart rates during sinus rhythm. This suggests that patients with a proximal lesion exhibit a more compromised hemodynamic status and, therefore, probably a less pronounced effect of AIVR on blood pressure than with patients a smaller area at risk in case of a distal location of the culprit lesion resulting in a significant larger effect of AIVR on blood pressure.

Clinical implications

The acute hemodynamic effects of AIVR, a reperfusion arrhythmia frequently observed in STEMI patients, underlines the importance of maintenance of atrioventricular sequential pacing and/or atrial contraction to maintain systolic blood pressure. Furthermore, it explains why atrial fibrillation in the setting of STEMI yields an important hemodynamic effect in particular due to loss of the atrial contribution to ventricular filling resulting in a consequent decrease in cardiac output.
Limitations
Echocardiography nor magnetic resonance imaging was performed to obtain information on left ventricular function. The data as assessed in our study are limited to continuous registrations of blood pressure and heart rate during primary PCI. We studied hemodynamic data only in the acute phase of reperfusion in STEMI patients. Furthermore, we did not evaluate the hemodynamic significance of the occurrence of AIVR in relation to clinical outcome.

Conclusion
We are the first to show that in STEMI patients with reperfusion-induced AIVR during primary PCI, AIVR is associated with marked reduction in both SBP and DBP, irrespective of infarct-related artery. Moreover, patients with a culprit lesion in the proximal LCA showed less reduction in blood pressure, probably due to a preexisting more comprised hemodynamic condition as indicated by a lower SBP and increased heart rate.

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References


hemodynamic effects of AIVR


