

Cardioprotected Percutaneous Coronary Intervention – A Case Study in a Stable Angina Patient

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Abstract

Fast and effective opening of epicardial arteries by thrombolysis or percutaneous coronary intervention (PCI) to restore perfusion to the jeopardised myocardium is considered the cornerstone in the treatment of patients with an acute ST-elevation myocardial infarction (STEMI) leading to a reduction in morbidity and mortality. However, despite successful opening of the coronary artery, myocardial reperfusion remains suboptimal (i.e. measured by blush grade) and results in up to 30–40% of the STEMI patients that is associated with a worse clinical outcome. In this case study of a stable angina patient we document for the first time that cardioprotected PCI (cPCI) using Pressure-controlled Intermittent Coronary Sinus Occlusion (PICSO®) has the potential to increase left anterior descending (LAD) wedge pressure and reduce ischaemia as measured by surface electrocardiogram (ECG). The next step in the development of cPCI will be to apply the PICSO technology in STEMI patients.

Keywords

Cardioprotected percutaneous coronary intervention (cPCI), ST-elevation myocardial infarction (STEMI), Pressure-controlled Intermittent Coronary Sinus Occlusion (PICSO®), acute myocardial infarction (AMI), Miracor

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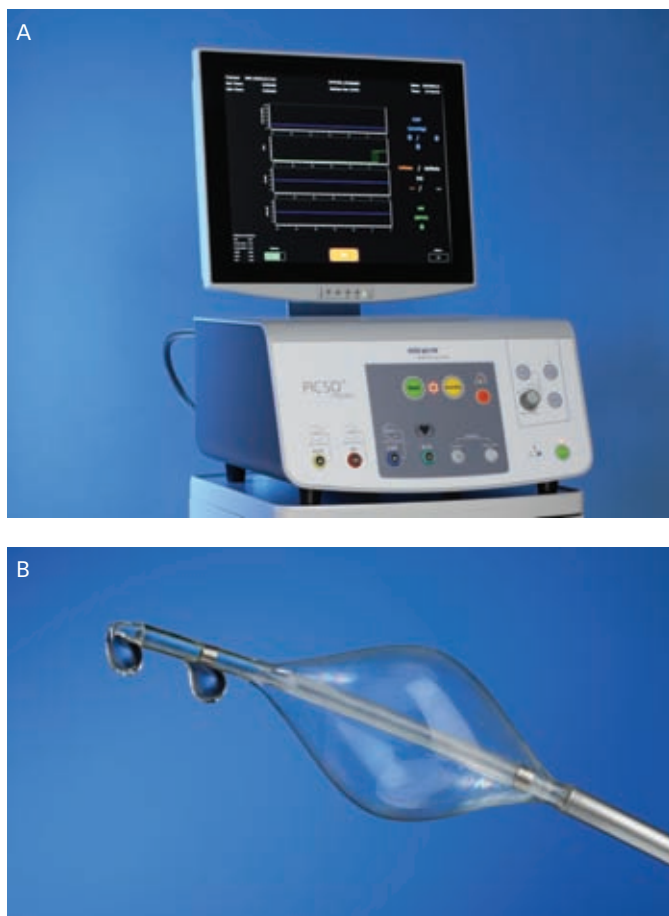
Fast and effective opening of epicardial arteries by thrombolysis or percutaneous coronary intervention (PCI) to restore perfusion to the jeopardised myocardium is considered the cornerstone in the treatment of patients with an acute ST-elevation myocardial infarction (STEMI) leading to a reduction in morbidity and mortality.¹ However, despite successful opening of the coronary artery, myocardial reperfusion remains suboptimal (i.e. measured by blush grade) and results in up to 30–40% of the STEMI patients that is associated with a worse clinical outcome.^{2–5} The sub-optimal myocardial reperfusion is a result of mechanical or functional obstruction of the coronary microcirculation by cellular debris and/or damaged endothelium, as a consequence of the primary epicardial event and/or of the reperfusion.^{6–8}

Due to the grave impact of microvascular dysfunction on patients outcome after STEMI and primary PCI, effective preventive and therapeutic strategies applicable in real-world, are needed. Retrograde perfusion of the coronary circulation is daily routine in cardiothoracic surgery in order to protect the myocardium during surgical cardiac arrest. Using the 'back door' of the heart to access deprived injured microvasculature is a fascinating idea and new developments of cardioprotective technologies so far unavailable in interventional cardiology are now available for further clinical testing. Our groups have therefore embarked on a development of a so called cardioprotected PCI (cPCI) which has the potential to address shortcomings of older technologies and approaches.

The fundamental requirements to achieve effective cPCI are that this adjunct technology supporting PPCI is applied quickly accessing the jeopardised microcirculation from the coronary sinus, clearing the microcirculation and thus creating a pressure gradient between the retroperfused venous compartment and the reperfused arterial bed. Coronary sinus interventions and especially the concept of intermittent coronary sinus occlusion (ICSO) introduced several decades ago meets this demand and has been evaluated in animal models^{9–11} and clinical observations showing profound reduction of ischaemic sequelae.^{12–14} This technology has the fundamental characteristics needed to fulfil the cPCI requirements and to fit into the current treatment scenario of STEMI patients. Pressure-controlled intermittent coronary sinus occlusion (PICSO®) is a modification of the original time-dependent technology and is applied through a recently Conformité Européenne (CE) marked (Miracor Medical Systems GmbH, Vienna). The proposed primary mechanisms of action are that intermittent pressure increase in the coronary venous circulation leads to:

- a retrograde perfusion of the border zone of the infarcted myocardium due to the coronary sinus pressure increase;
- a wash-out of noxious and embolic material across the microvascular bed when the occlusion balloon is triggered on the electrocardiogram (ECG) and rapidly deflated;
- a cardioprotective effect in the occluded artery by increasing the

Figure 1: The Miracor Pressure-controlled Intermittent Coronary Sinus Occlusion® Impulse Console and Catheter



- distal occlusion pressure on the arterial side; and
- a mechanical shear stress in the vasculature, inducing possible regenerative effects of the myocardium.

This case report documents the first experience of cPCI in a stable angina patient in order to document the safety and effectiveness of this new technology. Following studies will document the use of the PICSO technology in STEMI patients.

Product Description

The Miracor PICSO Impulse System consists of a console and a dedicated catheter. The Miracor PICSO Impulse Console includes a unique pneumatic circuit, which controls the inflation and deflation cycle of the occlusion catheter. The duration of the occlusion cycle is determined using the pressure increase that occurs in the coronary sinus when the vessel is occluded. After a given time, the coronary sinus pressure reaches a pressure plateau, which is unique for each patient. The pressure plateau is normally 40–60 mmHg and contributes to the redistribution of flow into the border zone of the ischaemic territory. The Miracor PICSO Impulse Catheter balloon is deflated when the pressure plateau is reached and the exact timing of the deflation is triggered by the ECG. This is done in order to achieve the maximum wash-out effect as previously described.

Several safety features are included in the devices in order to avoid any damage to the coronary sinus or the cardiac tissue. Most

Figure 2: Pre-procedural Angiogram



Figure 3: Venous Phase Angiogram to Visualise the Coronary Sinus (Left) and Successful Placement of the Guiding Sheath in the Ostium of the Coronary Sinus (Right)

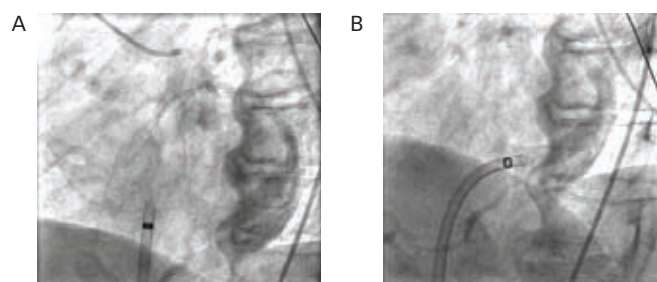
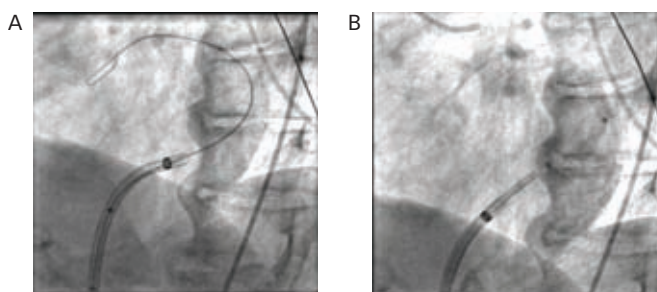


Figure 4: Coronary Sinus Wired with Terumo Guide Wire (Left) and Successful Placement of the Pressure-controlled Intermittent Coronary Sinus Occlusion® Impulse Catheter (Right)

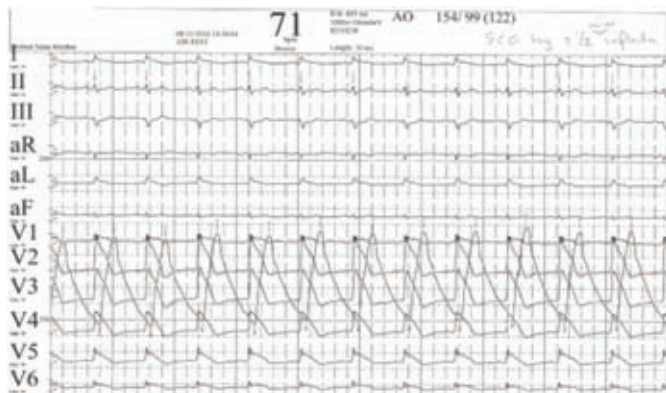


importantly, the balloon size is calibrated to each coronary sinus vessel size by multiple gradual inflations of the balloon. The console will automatically detect the volume needed to occlude the given coronary sinus and this volume will be shuffled between the console and the catheter balloon. In addition, the console continuously monitors specific pressure levels and timing to avoid prolonged occlusion of the coronary sinus. Safety features have also been integrated into the catheter design including a unique atraumatic distal tip.

Patient History

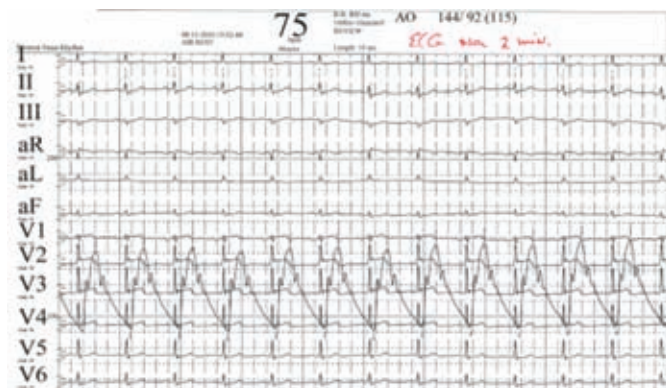
The patient was a 59-year-old male, his cardiac history notes a non-STEMI in February 2009 for which he received medical treatment. Family history is positive for coronary artery disease and no other risk factors are present. Recently, he returned to the outpatient clinic with progressive anginal complaints during daily activities. Exercise testing showed marked ST-segment depression

Figure 5: Surface Electrocardiogram Tracing with Left Anterior Descending Occlusion without Pressure-controlled Intermittent Coronary Sinus Occlusion®



Clear ST-segment changes in V2–V6. ECG = electrocardiogram.

Figure 6: Surface Electrocardiogram tracing with Left Anterior Descending Occlusion with Pressure-controlled Intermittent Coronary Sinus Occlusion®



ST-segment shift in V2–V6. ECG = electrocardiogram.

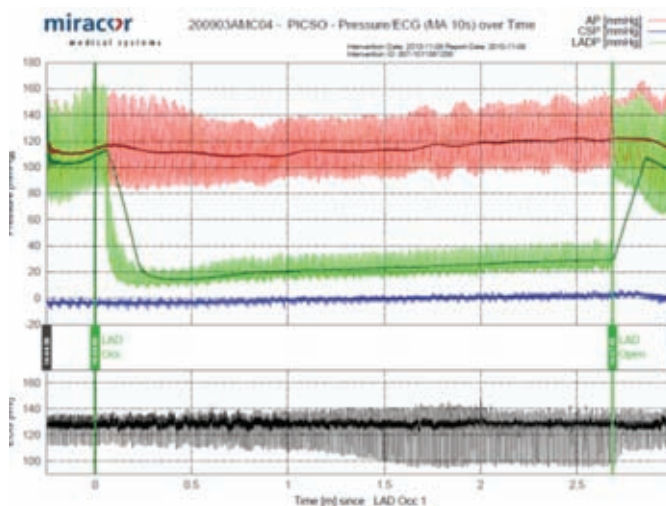
in the inferolateral leads and diagnostic coronary angiography revealed a significant left anterior descending (LAD) lesion just before the second diagonal branch and a significant stenosis in the large forked first marginal branch. There was no collateral flow visible on the diagnostic angiogram.

The Study Protocol

The study protocol consists of two LAD occlusions of a maximum three minutes, one without PICSO followed by one with PICSO, during which distal coronary pressure is recorded. The adjacent PCI is performed during PICSO and both the LAD and the obtuse marginal coronary arteries were successfully opened in this patient.

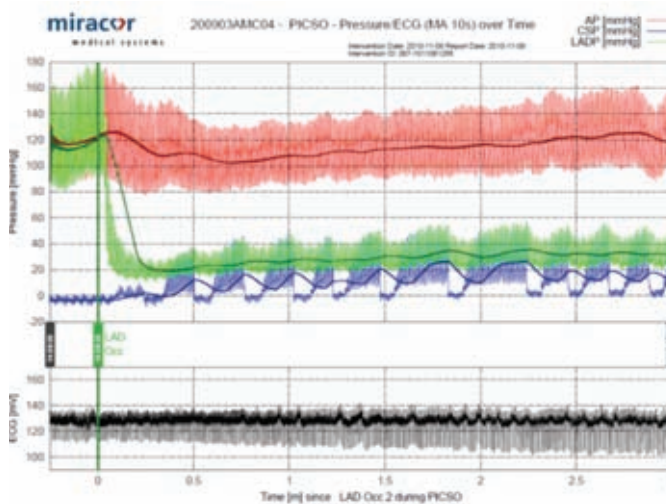
Cardiac catheterisation was performed using the right femoral artery with a standard 7 Fr sheath. The Miracor PICSO impulse catheter was introduced through the right femoral vein using a 9 Fr Bard's channel steerable sheath and a Terumo guide wire. Time from femoral vein puncture to PICSO catheter in place was six minutes and 57 seconds; PICSO catheter insertion time was three minutes. A Combewire (Volcano Corporation) was introduced in the LAD distal to the stenosis to record coronary flow velocity and pressure.

Figure 7: Distal Left Anterior Descending Occlusion Pressure (Green) without Pressure-controlled Intermittent Coronary Sinus Occlusion® (Blue), with Arterial Pressure (Red) and Electrocardiogram (Black)



AP = arterial pressure; CSP = coronary sinus pressure; ECG = electrocardiogram; LAD = left anterior descending; LADP = left anterior descending coronary artery pressure; PICSO® = Pressure-controlled Intermittent Coronary Sinus Occlusion®; Occ = occlusion.

Figure 8: Distal Left Anterior Descending Occlusion Pressure Modulation and Increase (Green) During Pressure-controlled Intermittent Coronary Sinus Occlusion® (Blue) with Arterial Pressure (Red) and Electrocardiogram (Black)



AP = arterial pressure; CSP = coronary sinus pressure; ECG = electrocardiogram; LAD = left anterior descending; LADP = left anterior descending coronary artery pressure; PICSO® = Pressure-controlled Intermittent Coronary Sinus Occlusion®; Occ = occlusion.

The first LAD-occlusion without PICSO resulted in transmural ischaemia on the surface ECG and lasted for three minutes, during which the patient suffered from mild anginal complaints (see Figure 5).

The second LAD occlusion during PICSO® resulted in markedly reduced ST-segment elevations (see Figure 6) and coronary wedge pressure increased almost twofold from 18 mmHg without PICSO to 35 mmHg with PICSO. Figure 7 and Figure 8 show distal LAD occlusion pressures without and with PICSO, respectively.

Conclusion

These initial results are promising and show the potential of the PICSO technique in increasing myocardial perfusion pressure during brief coronary occlusion, as well as a marked reduction of ischaemia as assessed by the surface ECG ST-segment shift, even in the absence of collateral flow. A key point remains precise placement of the Miracor PICSO impulse catheter in the coronary sinus to ensure optimal venous occlusion.

The marked reduction in ST-elevation during the second LAD occlusion is an interesting finding. This suggests that the increase in

myocardial perfusion during brief coronary occlusion, induced by PICSO, may reduce signs of ischaemia as assessed by surface ECG ST-segment shift. Nevertheless, the reduction observed may be partly explained by myocardial adaptation to ischaemia after the first coronary occlusion. The magnitude of the observed reduction, however, suggests an important effect of PICSO.

These initial findings are promising for the future development of cardioprotected PCI (cPCI) and the clinical application of the technology in STEMI patients is the next step to introduce the approach in patients with microcirculatory damage after PPCI. ■

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