

Case Report

Avoiding Hemodynamic Collapse During High-Risk Percutaneous Coronary Intervention: Advanced Hemodynamics of Impella Support

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The rate of performing primary percutaneous coronary intervention in patients with complex coronary artery disease is increasing. The use of percutaneous mechanical circulatory support devices provides critical periprocedural hemodynamic support. Mechanical support has increased the safety and efficacy of interventional procedures in this high-risk patient population. Predicting patient response to the selected intervention can be clinically challenging. Here we demonstrate a case where complete hemodynamic collapse during PCI was avoided by mechanical support provided by the Impella device. Further, we employ a comprehensive cardiovascular model to predict ventricular function and patient hemodynamics in response to the procedure. New computational tools may help interventionists visualize, understand, and predict the multifaceted hemodynamic aspects of these high risk procedures in individual patients. © 2016 Wiley Periodicals, Inc.

Key words: myocardial infarction; mechanical circulatory support; patient-specific computational modeling; patient hemodynamics

INTRODUCTION

Patients with complex coronary artery disease involving multiple vessels, total occlusions, left main lesions, and/or decreased ventricular function are being treated at increasing rates with primary percutaneous coronary interventions (PCI). Treatment may require prolonged balloon inflations which increases the exposure of at-risk myocardium to ischemia, exacerbation of ventricular dysfunction, and the potential for hemodynamic collapse. The safety and completeness of such high risk PCI (HR-PCI) procedures has been shown to be enhanced through the use of temporary percutaneous mechanical circulatory support (pMCS) [1]. Here, we present a case demonstrating the prevention of hemodynamic collapse in a complex HR-PCI patient supported by an Impella (Abiomed, Danvers, MA) device [1]. We also employ a previously validated comprehensive cardiovascular model, modified to include epicardial coronary artery blood flow and its regulation of myocardial contractility that replicates the recorded hemodynamics with high fidelity. The model demonstrates important hemodynamic principles of pMCS,

during HR-PCI not otherwise obtainable in the clinical setting, including ventricular dynamics [2,3].

CASE SUMMARY

An 85-year-old female was admitted with a non-ST segment elevation MI with preserved EF (60–65%). The patient had severe three vessel coronary disease

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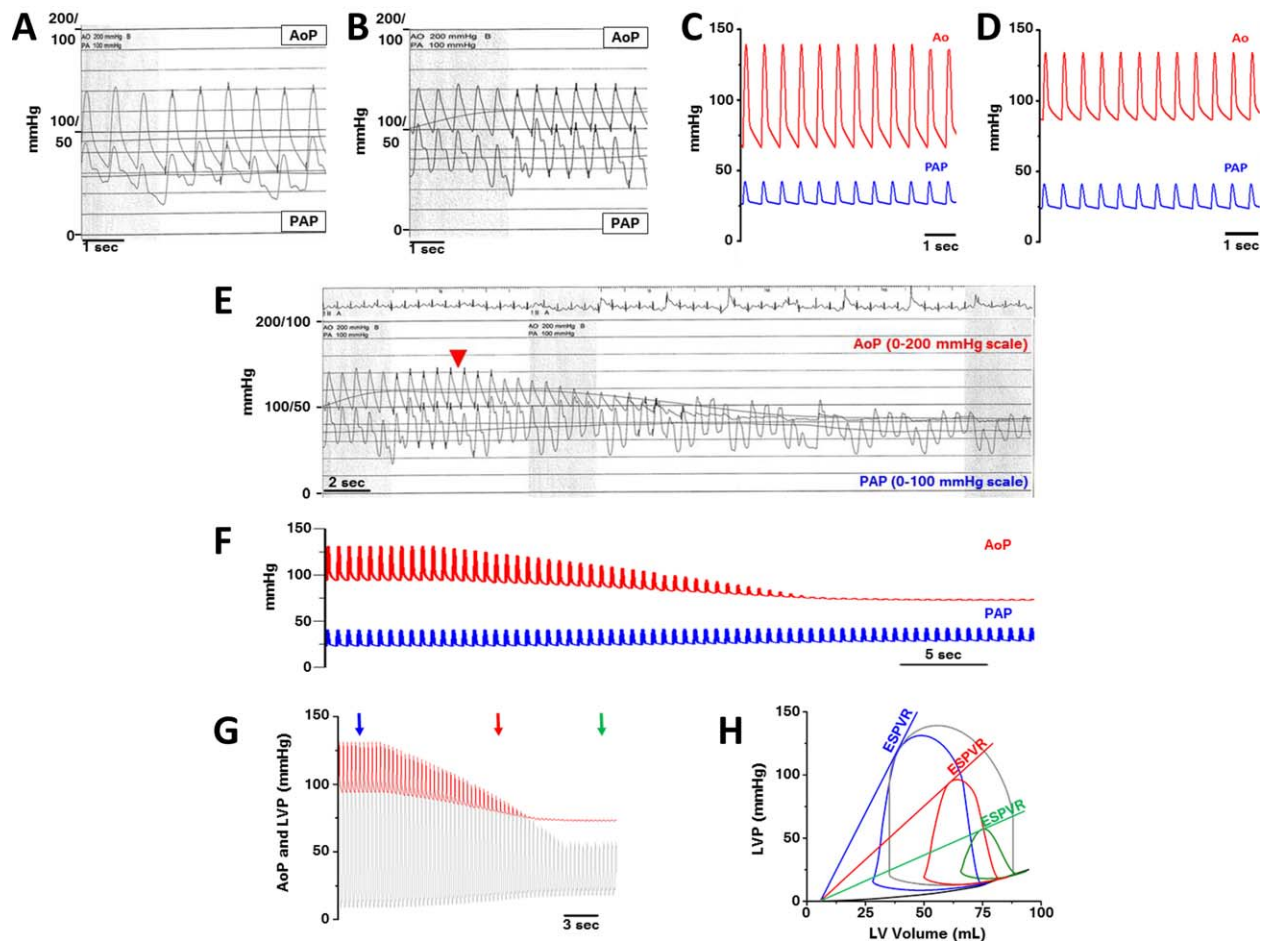


Fig. 1. Real and simulated periprocedural patient hemodynamics. (A) Actual aortic and PA pressures prior to initiation of mechanical support. (B) Actual aortic and PA pressures after initiation of mechanical support but prior to PCI. (Note the relative scales of Ao and PA pressures are 0–200 mm Hg and 0–100 mm Hg, respectively.) Patient hemodynamics, estimates of ejection fraction, heart rate, and cardiac output were used to simulate Ao and PA pressures prior to support (C) and (D) after initiation of support. (E) Actual aortic and PA pressures recorded just prior to and after angioplasty balloon inflation (marked with red arrowhead). Note the decline in aortic pulse pressure associated with balloon-induced ischemia. (F) Simu-

lated periprocedural hemodynamics behaved similarly. (G) Periprocedural LV (grey) and Ao (red) pressures were simultaneously simulated. The blue arrow indicates the simulated hemodynamics prior to PCI. The red arrow indicates that simulated hemodynamics during ischemia-induced contractile decline of the LV; the green arrow indicates the simulated hemodynamics where AoP is uncoupled from LV function and perfusion pressure is maintained by the Impella. (H) PV loops of ventricular dynamics corresponding to the colored arrows in Panel C. The progressive decline in contractility of the myocardium is reflected in the decreasing slope of the end-systolic pressure-volume relationship (ESPVR) line.

including 90% left main, 90% proximal left anterior descending, 90% proximal circumflex, and 100% right coronary artery occlusions. She was deemed to be a poor surgical candidate and PCI was planned. Because of the complex lesion involving a bifurcation of the left main artery, the patient was considered as high risk. Therefore, mechanical circulatory support was planned using the Impella CP.

At baseline, aortic pressure (AoP) was 140/65 (mean 95) mm Hg and end-expiratory pulmonary artery pressure (PAP) was 45/25 (mean 30) mm Hg (Fig. 1A). Upon initiation of Impella support (Fig. 1B), systolic

AoP did not change, but diastolic and mean pressures increased to 100 and 120 mm Hg, respectively. Data from the Automated Impella Controller indicated that the Impella provided an average of 3.5 L min^{-1} of support during the entire perioperative period. End-expiratory PAP increased by a small amount to 50/30 (mean 35) mm Hg. Baseline hemodynamics along with estimates of ejection fraction, heart rate, and cardiac output were used as inputs to the patient simulator of the cardiovascular simulation [3]. Simulated pressures compared well to corresponding actual patient pre- and post-support tracings (Fig. 1C and D).

Upon inflation of the angioplasty balloon in the left main coronary artery, the patient's AoP started to decline within a few beats, with progressively decreasing pulse pressure, until it stabilized at ~ 80 mm Hg. Figure 1E demonstrates that pressure was maintained for the >20 sec balloon inflation (Fig. 1E bottom), and the ECG remained normal (Fig. 1E, top). The lack of pulsatility in the AoP indicates there was no native output from the LV and that all blood flow was provided by the Impella. Importantly, PAP was stable during the entire inflation period indicating that the venous return, RV perfusion, and RV function were all normal in the setting of Impella support. Model-predicted AoP and PAP tracings during simulated left main coronary artery occlusion behaved similarly (Fig. 1F), again indicating that the Impella provided sufficient support to maintain adequate systemic pressure and perfusion and normal filling of the right ventricle.

The cardiovascular simulation provides further insights into periprocedural ventricular dynamics through examination of left ventricular (LV) pressures (Fig. 1G, grey) and pressure–volume (P–V) loops (Fig. 1H). P–V analysis provides a comprehensive framework for understanding ventricular energetics and mechanics. The P–V loop characterizes the events occurring during a single cardiac cycle [4,5] and is constructed by plotting the dynamic instantaneous relationship between left ventricular pressure and volume. The loop typically has a rounded trapezoidal shape (Fig. 1H, grey loop) and time proceeds in a counter-clockwise direction, starting with end-diastole at the bottom right corner. The four phases of the cardiac cycle are represented by the four sides of the loop: isovolumic contraction, ejection, isovolumic relaxation, and filling. The P–V loop is bound on its upper left corner by the end-systolic pressure–volume relationship (ESPVR) and on the bottom by the end-diastolic pressure volume relationship (EDPVR). Both the ESPVR and EDPVR reflect intrinsic properties of the myocardium. The ESPVR defines the maximum pressure that the ventricle can develop at and systole and at a given volume and thus indexes ventricular contractility. The EDPVR characterizes the passive diastolic ventricular properties. The area inside the loop is the mechanical energy (the stroke work, SW) generated by the ventricle with each beat and is measured in mm Hg·mL (aka, a joule).

The first point illustrated by the P–V loops in Figure 1H is that upon initiation of pMCS, the P–V loop is left-shifted toward smaller volumes and is more triangular in shape. The volume shift signifies the ventricular unloading provided by the Impella, and the triangular shape indicates loss of isovolumic periods since the device is always withdrawing blood from the

LV independent of the phase of the cardiac cycle [6]. Such phenomena have been confirmed by direct experimental recordings [6,7]. These P–V loops further illustrate that underlying the decline of aortic pressure following initiation of left main balloon inflation is a progressive reduction in LV contractility (i.e., a progressive decline in the LV end-systolic pressure–volume relationship, ESPVR) resulting from loss of coronary perfusion. The loss of pulsatility of aortic pressure is due to an uncoupling of LV pressure generation from LV function and the hemodynamic support provided by the Impella. The Impella maintains arterial pressure and provides systemic perfusion and collateral coronary flow so that LV contractility and pressure generation stabilizes at a lower, energy sparing level. In the P–V loop, this is observed as a significant decrease in the SW. The Impella unloads the LV and decreases the metabolic oxygen demand of the myocardium [4,5]. Without Impella support aortic pressure would have continued to be linked to ventricular pressure generation, and would have continued to decline to profound hypotension with induction of myocardial ischemia.

DISCUSSION

Arterial and pulmonary pressure measurements provide important information on patient hemodynamic status and guide clinical decision making in real time. However, they provide limited information about underlying right and left ventricular mechanics. Direct ventricular function monitoring in real-time during a PCI procedure is not clinically practicable. The potential importance of the immediate (short term) impact of an interventional procedure on ventricular function is often overlooked. While there is no doubt that restoration of normal coronary blood flow is the best treatment for coronary artery disease, the fact that PCI is a potential source of short term iatrogenic ventricular dysfunction cannot be discounted. Therefore, minimizing the risks of temporary procedural-dependent ischemia can be clinically important. The data presented here demonstrates the effect of PCI-dependent ischemia and the hemodynamic benefits of mechanical circulatory support.

This case demonstrates an example where occlusion of the left main coronary artery during PCI balloon inflation results in rapid loss of arterial pressure. In the presence of an Impella pMCS device, aortic pressure pulsatility also declines and eventually is completely lost, despite maintenance of a nearly normal mean arterial pressure value and almost no change in PAPs. Simulation of this case in a previously described and validated cardiovascular model [2,3] provides

additional insights into the underlying physiology of pMCS during HR-PCI. Specifically, simulation-derived LV pressure tracings and P-V loops illustrate the nature of unloading provided by the Impella device—consistent with prior experimental recordings [6]—the progressive reduction in left ventricular contractility to a new, stable, lower level without myocardial ischemia (supported by lack of ECG changes) and how the uncoupling between AoP and ventricular pressure generation results in loss of aortic pressure pulsatility [8].

Cardiovascular models have several limitations, and those of the current model have been described previously [9]. However, it is not required that such models simulate actual hemodynamics in great quantitative detail. Rather, their utility and value is in clarifying physiological principles not otherwise obtainable from clinical or experimental measurements.

CONCLUSION

Advances in interventional technologies and treatments have allowed clinicians to effectively treat patients with increasingly complex coronary artery disease. These patients present with numerous and profound challenges that need to be overcome by the treating physician if the patient is to have a positive outcome. Not least amongst these challenges is the need to be able to predict an individual patient's hemodynamic response to the intervention. With further development and validation, computational tools may help interventionists not only visualize and understand,

but also predict the multifaceted aspects of these high risk procedures in individual patients.

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