Pulmonary arterial pressure sensing in a patient with left ventricular assist device during ventricular arrhythmia

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Introduction

With the ever-increasing heart failure population, the use of left ventricular assist devices (LVAD) is becoming increasingly more accepted as a treatment for heart failure as destination therapy. In 2013, this became an American Heart Association recommendation as destination therapy.[1] Despite improved mechanical function, the problem of fluid management often persists for these patients. Patients can have problems with volume overload, but in addition they are sensitive to volume and filling pressure reduction. Readmissions among patients with an LVAD remain high, with heart failure as a common indication.[2] In addition, these patients are still prone to ventricular arrhythmias (VA).[3] Patients with LVAD have a high incidence of VA and have been shown to have an improved mortality when treated with an implantable cardioverter-defibrillator (ICD).[4]

Fluid management in patients with symptomatic congestive heart failure is improved when guided by pulmonary artery (PA) pressure sensor measurements.[5] There is scant data of this use in the LVAD population, but limited data suggest some utility.[6] Patients with an LVAD often have very depressed left ventricular function and blunted pulse pressure, as the left-sided cardiac output is driven by the LVAD. However, patients may still have intact right ventricular (RV) function that generates a significant pulse pressure in the pulmonary artery. Thus, ambulatory PA pressure measurements may serve as an ambulatory marker of RV function. We hypothesize that PA pressure measurements could be used as a surrogate for RV function and thus hemodynamic stability during VA. If so, could these data be utilized to enhance detection in patients with LVAD support and an ICD?

KEYWORDS Pulmonary artery pressure; Sensor; Ventricular arrhythmia; LVAD; Defibrillator; Heart failure (Heart Rhythm Case Reports 2017;3:348–351)

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Case report

A 73-year-old man presented to a community hospital emergency room following a syncopal episode while trying to get out of a car. The patient has had multiple episodes of syncope or presyncope in association with recurrent symptomatic and asymptomatic ventricular tachycardia (VT), with prior ablation both pre- and post-LVAD. His history is complicated with nonischemic cardiomyopathy first diagnosed in 2002, and history of a biventricular ICD (Guidant Corporation, St. Paul, MN, and Boston Scientific, Marlborough, MA), originally placed in 2002, and most recently replaced in January 2016. His heart failure continued to progress, requiring LVAD support as destination therapy with placement of a HeartMate II (Thoratec, Pleasanton, CA) in 2012. Following placement of his device, the patient had multiple episodes of VT. This was initially treated medically, and then first ablated in 2014. He was again hospitalized twice in 2015 due to recurrent VT with associated ICD shocks. The VT proved to be refractory to medical therapy. He underwent 2 subsequent ablations for VT in October 2015 and January 2016, though he remained inducible for very fast VA (rates $>$ 220 beats per minute [bpm]). Due to multiple episodes of sustained asymptomatic VT (rate 210 bpm) that was refractory to both antitachycardia pacing and shock therapy (VF 220 / VT 200 / VT1 140), all therapies were disabled to "monitor only" mode. With each hospitalization for VT, he was found to be fluid overloaded with a pulmonary arterial wedge pressure ranging from 20 to 26. A PA pressure sensor (CardioMEMS; St. Jude Medical, Sylmar, CA) was placed in January 2016 to aid in fluid management. The patient was maintained on oral amiodarone 200 mg twice daily.

Following placement of the PA pressure sensor, the patient was clinically stable and without hospitalization for 2 months. However, in March 2016 the patient suffered a witnessed syncopal event. Upon arrival to the emergency room, he was found to have a heart rate of approximately 210 bpm. Electrocardiogram revealed a wide complex tachycardia. A chest radiograph revealing his hardware, including the PA sensor, is shown in Figure 1. Device interrogation showed programmed DDDR pacing with RV-only pacing with clear VT that subsequently accelerated with continued A-V sequential pacing and brief classification in VT-1 zone,

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KEY TEACHING POINTS

- The care of heart failure patients is evolving to include multiple technologies as destination therapy. Familiarity with pulmonary artery pressure sensors, cardiac resynchronization therapy devices, and ventricular devices is essential to providing complete care to this patient population.
- Heart failure patients, including those treated with left ventricular assist devices (LVAD), have a greater incidence of arrhythmias. In the future, detection of these arrhythmias may not be limited to cardiovascular implantable electronic devices.
- Repeated assessment and monitoring of right ventricular function is required in patients treated with LVAD. Pulmonary artery pressure monitoring may prove to be a useful surrogate of right ventricular function.

demonstrating undersensing. At the time of the most recent episode, sensitivity was noted at 0.15 mV, with recent chest radiograph confirming no evidence of lead displacement in chronic RV apex lead.

At the time of transfer to our tertiary care center, the patient appeared minimally symptomatic while at bedrest and clinically stable with adequate support from the LVAD, with a mean arterial pressure of 65 mm Hg (systolic blood pressure). The patient was on intravenous lidocaine. On electrocardiogram the patient was seen to be in rapid VA, with initial bedside interrogation of the patient's PA pressures revealing only minimal and fast irregular pressure waveform with a mean of 22 mm Hg (Figure 2).

Given that the syncope was presumed to be secondary to loss of RV function, the patient was cardioverted to an A-V sequential biventricular-paced rhythm. Immediately after cardioversion, there was a return of normal waveforms on PA pressure recordings despite low signal strength, with a PA pressure of approximately 40/20 mm Hg (Figure 3). Blood pressure immediately improved to 100/80 mm Hg. The measurements recorded were not acute onset. In both retrospective (pulsatile devices) and prospective (continuous flow devices) studies, the LVAD patient population from ICD.[7] We speculate that VT in this population may cause an abrupt change in hemodynamic filling pressures to explain syncope but may eventually normalize with time. This may provide an explanation why the ICD is associated with improved outcomes in the post–ventricular assist device population.

With aggressive heart failure management, the patient has remained in sinus rhythm with only rare ventricular arrhythmias. The patient continues to have stable monitoring of PA pressures and has required occasional changes in diuretic therapy guided by sensor readings. He has not been hospitalized for the last 6 months.

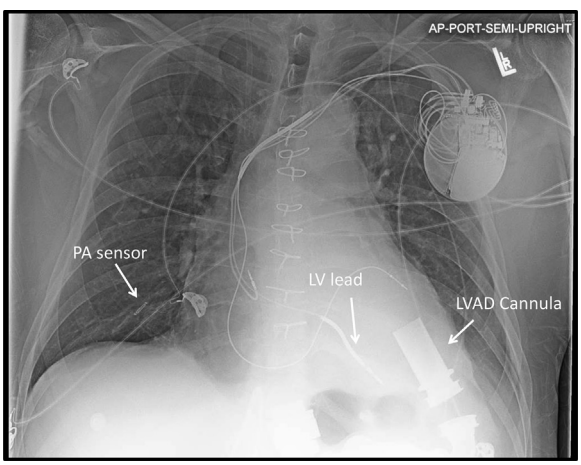

Figure 1 Chest radiograph revealing implanted devices, including pulmonary artery (PA) pressure sensor and chronic high-voltage lead. LV = left ventricle; LVAD = left ventricular assist device.

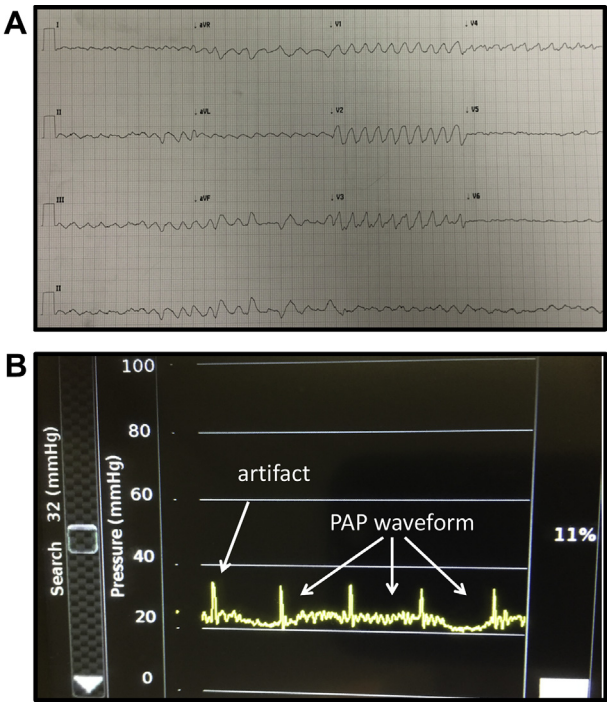

Figure 2 **A:** Twelve-lead electrocardiogram revealing wide complex tachycardia at the time of patient presentation to the emergency room following an episode of witnessed syncope. **B:** Initial interrogation of pulmonary artery pressure (PAP) sensor showing only minimal and fast irregular pressure waveform with a mean of 22 mm Hg.

Discussion

This case illustrates how physiologic PA pressure waveform as a marker of RV function could potentially be used to aid in determination of hemodynamically significant ventricular arrhythmia present in the presence of an ICD in a patient with an LVAD.

Inappropriate shocks continue to be a significant problem, related to supraventricular tachycardia, atrial fibrillation, or inappropriate noise/artifact detection. Despite improvements

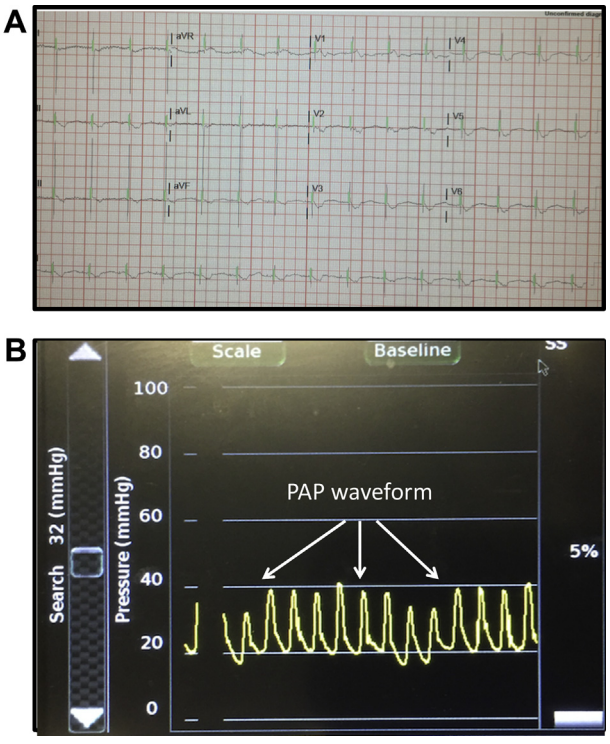

Figure 3 **A:** Electrocardiogram demonstrating an A-V sequential biventricular-paced rhythm post cardioversion. **B:** Postcardioversion interrogation of pulmonary artery pressure (PAP) sensor revealing normal pulmonary artery waveform.

in device detection algorithms and programming insights, ICD detection of life-threatening ventricular arrhythmias still may fail in current systems.[8] For the electrophysiologist, failure to detect a life-threatening arrhythmia is devastating, but one should not discount the negative consequences of inappropriate therapy, including mortality.[9] Reductions in inappropriate therapy with contemporary programming have been demonstrated to reduce mortality.[8]

Patients with an LVAD have varying degrees of preload sensitivity.[10] The mechanism of symptoms of syncope in this patient was presumably a reduction in preload during VA. Hemodynamic monitoring with a PA pressure sensor has the potential to differentiate between a hemodynamically stable and unstable arrhythmia, even in the patient with an LVAD. As preload sensitivity varies in each patient, not all VA may result in hemodynamic instability. Thus, it may be more appropriate to rely on hemodynamic parameters in this population. One might envision algorithms in the LVAD population that allow for defibrillation only if there are hemodynamic consequences.

Hemodynamic parameters have been demonstrated previously to predict VA in patients with advanced heart failure,[11] and hemodynamic sensors have been incorporated into ICD systems.[12,13] Recently, the CardioMEMS PA sensor readings have been incorporated into the same remote monitoring system as cardiac rhythm management devices (Merlin.net; St. Jude Medical, Sylmar, CA). Although this facilitates device and sensor ambulatory follow-up, it does not involve direct device–sensor communication. Patients with an LVAD may often tolerate VA for days before clinical deterioration, again presumably from loss of RV function. Thus, there could be easily programmable features that allow for the patient to withhold therapy until transmission of real-time hemodynamic assessment with the PA pressure. This concept of patient-driven withholding of therapy is currently used with the LifeVest wearable defibrillator.[14]

The CardioMEMS sensor contains a coil and capacitor that form a miniature electrical circuit sensitive to shifts in acoustic frequency.[15] The device coil allows for coupling to the sensor by an external antenna, measuring its resonant frequency, and converts the frequency shifts in a real-time pressure waveform. As modern ICDs communicate via radiofrequency, one could speculate that this could be incorporated into a cardiovascular implantable electronic device such as an ICD and allow for interaction between the sensor and the ICD, and then be incorporated into detection algorithms. Whether or not this is possible remains to be seen.

Conclusion

This clinical case demonstrates an association between the change in PA pressure waveform, indicating loss of RV function, and occurrence of symptomatic VA in a patient implanted with an LVAD. This technology has many potential applications, but if this technology could be linked with ICD therapy it would create an opportunity to differentiate between hemodynamically stable and unstable arrhythmias.

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