Use of Pressure-volume Conductance Catheters in Real-time Cardiovascular Experimentation

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Abstract

Background—Most applications of pressure-volume conductance catheter measurements assess cardiovascular function at a single point in time after genetic, pharmacologic, infectious, nutritional, or toxicologic manipulation. Use of these catheters as a continuous monitor, however, is fraught with complexities and limitations.

Methods—Examples of the limitations and optimal use of conductance catheters as a continuous, real-time monitor of cardiovascular function are demonstrated during inotropic drug infusion in anesthetised rats.

Results—Inotropic drug infusion may alter ventricular dimensions causing relative movement of a well-positioned catheter, generating artifacts, including an abrupt pressure rise at end-systole that leads to over estimation of indices of contractility (max dP/dt) and loss of stroke volume signal. Simple rotation of the catheter, echocardiography-guided placement to the centre of the ventricle, or ventricular expansion through crystalloid infusion may correct for these artifacts. Fluid administration, however, alters left ventricular end-diastolic pressure and volume and therefore stroke volume, thereby obscuring continuous real-time haemodynamic measurements.

Conclusions—Pressure-volume artifacts during inotropic infusion are caused by physical contact of the catheter with endocardium. Repeated correction of catheter position may be required to use pressure volume catheters as a continuous real-time monitor during manipulations that alter ventricular dimensions, such as inotropic therapy.
INTRODUCTION

Measurement of left ventricular pressure and volume is a valuable means for characterising cardiac function [1, 2, 3]. From these measurements and their derivatives, parameters of left ventricle (LV) function and energetics such as stroke volume (SV), stroke work (SW), ejection fraction, pre-load recruitable stroke work, arterial elastance, end-systolic pressure-volume relationship (ESPVR), maximum dP/dt, minimum dP/dt, and relaxation time constants can be obtained. In 1984, Baan and colleagues designed a conductance catheter that could acquire simultaneous pressure-volume (PV) measurements continuously in large animals [4]. This catheter eliminated the need for careful synchronisation of left ventricular manometric pressure readings with volume measurements from labour intensive, costly imaging methods like echocardiography, sonomicrometry, or MRI. A miniature PV catheter for mice was introduced in 1998 [5] and has been used to elucidate the haemodynamic implications of many rodent models of cardiovascular disease [6, 7, 8, 9].

The PV catheter passes a high-frequency low-amplitude current through two sets of electrodes that are ideally oriented along the longitudinal axis of the LV and simultaneously measures electrical potentials that are proportional to ventricular volume. With calibration, these signals can be converted to instantaneous LV blood volume measurements. Also integrated into the catheter is a pressure transducer allowing real time pressure-volume loop generation. While the PV catheter is easier to use and more direct than many cardiac imaging techniques, optimal position and orientation of the catheter in the ventricle at various haemodynamic states must be maintained for accurate measurements. Pacher and colleagues described a comprehensive guide for using this technology [1, 3]. They recommended that the PV catheter be adjusted and optimised prior to recording data, ensuring capture of the maximum LV blood volume signals.

Many applications of PV catheters assess cardiovascular function at a single point in time after genetic, pharmacologic, toxicologic, infectious, environmental or nutritional manipulation of an animal, and these data are compared to control animals without such interventions. These applications require delineation of steady state conditions before and after treatment or intervention, but neglect the time course of the transition. PV conductance catheters can be used as a continuous real-time monitor of cardiovascular function but such use is more complicated and somewhat more limited than at specific points in time. Continuous experiments are often pharmacologic interventions that produce acute haemodynamic changes, on the order of minutes to hours, and can be performed while the PV catheter remains in situ for the duration of the treatment [10, 11]. We have found that such real-time monitoring during pharmacologic treatment can only be performed with frequent manipulation to optimise position, which can lead to disruptions in continuous
signals. Some have presented real-time data from PV catheters [12, 13], but make no mention of the need for periodic catheter placement optimisation.

We demonstrate the limitations of continuous pressure and volume monitoring with PV catheters and methods to overcome them in a model of inotropic drug infusion, dobutamine. This beta adrenergic agonist increases SV and cardiac output through a beta-1 adrenergic mediated positive inotropic response on myocardium and beta-2 adrenergic mediated peripheral arterial and venous dilatation [14]. Dobutamine, therefore, increases contractility while, at the same time, decreases preload and afterload, a combination of effects that tends to decrease the volume of the ventricle. This drug also has the pharmacokinetic advantage of a short serum half-life of three minutes so that haemodynamics changes reach steady-state within 10 minutes of starting an infusion. Real-time continuous catheter measurements can accurately capture swift changes in the pharmacodynamic profile of dobutamine infusion. Our study suggests measures to be taken for optimal use of this technique as a real-time monitor. Researchers performing experimental interventions eliciting similar dynamic cardiovascular responses can benefit from this method of catheter use.

RESEARCH DESIGN AND METHODS

Rodent Preparation and Catheterization

All procedures were approved by the Institutional Animal Care and Use Committee at Steward St. Elizabeth’s Medical Center. Male Sprague-Dawley rats (400–450 grams; Charles River Laboratories, Wilmington, MA) were anaesthetised with pentobarbital (50 mg/kg, intraperitoneal, i.p.), weighed, and an intraperitoneal catheter was placed for continuous infusion of pentobarbital (30 mg/kg/hr). The neck, chest and inguinal areas were shaved and the animal placed supine on a heating pad (#TP-500; Gaymar Industries, Orchard Park, NY) set to maintain rectal temperatures between 36.7–37.0°C. A tracheotomy was performed with a 14-gauge cannula secured and connected to a custom ventilator system which delivered 153 mL/min O₂ through a solenoid valve controlled by a computer (Labview Express 7.0; National Instruments, Austin, TX) at a respiratory rate of 96 breaths per minute, an I:E ratio of 1:3, and an approximate tidal volume of 1.6 mL.

The right femoral artery and internal jugular vein were exposed and cannulated with polyethylene-50 (PE-50) tubing. Both cannulas were de-aired with heparinised saline (2 U/mL) and joined together by a single stopcock connected to a pressure transducer (#TRN050; Kent Scientific, Torrington, CT). Toggling the stopcock allowed acquisition of either the arterial or central venous pressures. A pressure-volume conductance catheter (#SPR-869; Millar Instruments, Houston, TX) was inserted via an arteriotomy in the right carotid artery and advanced retrograde across the aortic valve and into the left ventricle. The right femoral vein was cannulated with PE-50 tubing, and albumin (#A7906, 10% in normal saline; Sigma–Aldrich, St. Louis, MO) delivered through it at 0.25 mL increments until left ventricular PV loops showed the four distinct phases of the cardiac cycle. The sternum was removed through bilateral, anterolateral, vertical thoracotomies and a loop of suture was wrapped around the inferior vena cava (IVC). Preload on the heart was transiently decreased by pulling up on the suture over 7–10 heart beats to obstruct IVC blood flow.
Data Acquisition

Arterial and venous pressures were transduced to a signal amplifier (#TRN005; Kent Scientific, Torrington, CT), digitised (PowerLab/8SP; ADInstruments, Colorado Springs, CO) and recorded in LabChart software (version 7.3.3; ADInstruments, Colorado Springs, CO). Signals from the pressure-volume conductance catheter (MPVS-300 system; Millar Instruments, Houston, Texas) were similarly digitised and stored. At the end of each experiment, blood was collected and added to cuvettes of known dimensions (#910-1048; Millar Instruments, Houston, Texas). The conductance of blood in each cuvette was measured for calibration of ventricular volume.

Catheter Position in Drug Infusion

A drug infusion by syringe pump (#74900-00; Cole Parmer, Vernon Hills, IL) was connected to the internal jugular vein cannula via a stopcock which was toggled transiently between central venous pressure monitoring and drug infusion. After catheterisation and thoracotomy, the animal was allowed to stabilise for 15 minutes prior to starting dobutamine infusion (5 μg/kg/min I.V., 0.2mL/hr).

Prior to drug treatment, the position of the catheter was carefully adjusted until the SV signal was maximised and the PV loop showed four distinct phases corresponding to filling, isovolumetric contraction, ejection, and isovolumetric relaxation. This was done by twisting the catheter while simultaneously advancing into or withdrawing from the LV. Once steady-state drug effects have been reached, the catheter position was adjusted again in a similar manner to ensure optimal PV loop morphology and maximal SV.

Regression based measurements of heart function require transient reduction in preload [15]. For these measurements, the ligature around the IVC was transiently tightened to reduce preload on the LV. This allows regression-based analyses such as the ESPVR, preload recruitable stroke work, and arterial elastance [1, 3] (PVAN Ultra 1.1; Millar Instruments, Houston, Texas).

Echocardiography measurements

Transthoracic echocardiograms were taken during drug treatment. An HP Sonos 5500 echocardiogram unit (Agilent Technologies, Andover, MA) was utilised. Echocardiograms were acquired by using a pediatric neonatal transducer set with the acquisition frame rate of 89 Hz (Model#21380A S12 5–12 MHz Probe, Agilent Technologies). The LV short axis view was used to visualise the axial placement of the catheter in the mid-section of the LV.

RESULTS

PV-loop Morphologic Change by Catheter Manipulation

Intravenous infusion of dobutamine predictably increased SV (Fig. 1A). Approximately 15 minutes after the start of drug delivery, full drug effect was achieved as indicated by a plateau in heart rate (HR), SV and systemic vascular resistance (SVR) tracings. Whereas the morphology of the PV loop initially showed four distinct phases of the cardiac cycle (Fig. 1B), the steady state PV loop showed a loss of ventricular pressure during mid-systolic
ejection with an increase towards the end of systole (Fig. 1C). After manipulation of the catheter during steady state peak drug effect, the PV loop returned closer to its expected four-phase morphology and SV increased (Fig. 1D).

**Post-treatment Rise in Ventricular End Systolic Pressure and its Effect on Pressure-derived Measurements**

In another experiment, prior to dobutamine infusion the pressure during systolic ejection was constant (Fig. 2B). As the infusion progressed and changes in heart loading and inotropy moved the catheter relative to the heart wall, a late systolic ejection pressure peak formed (Fig. 2C). The arterial pressures, however, did not change, thus indicating an apparent new pressure gradient between ventricle and artery (Fig. 2A). Simple rotation of the catheter - up to 180° in either direction restored near normal PV loop morphology (Fig. 2D).

Echocardiographic imaging showed that the catheter had moved adjacent to a papillary muscle during dobutamine infusion. Echocardiographic video showed the displaced catheter pressure transducer being pushed by moving endocardium during systole (Supplemental Video 1). Corresponding PV loops revealed a sharp rise in end-systolic pressure (Fig. 3A). After the catheter position was manually centred in the ventricle, the transducer no longer contacted endocardium (Supplemental Video 2) and the PV loops were restored to normal four phase morphology (Fig. 3B).

The maximum in dP/dt signal normally occurs near the beginning of isovolemic contraction (Fig. 4A). Under conditions where the PV loop shows an end-systolic pressure peak, such as with inotropic drug infusion, two distinct peaks are seen in the dP/dt tracing during each cardiac cycle (Fig. 4B). Pressure at end-systole (P_{es}), stroke work (SW) and max dP/dt measured without eliminating the end systolic pressure (ESP) rise artifact with catheter manipulation are all overestimates (Table 1). Simple rotation of the catheter eliminated the bi-phasic artifact in dP/dt signal (Fig. 4C) and restored these indices of LV function to their correct levels. Inspection of the PV loops before and after repositioning the catheter showed that SW, which represents the energy exerted by the ventricle to eject a volume of blood into the aorta and calculated as the area inside the loop, is overestimated when the end-systolic pressure rise artifact is present. SW, in the example shown (Table 1, and Fig. 4B), is 65% greater than the actual SW calculated after catheter position correction.

**Regression-based Contractility Measurements**

PV loops with normal four-phase morphology responded to IVC occlusion with a steady decrease of end-systolic pressure and volume to give a measureable ESPVR (Fig. 5A). However, an ESP rise artifact forming in the middle of an IVC occlusion yielded artifactually high end-systolic pressures which disrupted the linearity of the ESPVR measurements (Fig. 5B). In another experiment, infusion of crystalloid prior to IVC occlusion improved the morphology of PV loops generated for regression analyses (Fig. 6). PV loops before saline bolus showed decreasing LV systolic pressures but only negligible changes in end diastolic or end systolic volumes during IVC occlusion (Fig. 6A). Since the volume at end systole did not fall with decreasing preload by IVC occlusion, the measured
ESPVR is near infinite. After addition of 1 mL of saline, the baseline end diastolic volume is increased. End systolic volumes shifted to the left with IVC occlusion, allowing calculation of the linear ESPVR (Fig. 6B). While intravascular resuscitation may facilitate measurement of ESPVR in one-time measurements, it also perturbed other haemodynamic parameters (Fig. 6C and Fig. 6D). In this example, the saline bolus (1 mL) increased SV, arterial pressure, and max dP/dt for more than 15 minutes (Fig. 6D).

DISCUSSION

PV conductance catheters are generally used as a convenient and reliable tool to assess cardiovascular function in vivo. When data are acquired at a single point in time after an experimental manipulation of an animal, this technology is simple to use. However, use of the PV catheters continuously in real-time is more complicated. We demonstrated some of their limitations and techniques to overcome these problems.

Correcting for Catheter Migration During Continuous Treatment and Measurements

Dobutamine infusion produced expected inotropic, chronotropic and vascular effects which reached steady state in approximately 15 minutes after the start of drug delivery (Fig. 1A). In this case, as in many previous experiments we have observed, the four distinct phases of the cardiac cycle seen prior to treatment were altered during drug infusion, with a loss of ventricular pressure during mid-systolic ejection and an increase toward end-systole (Fig. 1C). This suggests that the position of the catheter had changed relative to the walls of the ventricle. The catheter is most likely abutting the endocardium of the ventricular wall or papillary muscle. The migration of the catheter is predictable during infusion of beta-1 agonist drug as the preload is decreased and the contractility increased - two effects that decreased both end-diastolic and end-systolic volumes - and potentially moved the walls of the heart closer to the catheter. Manipulation of the catheter returned the PV loop to its expected four-phase morphology and increased measured SV (Fig. 1D). This corrected morphology suggests that the measured SV prior to catheter adjustment was grossly underestimated.

Others have noted that contact of the catheter with endocardium or mechanical deformation during the cardiac cycle can lead to sharp changes in impedance and erroneous volume signals [22, 23]. These observations preceded the advent of miniaturised, ultrafast pressure transducers and, therefore, the impact on pressure-volume loops was not demonstrated. After the introduction of pressure-volume conductance catheters, some have hypothesised that pressure signals can be artifactually inflated from the catheter striking the endocardium (“shock”) [24] and others recommended optimisation of the PV catheter position prior to a single-time analysis of the contractile state [1, 3]. These significant recommendations are applicable for the many studies of cardiac contractility that follow genetic, pharmacologic, toxicologic, infectious, environmental or nutritional manipulation of an animal. The example of inotropic drug infusion with simultaneous acquisition of pressure-volume and two-dimensional ultrasound data strongly supports the need for optimal catheter position (Fig. 3) and, most importantly, demonstrates the need to repeatedly adjust the catheter placement when used as a real-time monitor. Treatments altering haemodynamic function and ventricle...
geometry are likely to change the position of the PV catheter relative to the ventricular wall or papillary muscles. Whereas, initially, the conductance electrodes were centred along the axis of the ventricle, after inotropic drug infusion, the electrodes may be located closer to the myocardium where they might capture artifactual volume and pressure signals (Fig. 3). In addition, with the decrease in ventricular size driven by inotropic drug infusion, the catheter may move slightly out of the ventricle. Our data show that the ventricular volumes after drug infusion, but prior to catheter adjustment, were overestimated much more so at end systole than end diastole so that the measured SV is artifactually low (Fig. 1C and Fig. 1D). Therefore, reliable measurements require periodic adjustment of the catheter position.

**Optimising Pressure-derived Measurements After Obtaining End Systolic Pressure Rise Artifact**

Accurate pressure measurements are also dependent on catheter position. Several studies with PV conductance catheters have demonstrated a rise in ventricular pressure late in systolic ejection [4, 16, 17, 18, 19, 20, 21] and some have interpreted this morphology as an increase in SW, calculated as the area inside the PV loop [19, 20]. We replicated this end systolic pressure rise in our experiment (Fig. 2C) and found that the arterial pressures did not change, thus indicating an apparent new pressure gradient between ventricle and artery (Fig. 2A). This gradient is unlikely to acutely arise from the aortic valve. More likely, the rise in LV pressure late in systole was an intraventricular gradient or artifact. Restoration of normal PV loop morphology (Fig. 2D) after catheter manipulation demonstrated that the former pressure peak late in systolic ejection, seen in this example (Fig. 2B) and in prior studies, was in fact an artifact with no real intraventricular gradient.

Echocardiographic studies reveal that the mechanism of this pressure artifact is the pressure sensing orifice of the catheter approaching the endocardium or papillary muscle in a high pressure zone along the walls of the ventricle (Fig. 3). It is probably facilitated by geometric changes of the heart during inotropic treatment. The catheter pressure transducer has shifted position to be adjacent to the endocardium (Fig. 3A). Therefore, ventricular contraction and associated decrease in ventricular volume would result in endocardium contacting tissue and generating a high-pressure field on the transducer during systole. This is observed in echocardiographic video (Supplemental Video 1) and is evidenced by a sharp rise in end-systolic pressure in PV loops (Fig 3A). Twisting the catheter 180 degrees eliminated this pressure rise artifact (Fig. 3B). This manipulation moved the pressure sensing orifice away from the endocardium.

This end systolic pressure rise artifact may have led some researchers to overestimate the SW and the slope of the pressure-volume relationship (Emax) [24]. Furthermore, this artifact (Fig. 4) can also generate inaccurate contractility - max dP/dt - measurements (Table 1). While the maximum rate of change in ventricular pressure normally occurs at the beginning of isovolumetric contraction (Fig. 4A), the ESP rise artifact generated by dobutamine infusion (Fig. 4B) may yield a second peak in dP/dt signal whose maximum is higher than the peak corresponding to isovolumetric contraction. Automated algorithms that examine the maximum of dP/dt will report the higher of these two peaks and, in some cases, it will be from the ESP rise artifact. This artifact needs to be eliminated during experimentation by

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catheter repositioning (Fig. 4C). Alternatively, one could not use such an automated algorithm and only examine the peak of the dP/dt signal derived during the isovolumetric contraction phase of the PV loops and discard the peak during systolic ejection. Therefore, accurate max dP/dt may need to be manually verified. PV catheter users need to be cognisant that this pressure artifact can develop and the effects it can have on measured indices.

While these data attribute ESP artifact to contact of the catheter with the endocardium that can be easily eliminated, there are certain pathophysiological conditions that can also lead to legitimate, non-artifactual rises in ESP from reflected pressure waves [25, 26]. In particular, ESP rise has been associated with advanced age [27], shorter body length [28], and pathophysiologic conditions with increased arterial stiffness [29].

**Regression-based Contractility Measurements**

The ESPVR from a series of PV loops generated by an IVC occlusion is commonly used as a load-independent index of contractility [15] (Fig. 5A). An artifactual rise in ESP shown in our study can disrupt IVC occlusion-derived haemodynamic measurements due to an induced non-linearity in the pressure volume relationship (Fig. 5B). Since an IVC occlusion will transiently shrink the size of the ventricle, the pressure transducer on the catheter has an increased chance of striking the endocardium and producing an ESP rise artifact. This problem may be alleviated by reorienting the catheter so the pressure sensor is away from endocardium, or by expanding intravascular volume to enlarge the interior dimensions of the heart prior to IVC occlusion.

Under some conditions, a progressive decrease in LV systolic pressure without change in the end systolic volume during IVC occlusion (Fig. 6A) yields infinite ESPVR. Crystalloid infusion prior to IVC occlusion, as recommended by several investigators [1, 3, 4], increased both end-diastolic and end-systolic volumes, improved the morphology of PV loops generated, and allowed calculation of the ESPVR (Fig. 6B). Unfortunately, intravascular crystalloid administration affected other haemodynamic parameters (Fig. 6D), in some cases for more than 15 minutes, disrupting real-time haemodynamic measurements. Maintaining intravascular volume before and during experimentation will help minimise this limitation; however, the transient hypotension caused by IVC occlusion can illicit several reflexes that can disturb continuous measurement of haemodynamic indices. Therefore, regression analyses from IVC occlusions, while comprehensive and less sensitive to heart loading conditions, may not be compatible with continuous or ongoing measurements.

In conclusion, methods for optimising the use of PV catheters for continuous monitoring in small rodents have been described. Preventions and corrections for errant volume and pressure measurements will ensure optimal haemodynamic measurements. Awareness of these issues and procedures will shorten the learning curve for using the PV conductance catheter technology and may have implications on experimental results.

**Supplementary Material**

Refer to Web version on PubMed Central for supplementary material.
Acknowledgments

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References


Figure 1.
Catheter position adjustment to obtain optimal volume signals. (A) Heart rate (HR), mean arterial pressure (MAP), stroke volume (SV) and systemic vascular resistance (SVR) over the course of dobutamine infusion (5 μg/kg/min). Dashed line indicates the start of drug infusion. Grey shaded areas indicate where catheter position was adjusted. Time marked every 4 minutes. (B) The position of the pressure-volume (PV) conductance catheter in left ventricle (LV) is optimised prior to starting the infusion. At steady state drug infusion, HR and SV have increased, while MAP and SVR have fallen from baseline. (C) The steady state pressure-volume loop prior to manipulation for position optimisation. (D) Catheter repositioning at steady state produced step changes in SV and SVR. LV pressure and volume axes have same scales in panels B, C, and D.
Figure 2.
Correcting end-systolic pressure (ESP) rise artifact in pressure-volume (PV) loops. (A) PV conductance catheter position was adjusted (grey shaded areas) prior to start of dobutamine infusion (dashed line) and at steady state. Time marked every 4 minutes. PV loops (B) prior to dobutamine infusion (5 μg/kg/min). (C) At steady state the pressure during late systolic ejection rose sharply (ESP rise artifact). (D) After twisting the catheter up to 180° in either direction, the side-mounted manometer sensor escaped contact with beating endocardium to eliminate most, if not all, of the ESP rise artifact. LV pressure and volume axes have same scales in panels B, C, and D. Left ventricular pressure (LVP), arterial pressure (AP), stroke volume (SV), systemic vascular resistance (SVR).
Figure 3.
Echocardiograms at end diastole during drug delivery with corresponding PV loops. (A) At peak dobutamine infusion, the catheter (dashed arrow) was in contact with endocardium (non-dashed arrows). Corresponding PV loops revealed an end-systolic pressure (ESP) rise artifact. (B) After optimising catheter position, the catheter was relocated to the middle of the ventricle and PV loops showed normal four-phase morphology.
Figure 4.
End-systolic pressure (ESP) rise artifact in pressure-volume (PV) loops gave inaccurate max \( dp/dt \) values. (A) PV loops and corresponding \( dp/dt \) tracings before dobutamine infusion (5 \( \mu g/kg/min \)), (B) during steady state (exhibiting an ESP rise artifact), and (C) at steady state after elimination of artifact by catheter reorientation. The maximum in the \( dp/dt \) signal coincided with isovolumetric contraction (dashed line, *) The ESP rise artifact gave a second peak in \( dp/dt \) signal that coincided with mid to late systolic ejection (solid line, #) and may exceed the peak from isovolumetric contraction. After adjustment of the catheter, the maximum in \( dp/dt \) signal coincided again with isovolumetric contraction (dashed line).
and a lower measurement that is free from ESP rise artifact. Left ventricular (LV) pressure and volume axes have same scales in panels A, B, and C. Time marked every one-tenth second. Left ventricular pressure (LVP), arterial pressure (AP).
Figure 5.
End-systolic pressure (ESP) rise artifact during inferior vena cava occlusions affected end-systolic pressure-volume relationship (ESPVR). (A) Pressure-volume (PV) loops before dobutamine infusion did not show ESP rise artifact and allowed linear regression (dashed line) of end systolic pressures (*) to calculate ESPVR. (B) Dobutamine infusion (5 μg/kg/min) caused an ESP rise artifact which prevented linear regression of the pressures at end systole for ESPVR calculations.
Figure 6.
Normal saline bolus affects contractility assessment from inferior vena cava (IVC) occlusions. Pressure-volume (PV) loops with IVC occlusions (A) without saline bolus showed negligible shift in ventricular dimensions. (B) After saline bolus (1 mL) both diastolic and systolic ventricular dimensions fell steadily over 8 heart beats during IVC occlusion. Left ventricular (LV) pressure and volume axes have same scales in panels A and B. End-systolic pressures are denoted by * and line. (C) Zoomed-in view of haemodynamic indices including IVC occlusions before and after saline bolus. Time marked every 10 seconds. Dark shaded area = IVC occlusion, light shaded area = normal saline bolus. (D) Zoomed-out view of haemodynamic measurements. Adding 1 mL saline bolus increased arterial pressure, stroke volume (SV), and max dP/dt for over 15 minutes. Time marked every 2 minutes. Solid lines mark baseline haemodynamic values. Dashed lines mark elevated haemodynamic values after saline bolus. Left ventricular pressure (LVP), arterial pressure (AP), stroke volume (SV).
Table 1

Hemodynamic indices affected by ESP rise artifact

<table>
<thead>
<tr>
<th>PV Loops*</th>
<th>( P_{es} ) (mmHg)</th>
<th>SW (mmHg x ( \mu L ))</th>
<th>( E_a ) (mmg/( \mu L ))</th>
<th>Max dP/dt (mmHg/s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-drug delivery</td>
<td>93.06</td>
<td>6,302</td>
<td>1.08</td>
<td>4,730</td>
</tr>
<tr>
<td>Peak drug effect + ESP rise artifact</td>
<td>205.13</td>
<td>12,500</td>
<td>1.32</td>
<td>10,514</td>
</tr>
<tr>
<td>Peak drug effect after adjusting PV catheter</td>
<td>84.13</td>
<td>8,082</td>
<td>0.66</td>
<td>6,308</td>
</tr>
</tbody>
</table>

* PV loops are from Figure 3

\( P_{es} \) = end-systolic pressure, SW = stroke work, \( E_a \) = arterial elastance