Predicting Fluid Responsiveness During Infrarenal Aortic Cross-Clamping in Pigs

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Objective: **Infrarenal aortic cross-clamping (ACC) induces hemodynamic disturbances that may affect respiratory-induced variations in stroke volume and, therefore, affect the ability of dynamic parameters such as pulse-pressure variation (PPV) to predict fluid responsiveness. Since this issue has not been investigated yet to authors' knowledge, the hypothesis was tested that ACC may change PPV and impair its ability to predict fluid responsiveness.**

Design: **Prospective laboratory experiment.**

Setting: **A university research laboratory.**

Participants: **Nineteen anesthetized and mechanically ventilated pigs.**

Interventions: **Two courses of volume expansion were performed using 500 mL of saline before and during ACC. Animals were monitored using a systemic arterial catheter, and a pulmonary arterial catheter (stroke volume, central venous pressure, pulmonary arterial occlusion pressure). Animals were defined as responders to volume expansion if stroke volume increased ≥15%.**

Results: **Before ACC, 13 animals were responders. Fluid responsiveness was predicted by a PPV ≥14% with a sensitivity of 77% (95% CI = 46%-95%) and a specificity of 83% (95% CI = 36%-97%). The area under the receiver operating characteristic curve was 0.90(95% CI = 0.67-0.99) and was higher than those generated for central venous pressure and pulmonary arterial occlusion pressure. ACC induced an increase in PPV ($p < 0.0005$). During ACC, 8 animals were responders. An 18% PPV threshold discriminated between responders and non-responders to volume expansion, with a sensitivity of 87% (95% CI = 47%-98%) and a specificity of 54% (95% CI = 23%-83%). The area under the receiver operating characteristic curve was 0.72 (95% CI = 0.47-0.90) and was not different from those generated for central venous pressure and pulmonary arterial occlusion pressure.**

Conclusions: **ACC induced a significant increase in PPV and reduced its ability to predict fluid responsiveness.**

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PERIOPERATIVELY OPTIMIZING cardiac preload is paramount for precise hemodynamic management. It has now been demonstrated clearly that dynamic parameters resulting from heart-lung interactions are superior to static indicators for predicting fluid responsiveness.[1–4] Respiratory-induced variations in stroke volume may be assessed reliably from the arterial pressure waveform by determining pulse-pressure variations (PPV).[5–8] It has been shown that cardiac output (CO) optimization using PPV reduces the length of hospital stay, critical care admissions, and morbidity after major surgery in various settings.[9–11]

Intraoperative fluid management is very important during aortic surgery because it may impact the incidence of post-operative ischemic cardiac events, acute kidney injury and hypoxemia.[12] Aortic cross-clamping (ACC), which is required during open aortic abdominal surgery, results in profound hemodynamic disturbances including cardiac stress, increases in systemic vascular resistance (SVR), and mean arterial pressure (MAP), decreases in right ventricular preload and CO, and a blood volume redistribution.[13] Infrarenal aortic unclamping induces a significant decrease in MAP and physicians often perform volume expansion during aortic cross-clamping in order to reduce the risk of hypotension. Thus, clinicians could be tempted to use PPV to guide fluid administration during ACC. However, the impact of hemodynamic changes induced by ACC on PPV and its ability to predict fluid responsiveness remain poorly understood.

Therefore, the purpose of this experimental study was to evaluate and compare the ability of PPV to predict fluid responsiveness during infrarenal ACC in the pig. The authors tested the hypothesis that hemodynamic disturbances induced by ACC could induce variations in the absolute value of PPV and impair its ability to predict the effects of fluid administration.

METHODS

The experimental protocol procedures utilized in this study were approved by the Hospital Animal Care and Use Committee.

Twenty-four pigs (63 ± 8 kg) were studied. Animals had free access to food and water until 12 h before the beginning of the experiments. After premedication with intramuscular ketamine (30 mg/kg) and dexmedetomidine (10 μg/kg), a peripheral vein was cannulated. Anesthesia was induced intravenously with propofol (1.5 mg/kg), and the trachea was intubated after local anesthesia during spontaneous respiration. Anesthesia was maintained using propofol (10 mg/kg/h), sufentanil (bolus of 1 μg/kg followed by a continuous infusion of 10 μg/kg/h), and cisatracurium besilate (bolus of 0.2 mg/kg followed by a continuous infusion of 0.5 mg/kg/h). The animals were ventilated mechanically with 50% oxygen in air using a constant-volume respirator (ALYS 2000, Taema, Antony, France). Tidal volume was set at 10 mL/kg, the respiratory rate at 14 breaths/min, the positive

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end-expiratory pressure (PEEP) of 0 cmH_2O, and the inspiratory-to-expiratory ratio at 1:2. Respiratory gases were monitored continuously.

After a single dose of 50 IU/kg of heparin was given, the right internal jugular vein and right internal carotid artery were both cannulated with 8.0-French introducer sheaths. A flow-directed Swan-Ganz catheter (CCOmboV™, 7.5 French; Edwards Lifesciences, Irvine, CA) was introduced through the right internal jugular vein and advanced into the pulmonary artery. The correct position of the pulmonary artery catheter in West's zone 3 was verified using a previously described method.[14] The pulmonary artery catheter was connected to the Vigilance monitor (Edwards Lifesciences, Irvine, CA). Through the right carotid artery, a 7F dual-field pressure-volume conductance catheter (CD Leycom, Zoetermeer, Netherlands) was positioned in the left cardiac ventricle for continuous pressure and volume monitoring. Conductance catheter placement was guided by echocardiography and left ventricle pressure signals. Assessment of the individual segments of the pressure-volume loops confirmed the proper placement of the catheter. Blood resistivity rho (ρ) was measured at the beginning of the procedure. The theory of conductance volumetry previously has been described extensively.[15] Parallel conductance was measured twice by injecting 10 mL of hypertonic saline into the right atrium and blood resistivity was determined.[16] For each time point (T1-T6), CO measured by the conductance catheter was compared with CO as determined by a pulmonary arterial catheter in order to produce a constant of proportionality (α). Derived CO was corrected against CO measured by pulmonary thermodilution at each measurement step during the offline analysis. Arterial pressure tracings and MAP were measured by a left carotid arterial catheter (6-French) inserted into the descending thoracic aorta. Fluid-filled transducers were zeroed against atmospheric pressure at the anterior axillary level and calibrated with a mercury manometer. Continuous ECG monitoring was performed. Animals were placed on a fluid-filled heating pad to maintain rectal temperature between 37 and 38°C. A basal infusion of balanced electrolyte solution was administered at a rate of 5 mL/kg/h.

The volume arm of the conductance catheter was connected to a signal processor (Sigma 5 DF; CD Leycom, Zoetermeer, Netherlands), and the pressure sensor was connected to an electronic pressure interface (Sentron, CD Leycom, Zoetermeer, Netherlands). End-diastolic pressure and end-systolic pressure were obtained from the left ventricular waveform and determined by an algorithm from EMKA Technologies (Falls Church, VA). The maximal positive (dP/dt_{max}) and negative (dP/dt_{min}) left ventricular pressure derivatives were electronically derived from the left ventricular pressure signal. The continuous pressure signals were sampled digitally at 1,000 points/s. The data were displayed in real time using IOX® software version 2.4.7.3 (EMKA Technologies, Paris, France) and analyzed by an algorithm from EMKA Technologies. The left ventricular parameters (ie, end-systolic and end-diastolic pressure, $dP/dt_{max}$and dP/dt_{min}) were averaged over more than 20 successive heartbeats. The continuously recorded hemodynamic and respiratory parameters were stored at a sampling rate of 1000 Hz.

Cardiac output was measured by pulmonary artery thermodilution using the average of 3 consecutive measurements obtained by manual injection of 10 mL of iced saline (4°C) randomly during the respiratory cycle. Mixed venous saturation was measured continuously. CO and mixed venous saturation were displayed on the Vigilance monitor.

Pulse-pressure variation was defined as the difference between systolic and diastolic arterial blood pressures. Maximal (Pulse Pressure max) and minimal (Pulse Pressure min) values were determined over the same respiratory cycle. PPV then was calculated as: PPV = (Pulse Pressure max – Pulse Pressure min)/[(Pulse Pressure max + Pulse Pressure min)/2]*100 as previously described.[3,8] PPV was evaluated over each of 5 consecutive respiratory cycles. The mean values of the 5 determinations were used for statistical analysis.

Central venous pressure and *pulmonary artery occlusion pressure* were measured at the end of expiration and averaged over 3 consecutive respiratory cycles.

Systemic vascular resistance (dynes.s/cm^5) was calculated as [(mean arterial pressure – central venous pressure)/CO] × 80.

The *total positive end-expiratory pressure and plateau pressure* were measured using an end-expiratory and end-inspiratory occlusion maneuver of 5 seconds. Tidal volume was measured by the ventilator transducer. Static compliance of the respiratory system was calculated as follows: tidal volume / (plateau pressure – total positive end-expiratory pressure).

Arterial blood gas and *electrolytes analysis* were performed before the beginning of the protocol (after surgical preparation) and during infrarenal aortic cross-clamping.

Through a mid-laparotomy incision and following hemodynamic stabilization, the infrarenal abdominal aorta was dissected and isolated 1 cm above the origin of the left renal artery and loosely encircled with an umbilical tape snare. Infrarenal aortic cross-clamping was performed using an aortic cross-clamp (Cooley, Surtex Instruments Ltd, Surrey, United Kingdom). The abdominal incision was closed during the procedure to avoid liquid loss and hypothermia.

The protocol was initiated after at least 30 min of hemodynamic stability following surgical preparation. Six sets of measurements were

Table 1. Hemodynamic Variables Before and 10 Minutes After Infrarenal Aortic Cross-clamping (n = 19)

	Before Aortic Cross-clamping	10 Min After Aortic Cross-clamping	p Value
HR (bpm)	88 [77-101]	91 [73-101]	NS
	87 ± 17	87 ± 21	
MAP (mmHg)	87 [72-95]	93 [70-110]	<0.05
	85 ±18	91 ± 20	
CO (L/min)	4.2 [3.6-4.9]	3.5 [2.9-4.2]	<0.001
	4.2 ± 1.0	3.4 ± 0.9	
MPAP (mmHg)	20 [17-22]	18 [16-20]	<0.05
	20 ± 5	18 ± 5	
CVP (mmHg)	6 [5-9]	5 [4-8]	<0.005
	7 ± 3	6 ± 3	
PAOP (mmHg)	8 [7-10]	8 [6-9]	NS
	9 ± 3	8 ± 3	
SVR (dynes.s/cm^5)	1787 [1467-2321]	2583 [2001-3045]	<0.001
	1939 ± 651	2589 ± 766	
Ea (mmHg/mL)	2.0 [1.6-2.1]	2.3 [2.0-2.9]	<0.001
	1.9 ± 0.5	2.4 ± 0.6	
dP/dt/EDV	10 [8-12]	8 [7-11]	NS
	10 ± 4	8 ± 7	
Tau (ms)	30 [28-32]	33 [30-40]	<0.05
	30 ± 3	40 ± 17	
PPV (%)	12 [10-17]	22 [18-31]	<0.001
	14 ± 6	25 ± 9	
SvO2 (%)	65 [59-70]	57 [52-69]	<0.05
	63 ± 8	59 ± 9	

NOTE: Data are expressed as median [interquartile range 25%-75%] and mean ± standard deviation.

Abbreviations: CO, cardiac output; CVP, central venous pressure; dP/dt/EDV, maximal change in pressure over time/end-diastolic volume ratio; Ea, arterial elastance; HR, heart rate; MAP, mean arterial pressure; MPAP, mean pulmonary arterial pressure; NS, not significant; PAOP, pulmonary arterial occlusion pressure; PPV, pulse-pressure variation; SvO2, central venous oxygen saturation; SVR, systemic vascular resistance; Tau, left ventricular relaxation time constant.

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