

TRANSLATIONAL RESEARCH

Effects of arterial load variations on dynamic arterial elastance: an experimental study

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Abstract

Background. Dynamic arterial elastance ($E_{a_{dyn}}$), the relationship between pulse pressure variation (PPV) and stroke volume variation (SVV), has been suggested as a functional assessment of arterial load. The aim of this study was to evaluate the impact of arterial load changes during acute pharmacological changes, fluid administration, and haemorrhage on $E_{a_{dyn}}$.

Methods. Eighteen anaesthetized, mechanically ventilated New Zealand rabbits were studied. Arterial load changes were induced by phenylephrine ($n=9$) or nitroprusside ($n=9$). Thereafter, animals received a fluid bolus (10 ml kg^{-1}), followed by stepwise bleeding (blood loss: 15 ml kg^{-1}). The influence of arterial load and cardiac variables on PPV, SVV, and $E_{a_{dyn}}$ was analysed using a linear mixed-effects model analysis.

Results. After phenylephrine infusion, mean (SD) $E_{a_{dyn}}$ decreased from 0.89 (0.14) to 0.49 (0.12), $P<0.001$; whereas after administration of nitroprusside, $E_{a_{dyn}}$ increased from 0.80 (0.23) to 1.28 (0.21), $P<0.0001$. Overall, the fluid bolus decreased $E_{a_{dyn}}$ [from 0.89 (0.44) to 0.73 (0.35); $P<0.01$], and haemorrhage increased it [from 0.78 (0.23) to 0.95 (0.26), $P=0.03$]. Both PPV and SVV were associated with similar arterial factors (effective arterial elastance, arterial compliance, and resistance) and heart rate. Furthermore, PPV was also related to the acceleration and peak velocity of aortic blood flow. Both arterial and cardiac factors contributed to the evolution of $E_{a_{dyn}}$ throughout the experiment.

Conclusions. Acute modifications of arterial load induced significant changes on $E_{a_{dyn}}$; vasodilatation increased $E_{a_{dyn}}$, whereas vasoconstriction decreased it. The $E_{a_{dyn}}$ was associated with both arterial load and cardiac factors, suggesting that $E_{a_{dyn}}$ should be more properly considered as a ventriculo-arterial coupling index.

Key words: arterial pressure; cardiac output; pulse wave analysis; ultrasonography, Doppler

The effects of mechanical ventilation on heart–lung interaction and the usefulness of dynamic indices of preload for guiding fluid therapy have been extensively described.¹ Briefly, the magnitude of the swings in left ventricular stroke volume (SV) induced by IPPV defines the degree of preload dependence of a patient and can be used to predict whether or not cardiac output

will increase with fluids.² Given that the main determinant of arterial pulse pressure (PP) during a respiratory cycle is SV,³ changes in arterial PP have been proposed as a surrogate for the cyclic variations in left ventricular SV induced by mechanical ventilation.⁴ However, although arterial system properties are assumed to be relatively constant during a respiratory cycle,³

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Editor's key points

- Dynamic arterial elastance ($E_{a_{\text{dyn}}}$) describes the relationship between pulse pressure variation (PPV) and stroke volume variation (SVV) and may be useful as a functional measure of arterial load.
- The impact of arterial load changes on $E_{a_{\text{dyn}}}$ was measured in a rabbit model.
- Acute changes in arterial load affected $E_{a_{\text{dyn}}}$ and the impact of arterial and cardiac factors was different for PPV and SVV.
- $E_{a_{\text{dyn}}}$ may be considered as a ventriculo-arterial coupling index in this animal model.

circumstances such as sepsis, vasoactive therapy, or acute haemorrhage could alter the pressure–flow relationship,^{5–8} hence the PP/SV interaction.

The ratio of the pulse pressure variation (PPV) to the stroke volume variation (SVV) during a mechanical breath has been called dynamic arterial elastance ($E_{a_{\text{dyn}}}$).^{9–10} The $E_{a_{\text{dyn}}}$ depicts the slope of the pressure–flow relationship during a respiratory cycle, and it may be a useful parameter to predict arterial pressure response after fluid administration in preload-dependent patients.^{10–11} Rather than a steady-state index, $E_{a_{\text{dyn}}}$ represents the dynamic interaction between changes in arterial pressure and SV during a respiratory cycle and has been considered a functional parameter of arterial load.¹⁰ Given that the relationship between arterial PP and SV is determined by the interaction between the left ventricle and the arterial system,¹² the impact of changes in the arterial system could alter the relationship between PPV and SVV and consequently also $E_{a_{\text{dyn}}}$.

We therefore hypothesized that changes in arterial load would significantly alter the PPV/SVV relationship (i.e. $E_{a_{\text{dyn}}}$). In addition, we also aimed to determine the arterial and cardiac factors that could potentially influence this relationship, in order to understand the physiological mechanisms involved in $E_{a_{\text{dyn}}}$ changes. We tested this hypothesis in an experimental rabbit model of arterial load variations induced by vasoactive administration, fluid expansion, and acute bleeding.

Methods

Eighteen New Zealand rabbits 2.5 (0.3) kg, supplied by the Reproduction Laboratory of the University of Cadiz, were maintained at a controlled temperature (23 °C) in individual cages on a 12 h–12 h light–dark cycles with free access to food and water up to the time of experimental procedures. All procedures and protocols in this investigation were reviewed and approved by the Ethical Committee for Animal Experimentation of the School of Medicine of the University of Cadiz (license 07–9604). Animal care and use procedures conformed to European Ethical Standards (2012/707/EU) and Spanish Law (RD 53/2013) for the care and use of laboratory animals for experimental research. Relevant aspects of the ARRIVE guidelines were followed.

Animal preparation and instrumentation

Animals were premedicated with an i.m. dose of xylazine hydrochloride (10 mg kg^{−1}) and ketamine (40 mg kg^{−1}). A tracheostomy was performed, and animals were mechanically ventilated (Servo 900c; Siemens-Elcoma, Solna, Sweden) in volume-controlled

mode, with a tidal volume of 8 ml kg^{−1}, PEEP of 0 kPa, inspiratory-to-expiratory ratio of 1:2, fractional inspired O₂ of 0.6, and the respiratory rate adjusted to maintain an end-tidal CO₂ between 4.6 and 6 kPa. The right internal jugular vein was catheterized for a continuous administration of ketamine (15–20 mg kg^{−1} h^{−1}) and xylazine (6 mg kg^{−1} h^{−1}). Neuromuscular block was maintained with a rocuronium bromide infusion (1 mg kg^{−1} h^{−1}). Ringer's lactate solution (6 ml kg^{−1} h^{−1}) was administered as a maintenance fluid therapy. A 22-gauge sterile polyethylene catheter was inserted into the femoral artery and connected to a pressure transducer (TruWave; Edwards Lifesciences LLC, Irvine, CA, USA). The transducer was zeroed against atmospheric pressure, and optimal damping of the arterial waveform was checked by flushing the line before any recording. The left femoral vein was used to administer vasoactive agents and the fluid bolus. The adequacy of anaesthesia throughout the experiment was assessed by physiological responses to a nociceptive stimulus (tail clamping).

Haemodynamic monitoring

A paediatric oesophageal Doppler probe (KDP72; Deltex Medical, UK) was introduced into the oesophagus until the optimal outline and maximal peak velocity (PV) of the aortic blood waveform was obtained. Given that the aortic diameter was estimated by the Doppler system (CardioQ Combi, Deltex Medical, Chichester, UK) using a nomogram for human adults, we calculated cardiac output (CO) using the minute distance of aortic blood flow (MD) as follows: $CO = 1.158 \times (MD \times \text{animal height}^2 \times 10^{-7})^{0.785}$.¹³ The MD represents the distance travelled by a column of blood in 1 min and is calculated by the Doppler system as the product of heart rate (HR) and the velocity–time integral of the aortic flow waveform (i.e. stroke distance).

The arterial pressure signal was transferred from the multiparametric monitor (S/5; Datex-Ohmeda, Helsinki, Finland) to the Doppler system and automatically synchronized with the aortic blood flow waveform.

Arterial load assessment

A three-element Windkessel model was used for characterizing the arterial system,¹⁴ consisting of the following: systemic vascular resistance [$R = \text{mean arterial pressure (MAP)}/\text{CO}$]; net arterial compliance [$C = \text{SV}/\text{PP}$],¹⁵ and characteristic impedance (Z_c). The Z_c represents the arterial input impedance in the absence of arterial wave reflections.¹⁶ Assuming that reflections are minimal at the beginning of the systole, Z_c was calculated as the slope of the early ejection pressure–flow relationship, using the ratio between the maximal first derivative of pressure (dP_{max}/dt) and flow (dQ_{max}/dt) during 10 consecutive cardiac cycles.^{17–18} An example of this calculation is shown in the Supplementary material. The effective arterial elastance was computed as $E_a = \text{end-systolic pressure}/\text{SV}$, and used as a combined parameter that represents both resistive and pulsatile components of arterial load.¹² The dicrotic notch pressure was used as an estimate of end-systolic pressure.¹⁵

Pulse pressure variation, stroke volume variation, and dynamic arterial elastance

Pulse pressure variation was calculated as the percentage changes in PP during a ventilatory cycle as $[(PP_{\text{max}} - PP_{\text{min}})/(PP_{\text{max}} + PP_{\text{min}})/2] \times 100$, where PP_{max} and PP_{min} indicate the maximal and minimal arterial PP, respectively. Calculation of SVV was performed using a similar formula. Values of SV_{max} and

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