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Clinical Investigation

CLINICAL INVESTIGATION

Dynamic arterial elastance measured by uncalibrated pulse contour analysis predicts arterial-pressure response to a decrease in norepinephrine

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Abstract

Background: Dynamic arterial elastance (Ea_{dyn}) has been proposed as an indicator of vascular tone that predicts the decrease in arterial pressure in response to changes in norepinephrine (NE). The purpose of this study was to determine whether Ea_{dyn} measured by uncalibrated pulse contour analysis (UPCA) can predict a decrease in arterial pressure when the NE dosage is decreased.

Methods: We conducted a prospective study in a university hospital intensive care unit. Patients with vasoplegic syndrome for whom the intensive care physician planned to decrease the NE dosage were included. Haemodynamic and UPCA (VolumeView and FloTrac; Edwards Lifesciences, Irvine, CA, USA) values were obtained before and after decreasing the NE dosage. Responders were defined by a >10% decrease in mean arterial pressure (MAP).

Results: Of 35 patients included, 11 (31%) were pressure responders with a median decrease of 13%. Ea_{dyn} was correlated to systolic arterial pressure (SAP) (r=0.255; P=0.033), diastolic arterial pressure (r=0.271; P=0.024), MAP (r=0.310; P=0.009), heart rate (r=0.543; P=0.0001), and transthoracic echography cardiac output (r=0.264; P=0.024). Baseline Ea_{dyn} was correlated with MAP changes (r=0.394; P=0.019) and SAP changes (r=0.431; P=0.009). Ea_{dyn} predicted the decrease in arterial pressure with an area under the receiver-operating-characteristic curve of 0.84 (95% confidence interval: 0.70–0.97). The best cut-off was 0.90.

Conclusions: The present study confirms the ability of Ea_{dyn} measured by UPCA to predict an arterial pressure response to a decrease in NE. Ea_{dyn} may constitute an easy-to-use functional approach to arterial tone assessment regardless of the monitor used to measure its determinant.

Clinical trial registration: DRCIT95.

Keywords: hemodynamics; norepinephrine; vasoplegia

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Editor's key points

- Dynamic arterial elastance is a potential measure of arterial tone that may respond to changes in vasopressor drug dosage.
- Previous studies of dynamic arterial elastance have used cardiac-output-calibrated pulse contour analysis, but this has been shown to be an inaccurate measure of cardiac output.
- In this study, critically ill patients with vasoplegia were studied using uncalibrated pulse contour analysis.
- There was a correlation between dynamic arterial elastance and arterial pressure changes in response to a reduction in norepinephrine dose.
- However, the study was small, and the patients were heterogeneous, so the results should be interpreted with caution.

Norepinephrine (NE) is the first-line treatment for vasoplegic syndrome recommended by international guidelines.[1,2] Dynamic arterial elastance (Ea_{dyn}) has been proposed as an indicator of arterial tone that can predict NE-dependent arterial pressure.[3] Ea_{dyn} is calculated using the ratio of respiratory pulse-pressure variation (PPV) over the respiratory stroke volume variation (SVV). Guinot and colleagues[4] demonstrated a decrease in the duration of NE treatment with the use of Ea_{dyn}.[4] To date, studies that have validated Ea_{dyn} at bedside have used cardiac-output (CO) calibrated pulse contour analysis[3] (PiCCO™ and PULSION™) or oesophageal Doppler.[5] Such monitoring systems need dedicated and specific arterial line and venous access that may limit their use at bedside. In addition to CO calibrated pulse contour analysis, CO uncalibrated pulse contour analysis (UPCA) has been developed and is considered less invasive. Nevertheless, one limitation of the latter CO monitoring is inaccuracy of CO measurement in patients who are being treated with NE.[6,7] Mahjoub and colleagues[8] have demonstrated that CO measured by UPCA is inaccurate and unable to track CO trends with NE. These limitations may affect the predictability of Ea_{dyn}. To date, no study has evaluated the ability of Ea_{dyn} measured by UPCA to assess the arterial-pressure response to a decreased dose of NE.

We tested the hypothesis that Ea_{dyn} calculated from UPCA predicts the arterial-pressure response to an NE decrease in patients treated with NE.

Methods

Ethics

The study objectives and procedures were approved by the local ethics committee (Le Comité de Protection des Personnes Nord Ouest II CHU, Place Victor Pauchet, 80054 Amiens Cedex 1, France, RNI 2016-28). All subjects received written information about the study and provided their consent to participate. The present paper was drafted in compliance with the Strengthening the Reporting of Observational Studies in Epidemiology checklist for cohort studies.[9]

Patients

A prospective, observational study was conducted at the Amiens University Hospital intensive care unit (ICU) from July 2016 to March 2017. The inclusion criteria were all consecutive patients with a diagnosis of vasoplegic syndrome, treated with NE, for whom the attending physician decided to decrease the NE dosage. The vasoplegic syndrome was defined as persistent arterial hypotension with normal or high CO and low systemic vascular resistance (SVR).[10] The exclusion criteria were patients treated with epinephrine or dobutamine, arrhythmia, intra-abdominal hypertension, and patients younger than 18 yr. All patients were sedated with a continuous infusion of midazolam and sufentanil and were ventilated in volume-controlled mode. In our institution, the dosage of NE is decreased by 3.3 μg min^{-1} every 15 min as long as the mean arterial pressure (MAP) remains higher than 65 mm Hg. The indication for decreasing the dosage of NE was left to the physician's discretion. Only a one-step NE dose reduction was assessed in this study.[3]

Haemodynamic variables

We measured the UPCA CO (CO_{UPCA}) and SVV using UPCA (Monitor Vigileo, FloTrac™ pressure transducer software version 4.0; Edwards Lifesciences, Irvine, CA, USA), and obtained the average of five successive values.

We also measured the transthoracic echography CO (CO_{TTE}) by transthoracic echocardiography (CX50 Ultrasound System and S5-1 Sector Array Transducer; Philips Medical System, Suresnes, France). The diameter of the left ventricular outflow tract was measured on a long-axis parasternal view at the time of patient inclusion. The aortic velocity–time integral (VTIAo) was measured with pulsed Doppler on a five-chamber apical view. Stroke volume (SV; ml) was calculated as VTIAo × aortic area. CO_{TTE} (litres min^{-1}) was calculated as SV × heart rate (HR).

PPV was automatically calculated by the Philips monitoring system and represents the average of five successive values.[11] All SVV and PPV values were sampled at the same time on radial arterial line. Dynamic arterial elastance (Ea_{dyn}) was defined as the ratio of PPV/SVV.

We calculated SVR as SVR=MAP − CVP/CO_{TTE} * 80 (dynes s cm^{-5}). We calculated the static arterial compliance (C_A) by using transthoracic SV (SV_{TTE}) and SV Vigileo (SV_{UCP}) as $C_{A\ TTE}$=SV_{TTE}/arterial pulse pressure (PP) (ml $mmHg^{-1}$) and $C_{A\ UCP}$=SV_{UCP}/PP (ml $mmHg^{-1}$). We calculated the net arterial elastance (E_A) by using transthoracic SV (SV_{TTE}) and SV vigileo (SV_{UCP}) as $E_{A\ TTE}$=MAP/SV_{TTE} (ml $mmHg^{-1}$) and $E_{A\ UCP}$=MAP/SV_{UCP} (ml $mmHg^{-1}$).

Study protocols

The following clinical data were recorded: age, sex, surgical/medical history, indications for NE treatment, and Simplified Acute Physiology Score 2. The following haemodynamic measurements were recorded at baseline, with the patient connected to the ventilator: HR, systolic arterial pressure (SAP), MAP, diastolic arterial pressure (DAP), central venous pressure (CVP), SVV, PPV, CO_{UPCA}, CO_{TTE}, central venous saturation ($ScVO_2$), and arterial lactate. The dose of NE was decreased. After the stabilisation of haemodynamic variables, as assessed by the absence of variation of MAP by >10% over a 30 min period, a second set of measurements (HR, SAP, MAP, DAP, CVP, CVP, SVV, PPV, CO_{UPCA}, CO_{TTE}, $ScVO_2$, and arterial lactate) was recorded. The ventilator settings and sedations were kept constant throughout the study period.