Heart rate and the assessment of changes in venous return after phenylephrine

Alain F. Kalmar and Thomas W. L. Scheeren

Department of Anesthesiology, University Medical Center Groningen, University of Groningen, Groningen, The Netherlands

TO THE EDITOR: The article by Cannesson et al. (1) convincingly explains several analogous changes on dynamic preload variables, which we have only recently been able to monitor noninvasively in daily patient care. Although phenylephrine has been in clinical use for decades, measurements of advanced hemodynamic variables in a beat-to-beat fashion are regrettably much more laborious than simple blood pressure assessment, resulting in considerable bias among clinicians of its overall clinical effects.

Our observations using the Nexfin (Edwards Lifesciences BMEYE, Amsterdam, The Netherlands) monitor during routine clinical practice for continuous noninvasive monitoring of advanced hemodynamic variables manifestly confirms the distinct effects of phenylephrine on venous return and global hemodynamics as reported by Cannesson et al. However, an important issue to properly interpret the effects of phenylephrine on stroke volume (SV), stroke volume variation (SVV), or respiratory variations in pulse pressure (PPV) is considering its effect on heart rate (HR). In the Cannesson et al. report, there was no significant bradycardia—most probably because of atropine premedication—but this common effect of phenylephrine may conceivably result in an increase in SV resulting from an increased diastolic filling. In that case, an increase in SV is (at least) not an exclusive beneficial result of improved venous return. Equally, a decrease in SVV or PPV may be a result of decreased cardiac output (CO) with subsequent increased venous pooling.

Interestingly, atropine is often administered prophylactically in anticipation of vagal stimulating ophthalmic interventions, largely preventing the reflex bradycardia of phenylephrine.

As we now routinely monitor advanced hemodynamics in these patients using the noninvasive Nexfin device, we took the opportunity to evaluate the evolution of SV, SVV, and PPV in relation to HR and CO in patients who were parasympathetically blocked vs. non-parasympathetically blocked with atropine and received 100 µg phenylephrine. SVV and PPV decreased in both groups to a comparable amount, but in the non-parasympathetically blocked patients bradycardia results in an overall reduction in CO (2).

An undeniable increase in CO in concert with stable HR in the parasympathetically blocked patients confirms a recruitment of blood volume from capacitance vessels.

However, in the non-parasympathetically blocked patients, there was an equivalent effect on SV, SVV, and PPV, but a decreased CO, most probably because of the induced bradycardia. Although the decrease in SVV still reflects an optimization of the position on the Frank-Starling curve, this may be partly attributable to increased venous pooling as a result of decreased CO.

If a clinician were to rely only on a decreased SVV or decreased variation of pulse oximetric plethysmographic waveform amplitude (ΔPOP), this may falsely suggest an increased CO by virtue of increased SV, whereas the ultimate macrohemodynamic result is a decrease in CO and tissue perfusion. In addition to these negative systemic effects, the combination of pulmonary vasoconstriction and increased venous return by phenylephrine may put additional stress on the right heart (4).

We suggest that administration of phenylephrine in parasympathically blocked patients provides an increased perfusion pressure with positive effect on cardiac output. However, as higher doses of phenylephrine are well known for their negative effects on cardiac output and tissue perfusion (3) and because their effects are dependent on several complex interactions, we recommend making use of advanced (noninvasive) monitoring of CO and SVV during administration of this vasopressor in clinical practice.

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS

Author contributions: A.F.K. conception and design of research; A.F.K. analyzed data; A.F.K. and T.W.S. interpreted results of data analysis; A.F.K. drafted manuscript; A.F.K. and T.W.S. edited and revised manuscript; A.F.K. and T.W.S. approved final version of manuscript.

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Address for reprint requests and other correspondence: A. F. Kalmar, Dept. of Anesthesiology, Univ. Medical Center Groningen, Univ. of Groningen, Hanzeplein 1, 9700 RB Groningen, The Netherlands (e-mail: a.kalmar@umcg.nl).