Variability of aortic blood flow predicts fluid responsiveness in spontaneously breathing healthy volunteers

Roman Skulec[1, 3], Ondrej Cermak[2], Hana Skalicka[3], Jiri Kolar[3]

[1] Emergency Medical Service of the Central Bohemian Region, Beroun, Czech Republic

[2] Internal Department, Hospital Kolin, Kolin, Czech Republic

[3] 2nd Department of Internal Cardiovascular Medicine, General Teaching Hospital, Prague, Czech Republic

Abstract

Background: Assessment of fluid responsiveness is an important topic in acute cardiology. Echocardiographic measurement of respiratory variations of aortic blood velocity in ventilated shock patients can accurately predict the effect of volume expansion. On the other hand, it remains unclear whether this respiratory variability is a common physiological reaction to hypovolaemia and whether its measurement is applicable also in spontaneously breathing patients.

Aim: To assess whether respiratory variability of peak aortic blood flow velocity ($\Delta Vpeak_{ao}$) and of aortic velocity time integral (ΔVTI_{ao}) reflects preload-dependent changes of cardiac index (CI) and whether it predicts fluid responsiveness in healthy spontaneously breathing volunteers.

Methods: $\Delta Vpeak_{ao}$, ΔVTI_{ao} and CI were measured by transthoracic echocardiography in 20 volunteers at baseline and after intravenous administration of furosemide (0.5 mg/kg). Afterwards, volunteers were randomised to rapid intravenous volume expansion (group A) or no expansion (group B) and assessed finally.

Results: Hypovolaemia induction was associated with a decrease of CI (from 3.25 ± 0.50 to 2.28 ± 0.43 l/min/m^2, $p < 0.001$) which correlated with an increase of $\Delta Vpeak_{ao}$ ($r = -0.490$, $p = 0.028$) and ΔVTI_{ao} ($r = -0.554$, $p = 0.011$) in both groups. In group A, volume expansion was followed by a drop of $\Delta Vpeak_{ao}$ (from 16.04 ± 1.99 to 2.97 ± 1.65 %, $p < 0.001$) and ΔVTI_{ao} (from 20.43 ± 5.13 to 3.43 ± 1.68 %, $p < 0.001$) and CI increase (from 2.14 ± 0.47 to 3.29 ± 0.57 l/min/m^2, $p < 0.001$). This increase strongly correlated with the value of $\Delta Vpeak_{ao}$ ($r = 0.782$, $p = 0.008$) and ΔVTI_{ao} ($r = 0.770$, $p = 0.009$) before volume expansion. Conversely, there was no change of measured parameters in group B. Threshold values of 14% for ΔVao_{peak} and 17% for ΔVTI_{ao} were identified to predict fluid responsiveness (increase of CI > 15%) with a sensitivity of 89% and specificity of 100%.

Conclusions: $\Delta Vpeak_{ao}$ and ΔVTI_{ao} reflect preload-dependent changes of CI in healthy spontaneously breathing volunteers and predict fluid responsiveness.

Key words: fluid responsiveness, spontaneous breathing

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Introduction

Proper diagnosis and management of acute circulatory failure remain a daily challenge in coronary care units. Preload assessment is a very important part of this process. However, the key question should not be 'What is the preload?' but rather 'What will be the reaction to fluid administration?' A desirable response to the therapy is considered to be a clinically relevant increase in cardiac index (CI), usually ≥ 15%. Static parameters, such as central venous pressure (CVP) and pulmonary artery occlusion pressure (PAOP), are poor predictors of fluid responsiveness (FR).

Conversely, there have been identified several dynamic parameters of FR. Most of them are based on the quantification of the extent of their variability during the respiratory cycle [1-3]. All dynamic parameters were tested in the settings of mechanical ventilation. However, only limited data indicating the possible usefulness of dynamic parameters of FR in awake, spontaneously breathing patients are available [4-6]. Therefore, we designed a randomised clinical study to evaluate whether respiratory variability of aortic valve blood flow reflects preload-dependent changes of CI and whether it predicts FR in spontaneously breathing healthy subjects.

Address for correspondence:

Roman Skulec MD, 2nd Department of Internal Cardiovascular Medicine, General Teaching Hospital, U nemocnice 2, Prague 2, 128 08, Czech Republic, tel.: +420 777 577 497, 420 311 622 419, fax: +420 311 623 926, 420 311 625 248, e-mail: skulec@email.cz

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Methods

Study protocol

The clinical study was approved by the local ethics committee, in compliance with the Helsinki Declaration. We recruited 20 healthy volunteers who gave written informed consent to participate in the study. All volunteers had a normal medical history and physical examination, normal electrocardiogram and normal echocardiographic findings.

All investigations were performed in a quiet room, under standard room temperature, humidity and atmospheric pressure. During the study, all subjects were continuously monitored by electrocardiography, blood pressure was taken by a non-invasive plethysmographic method every 15 min and diuresis was measured. Volunteers remained supine or sitting in the bed and were conscious and breathing spontaneously. No food or fluid intake was allowed. Before echocardiography, they were asked to stay supine for at least 20 min. Five minutes before and during echocardiography, breathing of all subjects was guided by an optical and acoustical metronome with the breath rate of 6/min. Subjects were asked to expire passively. After the baseline echocardiographic assessment, 0.5 mg/kg of furosemide in one intravenous bolus was administered to induce a diuretic response and decrease preload. The second echocardiography was performed 90 min after furosemide administration. With that, volunteers were randomised by the envelope method to rapid intravenous volume expansion with normal saline in a dose equal to the diuresis during the period from furosemide administration to the second measurement (group A) or to further thirsting (group B). Randomisation was blinded for the echocardiographer. Finally, the third echocardiographic assessment was performed 30 min after randomisation in both groups.

Echocardiography

Transthoracic echocardiography was performed in standard apical four chamber view (A4C), apical five chamber view (A5C) and parasternal long axis view (PLAX), using a Toshiba Corevision (Tokyo, Japan). Parameters measured in every assessment were maximal ($Vao_{peak}max$) and minimal ($Vao_{peak}min$) value of peak blood flow through the aortic valve during the respiratory cycle, maximal (VTIao*max*) and minimal (VTIao*min*) value of velocity time integral of blood flow through the aortic valve during the respiratory cycle, all measured by continuous Doppler in A5C, maximal (VTIlvot*max*) and minimal (VTIlvot*min*) value of velocity time integral of blood flow through the left ventricular outflow tract during the respiratory cycle measured by pulse Doppler in A5C, and diameter of the left ventricular outflow tract (Dlvot) measured by M-mode in PLAX. The value of each parameter was measured in three consecutive respiratory cycles and three measurements were averaged for further analysis and calculations. Respiratory variability of peak flow velocity through the aortic valve (ΔVao_{peak}, %) was determined as $\{(Vao_{peak}max - Vao_{peak}min)/[(Vao_{peak}max + Vao_{peak}min)/2] \times 100\}$. Analogically, respiratory variability of velocity time integral of flow velocity through the aortic valve (ΔVTIao, %) was determined as {(VTIao*max* – VTIao*min*)/ [(VTIao*max* + VTIao*min*)/2] × 100}. Stroke volume (SV) was measured in the left ventricular outflow tract and calculated by the formula [(VTIlvot*max* + VTIlvot*min*)/2] × [(π × Dlvot^2)/ /4]. CI was determined as (SV × HR)/body surface area [7].

Statistical analysis

Mean values ± standard deviation (SD) or percentages were calculated for all variables. Differences between the groups were compared by χ^2 test and statistical significance was calculated by Fisher exact test for alternative variables. Statistical significance for continuous variables was determined by paired Student's t-test. Spearman's correlation index was used to estimate correlations of continuous variables. Data were analysed using JMP 3.2 statistical software (SAS Institute, Cary, NC, USA). A p value of < 0.05 was considered statistically significant. Before the study, we calculated inter-observer and intra-observer variability to assess proper calculation of Vao, VTIao, VTIlvot and Dlvot from recorded echo tracks. The former was calculated as the ratio (expressed as a percentage) of the amplitude of the difference between ten randomly selected values measured by each observer (expressed as absolute value) divided by the mean of the values of both observers. The latter was calculated in a similar manner intra-individually with a period of three days between two assessments.

Results

Changes in conventional haemodynamic and echocardiographic parameters

No significant changes of arterial blood pressure or heart rate were observed. In the whole cohort of 20 volunteers, furosemide administration was followed by accentuated diuresis (1663 ± 343 ml/90 min) and significant decrease of CI (from 3.25 ± 0.50 to 2.28 ± 0.43 l/min/m^2, $p < 0.001$). After randomisation, fluid administration led to a significant increase of CI in group A (from 2.14 ± 0.48 to 3.29 ± 0.58 l/min/m^2, $p < 0.001$), when compared with the second measurement in volunteers afterwards randomised to group A. In group B, no change of CI was observed between the second and third measurement (from 2.42 ± 0.34 to 2.31 ± 0.32 l/min/m^2, $p = 0.466$).

Inter-observer variability was 3.9 ± 2.3% for Vao_{peak}, 4.1 ± 2.1% for VTIao, 4.2 ± 2.8 for VTIlvot and 4.4 ± 3.2 for Dlvot measurements. Intra-observer variability was 2.5 ± 1.9% for Vao_{peak}, 2.8 ± 2.1% for VTIao, 2.9 ± 2.0% for VTIlvot and 3.1 ± 2.5% for Dlvot measurements.

Influence of manipulation with preload on ΔVao_{peak} and ΔVTIao

Furosemide administration led to a significant increase of both ΔVao_{peak} (from 3.94 ± 3.40 to 15.71 ± 2.93%, $p < 0.001$) and ΔVTIao (from 4.38 ± 2.69 to 20.82 ± 6.69%, $p < 0.001$) in the whole cohort. While rapid volume expansion with 1666 ± 227 ml of normal saline in group A provoked a dramatic decrease of ΔVao_{peak} and ΔVTIao to the baseline values, in group B both parameters remained comparable in the second and the third measurement (Figures 1, 2). Moreover, the change of CI after furosemide administration in the whole cohort negatively correlated with the increase of ΔVao_{peak} ($r = –0.490$, $p = 0.028$) and ΔVTIao ($r = –0.554$, $p = 0.011$).

ΔVao_{peak} and ΔVTIao and predictivity of fluid responsiveness

In group A, values of ΔVao_{peak} and ΔVTIao before volume expansion closely correlated with the increase of CI after volume expansion (Figures 3, 4). Threshold value of 14% for ΔVao_{peak} and 17% for ΔVTIao was identified to predict an increase of CI > 15% with sensitivity of 89% and specificity of 100%. Conversely, there was no correlation between ΔVao_{peak} and ΔVTIao in the second measurement and change of CI during the period between the second and the third assessment in group B.

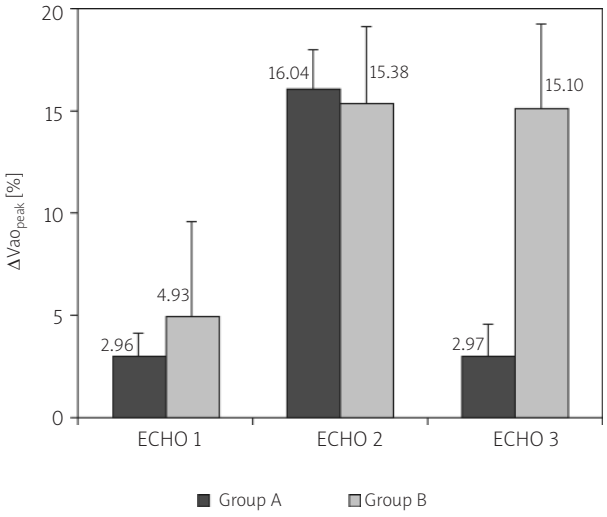

Figure 1. Values of ΔVao_{peak} in both groups

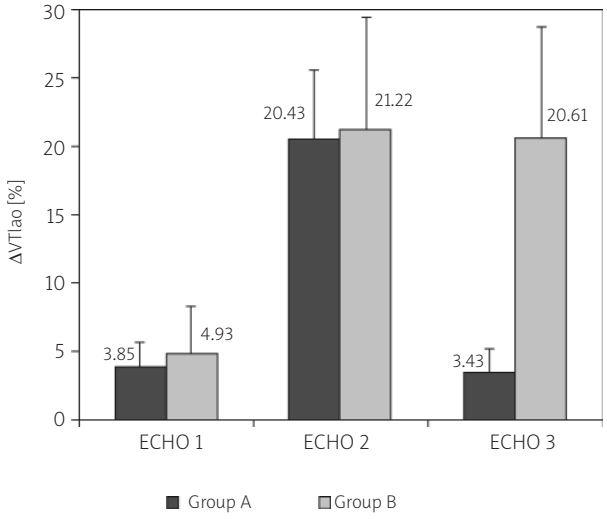

Figure 2. Values of ΔVTIao in both groups

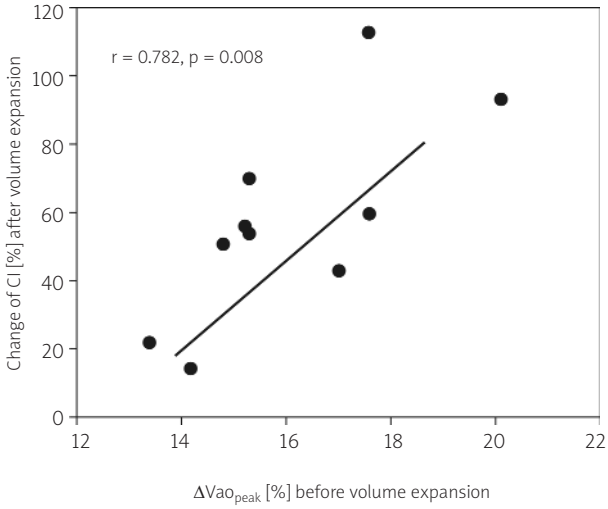

Figure 3. Correlation of ΔVao_{peak} values before volume expansion with change of CI induced by volume expansion in group A

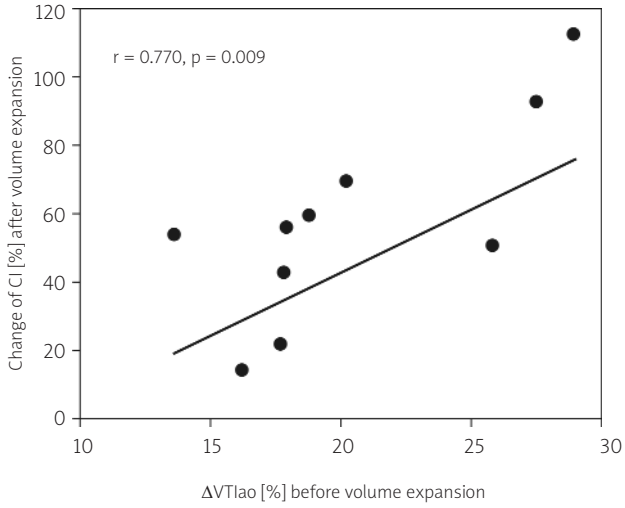

Figure 4. Correlation of ΔVTIao values before volume expansion with change of CI induced by volume expansion in group A

Discussion

The main finding of our study was that ΔVao_{peak} and ΔVTIao responded to provoked preload-dependent CI changes in awake spontaneously breathing healthy subjects. ΔVao_{peak} and ΔVTIao have been identified as very sensitive and specific predictors of FR in this group of subjects.

Volume expansion is the first-line therapeutic approach in critically ill patients with low CI. Unfortunately, approximately 50% of them do not respond to fluid administration. Moreover, in some of them circulatory failure can be worsened [1]. While traditional static preload measures (CVP, PAOP) fail in FR prediction, recently published experimental studies indicated several dynamic parameters as good predictors of FR (respiratory variability of arterial blood pressure, SV, blood flow velocity through aortic valve and vena caval diameter) [2, 8]. Respiratory variability of these parameters reflects on what portion of the Frank-Starling curve the heart ventricles work on. When the variability is high, the ventricles operate on the ascending part of the curve and patients will be responders to volume expansion. In contrast, low variability indicates ventricles operating on the flat portion of the curve and patients are supposed to be non-responders.

Nevertheless, there are some limitations. It is obvious that dynamic parameters are not applicable in patients with cardiac arrhythmias. All parameters have been evaluated in sedated and mechanically ventilated individuals. Spontaneous breathing efforts can affect measurements in several ways [9-11]. First, irregular uncontrolled spontaneous breathing makes dynamic parameters completely impracticable. Second, respiratory variations in intrapleural pressure are less intense than in mechanically ventilated subjects and active expiratory efforts can counterbalance the results. Third, a high respiratory rate can reduce the number of beats per respiratory cycle and affect the measurement. Fourth, CI can be affected by a variety of additional circumstances such as pain, noise, etc. However, many patients with acute circulatory failure breath spontaneously and prompt intervention can improve their prognosis. Accurate assessment of FR appears to be of extreme importance in this group of patients.

Only a limited number of studies evaluating FR in spontaneously breathing individuals have been presented so far. Madger et al. investigated predictive value of right atrial pressure (RAP) respiratory variations for FR in spontaneously breathing patients following bypass surgery. In more than 80% of them who had no fall of RAP while having a decrease in PAOC at least by 2 mmHg, CI did not increase after volume challenge. On the other hand, in the comparable proportion of patients exhibiting a fall of RAP during inspiration ≥ 1 mmHg, fluid administration increased CI significantly [4, 5]. Soubrier et al. demonstrated that respiratory variability of pulse pressure (ΔPP) of 12% predicted increase of SV in 32 spontaneously breathing patients [6]. Heenen et al. evaluated the role of ΔPP, inspiratory variations of RAP and of several static parameters in prediction of FR in 21 critically ill patients with spontaneous respiratory movements who required a fluid challenge. They did not find any predictive value for FR for any of the analysed parameters [9]. Moreover, Rooke et al. compared the effects of graded haemorrhage followed by volume substitution on respiratory variations of arterial systolic blood pressure (SPV) and expiratory decrease in arterial systolic blood pressure (Δdown) in the condition of mechanical ventilation or in awake, spontaneously breathing patients. While in ventilated patients haemorrhage significantly increased SPV and Δdown and hetastarch infusion decreased, in spontaneously breathing patients it did not [12].

The above-mentioned studies have brought discrepant results. Nevertheless, these results have not disqualified dynamic parameters from application in spontaneously breathing patients. First, it depends whether or not patients exhibit adequate inspiratory efforts. While the Magder et al. study included only subjects with sufficient efforts and without forced expiration, Heenen et al. included patients irrespective of this condition [13]. Second, in some studies the breath rate was uncontrolled and the length of breath cycle varied from breath to breath. Third, there were probably technical differences in measurement of analysed parameters. Fourth, volume expansion was performed with different solutions. Colloids, in contrast to normal saline, exhibit complex haemodynamic effects including improvement of contractility [5]. Finally, patients were not randomised in published studies.

From the panel of dynamic parameters we chose respiratory variations of blood flow through the aortic valve. These are common components of routine echocardiographic assessment and in awake cooperative patients are easily measurable with transthoracic echocardiography.

Respiratory changes of aortic blood velocity have been evaluated in experimental settings, but only in mechanically ventilated animals and individuals. Slama et al. analysed the predictive value of ΔVTIao for FR in 12 mechanically ventilated rabbits during blood withdrawal and reinfusion. During haemorrhage, ΔVTIao increased and after blood restitution returned to baseline values. Changes of ΔVTIao correlated with changes in CO. ΔVTIao in the last blood withdrawal step predicted FR [14, 15]. Feissel et al. measured ΔVao_{peak} in 19 sedated and mechanically ventilated patients with septic shock at the baseline and after volume expansion. The ΔVao_{peak} threshold value of 12% before volume expansion allowed discrimination between responders and non-responders with high sensitivity and specificity [15].

In our protocol, furosemide administration was followed by considerable increase of ΔVao_{peak} and ΔVTIao together with decrease of CI, which dropped significantly in 19 of 20 subjects. In one subject CI remained unchanged. This corresponded with the very mild increase of ΔVTIao and clinically irrelevant change of ΔVao_{peak} in this subject. After randomisation, we did not observe any significant change in CI, ΔVao_{peak} or ΔVTIao in group B. In contrast, the rapid volume expansion in group A induced a similar pattern of CI, ΔVao_{peak} and ΔVTIao reaction as was observed in studies in patients on mechanical ventilation. CI increased in all subjects and ΔVao_{peak} and ΔVTIao decreased to the baseline values. The close correlation between values of ΔVao_{peak} and ΔVTIao before volume expansion and change of CI after volume expansion indicates high sensitivity and specificity of ΔVao_{peak} and ΔVTIao in prediction of FR. These findings are consistent with those described by Slama and Feissel [14, 15].

In our study, breathing was completely spontaneous. Even though we did not measure tidal volumes and intrapleural pressure changes, we hope that through controlled, regular and slow breath rate associated with passive expiration we ensured sufficient and steady changes of intrapleural pressure with satisfactory length of breath cycle. All investigations were performed in quiet and stable conditions, in healthy individuals, without any unpleasant stimuli. Volume expansion was mediated by normal saline and patients were randomised to volume expansion or non-expansion.

We would like to stress that the results of our study do not automatically anticipate that ΔVao_{peak} and ΔVTIao are predictive for FR in all spontaneously breathing patients with acute circulatory failure. We only showed that in regularly and deeply spontaneously breathing, healthy volunteers, changes of ΔVao_{peak} and ΔVTIao significantly reflect changes of preload and preload-dependent variations of output, and both parameters predict FR in such a group of subjects. This indicates that the model could be potentially useful also in clinical practice. In any case, a very important condition is proper cooperation of patients to reach and maintain deep spontaneous breathing activity with controlled breath rate and passive expiration, which is very problematic in some critically ill patients. It is also questionable whether this concept works in patients with serious systolic and/or diastolic dysfunction. Anyway, we believe that the reported data could help to open the door for further studies in critically ill patients with acute circulatory failure without the necessity of ventilatory support.

In the last two years, there have been published a few studies assessing the validity of FR by careful analysis of haemodynamic response to passive leg raising. Passive leg raising is a standard method of increasing venous return to the right heart. Monnet et al. showed that continuous measurement of changes of descending aortic blood flow during passive leg raising predicts FR more reliably than change in pulse pressure in mechanically ventilated patients including those with spontaneous respiratory activity [16]. Similarly, Lamia et al. and Maizel et al. documented the positive predictive value for FR of stroke volume changes during passive leg raising in spontaneously breathing patients [17, 18]. Therefore, this approach can be another alternative to assessment of FR in such a group of patients [19].

We conclude that $\Delta Vpeak_{ao}$ and ΔVTI_{ao} reflect preload-dependent CI changes in healthy spontaneously breathing volunteers. Both parameters predict FR in this group of subjects. We consider tight control of breath rate as the key condition for acquiring valid data. Thus, we can speculate that simple single baseline measurement of ΔVao_{peak} and/or ΔVTIao without any other intervention such as fluid challenge or passive leg raising could predict FR also in spontaneously breathing, critically ill patients as well as in mechanically ventilated ones. Nevertheless, whether this is true awaits further studies.

References

1. Michard F, Teboul JL. Using heart-lung interactions to assess fluid responsiveness during mechanical ventilation. *Crit Care* 2000; 4: 282-9.
2. Pinsky MR. Assessment of indices of preload and volume responsiveness. *Curr Opin Crit Care* 2005; 11: 235-9.
3. Gunn SR, Pinsky MR. Implications of arterial pressure variation in patients in the intensive care unit. *Curr Opin Crit Care* 2001; 7: 212-7.
4. Magder S, Georgiadis G, Cheong T. Respiratory variations in right atrial pressure predict the response to fluid challenge. *J Crit Care* 1992; 7: 76-85.
5. Magder S, Lagonidis D. Effectiveness of albumin versus normal saline as a test of volume responsiveness in post-surgery patients. *J Crit Care* 1999; 14: 164-71.
6. Soubrier S, Saulnier F, Hubert H, et al. Respiratory changes in arterial pulse pressure and fluid responsiveness in spontaneously breathing patients. *Critical Care* 2005; 9 (Suppl. 1): 55.
7. Linhart A, Palecek T, Aschermann M. Measuring of output. In: Linhart A, Palecek T, Aschermann M (Eds.) Echocardiography for practice. Prague, Czech Republic: The Hague Audioscan, 2002; 96.
8. Feissel M, Michard F, Faller JP, et al. The respiratory variation in inferior vena cava diameter as a guide to fluid therapy. *Intensive Care Med* 2004; 30: 1834-7.
9. Heenen S, De Backer D, Vincent JL. How can the response to volume expansion in patients with spontaneous respiratory movements be predicted? *Critical Care* 2006; 10: 102-7.
10. De Backer D, Heenen S, Piagnerelli M, et al. Pulse pressure variations to predict fluid responsiveness: influence of tidal volume. *Intensive Care Med* 2005; 31: 517-23.

11. Weissman C, Kemper M, Damask MC, et al. Effect of routine intensive care interactions on metabolit rate. *Chest* 1984; 86: 815-8.
12. Rooke GA, Schwid HA, Shapira Y. The effect of graded hemorrhage and intravascular volume replacement on systolic pressure variation in humans during mechanical and spontaneous ventilation. *Anesth Analg* 1995; 80: 925-32.
13. Magder S. Predicting volume responsiveness in spontaneously breathing patients: still a challenging problem. *Critical Care* 2006; 10: 165-6.
14. Slama M, Masson H, Teboul JL, et al. Respiratory variations of aortic VTI: a new index of hypovolemia and fluid responsiveness. *Am J Physiol Heart Circ Physiol* 2002; 283: H1729-33.
15. Feissel M, Michard F, Mangin I, et al. Respiratory changes in aortic blood velocity as an indicator of fluid responsiveness in ventilated patients with septic shock. *Chest* 2001; 119: 867-73.
16. Monnet X, Rienzo M, Osman D, et al. Passive leg raising predicts fluid responsiveness in the critically ill. *Crit Care Med* 2006; 34: 1402-7.
17. Lamia B, Ochagavia A, Monnet X, et al. Echocardiographic prediction of volume responsiveness in critically ill patients with spontaneously breathing activity. *Intensive Care Med* 2007; 33: 1125-32.
18. Maizel J, Airapetian N, Lorne E, et al. Diagnosis of central hypovolemia by using passive leg raising. *Intensive Care Med* 2007; 33: 1133-8.
19. Teboul JL, Monnet X. Prediction of volume responsiveness in critically ill patients with spontaneous breathing activity. *Curr Opin Crit Care* 2008; 14: 334-9.

Ocena zmienności przepływu krwi w aorcie jako metoda przewidywania odpowiedzi na obciążenie płynami u spontanicznie oddychających zdrowych osób

Roman Skulec[1, 3], Ondrej Cermak[2], Hana Skalicka[3], Jiri Kolar[3]

[1] Pogotowie Ratunkowe Centralnej Bohemii, Beroun, Czechy
[2] Klinika Chorób Wewnętrznych, Szpital Miejski, Kolin, Czechy
[3] II Klinika Chorób Wewnętrznych i Naczyń, Szpital Główny, Praga, Czechy

Streszczenie

Wstęp: Ocena odpowiedzi na obciążenie płynami jest istotnym zagadnieniem. Wykazano, że na podstawie echokardiograficznych pomiarów oddechowej zmienności prędkości przepływu krwi w aorcie u chorych we wstrząsie podczas mechanicznej wentylacji można dość dokładnie przewidzieć skutki podania płynów. Nie wiadomo jednak, czy ta oddechowa zmienność jest typową fizjologiczną reakcją na hipowolemię i czy pomiar prędkości przepływu krwi w aorcie jest również użyteczny u chorych oddychających samodzielnie.

Cel: Ustalenie, czy oddechowa zmienność szczytowej prędkości przepływu krwi w aorcie (ΔV_{peak}ao) i całki tej wartości (ΔVTIao) odzwierciedla zależne od obciążenia wstępnego zmiany w rzucie serca (CI) i czy przewiduje odpowiedź na obciążenie płynami u spontanicznie oddychających zdrowych osób.

Metody: Wartości ΔV_{peak}ao, ΔVTIao i CI mierzono za pomocą echokardiografii przezklatkowej u 20 zdrowych ochotników przed i po dożylnym podaniu furosemidu w dawce 5 mg/kg. Następnie uczestnicy badania w sposób losowy zostali przydzieleni do jednej z dwóch grup: szybkiego dożylnego podania płynów (grupa A) lub bez obciążenia płynami (grupa B).

Wyniki: Wywołana furosemidem hipowolemia spowodowała spadek CI (z 3,25 ± 0,50 do 2,28 ± 0,43 l/min/m^2, p < 0,001), co korelowało ze wzrostem ΔV_{peak}ao (r = –0,490, p = 0,028) i ΔVTIao (r = –0,554, p = 0,011) w obu badanych grupach. W grupie A obciążenie płynami wiązało się z obniżeniem ΔV_{peak}ao (z 16,04 ± 1,99 do 2,97 ± 1,65%, p < 0,001) i ΔVTIao (z 20,43 ± 5,13 do 3,43 ± 1,68%, p < 0,001) oraz wzrostem CI (z 2,14 ± 0,47 do 3,29 ± 0,57 l/min/m^2, p < 0,001). Ten wzrost ściśle korelował z wartościami ΔV_{peak}ao (r = 0,782, p = 0,008) oraz ΔVTIao (r = 0,770, p = 0,009) ocenianymi przed obciążeniem płynami. W grupie B nie odnotowano zmian w wartościach tych parametrów. Wartości odcięcia 14% dla ΔV_{peak}ao i 17% dla ΔVTIao wyłaniały osoby z właściwą odpowiedzią na obciążenie płynami (wzrost CI > 15%) z czułością 89% i swoistością 100%.

Wnioski: Wartości ΔV_{peak}ao i ΔVTIao odzwierciedlają zmiany w CI zależne od obciążenia wstępnego u spontanicznie oddychających zdrowych osób i przewidują odpowiedź na obciążenie płynami.

Słowa kluczowe: obciążenie płynami, obciążenie wstępne, przepływ krwi w aorcie

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Adres do korespondencji:
dr n. med. Roman Skulec, 2nd Department of Internal Cardiovascular Medicine, General Teaching Hospital, U nemocnice 2, Prague 2, 128 08, Czech Republic, tel.: +420 777 577 497, +420 311 622 419, faks: +420 311 623 926, +420 311 625 248, e-mail: skulec@email.cz
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