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Comparison of automated vs. manual determination of the respiratory variations in the EKG R wave amplitude for the prediction of fluid responsiveness during surgery

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Abstract

Electrocardiogram (EKG) monitoring is a common standard of care across all operating rooms and intensive care units. Studies have suggested that respiratory variations in the EKG R wave amplitude (EKGv) can be used as an indicator of fluid responsiveness in mechanically ventilated patients under general anesthesia, but to date all calculations of variation have been done by hand. The aim of this study was to assess if a computer-automated algorithm could compute and monitor EKGv with the same precision as manual measurement. Batches of 30 s each of EKG lead II waveforms were recorded during surgical procedures with mechanical ventilation. R wave amplitude variability was assessed both manually and by automated algorithm. For both calculations, wave height was defined as R wave peak minus preceding Q wave trough, and the minimum and maximum amplitudes determined for each respiratory cycle. EKGv was calculated as $100 \times [(\mathrm{RDIImax} - \mathrm{RDIImin})/(\mathrm{RDIImax} + \mathrm{RDIImin})/2]$. Fifty-seven batches of waveforms were calculated. We found that our computer-automated algorithm calculation of EKGv was significantly correlated to manual measurements ($r = 0.968$, $P < 0.001$). Bland-Altman analysis also showed a strong agreement between automated and manual EKGv measurements (bias $0.13\% \pm 3.06\%$). The observed correlations between the manually and automatically calculated EKGv suggest that our current computer-automated algorithm is a reliable method for calculating EKGv. Validation in prospective volume expansion studies will be needed to assess the true clinical utility of this automated measurement.

Keywords: EKG; Brody effect; Monitoring; Noninvasive; Fluid responsiveness; Automated; Algorithm

Background

The first line of therapy for hypotensive patients in critical care settings is usually intravenous fluid infusion. Fluid responsiveness studies, however, suggest that more than 50% of the time fluid therapy does not result in the expected volume expansion response: an increase in stroke volume [1]. Such studies underline the need for predictors of fluid responsiveness in order to identify patients who can benefit from volume expansion and avoid the negative effects of ineffective or over-expansion.

Recently, more studies have been done on predictors of fluid responsiveness in response to a fluid challenge, or predictors of changes in stroke volume and cardiac output. Existing static predictors of fluid responsiveness such as central venous pressure (CVP), pulmonary capillary wedge pressure (PCWP), and left ventricular end diastolic area have been shown to be nonpredictive of fluid responsiveness [2,3]; while respiratory induced variation in circulatory-based variables such as pulse pressure (PP), stroke volume (SV), and plethysmographic waveform (PV) have been shown to be predictive [4]. Pulse pressure variation (PPV) and stroke volume variation (SVV) are already widely accepted and used in clinical settings for fluid optimization [5]. However, both predictors rely on invasive blood pressure measurements and are therefore not available for many low-to-moderate-risk surgical patients. While noninvasive dynamic predictors of fluid responsiveness such as plethysmographic waveform variation index (PVI) exist, PVI is a peripheral measure and is subject to changes in vasomotor tone and perfusion. There is an opportunity to improve currently available predictors of fluid responsiveness by providing a noninvasive dynamic predictor that is cheap, easy to use, effective, and a part of the standard care in all surgical patients.

Respiratory variation in the EKG lead II R wave amplitude (EKGv) has been shown to correlate with PPV and SVV in mechanically ventilated patients under general anesthesia and to reflect intravascular volume status [6-8]. EKGv relies on the 'Brody effect' theory [8], the idea that the left ventricular chamber size directly influences the R wave amplitude (i.e., an increase in ventricular preload induces an increase in R wave amplitude). During positive pressure ventilation, a preload-independent heart will not experience large variations in cardiac volume and thus the resistance across the heart tissue and, consequently, the QRS wave amplitude, will not have large variations [6]. For a preload-dependent patient, the large changes in cardiac volume induced by respiration will result in a corresponding large change in the measured QRS wave amplitude for the same reasoning. Since changes in R wave amplitude from lead II are induced by significant rapid LV preload changes and have been shown to correlate well with PPV and SVV [9,10], the EKGv for this study was calculated from lead II R wave amplitude (RDII) variations.

Currently, the only method for EKGv calculation is through manual measurement. In response to the need for an accurate and dynamic calculation of EKGv in the clinical environment, the aim of this study was to develop a computer-automated algorithm and assess its accuracy compared to manual measurements in correctly identifying R wave amplitude variability.

Methods

This study was approved by the University of California, Irvine research ethics committee with waived written informed consent. All patients in the authors' university hospital are informed that de-identified data may be collected for research purposes. All monitoring used in this study is considered part of standard care for patients.

Patient selection

The inclusion criteria for study patients were as follows: adult patients greater than 18 years of age, undergoing elective open abdominal surgery, classified as ASA 2 or 3, equipped with a standard 5-lead EKG, and undergoing mechanical ventilation with

constant respiratory frequency and in a volume control mode with at least 8 mL/kg tidal volumes. The major exclusion criteria were as follows: patients with cardiac arrhythmia, right ventricular failure, left ventricular ejection fraction <40%, PEEP >5 cmH_2O, and with a spontaneous breathing cycle detected on ETCO2 tracing. No modifications in anesthesia care were required for the study. A standard 5-lead electrocardiogram (EKG) was continuously displayed on a GE Healthcare monitor (GE Healthcare, Milwaukee, WI, USA) by standard monitoring electrodes (Novaplus, Irving, TX, USA). The standard 5-lead EKG placement used included the four limb electrodes (RA, LA, RL, LL) and the precordial lead V5. Extra care was taken to ensure that the standard 5-lead EKG placement was followed.

Data acquisition and recording

EKG waveforms were collected on a laptop computer using an Ethernet port of the Solar 8000i anesthesia workstation and multiparameter bedside monitor (GE Healthcare, Milwaukee, WI, USA). Utilizing the data collection software (Visual Studio 2005, Microsoft, Redmond, WA, USA) developed by our research team for communication with the GE workstation, EKG waveforms were simultaneously digitalized and continuously recorded on the laptop computer with a sample rate of 240 Hz for a total of 10,000 samples (42 s) per data batch.

Data analysis

From the recorded EKG waveforms, EKGv was calculated offline manually and by computer-automated algorithm. EKGv was defined as the relative difference in maximal (RDIImax) and minimal RDII (RDIImin) amplitude according to the following formula [6]:

$$\text{EKGv}\ (\%) = 100 \quad \times \quad \frac{\text{RDIImax}-\text{RDIImin}}{((\text{RDIImax} + \text{RDIImin})/2)}$$

For manual determination, the manual EKGv calculator assumed *implied* respiratory cycles based on observed variations in the RDII amplitude. RDII amplitude was defined as the difference between a distinct RDII peak and respective trough. The manual EKGv calculator looked at the changing slopes of the RDII amplitudes and assessed where the maxima (RDIImax) and minima (RDIImin) of the slopes were to the best of his ability. He then chose three distinct RDIImax amplitudes that best represented the data batch and their respective adjacent RDIImin to calculate a total of three EKGv values per data batch (Figures 1 and 2). The median value was used as the representative of the manually determined EKGv ($EKGv_{man}$).

For automated determination, all RDII peaks and troughs were automatically detected and used in the calculation of EKGv ($EKGv_{auto}$) by an algorithm written in Matlab (Matlab R2011a, MathWorks Inc., Natick, MA, USA) (Figure 3). The steps of the algorithm are detailed below:

Step 1. *Set upper and lower limits for R peak detection*: An automatic R peak detection algorithm is used to detect every R peak in a given data batch of 10,000 samples. Peak detection begins by creating upper and lower limits for potential R peaks. The algorithm first performs the local maxima detection using

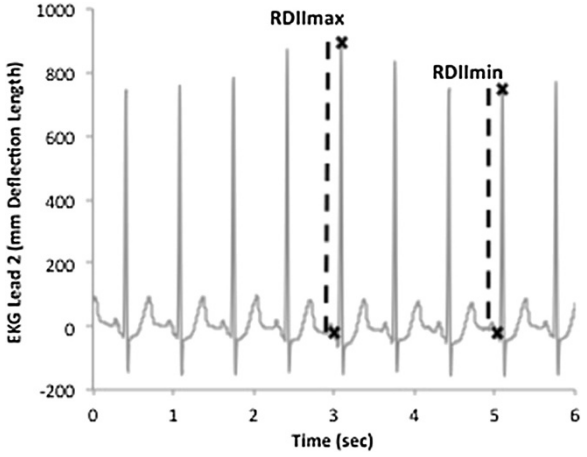

Figure 1 EKG lead II waveform recording over one respiratory cycle. RDIImin and RDIImax were defined as minimum and maximum R wave amplitudes of one implied respiratory cycle. These variables were then used to calculate a single EKGv.

MatLab's max function on every 100 data points for a total of 2,000 data points or 20 local maxima. The mean ± standard deviation of this list of local maxima is then calculated, and any local maxima in this list not within those limits are considered noise and removed from further analysis. The R peak upper and lower limits are then calculated as the mean ± standard deviation of the final list of the first local maxima.

Step 2. *Peak detection*: The algorithm searches for the local maxima, or R peak, in every 100 data points using the calculated R peak upper and lower limits and identifies the sample location corresponding to the R peak. Then the algorithm looks to the closest neighbors of the detected R peaks to ensure the correct local maxima was detected and corrects the R peak and location or eliminates the detected R peak if necessary. The R peak detection limits, as well as this additional step, help to eliminate the identification of incorrect R peaks due to noise. If greater than half the number of detected R peaks were eliminated

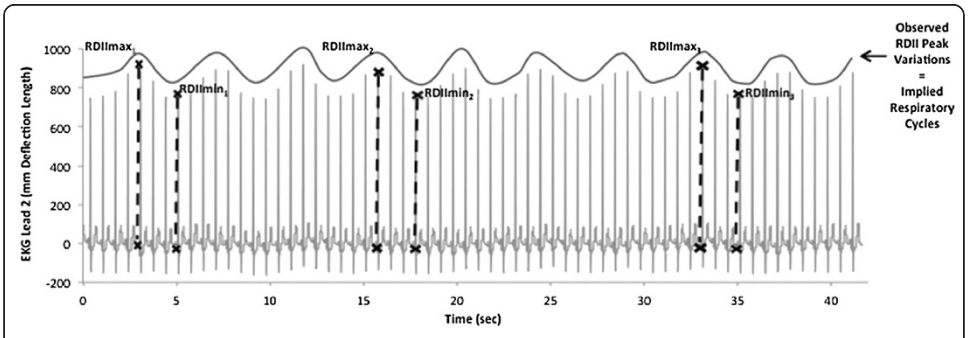

Figure 2 A full 10,000-sample (42-s) EKG lead II waveform recording in an illustrative patient. RDIImin and RDIImax were identified for three implied respiratory cycles across a 10,000-sample (42-s) data batch to calculate a total of three EKGvs ($EKGv_1$, $EKGv_2$, $EKGv_3$). The manual measurements of respiratory variations in EKG lead II R wave amplitude were calculated as (RDIImax – RDIImin)/[(RDIImax + RDIImin)/2]. The representative $EKGv_{man}$ was calculated as the median of $EKGv_1$, $EKGv_2$, …, $EKGv_n$.

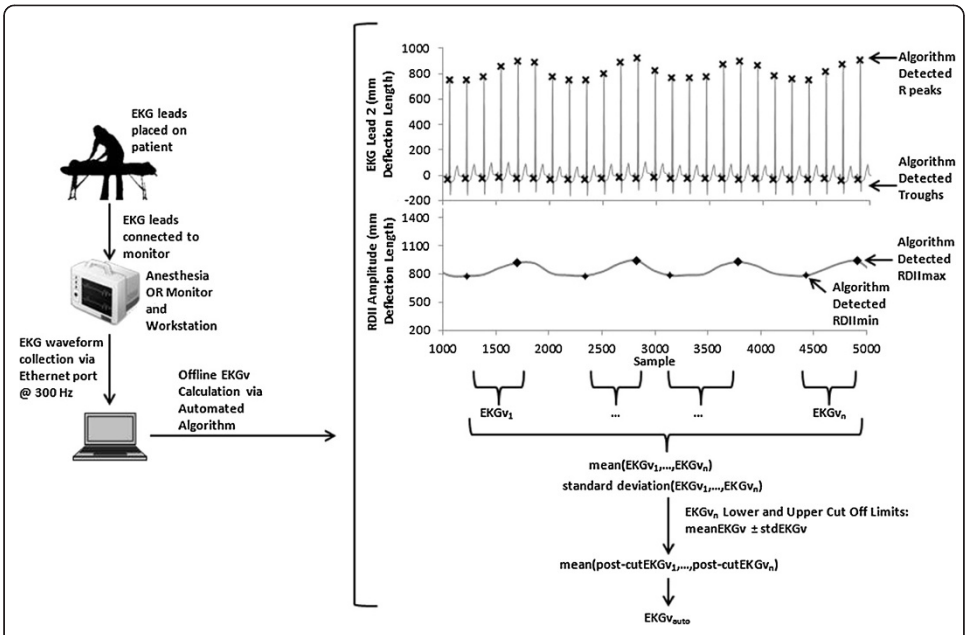

Figure 3 Schematic diagram of automatic calculation of EKGv. Algorithm steps in the automatic calculation of EKGv from the continuous recording of EKG lead II waveforms from a standard OR monitor.

during this step, the data batch was considered unanalyzable by the algorithm. Heart rate is estimated as the total number of R peaks divided by the length of sample time (42 s).

Step 3. *Trough detection*: Recalling the list of identified R peaks, another algorithm searches for the trough preceding the peak. The algorithm takes relative slope information backwards from the R peak; from right to left of the signal, this would consist of first a negative slope (R to Q), followed by a positive slope (Q to trough), then a zero or negative slope (trough). If a trough is not detected, then a nearby data point from the identified Q point is used as an estimate of the trough.

Step 4. *RDII amplitude calculation*: The RDII amplitude is calculated from the difference between R peaks and corresponding troughs. Upper and lower limits of RDII amplitudes are the mean ± standard deviation of all calculated RDII amplitudes. If the standard deviation of the RDII amplitudes is very large, the upper and lower limits are used to eliminate any values outside of the limits.

Step 5. *EKG variability estimation*: Relative amplitudes and slope information are used to identify the minima and maxima (RDIImin and RDIImax) of the RDII amplitudes to estimate the respiratory cycles and calculate EKGvs. The EKGvs for each respiratory cycle are then calculated from the previously stated formula using corresponding RDIImin and RDIImax. All calculated EKGvs were then filtered through an upper and lower cut-off limit of the mean ± standard deviation and averaged for a final reported EKGv. The averaged value was used as the representative computer-automated algorithm calculation of EKGv ($EKGv_{auto}$).

Statistical analysis

The normality of distribution of $EKGv_{man}$ and $EKGv_{auto}$ values was tested using the Kolmogorov-Smirnov test. In the case of a normal distribution, the parametric Pearson test was used to assess correlation. $EKGv_{man}$ and $EKGv_{auto}$ were also compared using Bland-Altman analysis [11]. A receiver operating characteristic (ROC) curve analysis was then performed for $EKGv_{auto}$ varying the discriminating threshold of this parameter to determine the ability of $EKGv_{auto}$ to discriminate between patients with an $EKGv_{man} > 15\%$ and $EKGv_{man} \leq 15\%$. This threshold was chosen based on a previous study by Lorne et al. which determined that the inconclusive limits of changes in stroke volume (ΔSV) ranged from 13% to 15%, and that $EKGv > 15\%$ was able to accurately predict $\Delta VTI > 15\%$ [10]. In all cases, a P value less than 0.05 was considered statistically significant. All statistical analyses were performed using SPSS (SPSS 13.0, Chicago, IL, USA). Data are represented as mean ± standard deviation.

Results

Fifty-seven distinct data batches were collected in this study from 15 patients. Eleven data batches were subsequently excluded: Nine batches were excluded due to high levels of noise that did not allow for reasonable analysis either by hand or by the algorithm, and two batches were excluded due to incomplete data. Noise was assessed visually and batches were determined unanalyzable if no distinct R peaks or troughs could be seen, as occurs during interference from electrical cautery devices. Incomplete data was defined as data batches with interrupted data collection and consisted of less than the complete 10,000 sample total.

Data description

Across all data batches, heart rate was 84 ± 17 beats per minute with a range of 40 to 128 beats per minute. Respiratory rate was 17 ± 4 breaths per minute with a range of 8 to 25 breaths per minute. Heart rate to respiratory rate ratio was 5 ± 1 beats per breath with a range of 2 to 11 beats per breath. Average $EKGv_{man}$ was 11.2 ± 5.4% with a range of 2.7% to 20.3%. Average $EKGv_{auto}$ was 11.0% ± 6.0% with a range of 1.4% to 21.4%.

Data analysis

Both $EKGv_{man}$ and $EKGv_{auto}$ were found to be normally distributed with $P = 0.478$ and $P = 0.468$, respectively. Consequently, the relationship between the two variables was tested using the Pearson correlation test. There was a statistically significant correlation between $EKGv_{man}$ and $EKGv_{auto}$ ($r = 0.968$, $P < 0.001$) (Figure 4). Bland-Altman analysis showed a strong agreement between $EKGv_{man}$ and $EKGv_{auto}$ (Figures 4 and 5). The bias for the automated algorithm was 0.13% with 95% confidence intervals of ± 3.06%. The receiver operating characteristic curve assessing the ability of $EKGv_{auto}$ to predict $EKGv_{man} > 15\%$ is shown in Figure 6. The area under the curve was 0.98 ± 0.01. An $EKGv_{auto}$ cut-off value of 15% accurately predicted $EKGv_{man} > 15\%$, with a sensitivity of 92% and a specificity of 94%.

Discussion

This study demonstrates that our custom algorithm for automated calculation of respiratory variations in the EKG waveform (EKGv) has a good agreement with manually

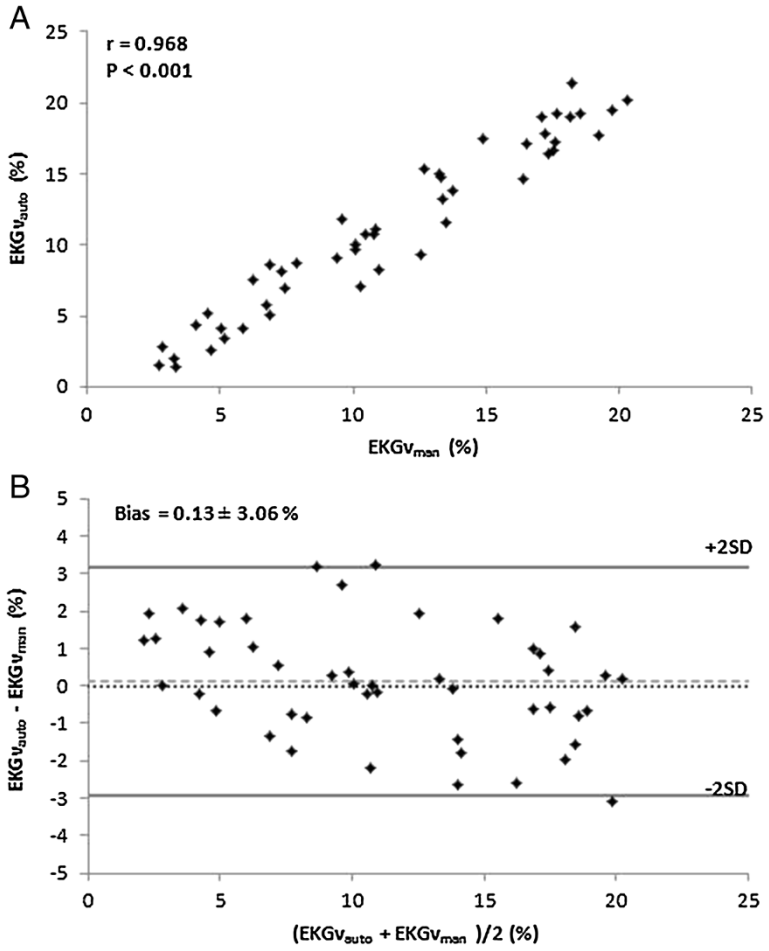

Figure 4 Statistical analyses. (A) Relationship between EKGv calculated manually and EKGv calculated by the automated algorithm. **(B)** Bland-Altman analysis showing the agreement between $EKGv_{man}$ and $EKGv_{auto}$.

calculated EKGv and has potential clinical applications for detection of fluid responsiveness in patients undergoing surgery with mechanical ventilation and general anesthesia.

Predictors of fluid responsiveness are important in anesthesia for the accurate identification of patients who can benefit from volume expansion, or cardiac output optimization, and avoid negative outcomes associated with hypovolemia or hypervolemia [12-15]. Studies have shown that cardiac output optimization cannot only decrease the cost of surgery but can also improve postoperative outcomes such as length of stay in the intensive care unit, length of stay in the hospital, and incidence of postoperative nausea and vomiting [4]. Presently, dynamic parameters of fluid responsiveness, such as PPV, SVV, and PVI, have been developed and can predict the need for fluid administration to improve cardiac output. However, PPV and SVV require invasive blood pressure measurements that are not necessary for all surgical patients, and PVI, while noninvasive, is limited by changes in vasomotor tone and perfusion [16]. EKGv presents a way to have an accurate predictor of fluid responsiveness, while being noninvasive, widely available, and independent of vasomotor tone. Our study here suggests this

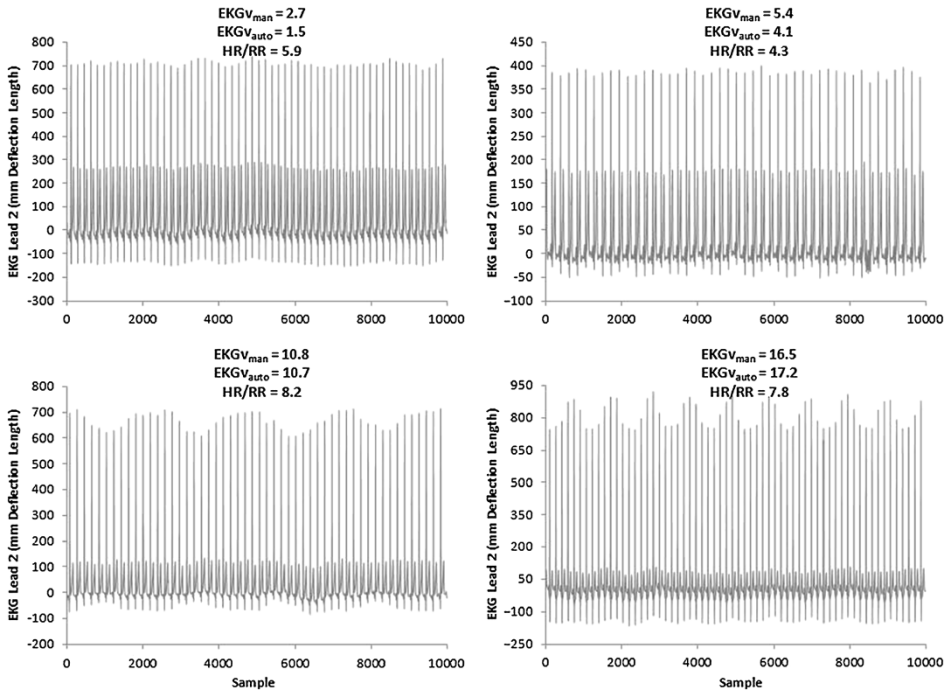

Figure 5 Illustrative examples of corresponding EKGv$_{man}$ and EKGv$_{auto}$ calculation results. EKGv$_{man}$ and EKGv$_{auto}$ calculation results for low-to-high respiratory variations in the EKG lead II R wave amplitude.

new index can be measured at the bedside in the clinical environment in patients undergoing surgery with mechanical ventilation and general anesthesia.

In 1956, Daniel A. Brody described the Brody effect [8]. The Brody effect suggests a direct relationship between intraventricular blood volume and R wave amplitude [8]. This is due to the electric inhomogeneity remote from the heart and its effect on the

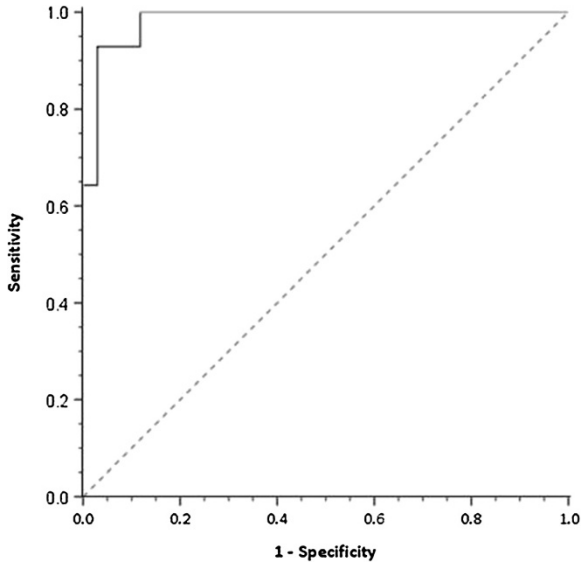

Figure 6 ROC curve analysis. Ability of automated algorithm calculation (EKGv$_{auto}$) of variation in R wave amplitude in lead II to predict a manual calculation (EKGv$_{man}$) of variation > 15% (receiver operating characteristics curve).

transmission of myocardium depolarization to the body surface. Intracardiac cavity blood mass has a resistivity of about 160 Ω cm, and the cardiac muscle resistivity averages about 250–550 Ω cm [17]. The heart is surrounded by the lungs which have a resistivity of about 2,000 Ω cm [17]. Thus, the conductivity increases tenfold from the lungs to the intracardiac blood mass, and the applied EKG lead field path includes the conductive intracardiac blood mass. As a consequence, the applied EKG lead field has an increased sensitivity to radial dipoles (when the progress of myocardial excitation is radial to the blood mass during the initial phases of ventricular depolarization) and decreased sensitivity to tangential dipoles (when the progress of myocardial excitation is tangential to the blood mass during the later phases of depolarization) in the cardiac muscle area. The effect has been further verified by other studies [18]. Then the volume of intracardiac blood may also affect the R wave amplitude, i.e., increased/decreased volume leads to an increase/decrease in the R wave amplitude. Thus, variations in ventricular end diastolic volume can affect variations in the R wave amplitude, which is generated at the end diastole when the intraventricular volume of blood is highest.

Several algorithms have been proposed for other dynamic predictors of fluid responsiveness, such as PPV, SVV, and PVI [19-23]. The presently proposed algorithm for EKGv was initially created to test if this automated algorithm measurement was feasible and as accurate as hand measurement. Using ideas similar to those used in the automated estimation of PPV from an arterial blood pressure signal [23], this proposed algorithm for EKGv automatically detects peaks and troughs of the lead II EKG waveform and then detects R wave amplitude minima and maxima to calculate EKGv. The automated algorithm EKGvs reported in this study are an average over a 42 s interval, and no physiological background such as the number of respiratory cycles or cardiac arrhythmia is considered in the automated EKGv calculation. We decided on this to keep our algorithm computationally nonintensive. Also, there are existing clinical devices that sample at a defined time interval without identifying respiratory cycles. For example, the PiCCO™ device (Pulsion Medical Systems, Eindhoven, The Netherlands) calculates SVV from the arterial pressure waveform using a 20-s moving window [24,25]. Similarly, the LiDCO pulseCO™ device (LiDCO Ltd, London, UK) measures PPV and calculates SVV from the arterial pressure also using a 20-s moving window [25]. In our algorithm, the continuous changes of the R wave amplitude for this algorithm were assumed to imply respiratory cycles. For example, Figure 3 shows four distinct R wave amplitude slopes over 4,000 samples, which under this assumption would correspond to an estimate of 4 breaths/17 s or 14 breaths/minute (at a sampling rate of 240 Hz, 4,000 samples corresponds to approximately 17 s). While this may be an overestimation of the respiratory rate of this patient, the representative EKGv used is an average of EKGvs filtered through mean ± standard deviation limits. The resulting EKGv then for a 42-s window with eight calculated EKGvs would most likely be the average of six to seven post-filtering EKGvs. This would correspond to a more reasonable estimate of 6 to 7 breaths/42 seconds or 9 to 10 breaths/minute. The Aboy et al. algorithm for PPV estimation utilizes a calculation over time intervals that is less dependent on respiratory phase shifts and eliminates the need of simultaneous airway pressure recording by utilizing a filtering method for respiratory frequency in the arterial pressure waveform [23]. It has also been shown, however, that PPV and

SVV analysis by time intervals may be consistent for respiratory rates >15 breaths/min, but unstable at respiratory rates <10 breaths/min [25]. This suggests that future work on the algorithm may need to be done to incorporate ways of discerning respiratory cycles using frequency filtering or to validate the use of R wave amplitude variations over time intervals to currently accepted dynamic parameters such as PPV and SVV.

There are some limitations to the current comparison between our algorithm and the hand-calculation method. The algorithm was not tested on any patients with left or right bundle branch block due to the inability of our current algorithm to analyze such patients for EKGv. Only patients with normal sinus rhythm, stable hemodynamics, and mechanical ventilation in volume-controlled mode were included in this study. While the current R wave peak and trough detection appears to be robust against noise like electrocautery and baseline drift, further work needs to be performed on the algorithm to include handling of abnormal heart rhythms, conduction abnormalities, and error catching. Moreover, it is likely that conduction abnormalities will disrupt the electromechanical relationships which give rise to EKGv such that EKGv may not even be predictive in this population.

Other limitations of this study include the lack of lung property measurements, the lack of confirmed respiratory cycles via airway pressure recordings for a stronger manual measurement of EKGv, and a lack of PPV data for comparison. We did not measure lung volume or lung mechanical properties, and it is possible that these could affect EKGv and its ability to assess fluid responsiveness. However, this effect has yet to be studied. Thus, further studies will need to be done to answer this question. On the other hand, EKGv is defined as respiratory variations in the EKG waveform per respiratory cycle to respiratory cycle. One would assume that lung volume and lung mechanics should not change during the period of one respiratory cycle, so the variation in these lung properties may not affect EKGv. PPV data was not collected in this study because fluid responsiveness was not the primary goal, but at some point a comparison to an established dynamic predictor as well as testing for actual prediction of fluid responsiveness will be needed. Also, only discrete EKGvs over 30-s intervals were compared for analysis and not continuous EKGvs over longer periods of time. Future studies will include accuracy of a real-time, continuous EKGv automated calculation and comparison of the algorithm to the currently accepted dynamic predictors of fluid responsiveness.

Conclusion

Based on the current data and potential for the automated continuous calculation of EKGv as a noninvasive predictor of fluid responsiveness, our goal is to continue to refine the current algorithm to calculate respiratory variations in the RDII waveform amplitude, with the long-term goal of using the algorithm in the clinical environment as an automated tool to predict fluid responsiveness during surgery. Initial analysis indicates a significant relationship and a strong agreement between manual and the automated algorithm calculation of EKGv.

Competing interests

Joseph Rinehart and Maxime Cannesson are co-inventors and co-owners of US patent 13/95,827: 'Method and apparatus for assessment of fluid responsiveness using the EKG waveform.' Other authors have no competing interests.

Authors' contributions

CL developed the algorithm, participated in waveform data collection, participated in the design of the study, performed the statistical analysis, and drafted the manuscript. JR conceived of the study, participated in algorithm development, participated in waveform data collection, participated in the design and coordination of the study, and helped to draft the manuscript. CC participated in the design and coordination of the study and helped to draft the manuscript. MC conceived of the study, participated in its design and coordination, and helped to draft the manuscript. All authors read and approved the final manuscript.

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