

Meeting abstract

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## I 138 Measurement of beat-to-beat variability of stroke volume

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### Introduction

Stroke volume variability (SVV) provides information about the activity of the autonomic nervous system, connecting heart rate variability (HRV) to blood pressure and venous return variabilities [1]. There is currently no non-invasive gold-standard for measuring stroke volume (SV). Recent MR methods can measure cardiac output by integrating flow volume through several cardiac cycles [2,3]. We propose a method that is capable of measuring changes in SV on a beat-to-beat basis.

### Methods

Real-time spiral phase-contrast [4-6] was used to measure through-plane velocities in the ascending aorta, achieving 3 mm spatial resolution and 57 ms temporal resolution. The ECG trigger was recorded every TR (7 ms). Frame rate was increased to 70 fps using view-sharing. Phase-offsets due to eddy currents were corrected based on variations within the chest-wall [7].

Thresholds are applied to the magnitude and phase-difference images. Region growing is used to automatically obtain a region-of-interest (ROI) for the ascending aorta. If an abrupt change in ROI size is detected, the operator is prompted to correct it.

A volume flow waveform is obtained from the time-velocity distribution within the ROI. ECG false negatives are automatically corrected based on peak-to-peak intervals measured on this waveform. The SV is calculated as the integral of the flow within each R-R interval. The respiratory motion is estimated from the position of the chest-wall.

Studies were performed on a GE Signa 3 T EXCITE HD system. Three healthy volunteers were imaged at rest, and while performing a Valsalva maneuver, a handgrip, and a breath-hold.

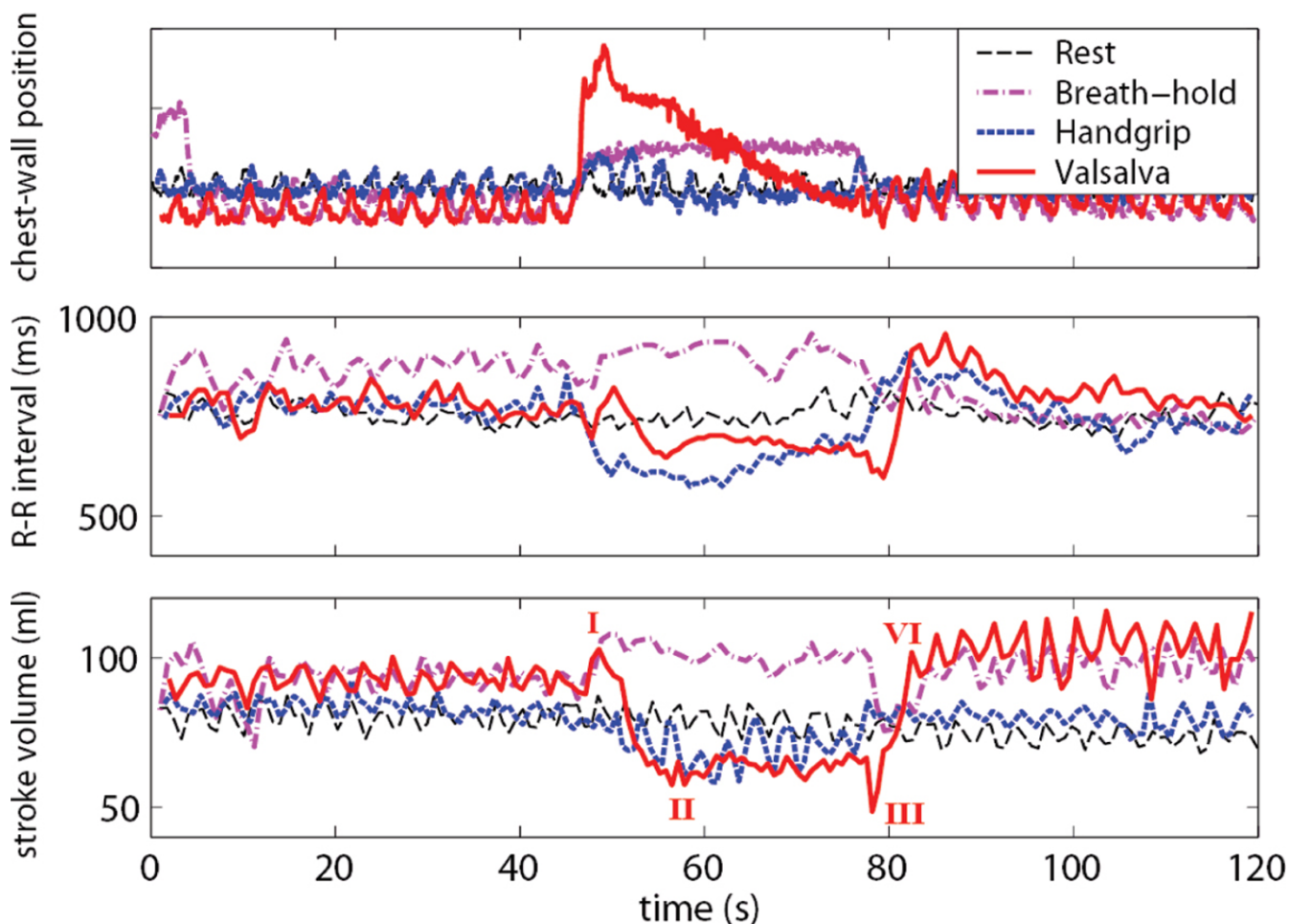
### Results and discussion

Fig. 1 shows representative results from one of the volunteers. Measured respiratory motion, R-R intervals, and stroke volume are presented.

The breath-hold was performed in the inhale position. We observed a slight increase in SV, and a reduction in HRV and SVV. This is consistent with the increase in venous return and sympathetic activation. A brief drop in SV was observed during the first exhale after the breath-hold, which can be attributed to a reduction in venous return.

During the handgrip, we observed a significant increase in heart rate, a moderate increase in SV, and a noticeable increase in SVV. These can be attributed to the balance between vascular sympathetic activation and cardiac vagal activation.

During the first few seconds of the Valsalva maneuver (phase I), we observed a slight increase in SV, associated with the increase in venous return due to inspiratory pressure. The significant drop in venous return associated with phase II explains the observed drop in SV and increase in heart rate. SVV and HRV are noticeably reduced, due to increased sympathetic activation. When breathing is resumed, heart rate briefly increases as the external compression on the aorta is removed (phase III), and a dip in SV was observed. The heart rate then drops due to



**Figure 1**  
Measured respiratory motion, heart rate variability, and stroke volume. The phases of the Valsalva maneuver are indicated (I–IV).

increases in aortic pressure, venous return and vagal activity (phase IV), and SV increases in response to the increased diastolic period [8].

### Conclusion

Beat-to-beat SV variability may be measured using real-time spiral phase contrast at 3 T. This approach proved capable of detecting dynamic changes in SV during different phases of stimuli such as the Valsalva maneuver, breath-hold, and handgrip.

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