

## UNIVERSITY OF SOUTHERN CALIFORNIA

#### 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. We demonstrate its use in healthy volunteers under different loading conditions.

### **Methods:**

Real-time spiral phase-contrast (Figure 1) [4,5,6] was used to measure through-plane velocities in the ascending aorta (Figure 2a-c). Scan parameters are listed in Table 1. The ECG trigger was recorded every TR. Phase-offsets due to eddy currents were corrected based on variations within the chest-wall [7].

Thresholds were applied to the magnitude and phase-difference images. Region growing was used to automatically obtain a region-of-interest (ROI) for the ascending aorta (Figure 2d). If an abrupt change in ROI size was detected, the operator was prompted to correct it.

A volume flow waveform was then obtained from the timevelocity distribution within the ROI (Figure 2e). ECG false negatives were automatically corrected based on peak-to-peak intervals measured on this waveform. The SV was calculated as the integral of the flow within each R-R interval. The respiratory motion was estimated from the position of the chest-wall.

Studies were performed on a GE Signa 3T EXCITE HD system. Three healthy volunteers were each imaged at rest, and also Beat-to-beat SV variability may be measured using real-time spiral phase contrast at 3T. This approach proved capable of detecting under each of the following conditions: (a) 20-second breathdynamic changes in SV during different phases of stimuli such as holdm (b) 30-second Valsalva maneuver, and (c) 2-minute handthe Valsalva maneuver, breath-hold, and handgrip stress. grip (~40% of maximum strength).

### **Results:**

Figure 3 shows the measured stroke volume variation from one of the volunteers. During a breath-hold, we observed a reduction in HRV and SVV, which is attributed to an increase in sympathetic activation, in response to the stimulus. A brief drop in SV was observed during the first exhale after the breath-hold, which is attributed to a reduction in venous return.

Figure 4 shows stroke volume variation during the Valsalva maneuver from a different volunteer. The results agree with the

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



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well-documented cascade of events during the Valsalva maneuver (phases I-IV) [8]. During the first few seconds (phase I), there is a slight increase in SV, attributed to 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 (HR). SVV and HRV are 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 increases in aortic pressure, venous return and vagal activity (phase IV), and SV increases in response to the increased diastolic period.

Figure 5 shows stroke volume variation during the handgrip ex-Figure 1: Spiral phase-contrast pulse sequence: (a) slice periment from a third volunteer. Handgrip exercise stimulates the selective excitation, (b) flow encoding bipolar gradient, (c) sympathetic nervous system, resulting in an increase in heart rate, spiral readout, and (d) refocusing and spoiler gradients. and a reduction in HRV. A reduction in SV is observed due to shorter diastolic periods.

### **Discussion:**

Potential sources of error of the method include partial volume effects, aliasing, off-resonance, and inaccurate segmentation of the lumen. Although low spatial resolution is used in order to maintain useful temporal resolution, we assume that the phase contrast velocity is the average velocity within each voxel, in which case partial volume would not be an issue. Pixels with static tissue have zero velocity/flow, therefore we prefer to overestimate the size of the lumen than to underestimate. In order to minimize errors due to off-resonance and aliasing, we use short readouts (4 ms), and reconstruct data from a single coil element.

### **Conclusions:**

### **References:**

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Figure 2: Stroke volume calculation. A scan plane is prescribed perpendicular to the ascending aorta (a). A region of interest corresponding the aorta's cross-sectional area (d) is obtained by applying a threshold to the magnitude (b) and phase-difference (c) images. A volume flow waveform is obtained from the time-velocity distribution within the ROI (e). The SV (indicated) is calculated as the integral of the flow within each R-R interval (red dots).

time (sec)

 Table 1: Scan parameters.

Flip angle: 10 <sup>0</sup> Slice thickness: 5 mm Venc: 200 cm/s	Spiral interleaves: 4 Readout: 4 ms FOV: 25~6 cm (variable-density)	TR: 7 ms Temporal res.: 56 ms After view-sharing: 70 frames/sec (14 ms)	
	(variable-density)	70 frames/sec (14 ms)	
	Spatial resolution: 3 mm		









**Figure 3:** Stroke volume measured during free-breathing (dashed) and breath-holding (solid). Sympathetic activation causes a noticeable reduction in SV variability.



Figure 4: Measured respiratory motion, heart rate variability, and stroke volume, during a Valsalva maneuver test. The different phases of the maneuver are indicated (I-IV).



Figure 5: Heart rate variability and stroke volume measured during handgrip stress (maintaining 40% of maximum grip). Note the slight increase in heart rate, which causes a reduction in SV in response to the shorter diastolic periods.