Assessment of stroke volume variability using real-time spiral phase contrast

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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. Recent MR methods can measure cardiac output by integrating flow volume through several cardiac cycles [2,3]. We propose using real-time spiral phase contrast (PC) for measuring changes in stroke volume (SV) on a beat-to-beat basis.

Methods: Real-time spiral PC [4,5,6] was used to measure through-plane velocities in the ascending aorta, with 3 mm spatial resolution and 57 ms temporal resolution [7]. The ECG trigger was recorded every 7 ms TR. Phase-offsets due to eddy currents were corrected based on variations within the chest-wall [8]. Thresholds are applied to the magnitude and phase-difference images, and region growing is used in each heartbeat to automatically estimate the aortic cross-sectional area. The SV is calculated as the integral of the flow (velocity x area) within each R-R interval.

Studies were performed on a GE Signa 3T EXCITE HD system. Seven healthy volunteers were imaged in acquisitions from 1 to 6 minutes long, covering a initial rest period, a stimulus period, and a recovery period. The responses to the following stimuli were evaluated: Valsalva maneuver (30 seconds); handgrip (approximately 40% of maximum strength, for 2 minutes); mental stress (consecutively subtracting 7 from the previous result for 2 minutes, starting from the number 901); facial cooling (cold compress on forehead for 2 minutes); and cold pressor (left hand submersion in icy water for 2 minutes). The measured dynamic changes in respiration, heart rate (R-R interval), and stroke volume were physiologically evaluated.

Results and Discussion: Fig. 1 shows the results of the Valsalva maneuver experiment from one of the volunteers. During the first few seconds (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 (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 [9].

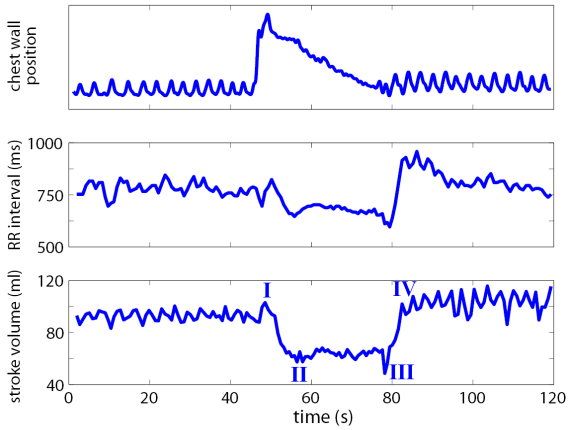

Fig. 1: Measured respiratory motion, heart rate variability, and stroke volume, during a Valsalva maneuver test. The different phases of the maneuver are indicated (I-IV).

Fig. 2 presents the results of the handgrip and facial cooling experiments performed on a second volunteer. The handgrip exercise stimulates the sympathetic nervous system, resulting in an increase in heart rate, and a reduction in HRV. A reduction in SV is observed due to shorter diastolic periods. Facial cooling, on the other hand, provides vagal stimulation, causing reduction in heart rate, and increase in SV (longer diastoles).

Fig. 3 presents the results of the mental stress and cold pressor experiments on a third volunteer. Both tests provide sympathetic stimulation. Mental stress tests typically cause an increase in respiratory rate. A change in HRV and SVV patterns due to increased sympathetic activity was observed in all volunteers, but variable results in HR and SV were observed in each volunteer, with these variables either increasing, decreasing, or not varying noticeably. Such variation is common, and is in part related to coping effects. In the result shown, HR and HRV decreased, and SV increased, with an increase in SVV frequency, which seems to be associated with the increase in respiratory rate (cardioventilatory coupling). In the cold pressor experiment, we observed a significant increase in HR, accompanied by a slight increase in SV, resulting in a substantial increase in cardiac output. Such response is caused by the increased sympathetic activity, due to local mediators in the muscles (humoral control) and from central stimulation of the sympathetic autonomic nervous system.

Conclusion: We used real-time spiral phase contrast imaging at 3T to measure dynamic changes in SV in response to five different stimuli. The observed results are in agreement with our expectations based on our current understanding of the physiology.

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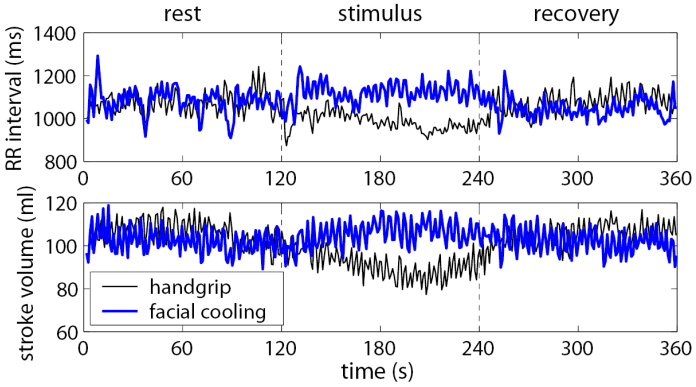

Fig. 2: Measured HR and SV, during handgrip and facial cooling tests.

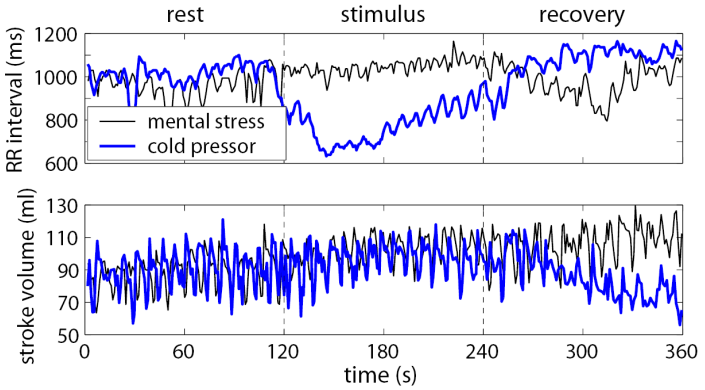

Fig. 3: Measured HR and SV, during mental stress and cold pressor tests.