**Objective:** To evaluate the selective contributions of cardiac sympathetic and vagal activity to the respiratory sinus arrhythmia (RSA) generated by rhythmic deep breathing.

**Background:** RSA is widely used clinically as a pure and independent estimate of cardiovagal outflow. However, cardiac sympathetic activity also exhibits strong respiratory modulation that should be reflected in the RSA. RSA is strongly dependent on the level of cardiac sympathetic activity and is enhanced by beta adrenergic blockade. RSA may also be generated by mechanical stretch of the atria induced by large fluctuations in tidal volume.

**Design/Methods:** In order to obtain further insight into the simultaneous changes in cardiovagal and cardiac sympathetic activity during RSA, we recorded beat-to-beat changes in RR intervals (RRI), and finger arterial pressure in 15 subjects breathing at 6 breaths/min. We applied a novel time domain method which exploits the inherent property of scale covariance of signals to analyze RRI in the time domain. This non-model driven methodology appears to decompose the beat-to-beat changes in RRI into velocities that reflect the parasympathetic and sympathetic components of cardiac autonomic activity.

**Results:** RSA typically consisted of a gradual decrease in RRI followed by a plateau phase and then a rapid increase in RRI. A clear pattern was observed in 10/15 subjects. In these subjects the initial tachycardia was due to decrease in vagal velocity while the plateau phase was associated with an increase in both vagal and sympathetic velocity. Abrupt bradycardia occurred as vagal velocity which continued to increase while sympathetic velocity diminished.

**Conclusions/Relevance:** Generation of RSA often reflects the complex interplay of changes in cardiovagal and cardiac sympathetic activity. In some cases RSA appears to be generated by rhythmic deep breathing in the absence of obvious fluctuations in autonomic activity.