

Cardiovagal and Cardiac Sympathetic Coactivation During the Valsalva Maneuver

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"If the glottis be closed after a deep inspiration, and a strenuous and prolonged expiratory effort be then made, such pressure can be exerted upon the heart and intrathoracic vessels that the movement and flow of the blood are temporarily arrested".

Antonio Maria Valsalva 1666 - 1723

INTRODUCTION

The cardiovascular response to the Valsalva maneuver reflects the complex central integration of baroreceptor, chemoreceptor, cardiopulmonary and somatic afferent inputs by the autonomic nervous system. The heart rate changes during the Valsalva maneuver are almost abolished by atropine but are not significantly altered by propranolol leading many to suggest that changes in parasympathetic cardiovagal outflow are almost completely responsible for both phase II cardioacceleration and phase IV cardioinhibition. This view presumes that each arm of the autonomic nervous system functions in an independent yet predetermined fashion that is unaffected by the pharmacological blockade, an assumption that is unlikely to be true.

METHODS

To obtain further insight into the simultaneous changes in cardiovagal and cardiac sympathetic activity during the Valsalva maneuver, we recorded beat-to-beat changes in heart rate and finger arterial pressure during Valsalva maneuver performed while supine against a 40 mmHg expiratory load for 15 seconds. RR intervals were analyzed using a renormalization-group approach which exploits the inherent property of scale invariance of the signal. Briefly, the current state of any dynamical system strongly depends on the previous states of that system. It is possible to apply non model driven feature extraction techniques that assign parameters that describe the current state of the system. When these techniques are applied to analysis of RR intervals only 2 parameters emerge.

Figure 1: Example of analysis of RR intervals at rest. Note the robust fluctuations in both presumed parasympathetic and sympathetic velocities (respectively, cyan and violet).

Figure 3 (below): The sympathetic index selectively increases during head-up tilt. The parasympathetic index is selectively abolished by atropine (data not shown).

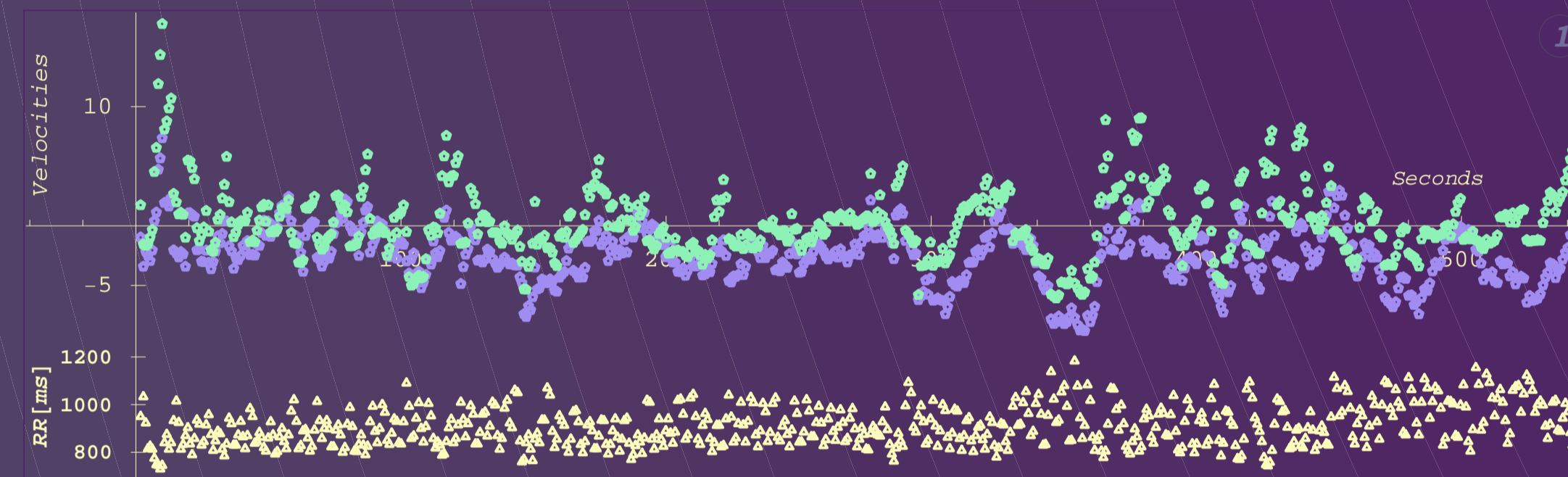
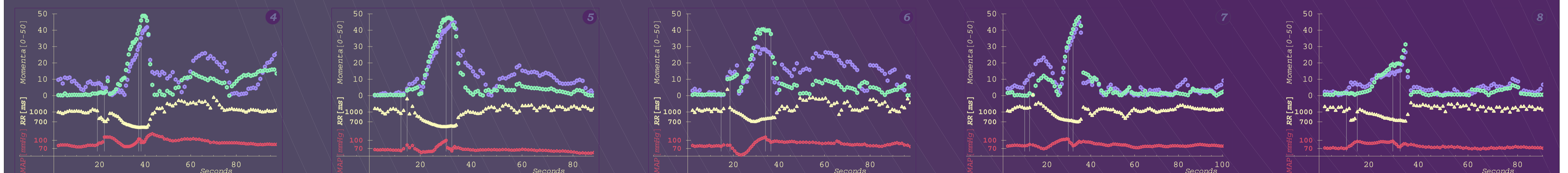
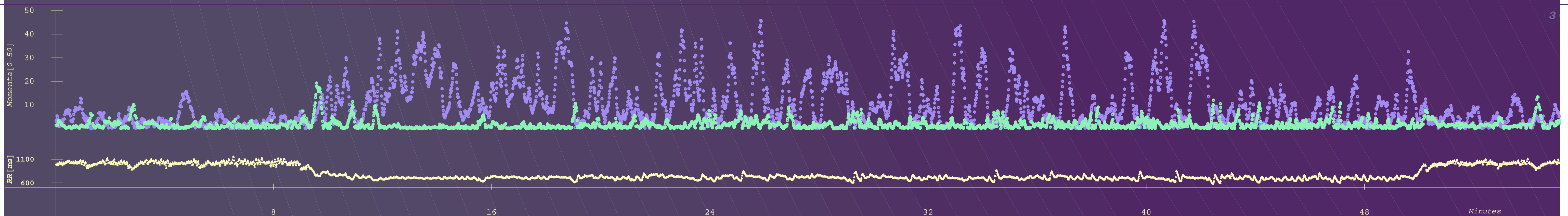
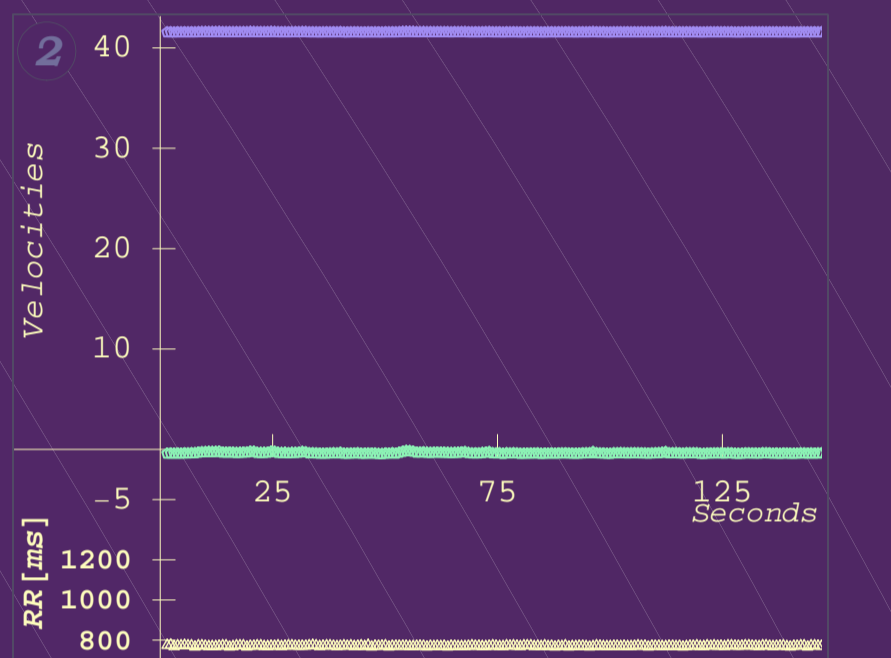


Figure 2: The transplanted denervated heart shows no fluctuations in these velocities.



Figures 4-8 (above): Five examples of Valsalva maneuvers from normal subjects are shown. Note in all cases the strong parasympathetic sympathetic coactivation during phase 2 of the maneuver. Note as well the relative predominance of parasympathetic activity during the plateau phase of the phase II tachycardia.

Figure 9: A sixth example from a patient with POTS.

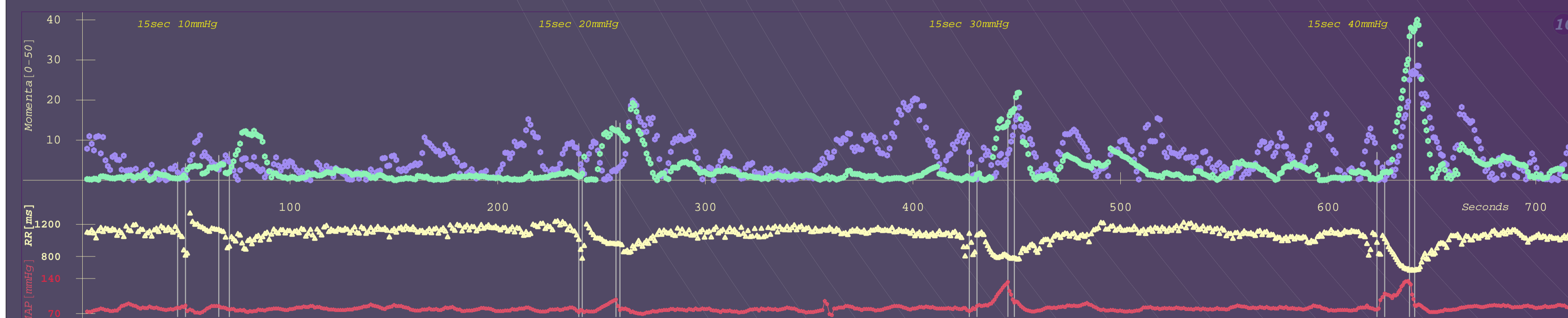
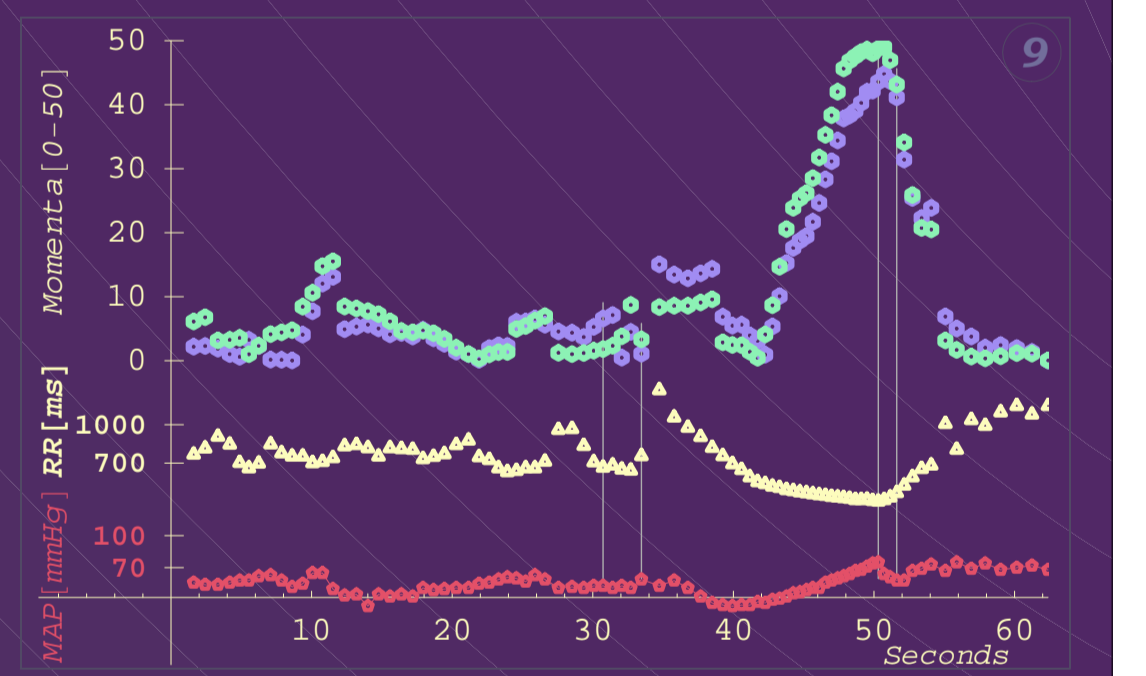
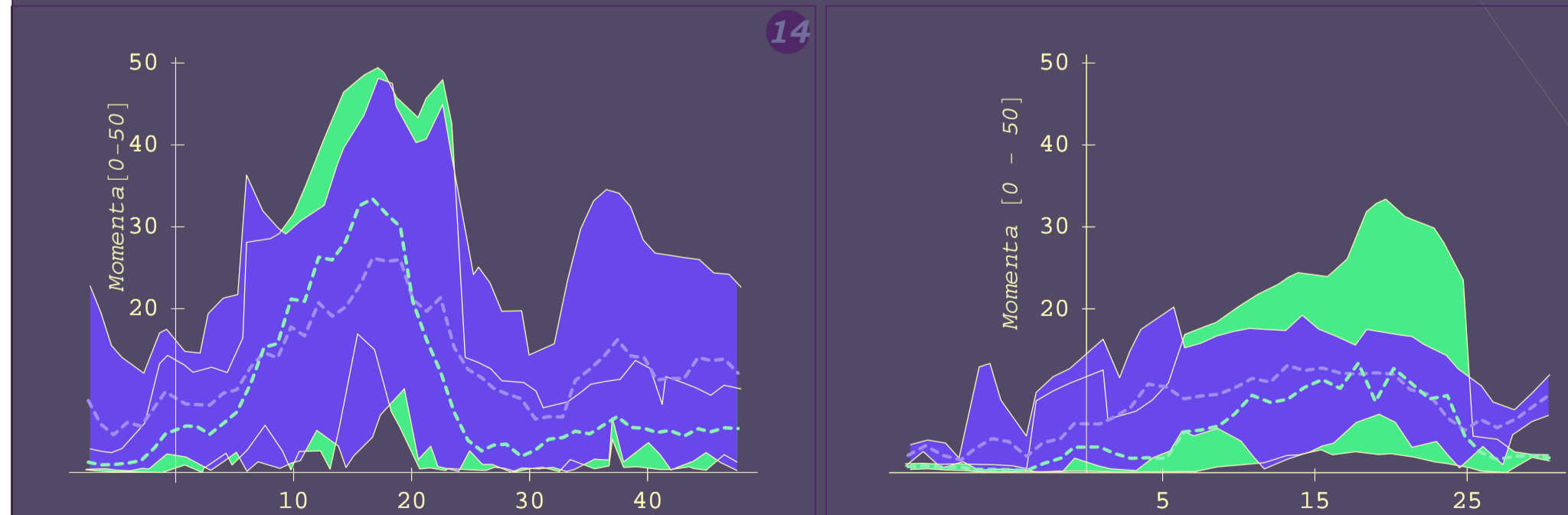
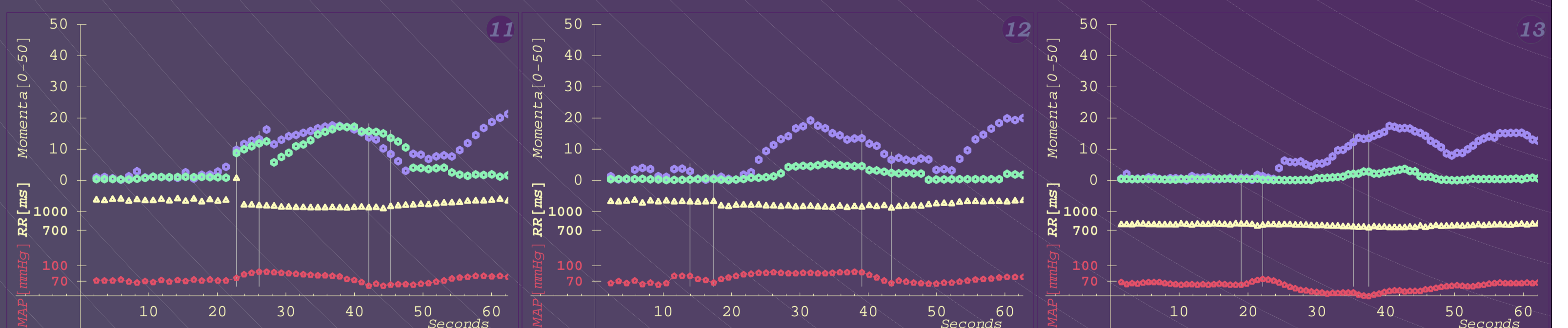


Figure 10: Graded Valsalva maneuvers at 10, 20, 30 and 40 mmHg expiratory load. Note that coactivation is most evident at higher expiratory loads.



Figures 14-15: Point-wise averages with all-cases minima and maxima. On the left, normal Valsalvas; on the right, cases of autonomic failure.



Figures 11-13: Three examples of Valsalva maneuvers in patients with autonomic failure. Note the flat sympathetic and parasympathetic indices prior to the Valsalva as well as reduced coactivation.

CONCLUSION

- There appears to be coactivation of parasympathetic and sympathetic cardiac activity during phase II of the Valsalva maneuver.
- This robust interaction is not present in patients with autonomic failure.
- Coactivation may serve to improve cardiac output during the strain phase of the Valsalva maneuver.

