Increases in Heart Rate Variability Low Frequency Power Due to Slow Yogic Breathing Are Vagally Mediated

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Aims

Examine effects of slow yogic breathing on HRV

Isolate vagal versus sympathetic contributions to HRV in LF range

Background

Variance in the timing of heart beats, known as heart rate variability (HRV), is a widely used measure within health psychology. High frequency (HF) changes in HRV (0.15-0.40 Hz) reflect changes in respiration and are mediated by parasympathetic (vagal) control. Low frequency (LF) changes (0.04-0.15 Hz) are often interpreted as reflecting a significant sympathetic component[1-2]. but cannot be interpreted as reflecting solely sympathetic influence. Slow yogic breathing is a non-invasive intervention that has relaxing effects and is thought to increase vagal control. Prescribed breathing rates (e.g. 6 bpm) commonly fall into the LF range and increase HRV in the LF band. This creates a paradox of interpretation for HRV LF power[3-4]: do the increases in LF power during low frequency yogic breathing reflect contributions from predominantly parasympathetic or sympathetic control?

REFERENCES

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4. Bernardi, L. *et al.* Effects of controlled breathing, mental activity and mental stress with or without verbalization on heart rate variability. *J. Am. Coll. Cardiol.* **35,** 1462–1469 (2000).
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Experimental Design

Procedure

- 6 healthy adults (2 females and 4 males, mean age 22)
- 1 minute of guided yogic breathing followed by 4 minutes of rest
- 11 different breathing rates between 4.0 and 9.0 breaths per minute (that span the LF and HF band above).
- Completed in a randomized order
- Breathing cadence was 4-4-6-2.

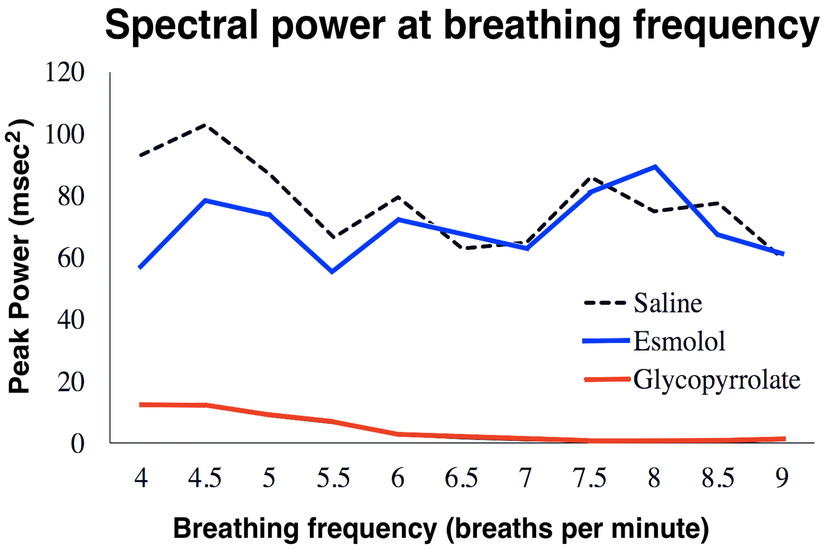
Experimental Conditions (within subjects)

HRV was calculated using spectral analysis of IBIs for each guided breathing trial on three different days corresponding to three different randomized conditions:

- Sympathetic blockade (Esmolol)
- Parasympathetic blockade (Glycopyrrolate)
- Placebo (Normal saline)

This design enabled a direct comparison between sympathetic and parasympathetic contributions to LF power in the context of a guided breathing manipulation.

Results

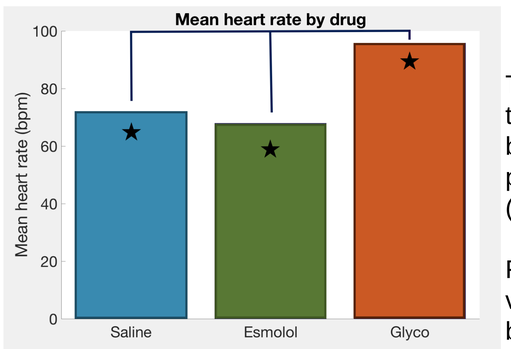


This figure demonstrates the significant effect of the drug (Wilks' λ= .181, approximate F(2,4) = 9.03, p<.05).

1) HRV at each breathing rate was virtually absent during Glycopyrrolate (p=.016 vs saline)

2) Esmolol had a minimal effect on HRV compared to saline (p=.671 vs saline)

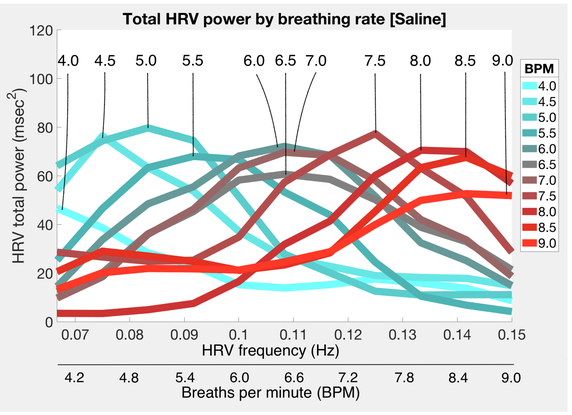
3) At the slowest breathing rate HRV showed a slight decline with Esmolol relative to saline (at 4.0 bpm, p<.005) and a slight elevation during Glycopyrrolate.



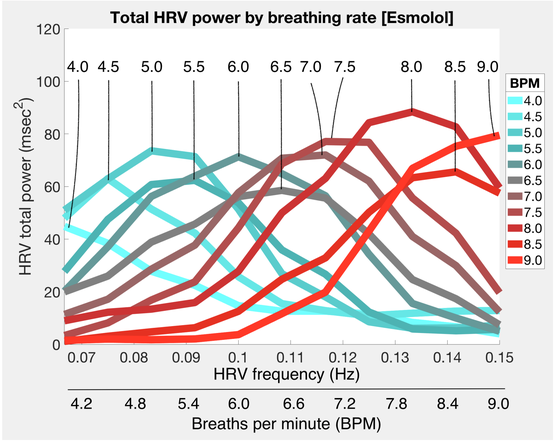
VALIDITY CHECKS

There was a significant effect of drug in the expected direction for sympathetic blockade (Esmolol) F(2,4) = 21.5, p<0.001 and parasympathetic blockade (Glycopyrrolate) F(2,4) = 512.5, p < 0.001.

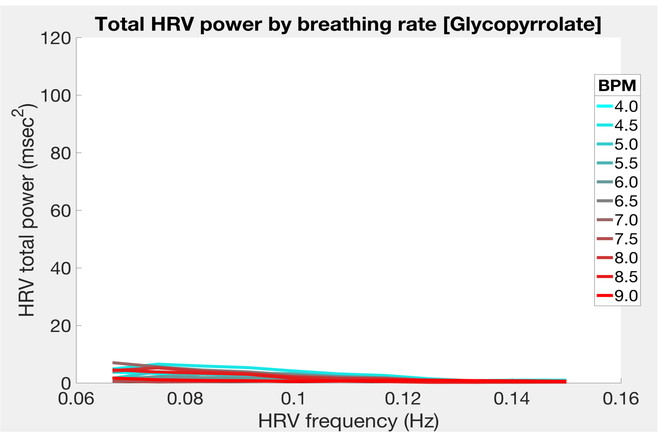
Fidelity to the guided breathing rate was verified by comparing it to the measured breathing rate (r = 0.94, p < 0.01).



Peak HRV total power mirrors breathing frequency for placebo.



This trend remains across all low frequency bands with sympathetic blockade.



HRV total power is nearly eliminated across all low frequency bands with parasympathetic blockade.

Discussion

Increases in HRV power in the low frequency range during slow yogic breathing are due to increased vagal activity and do not reflect changes in sympathetic activity. Yogic breathing offers a non-invasive method of increasing vagal control, which may improve sympathetic-parasympathetic balance[5]. Reducing chronically elevated sympathetic drive may improve health and reduce the risks associated with sympathetic predominance.