Noninvasive Testing: Insights Into Physiology/Pathophysiology (Oral Contributions)
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Tight Mechanism Correlation Between Heart Rate Turbulence and Baroreflex Sensitivity: Sequential Autonomic Blockade Analysis

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Background: Heart rate turbulence (HRT) is a de novo powerful risk predictor for patients (pts) surviving acute myocardial infarction. However, little is known about its underlying physiologic mechanism.

Methods: Hypothesizing that HRT be barorceptor reflex related, we studied the HR and blood pressure fluctuations at rest and following systematically introduced ventricular premature beat (VPB) in 16 pts without structural heart disease (10 male, 6 female, mean 45±17 y/o), before and after sequential sympathetic (esmolol, 4mg bolus followed by 120ug/kg/min iv), parasympathetic (atropine, 0.04 mg/kg iv) and total autonomic blockade (esmolol plus atropine). Turbulence onset (TO, %), and turbulence slope (TS, ms/beat) were averaged from 10 respective VPB's. Spontaneous baroreflex sensitivity (BRS, ms/mmHg) was calculated from 5 minutes of sinus rhythm recording. The results showed that TS decreased after atropine (0.71±0.50 ms/beat, vs. 5.17±3.96 ms/beat at baseline, p<0.01) and total autonomic blockade (1.23±1.02 ms/beat, p<0.01). TO increased after atropine (0.32±0.35%, vs. -0.45±0.94% at baseline, p<0.01) and total autonomic blockade (0.58±0.86%, p<0.05). TS was positively correlated with BRS at baseline (r=0.78, p<0.01) and after esmolol (r=0.8, p<0.05) and total autonomic blockade (r=0.31, p>0.05). TO was negatively correlated with BRS at baseline (r=-0.61, p<0.05) and after esmolol (r=-0.80, p<0.01). However, TO remained correlating with BRS after atropine (r=-0.53, p<0.05).

Conclusions: Heart rate turbulence of both TO and TS parameters is critically vagal-dependent and highly correlated with spontaneous baroreflex sensitivity, which underscores its clinical importance in cardiovascular risk stratification.