Instantaneous Time-Course of the Autonomic Cardiovascular Respiratory Response of Healthy Subjects to Moderate Hypoglycemic Stimulus

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The autonomic cardiovascular respiratory (ACR) response to hypoglycemia has been reported in a static and fragmentary fashion and with limited use of cardiovascular variability spectral analysis.

To characterize a dynamic and integrative ACR response to hypoglycemia we assessed, in 13 healthy subjects under control, peak hypoglycemia induced by administering 0.2 U/kg of insulin, and recovery conditions, instantaneous 5-min time-courses of: time series of RR intervals (RR), systolic (SP), diastolic (DP) and pulse (PP) pressures, tidal volume (V_T) and respiratory frequency (RF) obtained from ECG, arterial pressure (AP) and respiratory (Res) signals; their low-frequency powers (LF_{RR}, LF_{SP}, LF_{DP}, LF_{PP}), their respective central frequencies ($_{CF}LF_{RR}$, $_{CF}LF_{SP}$, $_{CF}LF_{DP}$, $_{CF}LF_{PP}$), and high-frequency powers (HF_{RR}, HF_{Res}), computed by a time-frequency distribution; baroreflex (BRS) and respiratory sinus arrhythmia (RSAS) sensitivities computed by alpha index and their coherences (cBRS, cRSAS) by a cross-time-frequency distribution. For statistical analysis, 1-min epoch means (EM) of dynamics were obtained.

With respect to control, peak hypoglycemia (48±6 mg/100 ml) provoked 1. decreases ($p<0.03$) in: five EM of HF_{RR}, LF_{RR}, BRS and RSAS dynamics, three EM of $_{CF}LF_{PP}$ and cBRS, two EM of $_{CF}LF_{RR}$ and $_{CF}LF_{SP}$; 2. increases ($p<0.02$) in: five EM of SP, DP, PP, V_T and RF, three EM of HF_{Res}, two EM of LF_{SP} and LF_{DP}, one EM of LF_{PP}; 3. no change in $_{CF}LF_{DP}$, RR and cRSAS.

Hypoglycemia modifies the basal fluctuating time-courses of ACR measures, specifically eliciting: sympathetic measures powers increase (except LF_{RR}) associated with central frequencies reductions; SP, DP and PP increments, indicating stroke volume elevation; BRS and cBRS decreases allowing AP to rise; vagal activity index and RSAS reductions determining greater RR regularity; and increased pulmonary ventilation. These effects integrate a dynamic mechanism of sympathetic, cardiovascular and respiratory activation, whose consequence is increased cardiac output with greater oxygen content to ameliorate the harmful effects of neuroglycopenia.