Low glucose and carotid body-mediated CO₂ chemosensitivity in the rat

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Superfusion with low glucose solution increases catecholamine release from isolated type I cells of the rat carotid body (Pardal & Lopez-Barneo, 2002) and insulin infusion increases baseline ventilation in a carotid body-dependent manner (Bin-Jaliah & Kumar, 2003). Whether this latter effect was due to hypoglycaemia per se or hypermetabolism was not determined. In this study, we hypothesized that insulin infusion should, like hypoxia, increase CO₂ chemosensitivity in vivo and we have tested the direct effect of low glucose on an in vitro carotid body preparation.

Ventilatory CO₂ chemosensitivity was measured from integrated airflow using a modified rebreathing technique (Read, 1967) in hyperoxia (P_{aCO₂} > 300 mmHg) in adult Wistar rats (300–350 g), anaesthetized with urethane (650 mg kg⁻¹, i.v.). Chemoreceptor afferent discharge was recorded in vitro from carotid bodies (Pepper et al. 1995) isolated from adult Wistar rats (120–150 g) anaesthetized with halothane (2–3 % in O₂). All animals were humanely killed at the end of the experiment. Data are expressed as means ± S.E.M. and significance (P < 0.05) was tested with ANOVA and, as appropriate, the post hoc Bonferroni/Dunn test.

Insulin infusion (0.4 U min⁻¹ kg⁻¹) lowered blood glucose from 6.7 ± 0.1 to 3.3 ± 0.1 mmol L⁻¹ (P < 0.0001) and in sham-operated, control animals (n = 6) increased CO₂ chemosensitivity from 12.58 ± 1.10 to 19.19 ± 1.51 ml min⁻¹ kg⁻¹ mmHg⁻¹ (P < 0.01). In contrast, CO₂ chemosensitivity in animals with bilateral carotid sinus nerve section (n = 6) remained unchanged (12.12 ± 0.92 to 13.78 ± 0.95 ml min⁻¹ kg⁻¹ mmHg⁻¹; P > 0.25). Chemoafferent recordings from few-fibre preparations of the carotid sinus nerve (n = 5) showed that, whilst all fibres responded to falls in P_{aO₂} or elevations in P_{aCO₂}, lowering superfusate glucose from 10 mM to 2 mM had no effect upon baseline discharge (0.45 ± 0.26 to 0.36 ± 0.20 Hz, respectively; P > 0.30) or upon CO₂ chemosensitivity measured as Δ discharge between 40 and 80 mmHg P_{aCO₂} at a P_{aO₂} of ca 400 mmHg (P > 0.15).

These data demonstrate that a lowered glucose concentration does not increase baseline carotid body chemoafferent discharge or CO₂ chemosensitivity during hyperoxia in vitro and suggest that the in vivo effects of insulin infusion upon baseline ventilation and CO₂ chemosensitivity are, therefore, more likely due to its effects upon metabolism rather than upon glucose concentration.


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All procedures accord with current UK legislation.
Insights into complexity and regularity of renal sympathetic nerve activity in response to haemorrhage in Wistar rats

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We recently utilised a power spectral technique and a cross-sample entropy (CSE) method (Rickman & Moorman 2000) to examine synchrony of the relationship between blood pressure (BP) and renal sympathetic nerve activity (RSNA) during right atrial stretch to mimic plasma volume expansion (Yang et al. 2002). CSE revealed that during the reflex inhibition there was more synchrony between the oscillating signals in the BP and RSNA sequences. In the present study we have used a similar analysis of these signals during a mild haemorrhage which reflexly causes an increase in RSNA in an attempt to maintain BP constant.

The experiments were performed on 10 anaesthetised (urethane 650 mg kg⁻¹, chloralose 50 mg kg⁻¹) Wistar rats. BP was measured from a femoral artery and RSNA from a branch of renal nerve after exposing the left kidney retroperitoneally. A 33 s high frequency (1 kHz) sampling of BP and RSNA was recorded and rectified. The trachea was cannulated and spontaneous respiration maintained. Rectal temperature was maintained at 37°C by a heating blanket. A femoral vein was cannulated and 1 ml of blood was removed into a pre-heparinised syringe over 1 min and 5 min later slowly reinfused. Data are expressed as means ± S.E.M., and analysed using repeated measures ANOVA. Statistical differences were considered significant when \( P < 0.05 \).

Rats were killed by overdose of urethane at the end of experiment.

A coherence measurement from power spectral analysis failed to detect significant changes between baseline and haemorrhage in either averaged coherence over the range 0–10 Hz (0.492 ± 0.01 to 0.491 ± 0.01) or coherence at heart rate frequency (0.94 ± 0.03 to 0.93 ± 0.02). However a non-linear dynamic analysis of the group data using CSE measurements showed that the relationship between BP signals and RSNA time series did change during haemorrhage, from 0.72 ± 0.05 at baseline to 0.79 ± 0.06 \((P < 0.05)\), revealing there was greater asynchrony.

The data suggest that cross-sample entropy calculations characterise the non-linearities underlying cardiovascular control signals and may reveal how homeostatic regulation is achieved by the autonomic nervous system.


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