PS C47

Low glucose and carotid body-mediated CO_2 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 CO_2 chemosensitivity *in vivo* and we have tested the direct effect of low glucose on an *in vitro* carotid body preparation.

Ventilatory CO_2 chemosensitivity was measured from integrated airflow using a modified rebreathing technique (Read, 1967) in hyperoxia ($P_{a,CO_2} > 300$ 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 O_2). 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 CO_2 chemosensitivity from 12.58 ± 1.10 to 19.19 ± 1.51 ml min^{-1} kg^{-1} mmHg^{-1} ($P < 0.01$). In contrast, CO_2 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_{O_2} or elevations in P_{CO_2}, 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 CO_2 chemosensitivity measured as Δ discharge between 40 and 80 mmHg P_{CO_2} at a P_{O_2} of *ca* 400 mmHg ($P > 0.15$).

These data demonstrate that a lowered glucose concentration does not increase baseline carotid body chemoafferent discharge or CO_2 chemosensitivity during hyperoxia *in vitro* and suggest that the *in vivo* effects of insulin infusion upon baseline ventilation and CO_2 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

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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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