

POSTER PRESENTATION

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# 0592. Metabolic acidosis induced by haemorrhage and hydrochloric acid generates different cardiorespiratory responses

G Sabbatini\*, A Dyson, M Singer

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## Introduction

Metabolic acidosis is classically thought to induce an enhanced ventilatory pattern, irrespective of the underlying aetiology.

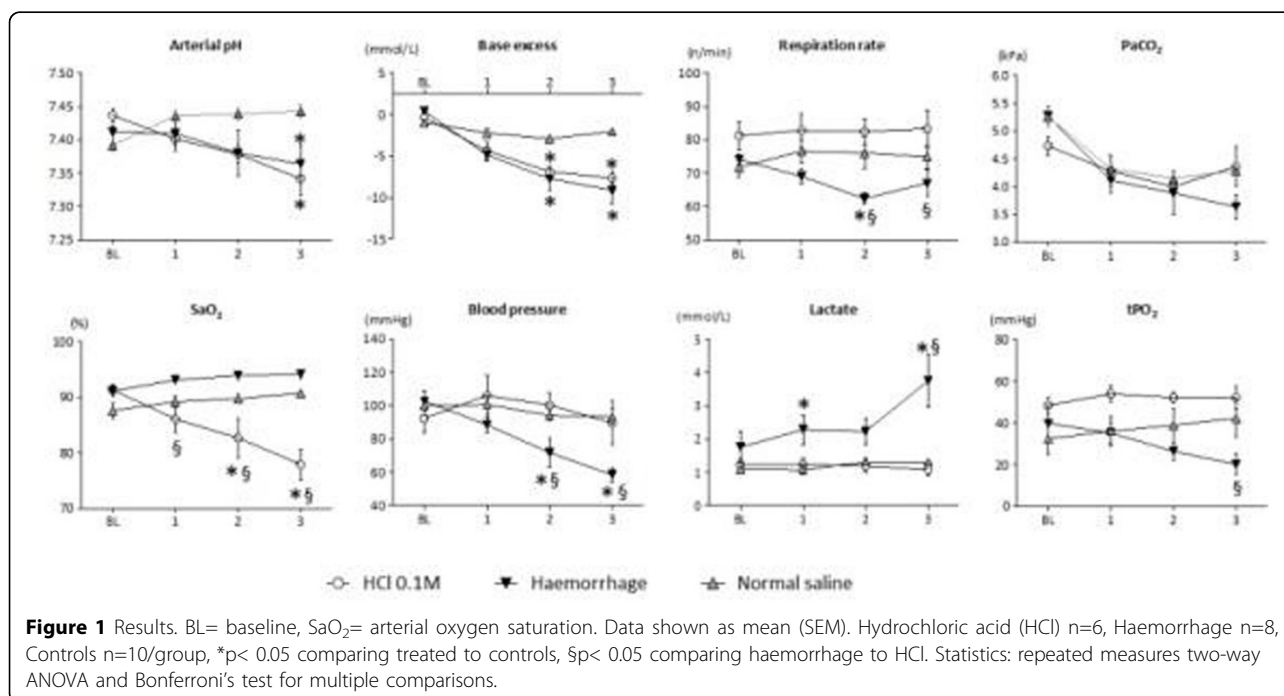
## Objectives

To induce a similar level of acidaemia in a rat model, by either infusion of an acidic solution or by blood withdrawal,

and to assess the physiological responses to these insults.

## Methods

Isoflurane-anaesthetised, tracheotomized rats were instrumented with left common carotid arterial and right jugular venous lines for blood sampling/BP monitoring and fluid/blood administration, respectively. Oxylite™ probes



**Figure 1** Results. BL= baseline, SaO<sub>2</sub>= arterial oxygen saturation. Data shown as mean (SEM). Hydrochloric acid (HCl) n=6, Haemorrhage n=8, Controls n=10/group, \*p < 0.05 comparing treated to controls, \$p < 0.05 comparing haemorrhage to HCl. Statistics: repeated measures two-way ANOVA and Bonferroni's test for multiple comparisons.

(Oxford Optronix, UK) placed in thigh muscle were used to monitor tissue oxygen tension ( $tPO_2$ ). Animals were subjected to either continuous 0.1 M hydrochloric acid (HCl) infusion or 60% withdrawal of estimated blood volume in six 10% steps over three hours to induce an equivalent fall in arterial base excess (BE). All animals (including a control group) received n-saline throughout. Hourly measurements were made of haemodynamics,  $tPO_2$  and arterial blood gas analysis.

## Results

See figure 1.

HCl induced a metabolic acidosis with arterial hypoxaemia yet a preserved muscle  $tPO_2$ , no tachypnoea nor fall in  $PaCO_2$ . By contrast, haemorrhage to achieve a similar acidaemia, resulted in significant falls in blood pressure and  $tPO_2$ , hyperlactataemia, a small rise in  $SaO_2$  and a decrease in respiration rate with a concomitant fall in  $PaCO_2$  probably related to higher tidal volumes.

## Conclusions

Tissue hypoperfusion (and not just acidaemia per se) is an important component that triggers an enhanced ventilatory drive.

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