

FURTHER PHYSIOPATHOLOGICAL FEATURES OF EXPERIMENTAL *HOMERIA GLAUCA* (WOOD & EVANS) N.E.BR. POISONING IN MERINO SHEEP

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ABSTRACT

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Three Merino sheep were given 3 g/kg of dried, finely-milled *Homeria glauca* (Natal yellow tulip) plant material intraruminally.

Plasma glucose, cortisol, catecholamines and lactate were measured hourly and also at the moment of death. Rising plasma glucose was shown to be associated with rising plasma cortisol and catecholamines, and the metabolic component of tulip-associated acidosis was shown to be the result of lactate accumulation.

INTRODUCTION

In a previous paper (Button, Reyers, Meltzer, Mülders & Killeen, 1983) we demonstrated that sheep dying of *H. glauca* intoxication suffered ventricular tachycardia, arterial hypertension, hypoxaemia, hypercarbia, acidemia, hypochloraemia and hyperkalaemia. These sheep were also hyperglycaemic and had raised serum creatinine concentrations. Rising serum enzyme concentrations suggested that there was damage of the liver and striated muscle.

We postulated that hyperglycaemia was the result of rising plasma cortisol and catecholamines and that the metabolic component of the acidosis was the result of hypoxaemia-induced lactate accumulation. In so far as sheep killed by *H. glauca* intoxication frequently die in ventricular fibrillation, we considered it important to measure plasma catecholamines. If plasma catecholamines were elevated, antiarrhythmic and β adrenergic blocking drugs could well have a protective effect on the myocardium. This trial was designed to confirm or disprove the above hypotheses.

MATERIAL AND METHODS

Three mature Merino sheep (2 wethers and 1 ewe) were used in this trial. The sheep were not fasted before the trial. Control venous blood samples were drawn before toxin administration into evacuated tubes containing 1,8 mg of EGTA [ethyleneglycol-bis-(β -aminoethyl ether)-N,N,N,N'-tetracetic acid] and 1,2 mg of glutathione per ml blood collected for catecholamine assay, into Anderson's solution (Anderson, 1969) for plasma glucose determination, into heparinized tubes for plasma cortisol assay and into EDTA/fluoride for plasma lactate determinations. Plasma was harvested as soon as practicable after bleeding and then deep frozen for later assay.

Plasma catecholamines were measured using a radioenzymatic assay kit*. Plasma glucose and lactate were measured using commercially available kits**. Plasma cortisol was measured by radioimmunoassay***.

After all control samples had been taken, the sheep were given finely-milled, dried *H. glauca* plant material suspended in water, at the rate of 3 g/kg by stomach tube.

* CAT-A-KIT, Upjohn Diagnostics

** Boehringer Mannheim Diagnostica

*** Solid Phase Radioimmunoassay, Diagnostic Products

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Further blood samples were collected hourly and also at the moment of death. After analysis, data were pooled for the control blood samples, 2 h pre-death, 1 h pre-death, and at the moment of death samples. Sheep No. 7, which lived somewhat longer, had the first sample after dosing omitted from the calculations.

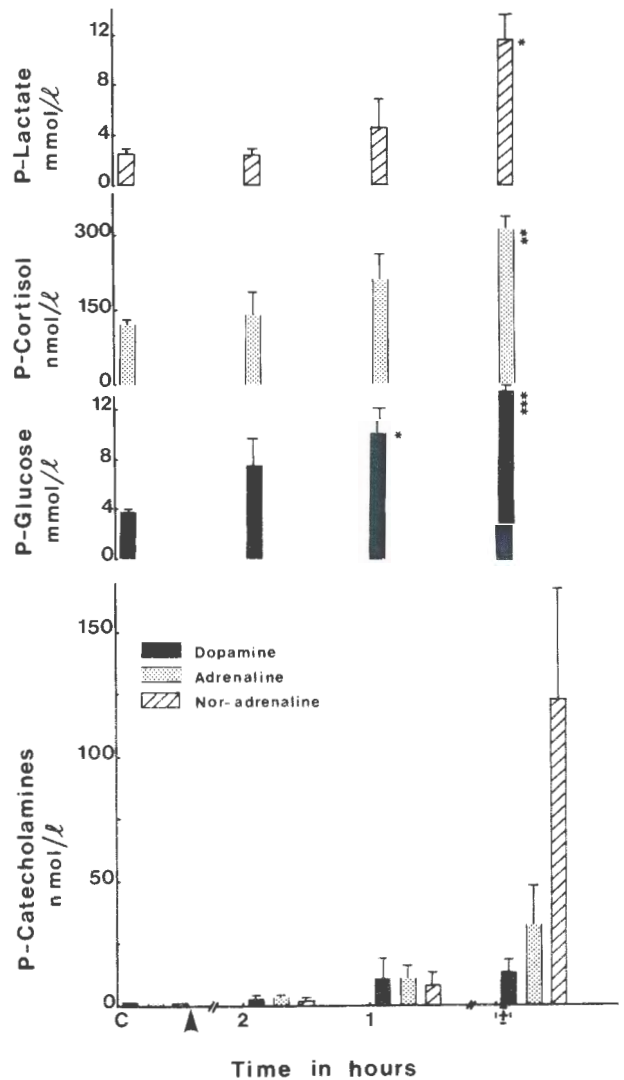


FIG. 1 Mean + SE of plasma lactate, cortisol, glucose and catecholamines for 3 sheep poisoned by *H. glauca*. Asterisks indicate that the mean was significantly different from control value (C) *P<0,05, **P < 0,01, ***P < 0,001. The arrow head indicates administration of toxin and the cross indicates death.

Means and standard deviations were calculated for the various pooled samples and the Student's *t* test (Steel & Torrie, 1960) was used to determine whether differences in means between the control data and data after administration of toxin were significant.

RESULTS

Plasma glucose increased progressively and significantly during the course of the trial. This was associated with a progressive and significant rise in plasma cortisol and increases in plasma adrenaline, noradrenaline and dopamine. The catecholamine increases were large, e.g. total plasma catecholamines increased 2,7, 11,9 and 167,0 times control values 2 h and 1 h pre-death and at the time of death, respectively. As a result of the small number of sheep and large standard deviations these differences did not reach significance (Fig. 1).

Plasma lactate also rose progressively to reach a value 4,6 times the control value at the time of death.

DISCUSSION

The progressive rise in plasma glucose can be ascribed to rising plasma cortisol and catecholamines. Rising plasma cortisol and catecholamine might, in turn, be ascribed to the hypoxaemia, acidosis and stress associated with intoxication along with other, as yet undetermined, factors.

Inasmuch as catecholamines and the bufadienolide toxin of *H. glauca* are well-recognized arrhythmogens (Naudé & Potgieter, 1971), and are active at the same

time as other arrhythmogenic influences, such as acidosis, hypoxaemia and hyperkalaemia, are becoming increasingly severe, protection of the myocardium by β blocking and/or antiarrhythmic agents should be attempted to prevent lethal ventricular dysrhythmias. In addition to oral activated charcoal, other therapies that might be tested included oxygen to counteract the hypoxaemia, systemic alkalinizers to combat the metabolic acidosis and measures to normalize hyperkalaemia.

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