Introduction:

The importance of external warming devices such as a heating mat has long been recognised to minimise the loss of body temperature in anaesthetised avian patients. It was assumed that the body temperature can decrease to 35°C during anaesthesia (Sinn, 1994). Verkest (1994) found an average body temperature loss of 4°C and development of acidosis in sulphur-crested cockatoos after four hours of Isoflurane anaesthesia. The importance of maintaining normal body temperature in avian anaesthesia was again emphasised in the clinical study of Verkest (1994) where one bird developed a cloacal temperature of 30.8°C during anaesthesia and subsequently died despite the ambient temperature being 30°C. In human paediatric medicine, humidified anaesthetic gas is used to minimise heat and energy loss from evaporation in the respiratory tract (Robinson 1979, Bengston et al 1989, Miyao et al 1992). Alternative methods of warming anaesthetized birds were investigated. The objective of the current study was to characterise the changes that occur during anaesthesia and to minimise anaesthetic risk factors in avian patients.

Materials and Methods:

Four sulphur-crested cockatoos, (*Cacatua galeria*), were anaesthetized on three separate occassions at least 7 days apart. The investigation was conducted in 3 parts. In the first part, (control study), the birds were anaesthetized with isoflurane for two hours with no external heat provision. In the second part, (heat mat study), a heating heating mat was used and maintained at 40oC. The third part of the experiment, (humidified gas study), the birds were anaesthetized with 40oC humidified anaesthetic gas. In both the heat mat and humdified gas studies, the birds were covered with a sheet of bubble plastic. The temperature of the heating mat was monitored by the placement of a continuous thermometer between the bird and the mat. In the humidified gas study, the anaesthetic gas was bubbled through a bottle containing hot water placed in a hot water bath. The anaesthetic gas was kept warm by a heating coil which was positioned between the oxygen supply and the delivery tube. The temperature of the air was monitored and maintained by a thermostat located at the endotracheal tube.

Blood was taken from the jugular vein at zero, one and two hours after induction for plasma biochemistry and blood gas analysis. The plasma parameters measured were sodium, chloride, potassium, calcium, phosphate, osmolality, glucose, aspartate aminotransferase (AST), creatinine

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kinase (CPK), gamma glutamyl transferase (GGT), glutamate dehydrogenase (GLDH), lactate dehydrogenase (LDH), urea, uric acid, and creatinine.

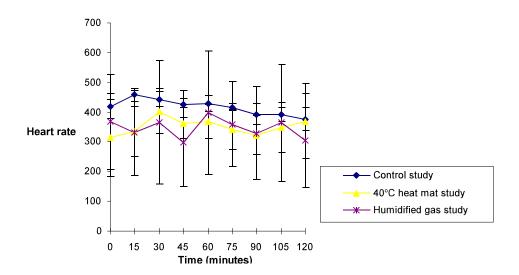
Cloacal temperature, heart and respiratory parameters were measured every 15 minutes during the studies. Cloacal temperature was measured by a thermistor probe, and validated with a standard clinical thermometer. Heart rate was obtained from Lead I of the electrocardiogram, at the speed of 50 mm per second. Respiratory rate was manually counted over one minute. Respiratory characteristics were qualitatively assessed according to the degree of chest and abdominal movements. Minute volume was measured with a ventilation monitor.

Results:

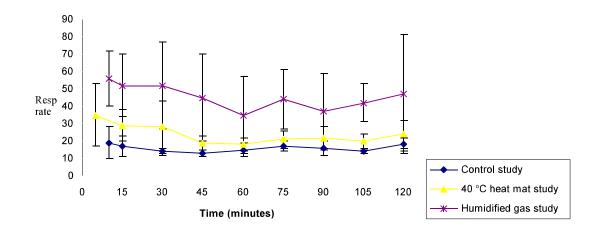
Table 1. Mean \pm SD of cloacal temperature (°C) measured by thermistor probe in four cockatoos during control, heat mat and humidified gas experiments.

Time (min)	Control	Heat Mat	Humidified Gas
0	41.2 ± 0.61	42.1 ± 0.27	42.6 ± 0.24
15	39.6 ± 1.08	41.9 ± 0.26	42.5 ± 0.25
30	39.2 ± 1.05	41.5 ± 0.31	42.2 ± 0.55
45	38.8 ± 1.06	41.2 ± 0.30	42.1 ± 0.82
60	38.0 ± 1.21	41.0 ± 0.43	42.0 ± 0.73
75	38.7 ± 1.43	40.9 ± 0.49	41.9 ± 0.74
90	38.2 ± 1.32	40.9 ± 0.53	41.8 ± 0.86
105	38.0 ± 1.32	40.9 ± 0.50	41.9 ± 0.70
120	37.8 ± 1.46	40.9 ± 0.54	41.8 ± 0.60

Mean ± SD of heart rate per minute in four cockatoos in control, heat mat and humidified gas studies



Mean \pm SD of respiratory rate per minute in four cockatoos in control, heat mat and humidified gas studies



The mean cloacal temperature decreased by 3.8°C in the control experiment, and 1.22°C and 0.78°C in the heat mat and humidified gas experiments, respectively. (Table 1) There was an average loss of 1 gram body weight in the control study, 2 grams in the heat mat study, and 3.2 grams in the humidified gas study.

Table 2. Mean \pm SD of acid-base measurements from four cockatoos in control, heat mat and humidified gas experiments.

Parameters/Hours		Control	Heat Mat	Humidified Gas
pН	0	7.5 ± 0.06	7.4 ± 0.09	7.4 ± 0.05
	1	7.3 ± 0.08	7.4 ± 0.04	7.4 ± 0.06
	2	7.1 ± 0.07	7.3 ± 0.05	7.4 ± 0.08
pCO ₂	0	42.1 ± 6.41	37.6 ± 1.41	33.3 ± 3.79
	1	67.6 ± 4.72	52.9 ± 4.72	42.0 ± 5.49
	2	96.1 ± 7.37	63.9 ± 8.89	43.7 ± 5.03
HCO ₃	0	27.1 ± 9.41	21.7 ± 4.62	21.1 ± 3.35
(mmHg)	1	32.8 ± 6.05	29.0 ± 1.79	26.2 ± 1.89
	2	32.2 ± 4.53	29.5 ± 1.88	27.2 ± 3.34
BE	0	2.5 ± 6.47	-2.7 ± 5.67	-2.1 ± 3.42
(mmol/L)	1	4.2 ± 6.65	2.8 ± 1.93	1.9 ± 2.57
	2	-0.5 ± 5.46	1.5 ± 1.8	2.4 ± 1.02

Venous blood gas measurement revealed that pH decreased by 0.32 and 0.08 in the control and heat mat experiments, respectively. (Table 2) However, the blood pH in the humidified gas study increased from 7.4 ± 0.045 at zero hour to 7.4 ± 0.076 at two hours. The partial pressure of CO₂ (pCO₂) increased from 42.14 ± 6.41 mmHg to 96.08 ± 7.37 mmHg in the control experiment, 37.56 ± 1.41 mmHg to 63.89 ± 8.89 mmHg in the heat mat study, and 33.31 ± 3.79 mmHg to 43.7 ± 5.03 mmHg in the humidified gas experiment. Bicarbonate (HCO₃) concentration increased by approximately 6mmol/L in all three experiments. Base excess (BE) decreased by 3 mmol/L in the control study, and increased by 4.16 mmol/L and 4.47 mmol/L in the heat mat and humidified gas studies, respectively. Oxygen saturation was greater than 93% in all the experiments.

Table 3. Mean \pm SD of plasma biochemistry from four cockatoos in control, heat mat and humidified gas experiments.

Parameters/Hours		Control	Heat Mat	Humidified Gas
Potassium	0	2.7 ± 0.73	3.6 ± 0	4.1 ± 0.40
(mmol/L)	1	3.1 ± 0.72	3.9 ± 0.35	4.3 ± 0.50
	2	3.6 ± 0.88	4.4 ± 0.42	4.2 ± 0.62
Calcium	0	2.3 ± 0.15	2.6 ± 0.39	2.3 ± 0.10
(mmol/L)	1	2.2 ± 0.39	2.5 ± 0.30	2.2 ± 0.13
	2	2.0 ± 0.11	2.5 ± 0.27	2.2 ± 0.15
Inorganic	0	0.7 ± 0.01	0.7 ± 0.06	0.7 ± 0.25
Phosphate	1	1.8 ± 0.31	1.9 ± 0.43	0.8 ± 0.21
(mmol/L)	2	2.3 ± 0.34	2.4 ± 0.42	1.3 ± 0.32
Total Solids	1	43.0 ± 8.89	40.0 ± 0	34.3 ± 3.40
(g/L)	2	40.3 ± 9.80	39.5 ± 2.12	31.5 ± 4.65
	3	35.0 ± 6.83	35.0 ± 1.41	29.7 ± 2.99
Albumin	0	13.7 ± 1.26	17.0 ± 0	15.3 ± 1.30
(g/L)	1	14.0 ± 2.83	16.5 ± 0.71	14.7 ± 1.26
	2	12.0 ± 2.45	15.0 ± 0	14.0 ± 2.16
Globulin	0	29.3 ± 7.37	23.0 ± 0	19.0 ± 2.90
(g/L)	1	27.0 ± 7.00	23.0 ± 1.41	16.7 ± 4.64
	2	23.0 ± 6.05	20.0 ± 1.41	15.7 ± 1.26
Lactic acid	0	-	5.1 ± 2.30	6.0 ± 2.83
(mmol/L)	1	-	1.7 ± 0.24	2.5 ± 1.68
	2	-	1.5 ± 0.20	1.7 ± 0.37

Changes occured in all the plasma electrolytes during the studies (Table 3), but they were most apparent in inorganic phosphate and potassium concentrations. Potassium concentration increased in all the studies, but there was less changes during the humidified gas study. Calcium concentration decreased in all the studies. Phosphate concentration increased in all the studies,

but less in the humidifed gas study. Total solids, albumin and globulin concentrations decreased uniformly in all the groups. Metabolites and most enzyme activities, including lactic acid, had decreased in all the experiments.

Discussion:

Attempts to maintain a normal body temperature during anaesthesia had resulted in body weight loss from dehydration. The methods by which heat, or water may be lost in these cockatoos are evaporation in the respiratory tract, conduction with the heating mat and convection. However, birds have no sweat glands. A subsequent study using just bubble plastic coverage resulted in only a slight improvement in temperature maintenence. Therefore, losses from evaporation of water in the respiratory tract is probably more important in avian species.

Phosphate concentration increased over the two hours of anaesthesia in all the studies. This increase may be a shift of ions from the intracellular compartment into the extracellular compartment. Phosphate is a weak acid and hyperphosphataemia can cause acidosis. (Whitehair *et al* 1995). Phosphate concentrations in the control and heat mat studies increased three fold, and did not change significantly in the humidified gas experiment. Therefore, the increase in phosphate concentrations may have contributed to the development of metabolic acidosis.

Potassium concentration increased in all the studies except in the humidified gas study where the initial and final potassium concentrations were almost the same. The changes in potassium concentration were directly related to the blood pH. Hyperkalaemia is known to occur in acidosis.

It is not understood why enzyme activities decreased during the two hours of anaesthesia in the studies. The total amount of blood taken was less than 1% of the cockatoos' body weight, and less than 10% of the total blood volume. The decrease in total solid, albumin and globulin concentrations and enzyme activities suggested dilution by a hypoproteineceous fluid.

There were great variations in heart rate within and between birds during the studies. The changes in heart rate did not appear to be related with the changes in plasma potassium and calcium concentrations. In these studies, the heart rate increased despite an increased potassium concentration. There was also wide variations in respiratory rates, especially in the humidified gas study. The birds in the humidified gas study had a faster respiratory rate and maintained a higher cloacal temperature than the birds in the control and heat mat studies. The heat mat group also had a faster respiratory rate and higher cloacal temperature than birds in the control study. Therefore, respiratory rate appeared to be related to the cloacal temperature.

Blood gas variables have not been well investigated in the avian species (Ritchie *et al* 1994). Verkest (1994) found pre-anaesthetic blood pH values of 7.4 ± 0.09 and 7.3 ± 0.05 in two groups of four sulphur-crested cockatoos. Assuming that the blood pH at zero hour in the current study are normal for anaesthetized birds, a pH range of 7.4 ± 0.045 was calculated and may be considered as the normal pH for these birds. Therefore, the changes seen in pH in the humidified

gas study are probably not significant. But respiratory acidosis occured in both the control and heat mat studies. The decrease in lactic acid concentration also indicated that it was not responsible for the acidosis seen in the control and heat mat studies.

Conclusion:

Both dehydration and acid-base balance are important, particularly in debilitated patients. This investigation suggested that humidified, heated anaesthetic gas is a suitable method for anaesthetising avian patients. Although these birds lost slightly more body weight than in the control or 40°C heat mat studies, there were less disturbances in body temperature, plasma biochemistry and acid-base balance. All birds recovered uneventfully. The birds panted heavily in the recovery period but no ill effect was seen within 90 days after the study.

Using heating mats alone, a temperature of 40 to 50°C is required to maintain normothermia (Lau, 1996, unpublished). However, constant monitoring and adjustment of the temperature of heating mat is essential to avoid overheating the birds. By comparison, the method of humidified anaesthetic gas is almost maintenence-free.

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