Anaesthetic Effects in Cockatoos

Kurt Verkest[*] and Lucio Filippich[*]

Cloacal Temperature Monitor

In a study to validate the use of the thermistor probe (A thermistor probe which can be used to monitor cloacal temperature continuously and can be constructed for around $15 plus the cost of a multimeter.) in clinical situations, the cloacal temperature of twenty birds was taken with a mercury-filled clinical thermometer and the thermistor probe. The thermistor probe consistently recorded a temperature which was 0-0.3°C higher than that recorded by the thermometer. This may be attributable to the smaller size of the tip of the thermistor and its lower thermal capacity, causing deeper penetration into the cloaca and less cooling of the tissues than would be caused by the thermometer.

The importance of non-specific, or supportive, therapy in avian veterinary care is widely recognised (Philip, 1981, Cannon, 1991). This has traditionally included placing the ill bird in a warm, semi-dark environment with minimal disturbance. If the goal of heat therapy is to restore body temperature to near normal, then the body temperature of the patient needs to be known initially and monitored during therapy. This would allow a rational treatment schedule to be formulated, and its effectiveness to be evaluated. Extremely low body temperatures (around 30°C) have been recorded in ill birds, which underscores the role of accurate appraisal of body temperature in birds (Verkest, 1994).

Birds presented to the University of Queensland Small Animal Hospital with suspected shock were given a clinical examination. Hypothermic were placed in a heated chamber. Cloacal temperature was monitored at the start and continuously during heat therapy using the thermistor probe. A blood sample was collected where possible and analysed for serum chemistries, especially urea, uric acid and osmolality.)

Conventional wisdom has maintained that hypothermia in birds is evident by the presence of fluffed feathers, this being a mechanism to increase the insulation properties of the feathers. This may not necessarily be the case, as budgerigars (with Megabacteria-associated disease) and a kookaburra included in the study exhibited feather fluffing behaviour, intermittently in the case of the budgerigars, but prolonged temperature recording revealed that hypothermia was not a feature of their condition. In fact, one of the budgerigars may have been hyperthermic or pyrexic (42.3°C) (Verkest, 1994). Heat therapy is contraindicated in such birds, as dehydration and heat stress may be induced or exacerbated.

It was further apparent from the data that six birds experienced a drop in body temperature of up to 4°C during and immediately after clinical examination. This supports anecdotal evidence that a thorough clinical examination is contra-indicated in a critically ill bird. It appears, then, that a bird with fluffed feathers is not necessarily hypothermic, nor necessarily in shock.

Some of the difficulties of assessing the state of shock in birds using clinical examination and serum chemistry analysis were encountered. A pelican with gastritis/peritonitis and a cloacal temperature of 31.4°C, pale mucosae and delayed filling of the ulnar vein exhibited acidosis and hypercapnia, but apparently normal serum urea, uric acid and urea:uric acid ratio. Prolonged filling time or lack of turgidity

[*] **Department of Companion Animal Medicine and Surgery, University of Queensland**

of the superficial ulnar vein are not necessarily present in birds where the laboratory data and other clinical signs are strongly suggestive of shock. A cyanotic pigeon with elevated serum urea and uric acid levels and moderate metabolic acidosis did not exhibit skin tenting, sunken eyes or delayed ulnar vein refill. Thus, a complete clinical examination, once the bird has been stabilised with appropriate supportive therapy, is essential.

Anaesthetic Study

Four Sulphur-Crested Cockatoos were twice anaesthetised, for a period of four hours, once at room temperature and once with heating supplied (heating mat underneath the bird and bubble-wrap plastic loosely covering the bird). Blood samples were collected at the start and end of this period and analysed for serum chemistries and blood gas and acid-base measurements. Cloacal temperature and heart rate were monitored continuously and half-hourly, respectively.

Temperature

The results showed that cloacal temperature decreases with anaesthesia, but this can be significantly ameliorated by providing heat. Cloacal temperature fell by an average of 4°C in unheated birds, while the temperature of heated birds initially declined by up to 1°C, but then rose by an average of 0.4°C. The increase in temperature of the heated birds suggests that care should be taken in administering heat to anaesthetised birds, as a rise in body temperature can be injurious

Laboratory Findings

Table One: Serum Chemistry Changes during Anaesthesia

Parameter	Heated Birds		Unheated Birds		Sign Diff
	Pre-An	Post-An	Pre-An	Post-An	
Chloride	121±0.4	116±14	117±5.9	115±9.7	N.S.
Osmalal	309±5.5	311±7.1	308±8.0	318±9.8	P<0.01
Sodium	153±10.1	154±12.9	147±5.1	155±8.5	N.S.
Urea:Uric	2.8±0.8	5.2±2.3	4.0±1.4	11.3±4.5	P<0.05
Urea	0.9±0.3	1.5±1.0	1.2±0.3	1.2±0.2	N.S.
Uric Acid	275±72	268±73	319±89	120±65	P<0.05

All results expressed as m±sd; urea and electrolyte expressed as mmol/l, uric acid concentrations expressed as umol/l, osmolality expressed as mosmol/kg; N.S. denotes no significant difference (Sign Diff) between heated and unheated birds in the behaviour of a parameter.

The effects of prolonged anaesthesia on serum biochemical parameters suggest that dehydration is an important effect, since the change in osmolality was accounted for mainly by an increase in sodium concentration. This hypothesis is not supported by the difference between heated and unheated birds, since it would be expected that a heated bird lose more water than an unheated one, especially in view of the increased respiratory rate observed in the heated birds.

The change in metabolite concentrations is obscure, since an increase in the U/UA may indicate a shut-

down of the renal arterial supply, causing urea concentrations to rise (due to reabsorption in the nephron) while uric acid continues to be excreted (due to continued renal portal perfusion). This may have been the case for the heated birds. However in the case of the unheated birds, the elevated ratio is due primarily to a decreased blood uric acid concentration. This could imply a decreased production of uric acid by hypothermic tissues.

Blood Gas and Acid/Base Analyses

Table Two: Blood Gas Changes During Anaesthesia

Parameter	Heated		Unheated	
	Pre-An	Post-An	Pre-An	Post-An
Hb	10±1.6	9.2±0.7	9.4±2.5	9.7±2.5
pH	7.375	7.495	7.331	7.010
pCO_2	29±5.6	36±5.3	37±5.8	140±71
pO_2	111±62	179±27	94±52	121±51
HCO_3	16±4.6	27±6.1	19±2.4	30±4.1

Results expressed in the form m±sd; partial pressures of carbon dioxide (pCO_2) and oxygen (pO_2) expressed as mmHg, bicarbonate concentration (HCO_3) as mmol/l, haemoglobin concentration expressed as g/dl.

An effect of anaesthesia that was well supported by the results was the evocation of acidosis in unheated birds, whereas heated birds experienced alkalosis. This was accompanied in all birds by an increase in blood bicarbonate concentration. The reason for this increase might be different in the two groups of birds. In the unheated group, which experienced marked hypercapnia, the increased bicarbonate is likely to be the result either of dissolved carbon dioxide being converted to carbonic acid, which dissociates to yield a bicarbonate radical, or of compensatory renal bicarbonate production. That is, the unheated birds exhibit compensated respiratory acidosis as a result of respiratory depression caused by anaesthesia. In the heated birds, the markedly increased blood oxygen and the moderate increase in blood carbon dioxide (compared to the unheated birds) suggest a less marked decrease in ventilation than was observed in unheated birds. Thus, the source of the extra bicarbonate must be renal carbonic anhydrase activity. Using isopleth curves for whole chicken blood (Tazawa, 1986), it can be seen that the increased CO_2 tension, which tends to decrease pH, does not alter blood pH as much as the increased bicarbonate concentration, which tends to increase pH. Thus, the results suggest that the heated birds are undergoing a compensated metabolic alkalosis. Respiratory alkalosis would result in reduced blood CO_2 tension which was not observed (Marder and Arad, 1989).

These results suggest that, especially during prolonged surgery, cloacal temperature should be monitored, as maintenance of core body temperature appears to minimise the physiological changes during anaesthesia. Further, monitoring temperature during anaesthesia will reduce the possibility of the development of hyperthermia and concurrent alkalosis.

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