

## Effect of Intermittent Positive Pressure Ventilation (IPPV) on Acid-Base Balance and Plasma Electrolytes during Isoflurane Anaesthesia in Sulphur-Crested Cockatoos (*Cacatua galerita galerita*)

Saul Chemonges

University of Queensland, Critical Care Research Group Laboratory, 3rd Floor, Clinical Sciences Building, the Prince Charles Hospital, Rode Road, Chermside, QLD 4032, Australia

**Abstract:** The objective of the study was to investigate the effect of Intermittent Positive Pressure Ventilation (IPPV) on acid-base balance and plasma electrolytes during isoflurane anaesthesia in sulphur-crested cockatoos (*Cacatua galerita galerita*). Anaesthesia was induced in six birds by mask using a T-piece with 3.0% isoflurane. Blood gases, plasma electrolytes, PCV and Total Protein (TP) were monitored for one hour during Spontaneous Ventilation (SV) and IPPV. IPPV was instituted by engaging the pop-off valve (IPPVa) of the circle absorber or by squeezing the breathing bag (IPPVb). Results showed that during SV, pCO<sub>2</sub>, pO<sub>2</sub>, [HCO<sub>3</sub><sup>-</sup>], BE, C+CO<sub>2</sub> and PO<sub>4</sub><sup>-</sup> increased significantly, while [Na<sup>+</sup>], [K<sup>+</sup>] and [Ca<sup>2+</sup>] did not change significantly. During IPPV, pCO<sub>2</sub> and pO<sub>2</sub> decreased, while C+CO<sub>2</sub> increased during the initial 30 min. [HCO<sub>3</sub><sup>-</sup>] increased during IPPVa only in the first 30 min. BE increased only in the first 30 min of IPPV. There was a marginal increase and decrease in PO<sub>4</sub><sup>-</sup> during SV and IPPV, respectively. [Na<sup>+</sup>], [K<sup>+</sup>] and [Ca<sup>2+</sup>] remained stable during both SV and IPPV. Subtle decreases were noted for [Cl<sup>-</sup>], TP and PCV during SV. It was concluded that mixed metabolic and respiratory acidosis occurs during SV in isoflurane-anaesthetised cockatoos. Metabolic acidosis that develops during isoflurane anaesthesia in spontaneously ventilating birds is reversible to some extent by IPPV, possibly through a mixed acidosis-alkalosis, respiratory alkalosis and a non-respiratory contribution to alkalosis mechanism. Reversal of Bohr Effect occurs during IPPV in isoflurane-anaesthetised cockatoos. Studies are indicated to understand the causes of decreased oxygen saturation in apparently alkalotic birds.

**Keywords:** Acid-base, anaesthesia, cockatoos, IPPV, isoflurane, sulphur-crested

### INTRODUCTION

It is common for veterinarians to be presented with an avian patient that requires general anaesthesia for procedures including complete physical examination, venipuncture, diagnostic workup or medical treatments (Chemonges and Filippich, 1999). As in mammals, plasma electrolyte levels in birds are of concern in every candidate for anaesthesia (Sinn, 1994). Evaluation of acid-base status, electrolyte levels, blood gas analysis and the monitoring of these parameters in anaesthetised birds is essential to be able to recognise and promptly correct any complications that may affect normal body functioning (Chemonges and Filippich, 1999; Jaensch *et al.*, 2001).

For a complete evaluation of acid-base disorders, plasma sodium (Na<sup>+</sup>), potassium (K<sup>+</sup>), chloride (Cl<sup>-</sup>), Total Protein (TP), inorganic phosphate (PO<sub>4</sub><sup>-</sup>), blood gas analysis with hydrogen ion concentration (pH), partial pressure of carbon dioxide (pCO<sub>2</sub>), total carbon dioxide (C+CO<sub>2</sub>), bicarbonate (HCO<sub>3</sub><sup>-</sup>) and Base Excess (BE) levels are required (Bouda *et al.*, 2009; Russell *et al.*, 1996). Evaluation of acid-base status is accomplished by the analysis of blood gases and the application of the Henderson-Hasselbach equation to

describe the relationship between pH, pCO<sub>2</sub> and HCO<sub>3</sub><sup>-</sup> (Russell *et al.*, 1996). Total carbon dioxide is used to classify acid-base imbalances (Russell *et al.*, 1996; Steinmetz *et al.*, 2007). A significant decrease in C+CO<sub>2</sub> indicates metabolic acidosis while an increase is a mixed acid-base imbalance (Bailey and Pablo, 1998). The relative concentration of chloride in extra cellular fluid affects acid-base status, and changes in bicarbonate can occur if chloride, an independent variable, is altered (Russell *et al.*, 1996).

Plasma protein and various electrolytes including phosphate serve as physiological buffers (Bailey and Pablo, 1998; Steinmetz *et al.*, 2007). Changes in [Na<sup>+</sup>] can have a significant impact on plasma osmolality (Marks and Taboada, 1998). Kidneys regulate [Na<sup>+</sup>] and water balance in the body (Di Bartola, 1998). Potassium plays a critical role in neuromuscular transmission. Imbalances in [K<sup>+</sup>] manifest as skeletal muscle dysfunction (Phillips and Polzin, 1998). Calcium is a regulatory ion and plays a role in neuronal excitability, muscle contraction and blood coagulation (Dhupa and Proulx, 1998). Phosphorous is the major intracellular anion existing in organic or inorganic phosphate and plays an integral role in many metabolic processes (Rosol and Capen, 1996).

Endotracheal intubation and the use of IPPV is recommended in anaesthetised birds (Edling *et al.*, 2001; Sedgwick, 1980); however, there is little information in the literature regarding the effect of IPPV on plasma electrolytes during isoflurane anaesthesia. The objective of this study was to evaluate the acid-base balance and plasma electrolyte changes during isoflurane anaesthesia in sulphur-crested cockatoos undergoing Spontaneous Ventilation (SV) and IPPV. Isoflurane was used in this study because it is the commonly used anaesthetic agent in birds (Chemonges and Filippich, 1999; Curro, 1998; Heard, 1997a, b).

## MATERIALS AND METHODS

Six sulphur-crested cockatoos (*Cacatua galerita galerita*) were subjected to the experiments. The University of Queensland Animal Ethics and Experimentation Committee (Permit No. CAS/118/98/D) and the Department of Environment (Permit No. E4/000229/98/SAA).

The study was conducted at Veterinary Teaching Hospital, School of Veterinary Science, the University of Queensland St. Lucia in Australia. Experiments were conducted in a room preheated with an electric air heater to 28°C. Anaesthesia was induced using a T-piece and 3.0% isoflurane (Forthane® Abbott, Australia), before connecting the bird to a circle absorber (CIG, Australia). After allowing the anaesthesia to stabilise for 5 min, zero (0) time was recorded. The bird was then placed in dorsal recumbency, kept warm using an electric heating pad (Breville®, China) and covered with bubble plastic to prevent heat loss. The temperature of the heat pad was maintained between 40 and 45°C.

The right thigh of the bird was depilated and a paediatric digital blood pressure inflatable cuff (Hokanson, WA) was placed around the proximal third of the tibio-tarsal region. A Doppler probe was wetted with a conductive gel (SS250C®, Seal Systems, Australia) and secured distally to the inflatable cuff over the medial, mid tibiotarsal region with a cohesive flexible bandage (Co-flex®, Andover, US) to measure blood pressure by an ultrasonic Doppler flow detector (811-b, USA). The heart rate was determined by counting the pulse sound amplified by the partially inflated cuff of the Doppler flow detector. Cloacal temperature was read from a clinical mercury thermometer (Nesco®, Stubby C., Japan). The respiratory rate was determined by counting abdominal movements during spontaneous ventilation.

A 23 gauge indwelling IV catheter was placed and secured into the left jugular vein. Immediately before a blood sample was collected, the catheter was flushed by drawing blood into a separate 1-mL syringe and then back into the vein five times. Blood (1.5 mL) was collected into a 2.5 mL heparinized syringe at time 0, 30 and 60 min. Blood for PCV and Total Protein (TP) determination was immediately placed into a microhaematocrit tube and centrifuged. The PCV was read directly and TP was read by a hand-held refractometer. About 1-mL of blood was put in a serum separator tube (Vacutainer®, UK) and centrifuged for 3 min at 2,500 revolutions/min before being submitted to the laboratory for measurement of plasma electrolytes. A heparinized syringe containing 0.5 mL of blood was capped, placed in ice, and blood gas and acid base parameters measured within 30 min using a blood gas machine (Chiron Diagnostics, Australia). Blood gas analysis was also measured using a portable blood gas machine (i-STAT® System, Abbott) in some studies.

Each bird was subjected to three different 60-min anaesthetic experimental sets in a crossover pattern with three days between each set of the experiments. Each set of experiments involved anaesthesia when birds Spontaneously Ventilated (SV) and with two types of IPPV, designated as IPPVa and IPPVb. During IPPVa, the bird was subjected to intermittent pressure ventilation of 4 cmH<sub>2</sub>O at 12 breaths/min by engaging the pop-off valve of the anaesthetic machine to create positive pressure ventilation within the breathing circuit. The positive pressure was read from the manometer of the IPPV machine (Campbell Respirator®, Australia). During IPPVb, the bird was subjected to pressure at 12 breaths/min by gently squeezing the 500 cm<sup>3</sup>-breathing bags (Ohmeda®, Australia) between the hand and the thumb until the bird's abdomen rose, and the bag was then released.

After each set of experiments, the intravenous catheter was removed, the endotracheal tube cuff deflated and the bird was disconnected from the anaesthetic machine and weighed. The bird was wrapped in a towel and placed in a bird cage to recover.

## RESULTS

The mean body weight of the birds subjected to SV experiments was 826.5±53.1 g. The respiratory rate progressively decreased over time. Respiratory rate readings taken at 15 min intervals differed significantly ( $p<0.05$ ) during the hour of anaesthesia in the spontaneously ventilating birds (Table 1). The mean heart rate did not change initially but had significantly

Table 1: Mean values  $\pm$ S.D. for Respiratory Rate (RR), Blood Pressure (BP), Heart Rate (HR) and cloacal temperature (Temp) in 6 cockatoos during spontaneous ventilation with 3.0% isoflurane anaesthesia

Time (min)	RR (breaths/min)	HR (beats/min)	BP (mmHG)	Temp ( $^{\circ}$ C)
0	66.7 $\pm$ 53	245 $\pm$ 49	173.5 $\pm$ 23	40.5 $\pm$ 0.2
15	62.7 $\pm$ 42	245 $\pm$ 46	176.8 $\pm$ 28	40.4 $\pm$ 0.4
30	43.3 $\pm$ 27	231 $\pm$ 42	178.7 $\pm$ 28	40.0 $\pm$ 0.5
45	46.7 $\pm$ 32	225 $\pm$ 32	175.0 $\pm$ 27	39.7 $\pm$ 0.7
60	38.7 $\pm$ 30	170 $\pm$ 93	181.3 $\pm$ 33	39.4 $\pm$ 0.6

Table 2: Mean values $\pm$ S.D. for Packed Cell Volume (PCV) and Total Plasma protein (TP) in 6 cockatoos during spontaneous ventilation with 3.0% isoflurane anaesthesia

Time (min)	PCV (%)	TP (g/L)
0	44 $\pm$ 2.3	35 $\pm$ 3.7
30	42 $\pm$ 3.7	35 $\pm$ 4.0
60	42 $\pm$ 2.5	33 $\pm$ 3.0

( $p < 0.05$ ) decreased by 60 min, while blood pressure remained steady. Cloacal temperature decreased, but not significantly during SV.

The PCV and Total Protein (TP) did not change significantly during SV, although TP decreased significantly by 2.17 g/L during the 30 to 60 min period (Table 2).

During SV, mean blood pH and haemoglobin O<sub>2</sub> saturation did not change significantly during the 60 min of anaesthesia, although pCO<sub>2</sub> and pO<sub>2</sub> progressively and significantly ( $p < 0.05$ ) increased. The mean Base Excess (BE) changed significantly ( $p < 0.05$ ) in the first 30 min, then dropped thereafter. Overall, there was a significant ( $p < 0.05$ ) increase in blood gas and acid-base values (Table 3).

Although mean values for plasma [Na<sup>+</sup>, [K<sup>+</sup>, [Cl<sup>-</sup>] and Ca<sup>2+</sup> did not significantly change, mean serum PO<sub>4</sub><sup>-</sup> progressively and significantly ( $p < 0.05$ ) rose from 0.6 $\pm$ 0.2 mmol/L at 0 min to 1.3 $\pm$ 0.3 mmol/L at 60 min of SV anaesthesia (Table 4).

The PCV remained steady throughout IPPVa, however, during IPPVb, PCV progressively dropped (Table 5). The PCV during IPPVa did not differ

significantly from those of IPPVb. The total plasma protein progressively decreased during IPPVa and IPPVb. The change in total plasma protein was not significant for IPPVa, but was significant ( $p < 0.05$ ) at 0, 30, and 60 min during IPPVb.

The pH during IPPVa and IPPVb were not significantly different. The pH increased in the first 30 min and remained steady thereafter for both IPPVa and IPPVb. There was a significant decrease in pCO<sub>2</sub> ( $p < 0.05$ ) during the initial 30 min of IPPVb. The pCO<sub>2</sub> decreased significantly ( $p < 0.01$ ) in the initial 30 min; however, the changes were not significantly different after 30 and 60 min for either IPPVa or IPPVb. The C+CO<sub>2</sub> increased after 30 and 60 min during IPPVa; however, it did not change significantly during IPPVb. The HCO<sub>3</sub><sup>-</sup> increased during IPPVa in the first 30 min and did not change significantly thereafter. HCO<sub>3</sub><sup>-</sup> remained steady during IPPVb. The O<sub>2</sub> saturation did not change during IPPVa; however, during IPPVb it declined progressively, though not significantly. The O<sub>2</sub> saturation for IPPVb after 60 min was much lower than that of IPPVa at the same time. Overall, there was a rise in pH and BE, whereas there was a fall in pCO<sub>2</sub> and O<sub>2</sub> saturation during the 60 min (Table 6).

Total CO<sub>2</sub> decreased, but not significantly for blood gas readings taken by the i-Stat blood machine (Table 7). Although the pH did not change significantly, there was a significant difference ( $p < 0.05$ ) between the pCO<sub>2</sub> at 0, 30 and 60 min, whereby, it decreased significantly during the first 30 min and remained steady after 30 and 60 min. pO<sub>2</sub>, decreased significantly ( $p < 0.05$ ) during the first 30 min. HCO<sub>3</sub><sup>-</sup>, C+CO<sub>2</sub> and PCO<sub>2</sub> decreased during the first 30 min and steadied thereafter during IPPV. Blood gas changed significantly between the two readings at 0 and 30 min and the difference was significant ( $p < 0.01$ ). Overall, there was a rise in pH, and BE, and a fall in C+CO<sub>2</sub>, pCO<sub>2</sub> and O<sub>2</sub> saturation during the hour of IPPV.

Table 3: Mean values $\pm$ S.D. for venous pH, carbon dioxide partial pressure (pCO<sub>2</sub>), oxygen partial pressure (pO<sub>2</sub>), bicarbonate (HCO<sub>3</sub><sup>-</sup>), Base Excess (BE), total carbon dioxide (C+CO<sub>2</sub>) and haemoglobin oxygen saturation (O<sub>2</sub> sat) during spontaneous ventilation in 6 cockatoos and 3.0% inhaled isoflurane anaesthesia

Time (min)	pH	PCO <sub>2</sub> (mmHg)	pO <sub>2</sub> (mmHg)	C+CO <sub>2</sub> mmol/L	HCO <sub>3</sub> <sup>-</sup> mmol/L	BE (mmol/L)	O <sub>2</sub> Sat. (%)
0	7.4 $\pm$ 0	38.3 $\pm$ 5	101.8 $\pm$ 12	25.5 $\pm$ 1.8	24.3 $\pm$ 1.6	0.30 $\pm$ 1	98 $\pm$ 0.6
30	7.4 $\pm$ 0	52.4 $\pm$ 7	114.8 $\pm$ 9	31.4 $\pm$ 1.8	29.8 $\pm$ 1.7	11.2 $\pm$ 2	98 $\pm$ 0.2
60	7.4 $\pm$ 0	61.8 $\pm$ 14	120.9 $\pm$ 56	33.4 $\pm$ 1.8	31.5 $\pm$ 3.4	3.7 $\pm$ 1.8	99 $\pm$ 0.4

Table 4: Mean plasma electrolyte concentration of sodium (Na<sup>+</sup>), potassium (K<sup>+</sup>), chloride (Cl<sup>-</sup>), phosphorous (PO<sub>4</sub><sup>-</sup>) and calcium (Ca<sub>2+</sub>)  $\pm$ S.D. during 3.0% inhaled isoflurane anaesthesia in 6 cockatoos during spontaneous ventilation

Time (min.)	Na <sup>+</sup> (mmol/L)	K <sup>+</sup> (mmol/L)	Cl <sup>-</sup> (mmol/L)	PO <sub>4</sub> <sup>-</sup> (mmol/L)	Ca <sub>2+</sub> * (mmol/L)
0	145 $\pm$ 1.6	4 $\pm$ 0.3	118 $\pm$ 4	0.6 $\pm$ 0.2	1.1 $\pm$ 0.4
30	146 $\pm$ 2.2	4 $\pm$ 0.2	115 $\pm$ 4	0.9 $\pm$ 0.2	0.9 $\pm$ 0.1
60	147 $\pm$ 1.6	4 $\pm$ 0.1	116 $\pm$ 3	1.3 $\pm$ 0.3	1.0 $\pm$ 0.1

\*: Adjusted to pH 7.4

Table 5: Mean Packed Cell Volume (PCV) and Total Protein (TP)  $\pm$ S.D. during anaesthesia in 6 cockatoos during intermittent positive pressure ventilation generated by engaging the pop-off valve (IPPVa) and by squeezing the breathing bag (IPPVb) at 12 respirations/min at 3.0% inhaled isoflurane

Time (min)	PCV (%)		TP (g/L)	
	IPPVa	IPPVb	IPPVa	IPPVb
0	44 $\pm$ 3	44.5 $\pm$ 2	36.7 $\pm$ 3	36.7 $\pm$ 4
30	44 $\pm$ 3	43 $\pm$ 2	36.8 $\pm$ 4	34.8 $\pm$ 3
60	44 $\pm$ 3	42 $\pm$ 3	35.3 $\pm$ 4	33.5 $\pm$ 3

Table 6: Mean values $\pm$ S.D. for venous hydrogen ion concentration (pH), carbon dioxide partial pressure (pCO<sub>2</sub>), oxygen partial pressure (pO<sub>2</sub>), bicarbonate (HCO<sub>3</sub><sup>-</sup>), Base Excess (BE), total carbon dioxide (C+CO<sub>2</sub>) and haemoglobin oxygen saturation (O<sub>2</sub> Sat) during intermittent positive pressure ventilation generated by occluding the pop-off valve (IPPVa) and by squeezing the breathing bag (IPPVb) at 12 respirations/min at 3.0% inhaled isoflurane in 100% oxygen

Time (min)	pH		pCO <sub>2</sub> (mmHg)		pO <sub>2</sub> (mmHg)		C+CO <sub>2</sub> (mmol/L)		HCO <sub>3</sub> <sup>-</sup> (mmol/L)		BE (mmol/L)		O <sub>2</sub> Sat. (%)	
	a	b	a	b	a	b	a	b	a	b	a	b	a	b
0	7.4 $\pm$ 0	7.4 $\pm$ 0	37.3 $\pm$ 6	40 $\pm$ 2	110 $\pm$ 24	104 $\pm$ 7	23 $\pm$ 4	26 $\pm$ 1	22 $\pm$ 3	24 $\pm$ 1	-2.8 $\pm$ 3	-0.2 $\pm$ 2	98 $\pm$ 1	98 $\pm$ 0.2
30	7.5 $\pm$ 0	7.6 $\pm$ 0	33.1 $\pm$ 3	25 $\pm$ 3	72 $\pm$ 9	64 $\pm$ 8	27 $\pm$ 2	25 $\pm$ 1	26 $\pm$ 2	24 $\pm$ 1	3 $\pm$ 2	3.7 $\pm$ 2	96 $\pm$ 1	96 $\pm$ 1
60	7.5 $\pm$ 0	7.6 $\pm$ 0	34.3 $\pm$ 4	26 $\pm$ 6	82 $\pm$ 12	59 $\pm$ 6	27 $\pm$ 2	25 $\pm$ 3	26 $\pm$ 2	24 $\pm$ 2	3.6 $\pm$ 2	4 $\pm$ 1	97 $\pm$ 1	94 $\pm$ 15

Table 7: Mean values $\pm$ S.D. for venous hydrogen ion concentration (pH), carbon dioxide partial pressure (pCO<sub>2</sub>), oxygen partial pressure (pO<sub>2</sub>), bicarbonate (HCO<sub>3</sub><sup>-</sup>), Base Excess (BE), total carbon dioxide (C+CO<sub>2</sub>) and hemoglobin oxygen saturation (O<sub>2</sub> sat) read from the i-Sat<sup>®</sup> portable blood gas machine during intermittent positive pressure ventilation generated by squeezing the breathing bag (IPPVb) at 12 respirations/min at 3.0% inhaled isoflurane in 100% oxygen at a flow rate of 2.0 L/min

Time (min)	pH	pCO <sub>2</sub> (mmHg)	pO <sub>2</sub> (mmHg)	C+CO <sub>2</sub> (mmol/L)	HCO <sub>3</sub> <sup>-</sup> (mmol/L)	BE (mmol/L)	O <sub>2</sub> Sat. (%)
0	7.4 $\pm$ 0	40 $\pm$ 4	103 $\pm$ 13	23.8 $\pm$ 1	23 $\pm$ 0.8	-2.7 $\pm$ 1	98 $\pm$ 0.8
30	7.6 $\pm$ 0	22.6 $\pm$ 3	62 $\pm$ 10	21.0 $\pm$ 2	21 $\pm$ 1.6	-1.5 $\pm$ 2	94 $\pm$ 2.7
60	7.6 $\pm$ 0	23.6 $\pm$ 6	58 $\pm$ 8	21.0 $\pm$ 3	21 $\pm$ 3.0	-1.7 $\pm$ 2	93 $\pm$ 2.5

Table 8: Mean $\pm$ S.D. for blood electrolytes: sodium (Na<sup>+</sup>), potassium (K<sup>+</sup>), chloride (Cl<sup>-</sup>), phosphorous (PO<sub>4</sub><sup>-</sup>) and calcium (Ca<sup>2+</sup>) during anaesthesia in 6 cockatoos during positive pressure ventilation by occluding the pop-off valve (IPPVa) and by squeezing the breathing bag (IPPVb) at 12 respirations/min at 3.0% inhaled isoflurane

Time (min)	Na <sup>+</sup> (mmol/L)		K <sup>+</sup> (mmol/L)		Cl <sup>-</sup> (mmol/L)		PO <sub>4</sub> <sup>-</sup> (mmol/L)		Ca <sup>2+</sup> (mmol/L)*	
	a	b	a	b	a	b	a	b	a	b
0	146 $\pm$ 2	147 $\pm$ 2	4 $\pm$ 0.3	3.7 $\pm$ 0.3	120 $\pm$ 3	118 $\pm$ 1.2	0.6 $\pm$ 0.2	0.6 $\pm$ 0.3	0.9 $\pm$ 0	1.0 $\pm$ 0
30	146 $\pm$ 1	145 $\pm$ 2	4.3 $\pm$ 0.2	4.0 $\pm$ 0.3	119 $\pm$ 2	118 $\pm$ 1.2	0.5 $\pm$ 0.2	0.4 $\pm$ 0.2	1.0 $\pm$ 0	1.1 $\pm$ 0
60	146 $\pm$ 1	146 $\pm$ 2	4.4 $\pm$ 0.2	4.0 $\pm$ 0.2	119 $\pm$ 2	118 $\pm$ 0.5	0.6 $\pm$ 0.2	0.4 $\pm$ 0.1	0.9 $\pm$ 0.6	1.0 $\pm$ 0

\*: Adjusted to pH 7.4

Although there was a rise in PO<sub>4</sub><sup>-</sup> in IPPVa and a decrease during IPPVb, the change was not significant. Overall, serum electrolytes did not change significantly during the IPPV periods, except for a marginal increase in PO<sub>4</sub><sup>-</sup> during IPPVb (Table 8).

## DISCUSSION

The value of plasma biochemistry, TP and PCV measurements as complementary tools in the diagnosis of disease and in monitoring the progress of a clinical condition of birds is widely recognized (Polo *et al.*, 1998; Roskopf *et al.*, 1982; Woerpel and Roskopf, 1984). In this study, PCV was in the same range as reported for other cockatoos (Polo *et al.*, 1998). Cockatoos, however, have been reported to have the lowest normal PCV range of the psittacine species (Woerpel and Roskopf, 1984). The slight drop in PCV and TP observed in this study was probably due to blood loss from repetitive blood sampling.

Maintenance of plasma pH within a narrow range is critical to survival. In health, acid-base status is maintained through a complex regulation of hydrogen

ion concentration and is accomplished by buffer systems in plasma and blood responses of the respiratory and renal systems (Russell *et al.*, 1996). During SV the pH remained constant despite an increase in pCO<sub>2</sub>, implying that there was a metabolic acidosis that was buffered. The increase in pH during IPPV indicated metabolic alkalosis.

The pCO<sub>2</sub> was higher than the normal values reported in other studies (Sinn, 1994). The decrease in pCO<sub>2</sub> during IPPV means birds were better ventilated and tended to be hypocapnoeic. The pCO<sub>2</sub> during IPPVb had a faster pCO<sub>2</sub> decrease compared to IPPVa, suggesting that squeezing the breathing bag was more efficient at decreasing pCO<sub>2</sub> than engaging the pop off valve during assisted ventilation. Venous blood samples used to assess blood gases may not have reflected the level of oxygenation as compared to arterial blood, although studies suggest that the jugular vein is appropriate for blood sampling (Cooper, 1988).

The increase in pCO<sub>2</sub> and bicarbonate without change in pH during SV suggests that there was a

mixed respiratory acidosis and metabolic alkalosis. Although serum  $\text{HCO}_3^-$  decreased after the start of IPPV for blood that was analyzed immediately after collection (i-STAT<sup>®</sup> blood gas machine), the steady  $\text{HCO}_3^-$  concentration during IPPV was probably due to an acidosis-alkalosis equilibrium that had been reached with a marginal shift towards alkalosis, as pH did not change.

Base excess is used to determine the presence of metabolic acidosis or alkalosis (Russell *et al.*, 1996; Steinmetz *et al.*, 2007). Positive values of BE indicate a deficit in non-carbonic acids and negative values indicate an excess of non-carbonic acids (Russell *et al.*, 1996). An increase in BE above normal range implies respiratory acidosis and a decrease below normal range implies metabolic acidosis (Bailey and Pablo, 1998). Base excess is used to assess the non-respiratory component of acid-base disorder. In this study, during SV, the large increase in BE in the first 30 min of anaesthesia suggests respiratory acidosis which later dropped, probably due to metabolic acidosis. This observation confers with other studies that metabolic acidosis occurs in spontaneously ventilating birds during anaesthesia (Phalen *et al.*, 1997). The metabolic acidosis would probably increase during prolonged periods of anaesthesia. Base excess increased during IPPV, suggesting that there was a contribution to respiratory acidosis. The duration between collection and blood gas analysis could have contributed to the higher BE values for blood analyses by the Chiron blood gas machine compared with the i-STAT<sup>®</sup> blood gas machine. The negative BE values at the time of first blood sampling during IPPV, and the subsequent shift towards positive values, implies that the birds became metabolically acidotic soon after induction of anaesthesia. It is therefore evident that IPPV reversed the further development of metabolic acidosis because of the gradual positive shift in BE-an indication of respiratory acidosis.

Most laboratories measure the total  $\text{CO}_2$  levels as indicators of bicarbonate levels. Normal values of the total  $\text{CO}_2$  levels are available for many species which are primarily used for determining the patient's acid base status. Increased bicarbonate levels indicate metabolic alkalosis and decreased levels indicate metabolic acidosis. The increase in  $\text{C}+\text{CO}_2$  during SV suggests a metabolic alkalosis. The  $\text{C}+\text{CO}_2$  was constant during IPPV probably because the  $\text{HCO}_3^-$  was steady, as  $\text{HCO}_3^-$  and  $\text{C}+\text{CO}_2$  have a fixed ratio. For this reason,  $\text{C}+\text{CO}_2$  is used to estimate  $\text{HCO}_3^-$  (Bailey and Pablo, 1998).

The  $\text{pO}_2$  was lower than the normal arterial values of birds reported in other studies (Desmarchelier *et al.*,

2007; Edling, 2003; Sinn, 1994; Steinmetz *et al.*, 2007). Since venous  $\text{pO}_2$  is usually lower than the arterial  $\text{pO}_2$ , the  $\text{pO}_2$  in this study was probably within the normal range for cockatoos. The gradual increase in  $\text{pO}_2$  during spontaneous ventilation was most likely an attempt to correct the initial hypoxaemia that may have occurred during restraint and struggling at the time of anaesthetic induction. The decrease in  $\text{pO}_2$  during IPPV could have clinical implications because the avian oxygen dissociation curve resembles that of mammals (Heard, 1997b). Haemoglobin affinity for oxygen is affected by pH, temperature and some organic phosphate compounds. Acid-base status influences the organic/inorganic phosphate equilibrium; decreased pH favours the formation of organic phosphate, and increased pH favours formation of the measured inorganic phosphate (Rosol and Capen, 1996). Respiratory alkalosis causes hypophosphataemia (Rosol and Capen, 1996). High concentrations of inorganic phosphate prevent the release of oxygen from oxyhaemoglobin (Rosol and Capen, 1996). In the present study, the gradual increase in serum phosphorous during SV is consistent with metabolic acidosis. Intermittent positive pressure ventilation caused a decrease in serum  $\text{PO}_4^-$  (an indication of alkalosis) and this was more marked during IPPVb. Plasma phosphate was; however, lower than that of studies in other psittacine birds (Polo *et al.*, 1998).

Since the pH tended to increase during IPPV, the reduced affinity of haemoglobin for oxygen with decreased pH (Bohr Effect) was expected to occur. Although the haemoglobin oxygen saturation decreased and paralleled the  $\text{pO}_2$  during IPPV, this change was not expected as the pH also increased. Oxygen saturation is expected to be higher (left displacement of the oxygen dissociation curve) at high pH. Therefore, the reversal of the Bohr Effect was observed during IPPV. This observation is unusual and further studies are suggested to understand the causes of the decreased oxygen saturation in apparently alkalotic birds.

Other than the differences likely to be caused by a delay in blood sample analysis for  $\text{HCO}_3^-$  and BE, the i-STAT and Chiron blood gas machines gave similar readings for other blood gas parameters. The time between collection and measurement is critical for  $\text{HCO}_3^-$  and BE indices. It is thus important to perform blood gas analysis immediately after collection.

Plasma  $[\text{Na}^+]$ ,  $[\text{K}^+]$ ,  $[\text{Ca}^{2+}]$  and  $[\text{Cl}^-]$  values fell within the ranges published for other studies (Jaensch *et al.*, 2001, 2002; Woerpel and Roskopf, 1984) and other psittacine birds (Polo *et al.*, 1998). Apart from change in  $\text{PO}_4^-$ , plasma electrolyte during SV and IPPV were comparable. Again, this implies that IPPV reversed the effects of metabolic acidosis, and IPPV

instituted by squeezing the breathing bag caused a greater change in reversing metabolic acidosis.

The progressive decrease in plasma chloride during spontaneous ventilation in anaesthetised cockatoos suggests that there was a tendency to hypochloraemic acidosis (Bailey and Pablo, 1998). This further underscores the development of metabolic acidosis during anaesthesia and is in agreement with other observations (Phalen *et al.*, 1997).

### CONCLUSION

In conclusion, during isoflurane anaesthesia in spontaneously ventilating cockatoos, birds become metabolically acidotic soon after induction of anaesthesia. Intermittent positive pressure ventilation facilitates the reduction in pCO<sub>2</sub> and reverses the metabolic acidosis by causing a mixed acidosis-alkalosis, respiratory alkalosis and a non-respiratory contribution to alkalosis. Artifactual increase in base excess that mimics respiratory acidosis happens if there are delays in blood gas analysis. Within an hour of isoflurane anaesthesia, sodium potassium and calcium changes are minimal. Reversal of the Bohr Effect occurs during intermittent pressure ventilation. Further studies are indicated in order to understand the causes of decreased haemoglobin oxygen saturation in apparently alkalotic birds.

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