Blood Gas Analysis of Raptors

Kurt Verkest[1], Peter Frater[2] and Lilliana Frater[2]

Blood Gas Analysis of Raptors

Blood gas analysis is recognised as a useful technique in the clinical assessment of in human and other mammals. Lung function, renal perfusion and function, tissue perfusion and electrolyte status can be assessed through knowledge of a patientÆs acid/base and blood gas status. Thus, blood gas analysis has found a place in the monitoring of anaesthesia, fluid therapy and cardio-respiratory function. Whilst arterial blood gas analysis provides information about respiratory compensation not provided by venous samples, central venous blood gas analysis provides information about tissue oxygenation, bicarbonate status and acid-base balance (Hauptman and Chaudry, 1993).

This paper seeks to establish the possibility of using blood gas analysis in avian medicine, both as a diagnostic tool and to assess therapeutic regimens. Suggested fluid and heat therapy protocols are more numerous and varied than the papers in which they appear (Redig, 1984; Martin and Kollias, 1989; Cannon, 1991; Philip 1981). Heat therapy can easily be assessed using an unobtrusive cloacal thermometer linked to a continuous monitoring device (Verkest, 1995). Likewise, fluid therapy can be monitored through the judicious use of serial blood gas analysis.

Whilst the cost of obtaining and maintaining an acid base laboratory is too onerous a burden for most veterinary practices, it is often possible to make use of a locally available blood gas analyser. This study was undertaken to assess the changes that occur in an avian blood sample with respect to blood pH and blood gases.

Materials and Methods

The birds in this study were clinically healthy birds of the following species; four Peregrine Falcons (*Falco peregrinus*), four Square-tailed Kites (*Lophoictinia isura*), one White-breasted *Sea-eagle (Haliaeetus leucogaster*) and one Wedge-tailed Eagle (*Aquila audax*). They were housed in outdoor accommodation near Gympie, Qld, and were either in the process of rehabilitation or permanently incapacitated. Birds were caught early in the

[1] South Beech Veterinary Surgery, 40 Southend Road, Wickford, Essex, SS11 8DU, England

[2] 20 Cliff Jones Court, Curra via Gympie, QLD 4570

morning and were transported to Gympie General Hospital where they were held in ventilated cardboard boxes in an air conditioned room for at least 30 minutes prior to blood sampling. Birds were fasted for 36 hours prior to transportation. Each bird was sampled once, except the White-breasted Sea-eagle, which was sampled twice.

Blood samples (between 1 and 2 ml) were collected, as part of a routine health check, from the superficial ulnar vein of conscious, unsedated birds into heparinised plastic syringes (Preza-Pak II 2.5ml Arterial Blood Sampler, Terumo Corporation, Tokyo, Japan). Haematoma formation was prevented by digital pressure for at least 60 seconds after venipuncture. The sample was mixed gently in the syringe and air bubbles were expelled. A portion of each sample was centrifuged and submitted for blood biochemical analysis. The remainder of the sample was processed by an Acid Base Laboratory (ABL330, Radiometer, Copenhagen) within 30 seconds of collection and at intervals of 15 minutes up to two hours after sample collection. A drop of blood was expelled from the syringe prior to introduction of a sample into the machine.

The results of blood gas analysis were accepted where error messages or question marks were not displayed with the results. Early results indicated that avian blood samples required an additional rinse cycle to be performed, or that rinse solution be introduced to the machine in place of a sample, between samples.

Results of the measurements at 30 seconds and 60 minutes after sampling were analysed for significance using Student's t-test for paired observations. Significance was assumed when $p < 0.05$.

Results

N.B. As results are rounded to the nearest significant figure, stated average increases and decreases do not always match the change in averages. ôSignificantö is used to mean statistically significant at the 95% level of significance.

Blood pH decreased, significantly, by an average of 0.02 pH units, from 7.45 to 7.43 pH units. Carbon dioxide partial pressure (pCO_2) increased significantly, by an average of 2.4 mmHg from 32 mmHg to 34.3 mmHg. Bicarbonate (HCO_3) concentration increased an average of 0.4 mmol/L from 22mmol/L to 22.5 mmol/L. This change was not significant. Calculated total carbon dioxide increased, but not significantly, by 0.6 mmol/L from 23 to 23.5 mmol/L. Blood oxygen partial pressure (pO_2) decreased significantly, by an average of 7.7 mm Hg from 62.6 to 55 mm Hg.

Discussion

The above results show that avian blood changes during storage in terms of its oxygen and carbon dioxide partial pressures and in its pH. The direction of the changes suggests metabolic activity. The changes in gas partial pressures cannot be explained by diffusion, since the sample pO_2 declined with time but was in all cases lower than ambient pO_2 (app.

150 mm Hg), and the pCO_2 increased with time but was always greater than ambient pCO_2 (approximately 0 mm Hg). It follows that oxygen was consumed or that more oxygen was bound to haemoglobin, perhaps as a result of pH alterations. The results also suggest acid production during storage. It is not clear whether net carbon dioxide production occurred, or whether net acid production occurred, causing carbonic acid dissociation to carbon dioxide and water.

Closer analysis reveals that, although the changes present are statistically significant, they are not necessarily clinically significant. For example, an acidotic bird recovering through appropriate use of fluid and heat therapy might increase its blood pH from 7.2 to around 7.4. This change will be detectable even in the face of a 0.02 unit change in pH during storage. Moreover, if consecutive samples are stored for similar periods of time, changes due to storage can be expected to cancel out, leaving the clinician with a clearer idea of a patient's decline or recovery. However, a clinician may not be able to rely on measured pO_2 values of stored blood, as the storage changes are sufficient to obscure clinical changes.

Venous blood gas analysis may therefore present the clinician with a way of assessing the treatment protocol in place for the shocked avian patient. Clinical data obtained in this way may be of value in supplementing the scant data on avian cardiovascular shock. There is considerable evidence that birds differ with regard to the shock mechanism from mammals. For example, they possess a renal portal system of unclear compensatory function (Sturkie, 1986), they are capable of enormous compensation to osmotic (Arad, 1989) and haemorrhagic (Wyse and Nickerson, 1971) insults, and they exhibit paradoxical hypolactacidaemia during anaesthetic acidosis (Lau, 1996 unpublished observations). Well documented information about serial blood gas and lactic acid measurements in clinical patients may help elucidate some of these peculiarities.

Finally, routine acid/base analysis may shed light on the value of correcting bicarbonate levels and acidoses with bicarbonate. This practice was advised in the often quoted paper by Redig (1984). However, mammalian practice suggests that this procedure is accompanied by several hazards. Most obvious is the possibility of overshoot alkalosis. However, the additional risks of hypokalaemia, paradoxical cerebral acidosis and shifts in the oxygen dissociation curve of haemoglobin make bicarbonate supplementation a hazardous procedure (Hauptman and Chaudry, 1993). The change in oxygen dissociation is worthy of particular note, since acidosis causes a right shift in the dissociation curve. This improves tissue oxygenation. Too rapid reversal of acidosis is likely to reduce tissue oxygenation, to the detriment of the patient. Although avian haemoglobin differs from mammalian haemoglobin, similar patterns might exist.

Ethical approval for this project was obtained from the University of Queensland Animal Experimentation Ethics Committee, approval number CAMS/706/5/SF.

Acknowledgements

Pat Hore and the Clinical Pathology laboratory of Gympie General Hospital generously made their time, facilities and expertise available to this study in order that knowledge about raptors and their health might be extended. The authors thank them for their far-sightedness, interest and devotion.

References

Arad, Z, M Horowitz, U Eylath and J Marder, 1989, Osmoregulation and Body Fluid Compartmentalisation in Dehydrated, Heat-Exposed Pigeons. *American Journal of Physiology 257 (Regulatory integrative comparative physiology)*: R377-R382.

Cannon, MJ, 1991, Care of the Ill Bird, in *Avian Medicine*, Proceedings 178, Postgraduate Committee in Veterinary Science, University of Sydney, pp 13-17.

Hauptman, J and IH Chaudry, 1993, Shock: Pathophysiology and Management of Hypovolaemia and Sepsis, in *Textbook of Small Animal Surgery* (Slatter, D, Ed), W B Saunders Company, Philadelphia, pp 1-11.

Lau, MT-M, 1996, Master of Veterinary Science candidature work, Department of Companion Animal Medicine and Surgery, University of Queensland.

Martin, HD and GV Kollias, 1989, Evaluation of Water Deprivation and Fluid Therapy in Pigeons. *Journal of Zoo and Wildlife Medicine* **20**(2):173-177.

Philip, CJ 1981, Emergencies, Shock and Post-Surgical Care, in *Aviary and Caged Birds*, Proceedings 55, Postgraduate Committee in Veterinary Science, University of Sydney, pp 256-296.

Redig, PT, 1984, Fluid Therapy and Acid-Base Balance in the Critically Ill Avian Patient, *Proceedings of the Association of Avian Veterinarians*, pp 59-73.

Sturkie, PD, 1986, Heart and Circulation: Anatomy, Haemodynamics, Blood Pressure, Blood Flow, in *Avian Physiology* (Sturkie, PD, Ed), Springer-Verlag, New York.

Verkest, KR, 1994, Shock and Compensatory Mechanisms in Birds, Bachelor of Veterinary Biology thesis, University of Queensland.

Wyse, DG and M Nickerson, 1971, Studies on Haemorrhagic Hypotension in Domestic Fowl. *Canadian Journal of Physiology and Pharmacology* **49**: 919-926.