Avian Anaesthesia

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Avian anaesthesia or anaesthesia of birds is an enormous topic. There is a tendency to forget that we are dealing with an entire class as well as the orders, families, genera, and species within that class.

Can this make any difference? I believe it can. If nothing else, the control of respiration is likely to be different in birds which frequent different habitats just as it is in mammals. So we can expect to see respiratory behaviour in some diving water birds which is different from that of land based birds. And if one major piece of physiology can be variable between species then why not others? Indeed thermoregulation must be different in the antarctic penguins when compared with say tropical birds. Does any of this rate a mention in the avian anaesthetic literature? No!

What does the literature say about anaesthetising birds? It is interesting to remember that one of the earliest, if not the earliest, recorded anaesthetic reports is from Paracelcus ca. 1540 where he notes that chickens could be anaesthetised with ether.

Slightly more recently but not much further advanced Blount in 1949 (Blount 1949) states that ostriches and pigeons can be anaesthetised with chloroform but ether is recommended for other species. Other regimes mentioned are rectal chloral hydrate, intraperitoneal pentobarbitone and local anaesthesia with procaine, which is stated to be "without exception, perfect in birds." Presumably this means chickens not psittacines. Blount also mentions morphine in poultry and states that a 3.2 kg bird was given 360 mg morphine by mouth which had no effect and the following day received 180 mg I/P and a further 120 mg I/V "but no anaesthetic effect occurred."

In 1961 Grono (Grono 1961) wrote on anaesthesia of budgerigars using a variety of regimes. Local anaesthesia was almost invariably fatal as were thiambutene and pentobarbitone whilst ether and fluothane gave good results. This was no mean feat considering the equipment used for halothane administration " The inhalation anaesthetics were administered on cotton wool in a glass tube 22.5 cm long and 2.5 cm in diameter. The birds were placed on their backs and the glass tube passed over the heads and down to the wings permitting observation of respiration."

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By 1977 Jones (Jones 1977) reported difficulty using volatile agents. Once again the technique was to use the agent on cotton wool in a jar or box. In 1978 Camburn and Stead (Camburn and Stead 1978) reported good results with halothane and nitrous oxide using a Magill attachment and a controlled concentration of the volatile agent. They also report that the birds were acclimatised and ensured that the plane of nutrition was adequate before anaesthesia. They report poor results with metomidate and saffan but better results with ketamine.

In 1986 Hartsfield and McGrath (Hartsfield and McGrath 1986) point out that gentle handling will benefit the anaesthetic, that visceral and somatic pain is similar in birds to mammals, and, that for the delivery of volatile agents the use of accurate delivery systems is the most appropriate.

Is there a problem with anaesthetising birds? No, the problems start when you want to wake them up again, and failure to recover is the major hazard. Why do they fail to wake up?

Anaesthetic mortality among the avian patients would seem to be excessively high. This can only be an impression because there is little or no documented data available. The impression is that these patients suffer respiratory arrest. A particular drug is often cited as the, or a, cause.

Hypothermia particularly in small birds is a real and serious problem. If a bird weighs 25 g and assuming the heat loss as for water 1 cal g^{-1} degree C^{-1} and assuming the latent heat of vaporisation of water at 40° C to be about 550 cal g^{-1} then for each gram of water lost 550 cal would be lost, that is 0.55 cal. mg^{-1} If one mole of water vapour weighs 18 gm and occupies 25 l at 38° C then 1 ml weighs 0.72 mg. If the minute volume of the bird is about 150 ml and the water content is 5% then 7.5 ml of water vapour is lost per minute. This is about 3 cal min⁻¹ or about 1° C each 10 min for respiratory water vapour alone. They can cool very quickly.

Blood loss is another serious concern. Total blood volume is around 100 ml Kg⁻¹ and for a 25 gm bird this comes to 2.5 ml. At 15 drops per ml, 2.5 ml represents 37.5 drops. That is to say if you spill 10 drops of blood you have lost 25% of the birds blood volume which is a substantial loss.

How much agent is enough? What is the Minimum Alveolar Concentration for Anaesthesia (MAC) in the species at issue? What is the MAC in the bird? Quasha et al. (Quasha, Eger et al. 1980) have looked at the MAC for various agents in a range of species and if we look at halothane the range is from 0.67% to 1.17% a little over a 100% variation. These figures are not available for many of the avian species. There is a figure for ducks with isoflurane (Ludders 1990) with a MAC of 1.3 " 0.23%. These authors also point out that isoflurane is a potent respiratory depressant and is markedly more so in the duck than in the dog or cat. Who knows what may happen in other species of bird? Isoflurane MAC in Sandhill Cranes is given as 1.34 " 0.14% (Ludders 1989) and the MAC for halothane in

chickens is given as 0.85 " 0.09% (Ludders, Mitchell et al. 1988). These figures suggest that the potency of the volatile agents is similar in birds to that seen in mammals but much more work remains to be done.

There are economic constraints on avian anaesthesia. Many people are now using isoflurane for routine avian anaesthesia and isoflurane is expensive. A vaporiser for isoflurane is also expensive, but if you don't use a properly calibrated vaporiser then do you get the real benefits of the agent? There is a feeling that isoflurane is a safer or more forgiving agent than halothane. If we look at the two agents, isoflurane has a lower blood gas solubility co-efficient, a higher MAC, and is a less potent cardiac depressant than halothane although this is not a great factor at 1 MAC, it becomes more significant as alveolar concentration increases. The difference in MAC however may be very significant. If we assume a MAC for halothane of 0.8% and for Isoflurane of 1.4% then the 1.5 MAC level which we should probably be trying not to exceed is 1.2% for halothane and 2.1% for isoflurane and the 3 MAC figures where the game is getting quite dangerous are halothane 2.4% and isoflurane 4.2% so the greater MAC of isoflurane confers more leeway in terms of the upper limit of vaporiser setting. But if we look at the vaporisers themselves the graduations are almost the same because the halothane vaporiser was made first and the scale chosen for convenience in dealing with a range of flow rates in humans. Perhaps if a halothane vaporiser had a greater range of dial movement for each 1% increment in output then halothane may seem a safer agent.

Monitoring the anaesthetised bird is to my mind the greatest difficulty. What should we monitor and how to do it. Many regimes and systems have been suggested over the years but I believe now that the single most valuable parameter to monitor is the presence of arterial blood flow, and the easiest and possibly most reliable way to monitor that is with a Doppler blood flow detector. This will work reliably on a wide range of sizes and gives an audible indication of flow. They are not cheap but they do work.

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