Intensive care in an unusual setting: management of pneumonia in a chimpanzee

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ABSTRACT

We report a case in which intensive care doctors and nurses became involved in the care of a young chimpanzee who required ventilation for pneumonia at Wellington Zoo, New Zealand. This required staff to work outside the usual protected environment of a hospital intensive care unit. The chimpanzee, Bahati, was ventilated for 3 days, replicating intensive care practice, but died. Logistical challenges included equipment procurement, environment, electrical safety, gas supply and infection control. Other difficulties included differences in physiology, nursing care and therapeutics. End-of-life processes were similar, with zoo staff responding as if they were immediate family. Euthanasia was an unfamiliar process to ICU staff. Bahati's death received national media attention and some criticism of the involvement of intensive care staff. The zoo staff were overwhelmed and grateful that everything possible was done for Bahati.

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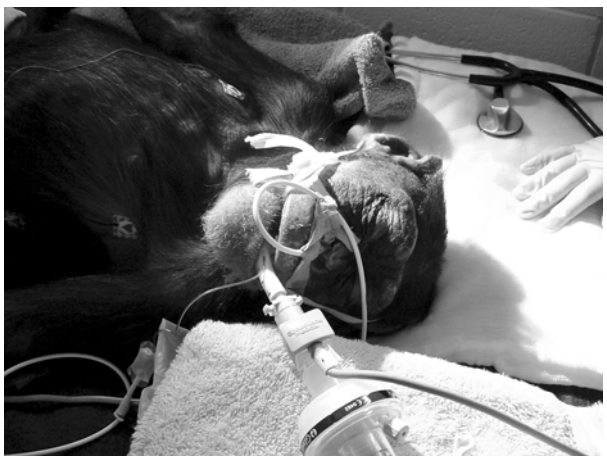
Intensive care medicine is a discipline dependent on the skill of specialised staff, working with variable quantities of equipment in a tightly controlled environment. It is uncommon for these skills to be required outside a hospital, and even less common for this need to continue over a period of days. Further, most intensivists limit their practice to *Homo sapiens*. We discuss a case of intensive care applied outside the usual environment and genus.

Clinical record

In October 2005, the director of the intensive care unit in Wellington Hospital, New Zealand, received an unusual request for assistance from the veterinary department of Wellington Zoo. A 4-year-old, 19 kg male chimpanzee named Bahati (Swahili for "luck") had become unwell over the preceding few days with anorexia, lethargy and nasal discharge. He was initially treated with amoxycillin–clavulanic acid and paracetamol, but his condition had continued to deteriorate. He was separated from the chimpanzee group and taken to the veterinary hospital for a chest x-ray, which routinely requires anaesthesia and intubation.

After the x-ray had been taken, it became apparent that Bahati was unable to be extubated. He remained breathing spontaneously through the endotracheal tube overnight on an anaesthetic circuit with isoflurane supplied from a draw-over vapouriser. The veterinarians could only provide a fixed concentration of 100% oxygen and had no means of delivering positive end-expiratory pressure (PEEP). Intravenous access was obtained, and the animal was treated with cefuroxime, flucloxacillin and amoxycillin–clavulanic acid. His condition continued to deteriorate, culminating in an episode of apnoea associated with a loss of palpable pulse. Cardiopulmonary resuscitation was performed, including external cardiac massage and direct intracardiac adrenaline injection, before cardiac output was restored after 3 minutes. As he had survived until morning, and with no facilities for prolonged non-manual ventilation, the veterinarians contacted Wellington Hospital for help. The ICU director and two senior nurses went to the Zoo to see what assistance could be provided.

Figure 1. Bahati undergoing ventilation



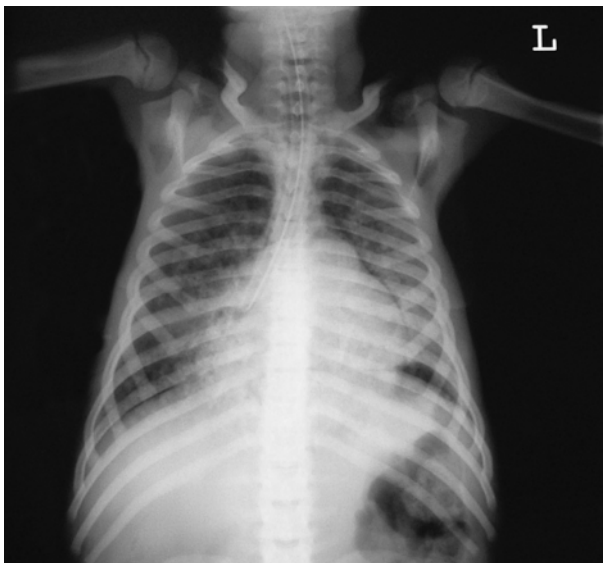
Bahati (Figure 1) was breathing spontaneously through an anaesthetic circuit with fresh gas supplied from a size E oxygen cylinder. No electronic monitoring was available other than a pulse oximeter. He was being nursed on a mattress on the floor of a concrete area in the Zoo hospital.

On examination, he was well perfused with a strong pulse and adequate blood pressure. His breathing was shallow and rapid. Auscultation revealed bilateral coarse

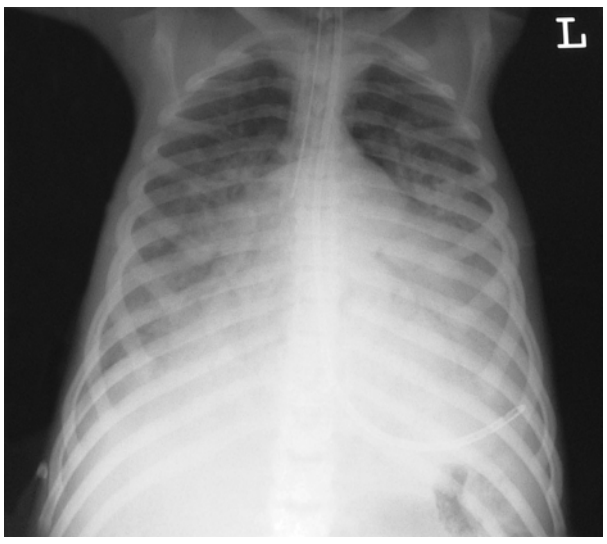
chest sounds, and the lungs appeared stiff on manual ventilation. The chest x-ray from the previous day showed patchy left parahilar and basal airspace consolidation with a right main bronchial intubation (Figure 2A). Initial blood tests showed leukocytosis with neutrophilia and a rise in creatine kinase concentration which was presumed secondary to the intracardiac injection during resuscitation. Renal function was normal, and there were no electrolyte abnormalities.

We diagnosed significant respiratory failure from pneumonia, without septic shock. We considered his breathing might improve over several days and that we could try to support him over that period.

Figure 2. Serial chest x-rays of Bahati



A. *On Day 0, the chest x-ray showed patchy left parahilar and basal airspace consolidation.*



B. *On Day 3, increasing bilateral mid and lower zone consolidation were apparent.*

Our first task was to procure a ventilator and monitoring equipment, and volunteers to manage both. An old portable Spacelabs monitor (Spacelabs Healthcare, Wash, USA) and Bird Avian Transport Ventilator (Crestline Medical, Utah, USA) were borrowed from the patient simulation centre. Some consumable supplies, drugs and paperwork were collected. Staffing was the main challenge. We asked selected staff nurses whether they would volunteer time to provide 6-hour shifts of care. Those with a flight-nursing background were asked as they were more familiar with providing intensive care support in an unfamiliar environment using portable equipment. Medical support was to be provided by an intensive care consultant (P R H) and a registrar (A J P). It was decided that a maximum of 3 days' assistance was possible, after which treatment would be withdrawn. We also decided not to provide inotropic support, central venous or arterial access, nor further cardiopulmonary resuscitation, should it be required.

On returning to the Zoo, we began electrocardiography and end-tidal capnography monitoring. A paediatric urinary catheter and nasogastric tube had been inserted, and nasogastric feeding begun with date-expired strawberry flavoured Ensure Plus (Abbott). It was not possible to use the Bird Avian Transport Ventilator, as we could not work out how to set the PEEP nor stop the high airway alarm. We were offered a new Oxylog 3000 transport ventilator (Draeger Medical, Telford, Pa, USA) from the nearby Life Flight Trust flight retrieval service. Three veterinary nurses and two veterinarians worked in shifts with seven intensive care nurses to provide the care needed.

Over the next few hours, ventilation was optimised using sedation (diazepam plus tiletamine–zolazepam) administered by the veterinarians, and pancuronium boluses to paralyse. This allowed the inspired oxygen fraction to be reduced from 100% to 60%, with pulse oximetry saturation now around 90% on 10 cm PEEP. The patient was ventilated on a mandatory mode using low tidal volumes (5 mL/kg) which gave an end-tidal CO_2 partial pressure of 70 mmHg. His peak airway pressure was 35 mmHg, which we concluded was due to poor lung compliance.

The initial anuria responded to a fluid bolus, and he began to pass urine at a rate of around 100 mL/h. His mild hypothermia decreased with passive warming, and he subsequently became febrile, with the temperature spiking at 38.8° C.

Overnight, with respiratory toilet and regular turning, the inspired oxygen fraction was further reduced to 40%. Hourly observations were recorded on intensive care flow sheets, and drug prescriptions were written on standard hospital drug charts.

The next day, medical review found Bahati's clinical condition to be improving slowly, while a second chest x-ray (Figure 2B) showed increasing bilateral mid and lower zone consolidation. An endotracheal tube aspirate and nasal swab grew *Escherichia coli*; a throat swab also grew *E. coli* plus β-haemolytic streptococci. Results of repeat blood tests processed at a local private laboratory were unremarkable.

End-tidal CO_2 partial pressure was 60–70 mmHg, and an arterial blood gas sample showed a P_{CO_2} of 105 mmHg, which may have explained the occasional periods of dilated but reactive pupils. An increase in the rate and volume of ventilation produced an end-tidal CO_2 partial pressure of 40 mmHg with no increase in peak airway pressure.

Ventilation, feeding, turning and regular pulmonary toilet were continued by intensive care and veterinary nurses for most of the following 24 hours.

By the morning of Day 3, Bahati's respiratory function was much improved. He had needed very little sedation and no paralysis overnight. We suspected an ischaemic brain injury, and sedation was ceased. On review at 17:00, Bahati intermittently chewed and blinked, with some limb movements consistent with myoclonic jerks. His pupils remained dilated, and chewing was also seen in response to painful stimulation of his face. Central chest stimulation produced an extensor limb response. After discussion with the veterinary staff, it was decided to euthanase him, which was carried out with pentobarbitone. His body was shown to his mother (another member of the chimpanzee group at Wellington Zoo) so that she was aware of his fate.

Postmortem examination showed marked pulmonary congestion with organising diffuse alveolar damage, superimposed acute bronchiolitis and bronchopneumonia.

Table 1. Laboratory results for Bahati (all values in mmol/L unless otherwise stated)

Parameter	Bahati	Human reference range
Sodium	141	136–146
Potassium	5.2	3.5–4.9
Creatinine	66	62–103
Magnesium	1.54	0.76–0.99
Phosphate	1.50	0.95–1.60
Corrected calcium	2.38	2.25–2.49
Bilirubin	2	4–22
Alkaline phosphatase	230	36–100
Haemoglobin (g/L)	151	130–175
Platelet count ($\times 10^9$/L)	319	160–460
White cell count ($\times 10^9$/L)	18.0	4–10.5

Discussion

There are few reported cases of the involvement of intensive care physicians and nurses in the management of primates or indeed any other animals. Paediatric medical and nursing staff were involved in the care of a 50-day-old chimpanzee in China,[1] and neonatal intensivists treated a small-for-dates infant chimpanzee with severe cardiorespiratory distress from pneumonia in Germany.[2] No previous involvement of non-paediatric intensive care staff has been described.

Transmission of infectious diseases from humans to non-human primates in a zoo setting is recognised as contributing to animal deaths.[3] Control of infection is difficult because zoos aim to maximise the exposure of animals to humans. Common human pathogens, including *Streptococcus pneumoniae* and *Klebsiella pneumoniae*, have been described as causative agents for fatal respiratory disease in a chimpanzee colony in North America,[4] and a *Klebsiella* species in an outbreak in a chimpanzee nursery.[5]

There was concern about the potential for transmission of infection to the staff treating Bahati. In the wild, there are documented cases of primate-to-human transmission, the most lethal being Ebola haemorrhagic fever.[6] Bahati, who had been born in captivity, had no risk of this and succumbed to a pathogen of human origin.

The Zoo environment presented several challenges to hospital staff used to working in a controlled area, with all requirements immediately to hand. No piped oxygen was available, the only supply being from size E oxygen cylinders (680 L). The intensive care flow sheet was adapted to include hourly readings of remaining capacity, so that exhaustion of the supply could be anticipated well in advance. Suction was available only from a transport device, to which a standard in-line suction catheter was connected. A hospital sharps bin had to be supplied. Nursing Bahati on a mattress on the concrete floor of a cage was impractical, so he was transferred to a waist-high table. Electrical circuit-breakers were introduced at the wall to prevent damage to equipment and electrical injury to staff and patient in an area that was designed to be hosed down after use. The absence of on-site laboratory analysis meant that ventilator settings had to be titrated to end-tidal rather than arterial CO_2 partial pressures. Daily x-rays were performed by a radiographer from a nearby private radiology service, and blood tests by a private laboratory.

The Zoo management requested that Bahati's illness be kept private to avoid concerned members of the public offering help. Shortly after he died, the media discovered the story, and it received prime-time news coverage. The ICU involvement became public at a time that criticism of

long waiting times for cardiac surgery was prevalent in the media. The story received a mixed response, from support to concern that we were "more interested in looking after animals than humans". Within the ICU, views were divided, and seemed to correlate with pet ownership.

Other issues common in intensive care cases were absent. Family meetings were not conducted, documentation was limited, and we did not have to complete an assessment of fall risk or pressure area risk score, nor collect data for an APACHE score.

The end-of-life process had some interesting differences and similarities to that of humans receiving intensive care. When it became apparent that Bahati had suffered severe brain injury, the medical team suggested withdrawing active treatment — "the nicest thing we can do now is to stop the intensive care, take out his breathing tube, and let nature take its course". The veterinary team considered this unnecessarily cruel and proposed that the most "humane" treatment would be euthanasia, which was duly performed. Bahati had severe brain damage, but even moderate impairment of a chimpanzee would result in starvation or attack by other members of the group. Regular meetings were held with the zoo staff who usually care for the chimpanzees, and we explained matters to them in the same manner we would to an immediate human family. In the last hours, we handed care back to Zoo hospital staff, as we regarded this as Zoo "family" business.

Physiologically, it became apparent that the decision to ventilate on a per-weight human basis was incorrect. In the absence of available guidelines on chimpanzee ventilation, we were initially unclear whether to ventilate Bahati as a 4-year-old child (per weight) or as a 15-year-old adult (per human-adjusted age). A single arterial stab on Day 2 showed CO_2 partial pressure in excess of 100 mmHg despite ventilation with a minute volume of 4.5 L (12 mL/kg $\times$ 19 kg $\times$ 20 breaths per minute). Hence, it became apparent that permissive hypercapnia was as much by error as design. A possible reason for our underestimation was his comparatively large muscle mass in relation to weight. All other laboratory test results were compared with reference ranges for primates, most of which were noted to be remarkably close to those of their human counterparts (Table 1). Drug doses were calculated and given on a per-weight basis, including antibiotics and nasogastric feed. Both deep vein thrombosis prophylaxis and gastrointestinal haemorrhage prophylaxis were considered but not used. Regular turning was instituted by the nursing staff, to the surprise of the veterinary nurses who had no experience of pressure area care or postural drainage.

Daily sedation hold for assessment was not advisable, even if it had not been precluded by ventilatory difficulties. Despite appearances, chimpanzees are considered the most dangerous animals in the zoo; they are very strong and have a good understanding of humans. The normal procedure for waking anaesthetised chimpanzees is to put them on the floor, turn everything off, remove the endotracheal tube, and walk very briskly away with all the equipment, shutting the cage door. Oxygen therapy postextubation would not be possible.

It was an emotionally difficult time. The Zoo staff had cared for Bahati since his birth, so that treating him was almost akin to treating a member of their own family. The veterinary staff were overwhelmed that the ICU staff offered to help and very grateful that everything had been done that could be done.

The memory we will take away is being repeatedly chastised by Zoo staff for calling Bahati a monkey — monkeys have tails, chimpanzees do not.

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