THE MANAGEMENT, TREATMENT AND PHYSIOLOGY OF HEAT STROKE IN FLYING FOXES.

(Pteropus spp.)

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INTRODUCTION

- Heat stress events and therefore heat stroke in flying foxes is an ever increasing problem as global warming continues to increase mean temperatures.

- In January of 2014 an estimated 45500 flying foxes fell over 52+ camps over SEQ fell out of the skies as an temperatures soared above 41-42 degrees.

- It is predicted that more and more of these events are likely to happen, and have serve as a warning that global warming is well and truly upon us, real and will have wide-spread environmental effects worldwide.

INTRODUCTION

- Black flying foxes were most affected proportionally (95.9% total)

- Then grey headed flying foxes (*P.poliocephalus)* (3.6%)

- And for the first reported time; little red flying foxes (0.5%) (*P.Scapulatus*) (Welbergen, Martin, Booth et al.,*unpublished)*

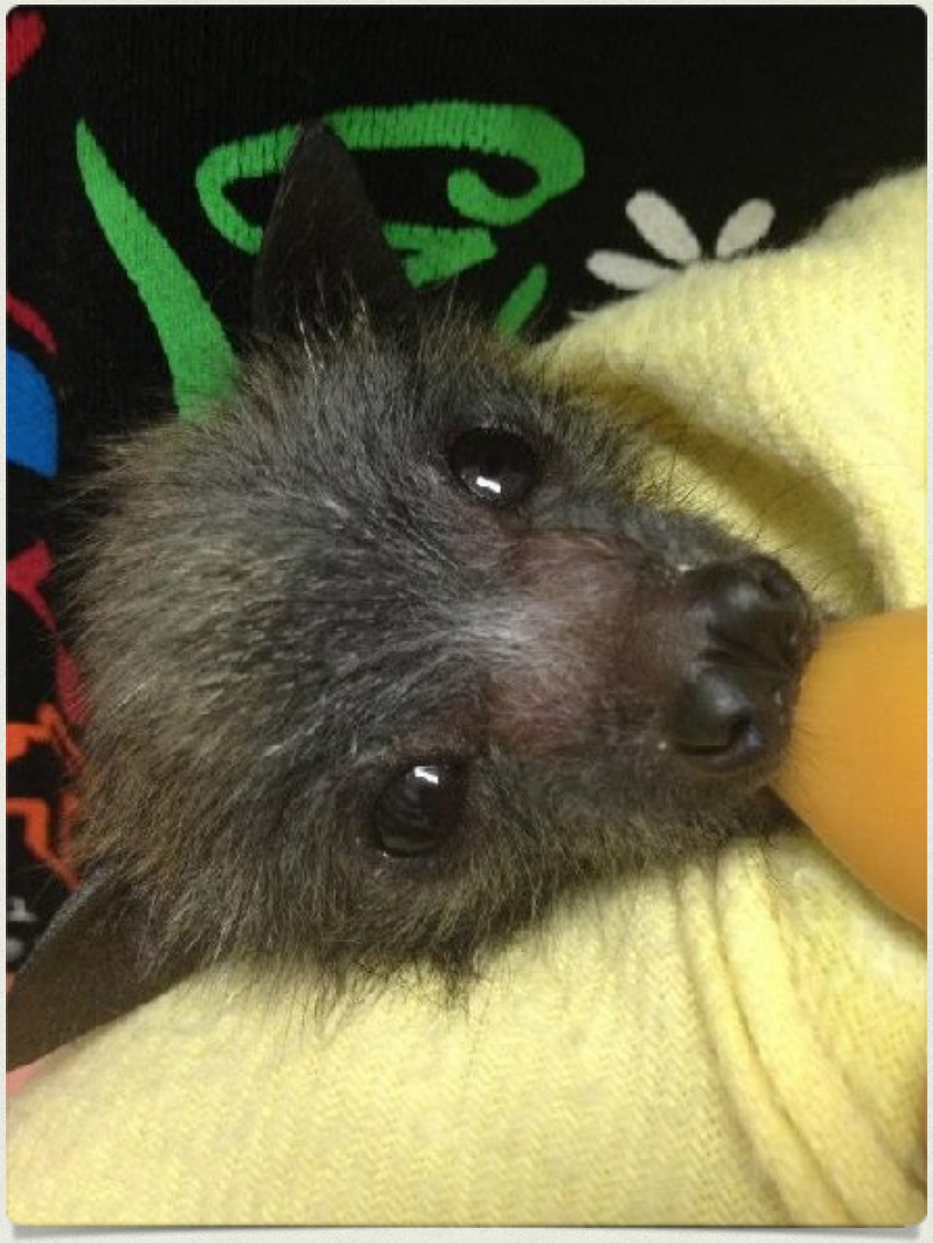
Map of affected camps across SEQ Jan 4 2014

(Welbergen, Martin, Booth et al.,*unpublished)*

INTRODUCTION

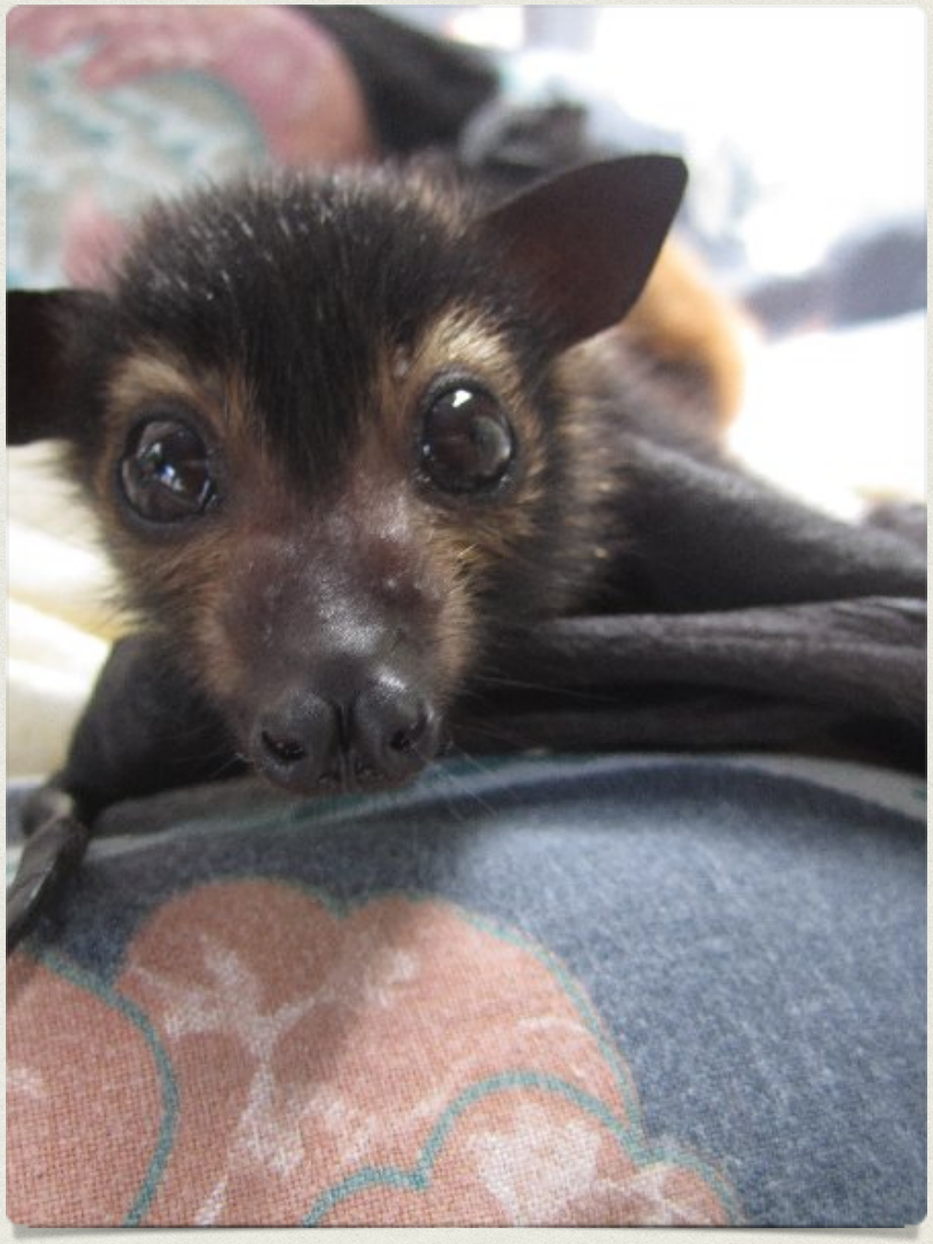
- As threatened endemic species, as grey headed flying foxes are forced into higher latitudes in response to increased mean temperatures, paradoxically this will expose them to a greater frequency of extreme heat events. (http://theconversation.com/killer-climate-tens-of-thousands-of-flying-foxes-dead-in-a-day-23227

- In species such as flying foxes with a low natural capacity for population increase, factors that undermine recruitment and the effective breeding population are expected to exacerbate the threat to species survival in addition to significant and increasing anthropogenic factors. (Welbergen et al., 2008)



INTRODUCTION

- Both Welbergen et al., 2008 and Snoyman et al., 2012 identified that lactating females and dependent young are the first to be affected by heat stress, potentially acting as an early warning indicator for management purposes but also indicating that temperature extremes can have disproportionate effects on the effective breeding population and recruitment which are two key parameters of conservation.



INTRODUCTION

- Based on data from heat stress events of the last summer, P.Alecto, were far more affected than P. Poliocephalus or P.scapulatus and that females with dependent young were far more susceptable to heat stroke. (Welbergen et al., 2008)

- Differences in mortalities seen were due to species specific physiological mechanisms to deal with heat stress as well as the breeding status of the different species.

J.Welbergen

INTRODUCTION

- Females with dependent young had higher metabolic demands due to lactation,had higher heat radiation from clustering with young spent less time resting and more time fanning at lower temperatures, therefore had less time to recover in continual days of high temperatures, therefore exhibited higher mortalities in heat stress events. (Snoyman et al., 2012)

- Females with dependent young also show behavioural traits making them more susceptible such as roosting closer to the outer, less-shaded areas of the camp.

HEAT STRESS V'S HEAT STROKE

- Management of heat stress events, both preventative and medical, could significantly act to reduce the significant mortalities and suffering seen in the last few years.

HEAT STRESS V'S HEAT STROKE

- Steven et al, 1991 stated that because 75% of an exercising animal's metabolic energy appears as heat within the body, it follows that flying vertebrates must be capable of dissipating heat at about twice the rates of their exercising, non-flying mammalian counterparts.

- Thus, flying foxes are highly prone to heat stress as they are unable to sweat.

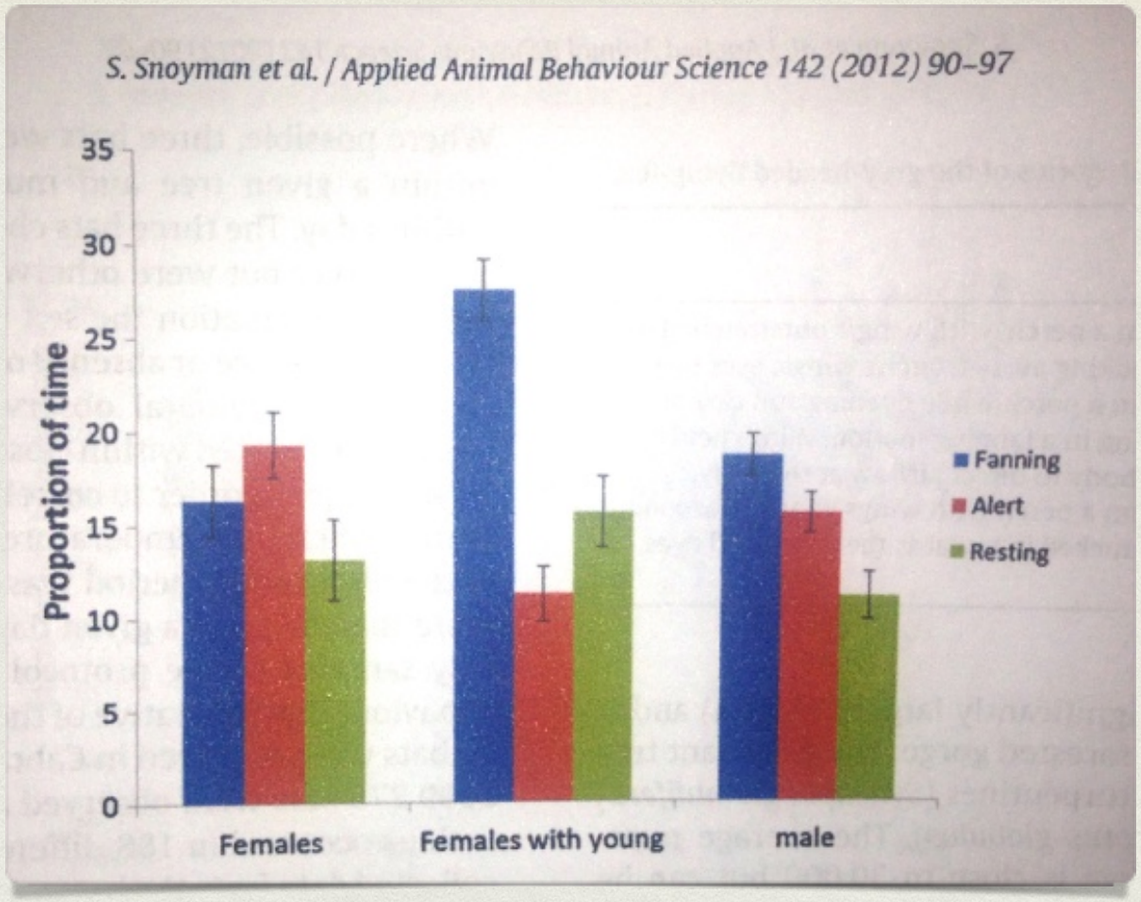
Body temperature of bats meeting ambient temperature as they can Respiratory system

HEAT STRESS V'S HEAT STROKE

- Heat stress occurs when the ambient temperature reaches a point above the thermoneutral zone of a particular individual.

- The point at which an individual starts to experience heat stress or progress on to develop heat stroke, varies with metabolic rate and individual behaviours.

- According to Welbergen et al., 2008 and Snoyman et al., 2012, all species exhibit a predictable sequence of behaviours indicating the progression from heat stress through to heat stroke and can act as early warning signs for a need for intervention.

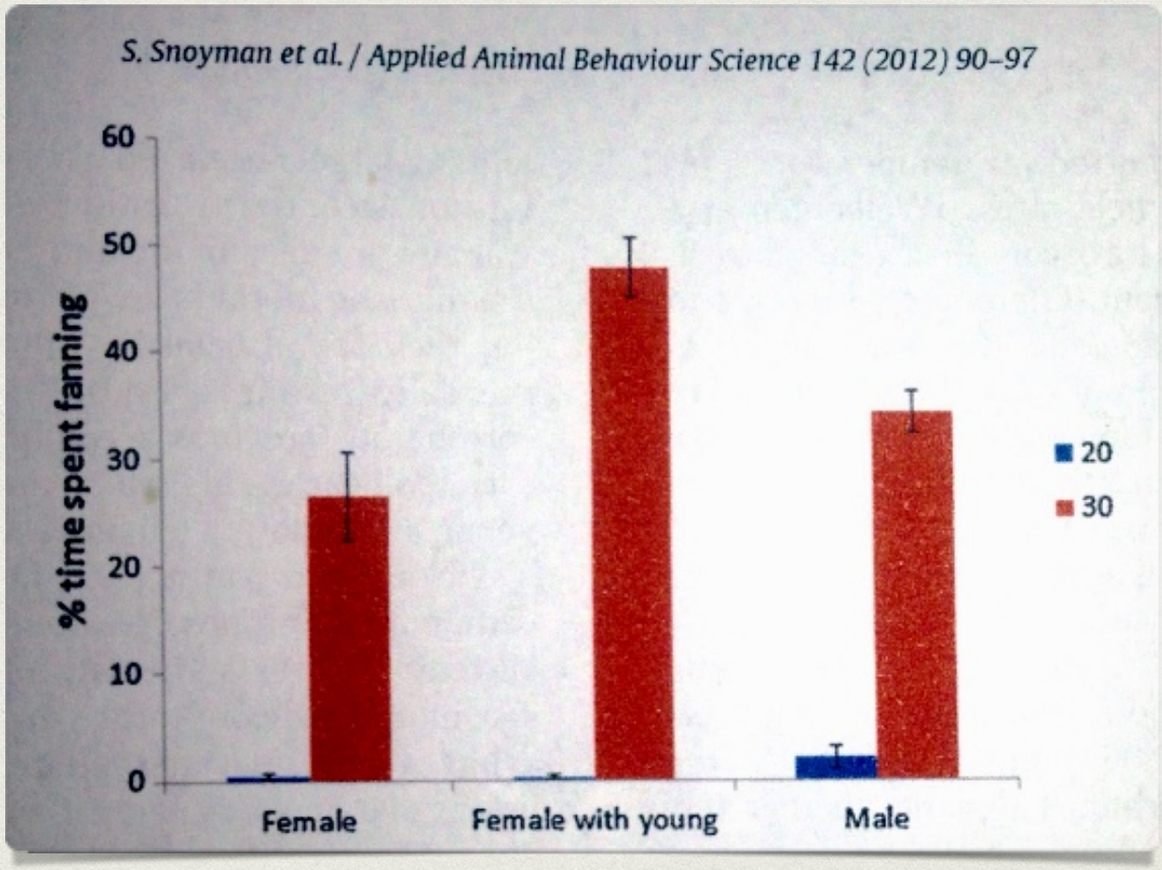


Snoyman et al., 2012 indicated that as ambient temperature rises above the thermoneutral zone the amount of time required to actively take measures to reduce body temperature increases significantly also with a notable demographic difference.

HEAT STRESS V'S HEAT STROKE

BEHAVIOURS INDICATING HEAT STRESS

- Wing fanning and shade seeking are usually the first signs of heat stress, where the temperature has reached above the level where the flying fox must actively reduce body heat.
- Wing fanning occurs in females with dependent young.
- Contributes to thermoregulation by forced convection, but very energetically taxing.
- Wing fanning usually starts at a temperature of 23 degrees or above and increases significantly at temperatures of over 30 degrees.
- Females with dependent young started fanning far earlier than males and then females without young.



Snoyman et al, 2012 indicated that around the thermoneutral zone (23°C), there was minimal fanning
however above the thermoneutral zone significant fanning startedand a definite population demographic difference becomes apparent.

HEAT STRESS V'S HEAT STROKE

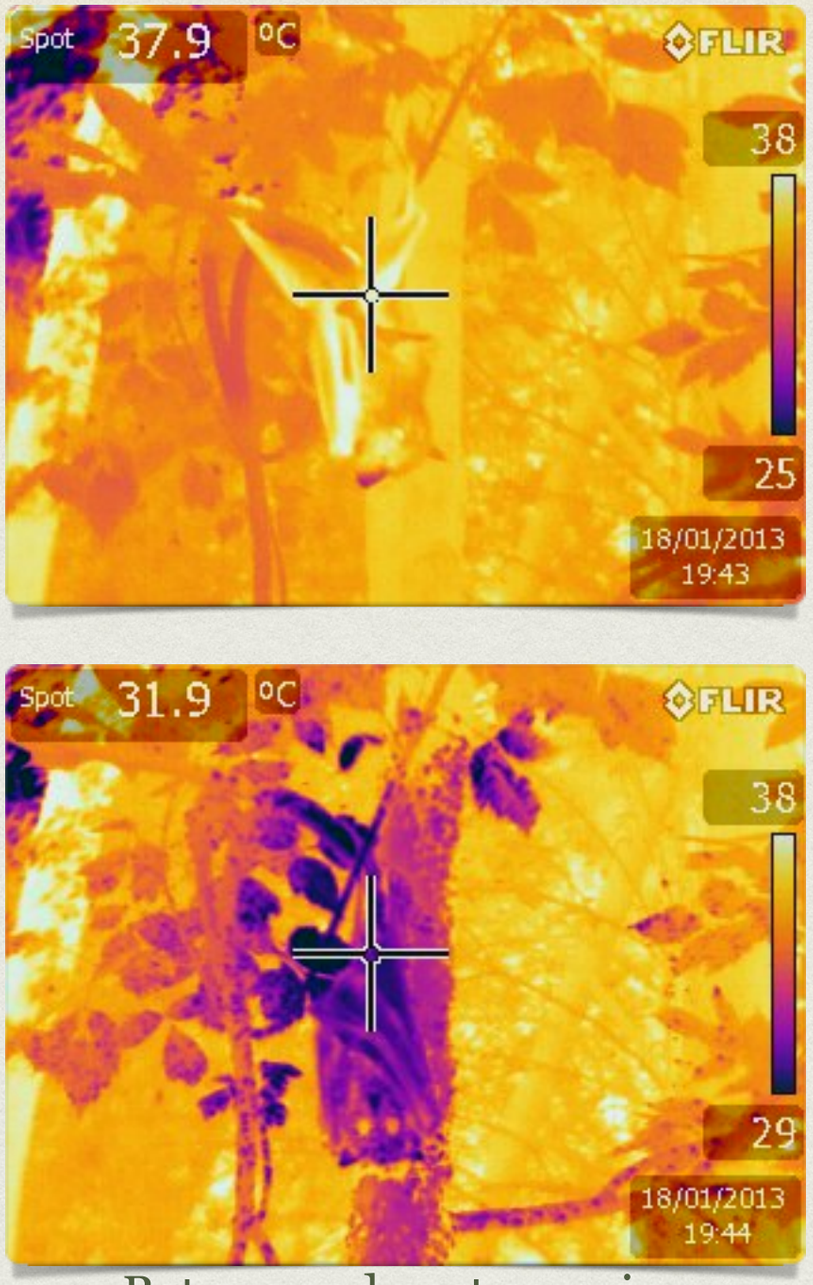
BEHAVIOURS INDICATING HEAT STRESS

- Once the ambient temperature is greater than body temperature, fanning and shade seeking are no longer effective and panting and saliva spreading are required in a desperate attempt to reduce body temperature.

- This results in considerable valuable bodily fluid loss. Constant fanning and moving to seek shade also contributes to exertional heat stroke, where myopathy or rhabdomyolysis can result.

- Beyond this point the animal is considered to be suffering from heat stroke.

- It is at this stage the most preventative changes can take place, such as spraying of colonies to aid evaporative cooling.



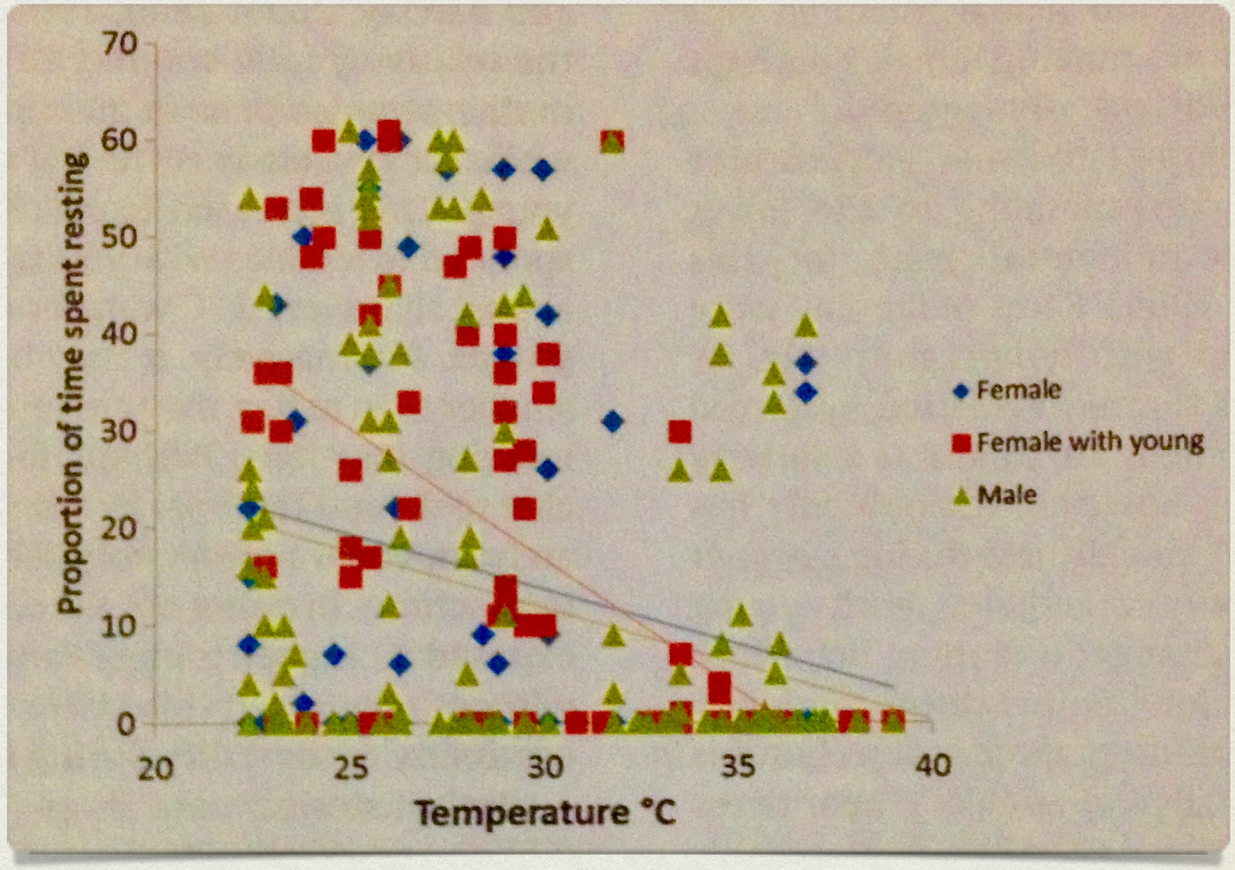
Bat pre and post spraying
J.Welbergen

https://www.dropbox.com/s/z61igrkm6wsr46r/MOV_2775.avi

HEAT STROKE

HEAT STROKE

- Once ambient temperature is greater than body temperature, fanning and shade seeking are no longer effective and panting and saliva spreading are required in a desperate attempt to reduce body temperature
- This results in considerable bodily fluid loss.
- Panting was initiated at temperatures of greater than 35degrees. In temperatures of greater than 35 degrees, no difference in behaviour between the sexes was noted.
- The licking of wrists occurred when temperatures were close to lethal limits.
- It is at this point that triage of any attainable bats must be initiated, as significant multi-organ damage has already started. *See Treatment for further information.*



HEAT STROKE

- Heat stroke is the cell damage that occurs from a marked increase in body temperature when the body has exhausted every ability to dissipate heat effectively.

- Heat stroke affects almost all body systems and the physiologic effects are directly attributable to cellular thermal injury.

- A flying foxes main mechanism of heat dissipation is via evaporative cooling, thus is also affected by humidity also. When dry this can occur far easier than in more humid conditions, meaning that heat stroke can occur at lower temperatures in more humid conditions.

- Direct thermal injury occurs at 42.7 °C causing enzymatic alterations from the denaturation of proteins, however multi-systemic damage can occur with sustained temperatures as low as 40.6°C especially in conditions of higher humidity.

HEAT STROKE

- Heat stroke can be exertional, caused by prolonged forced exercise under increased temperatures, or non-exertional, caused by an animal being exposed to prolonged high temperatures.

- Flying foxes usually exhibit a mixture of exertional and non-exertional heat stroke due to the physical efforts involved in the desperate effort to dissipate heat.

https://catalyst.library.jhu.edu/catalog/bib_2649264

HEAT STROKE

- It is because of this, that the treatment of myopathy (muscle damage) or rhabdomyolysis, is also required in the treatment of heat stroke in flying foxes.

- Damage to muscles can occur via direct thermal injury or as a result of myopathy secondary to prolonged fanning.

- Marked necrosis of muscle cells results in hyperkalaemia, hypocalcaemia, lactic acidosis and myoglobin release into the blood stream resulting in the death of proximal tubular cells of the kidneys.

- This also contributes to deaths from acute and chronic renal failure discussed later.

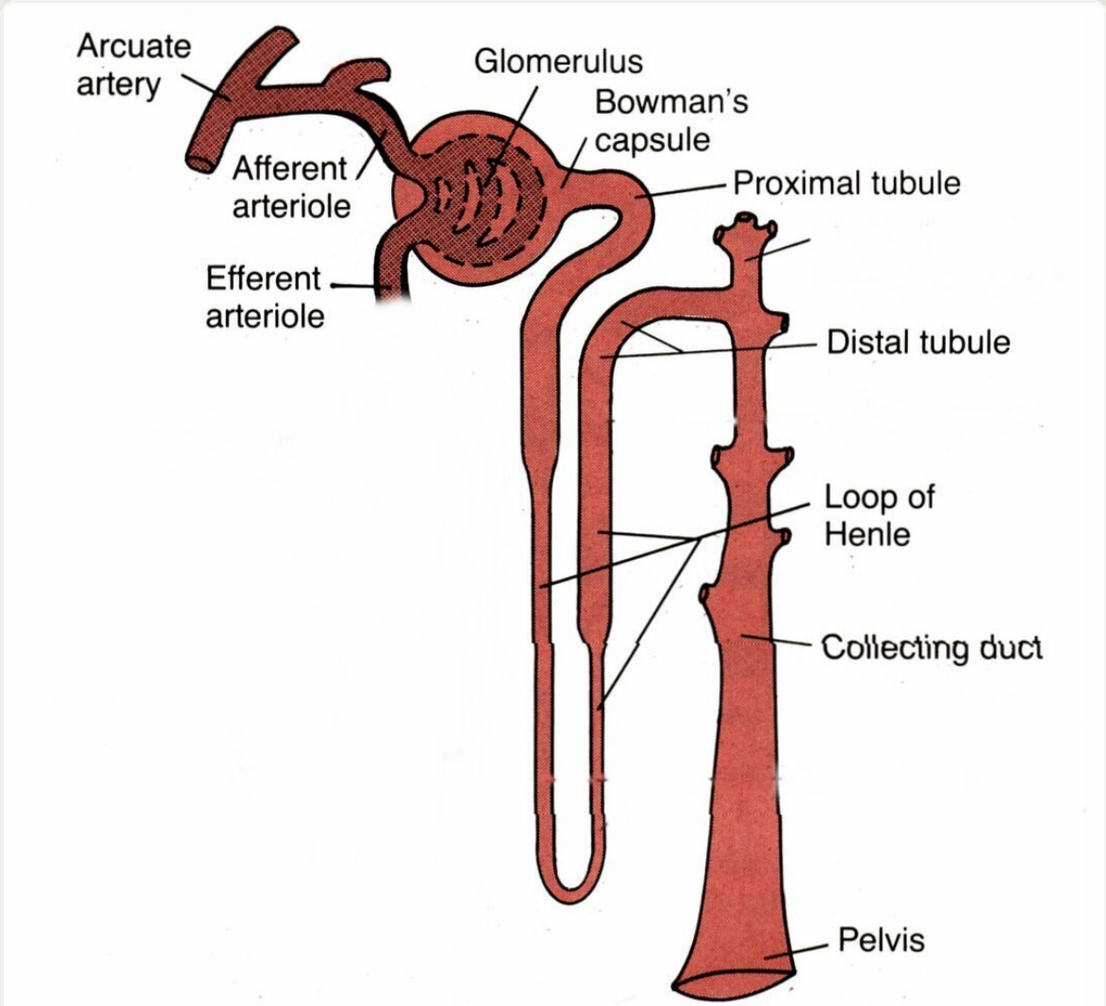


Figure 26–3. The functional nephron.

Guyton A, 1991. pg 288

HEAT STROKE

- The most distressing aspect of trying to triage and treat animals affected by heat stroke in the field is that many of these complications are not seen immediately.

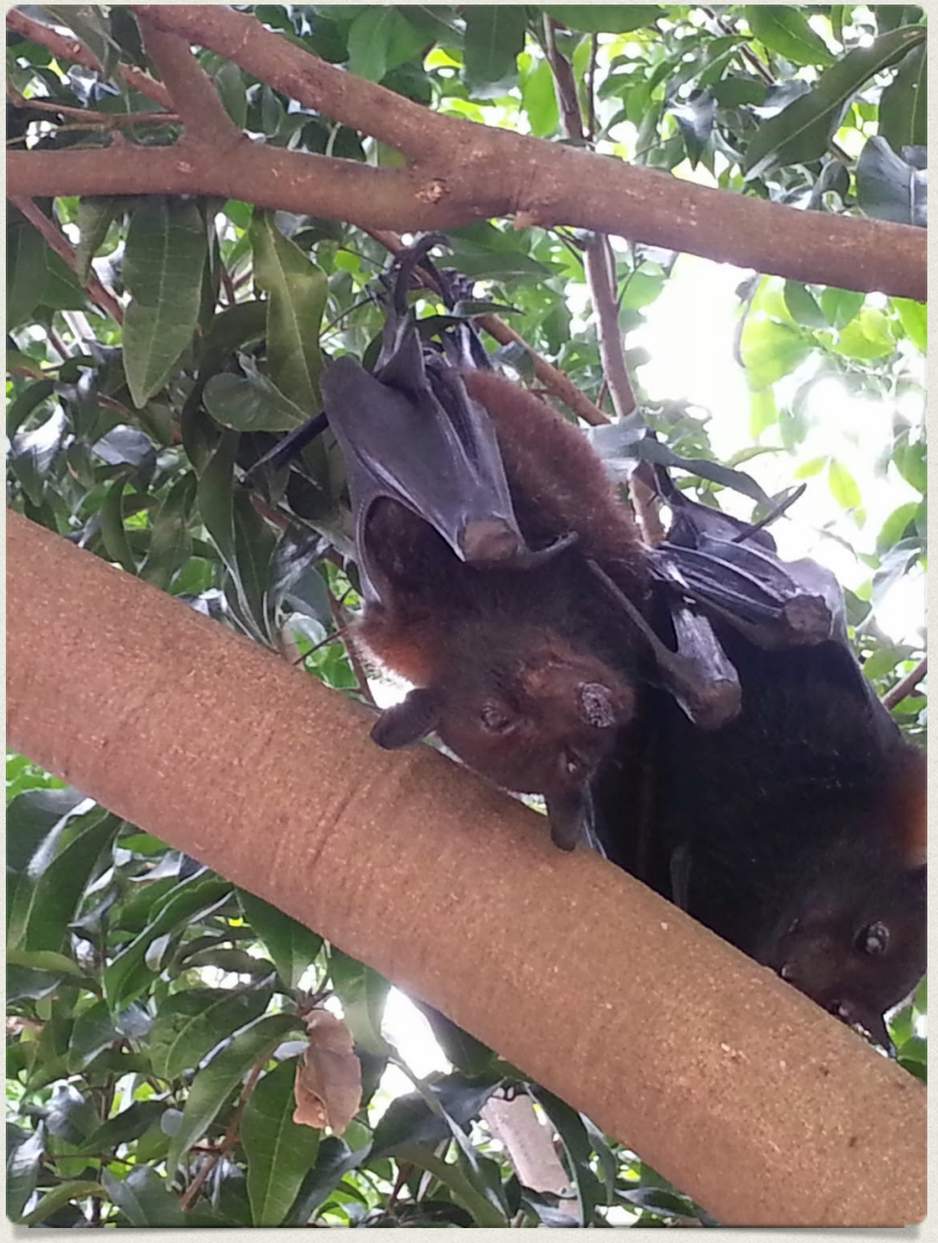
- A badly affected animal may fly away after being given fluids SC, orally or IP, and surviving the initial insult but further complications may appear days to weeks later.

- The consequence of this, is that any animal that doesn't respond to spraying prior to wrist licking or collapse or immediately to oral fluids, really needs to be taken into at least care or to a veterinary hospital.

HEAT STROKE

GASTROINTESTINAL TRACT

- The GI tract is profoundly affected by heat stroke.
- This causes profuse bloody diarrhoea.
- Sloughing of the mucosal lining.
- Endotoxaemia from bacterial translocation into the blood stream.
- Profound protein, fluid and electrolyte losses on top of those already present from earlier attempts to reduce body temperature.
- Severe hypovolaemic shock as a sequelae, meaning that, at this stage only IV fluids have a chance of reversing damage as all peropheral blood vessels have contracted in order to keep vital organs functioning.



HEAT STROKE

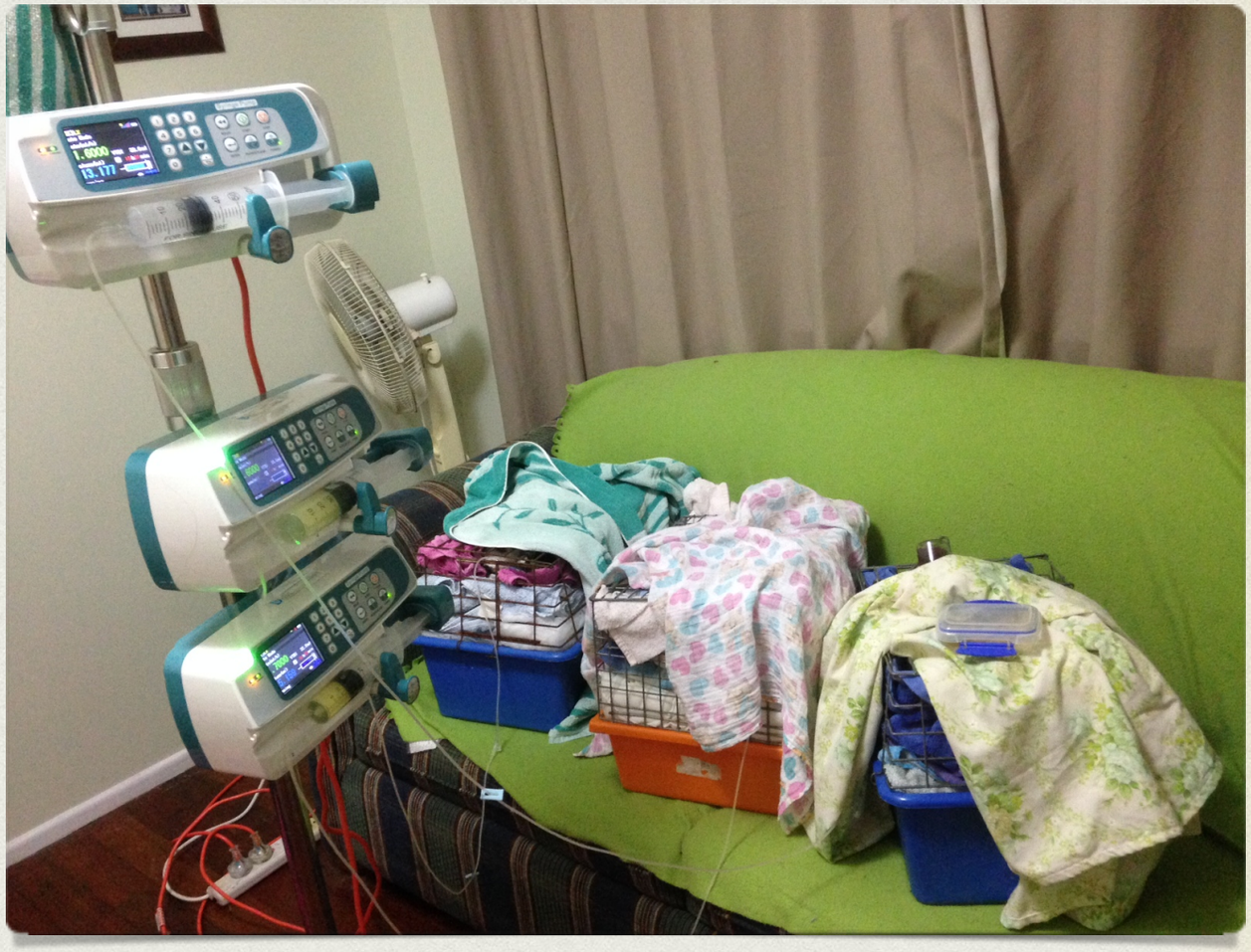
KIDNEYS

- Acute renal failure is common in heat stroke and can be fatal.
- Contributors to this are profound hypoperfusion from severe dehydration.
- Hypovolaemic and cardiogenic shock.
- Direct thermal injury.
- Cell damage due to rhabdomyolysis and the release of myoglobin from ruptured muscle cells into the blood stream.
- Signs of acute renal failure may show up earlier post insult around 24hr, but can develop into chronic renal failure after >65% of all nephrons are destroyed more than 3 weeks after the initial insult.

HEAT STROKE

BLOOD VESSELS

- Cell membrane stability is altered all over the body.
- Vascular beds all throughout the body are directly damaged by thermal injury, causing the random triggering of coagulation factors often resulting in disseminate intravascular coagulation.
- Coagulation factors are often depleted due to these damaged vascular beds, decreased production by the liver and direct thermal injury.
- Also once an animal is in shock from dehydration etc, all peripheral blood vessels constrict to maintain flow of what blood volume remains to vital organs.
- This is why neither oral , SC or IP fluids are not effective at this point.



HEAT STROKE

NERVOUS SYSTEM

- Cerebral oedema from direct thermal injury.
- cerebral haemorrhage in severe cases.
- Both of these can lead to changes in behaviour and mentation to seizures and even a comatose state in some cases.
- In some cases, damage to the hypothalamus can predispose to further cases of heat stroke due to impaired thermoregulatory mechanisms in the future.

HEAT STROKE

RESPIRATORY SYSTEM

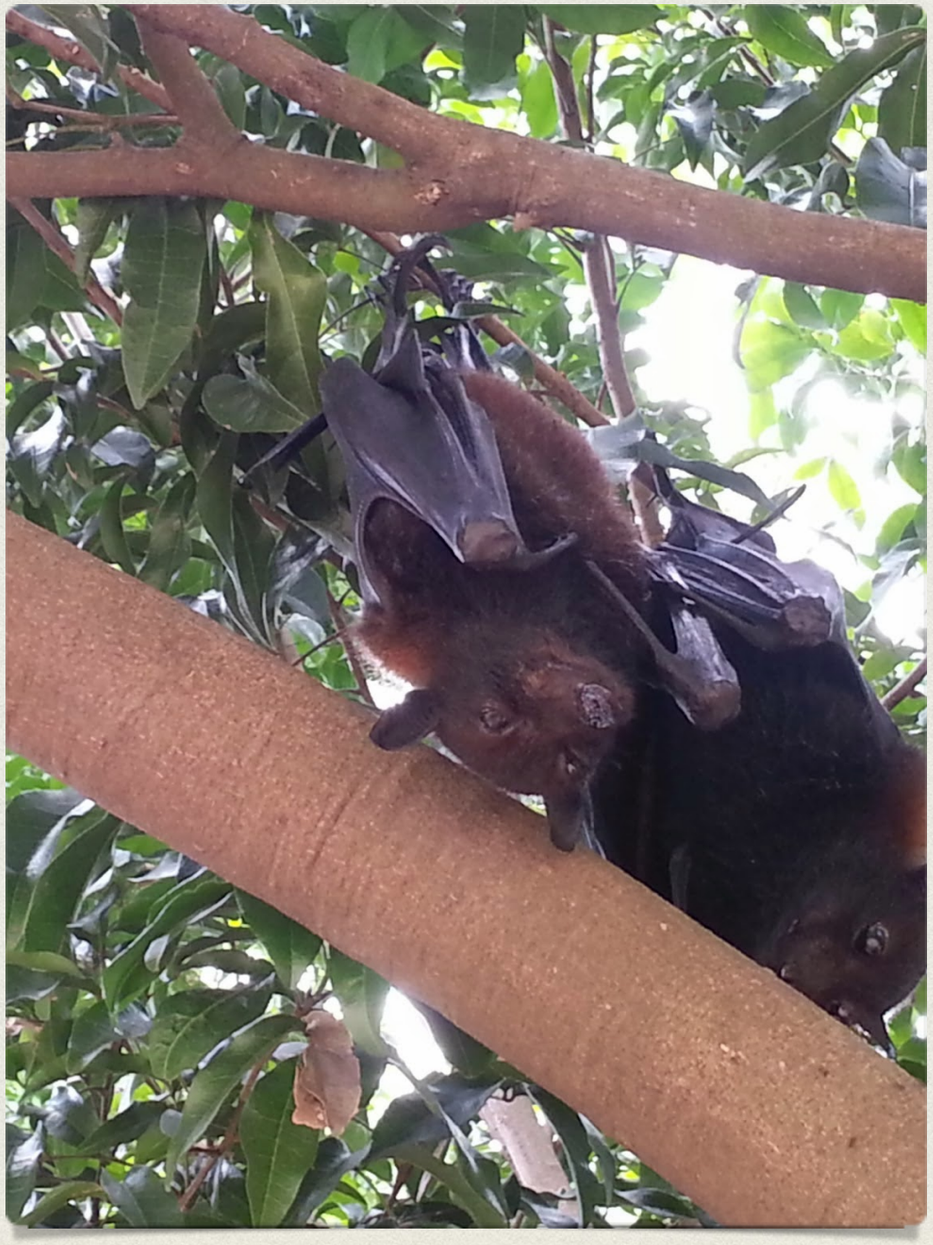
- Pulmonary oedema is most common usually days after the event.
- Can result from cardiac dysfunction.
- Damage to the vascular epithelium of the lungs.
- Or as a result of hypoalbuminaemia secondary to protein loss from the GI system or renal loss of protein or lack of production from a damaged or failing liver.

HEAT STROKE

LIVER

- Liver damage is responsible for loss of coagulation factor production.
- Blood protein loss and persistent hypoglycaemia.
- Damage to the monocyte-macrophage systems of the liver can also contribute to the development of endotoxaemia and sepsis resulting in SIRS. (Systemic inflammatory response syndrome)
- If liver damage is too extensive, liver failure can result and worsen neurological signs and lead to death from 24hrs post insult to weeks post the initial insult.

CLINICAL SIGNS OF HEAT STROKE



- The clinical signs seen are related to the body systems affected, the degree of injury to those organs and the time after initial insult.

- The clinical signs seen evolve as the animal survives insult to some organs and the resultant damage of other organ systems becomes apparent.

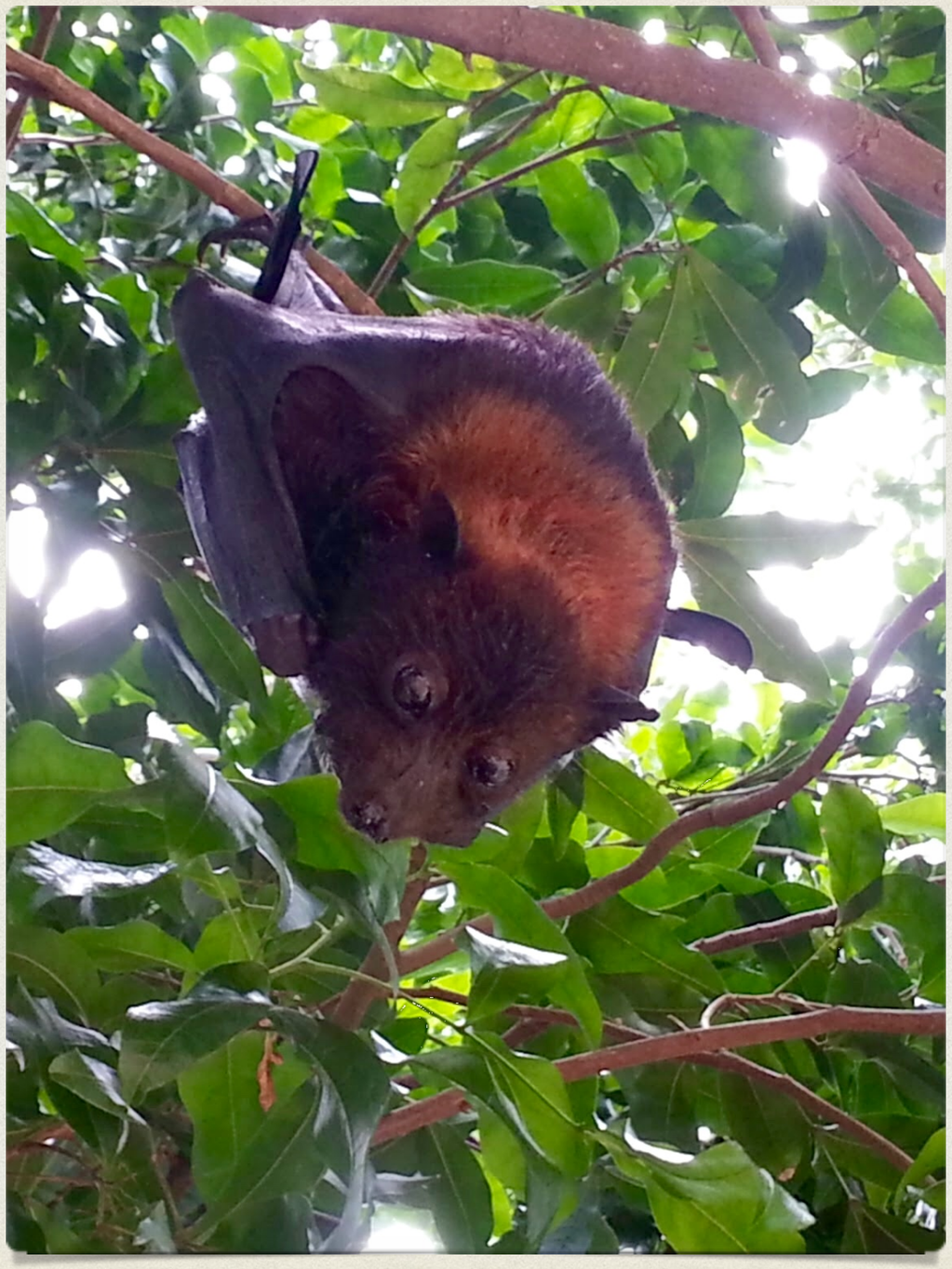
CLINICAL SIGNS OF HEAT STROKE

- Often animals that survive the initial insult, may then suffer from renal failure or complications associated with the loss of the lining of the gastrointestinal tract.

- These can then cause short and longer term complications from dehydration and electrolyte imbalances; infection and endotoxaemia to malnutrition from malabsorption which can take weeks after the initial insult to resolve as the lining of the gastrointestinal tract repairs itself.

- An understanding of the underlying processes involved, helps guide not only treatment but also rehabilitation and viability for release.

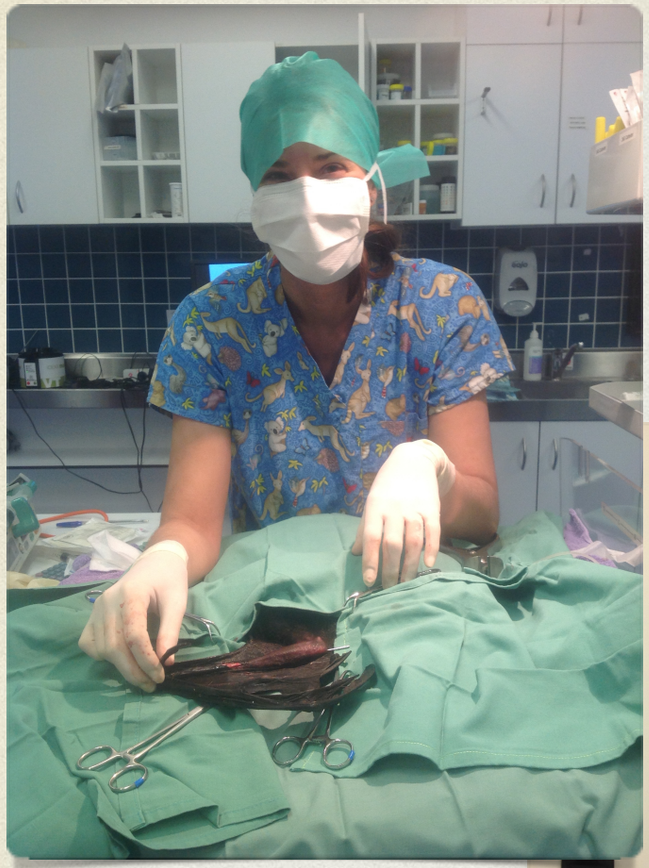
CLINICAL SIGNS OF HEAT STROKE



EARLY SIGNS

- Panting
- Hyper salivation
- Tachycardia - Fast heart beat.
- Hyperaemic mucous membranes - very red / darker than normal.
- A shortened OMMCRT - Oral mucous membrane capillary refill time or OMMCRT is defined as the time taken for a mucous membrane to return from white to original colour when firm pressure is applied and then removed.
- bounding pulses - best felt over the femur or thigh of a flying fox or just above the elbow.
- increased blood pressure
- often excited or hyperactive / distressed.
- rectal temperature of >40.5

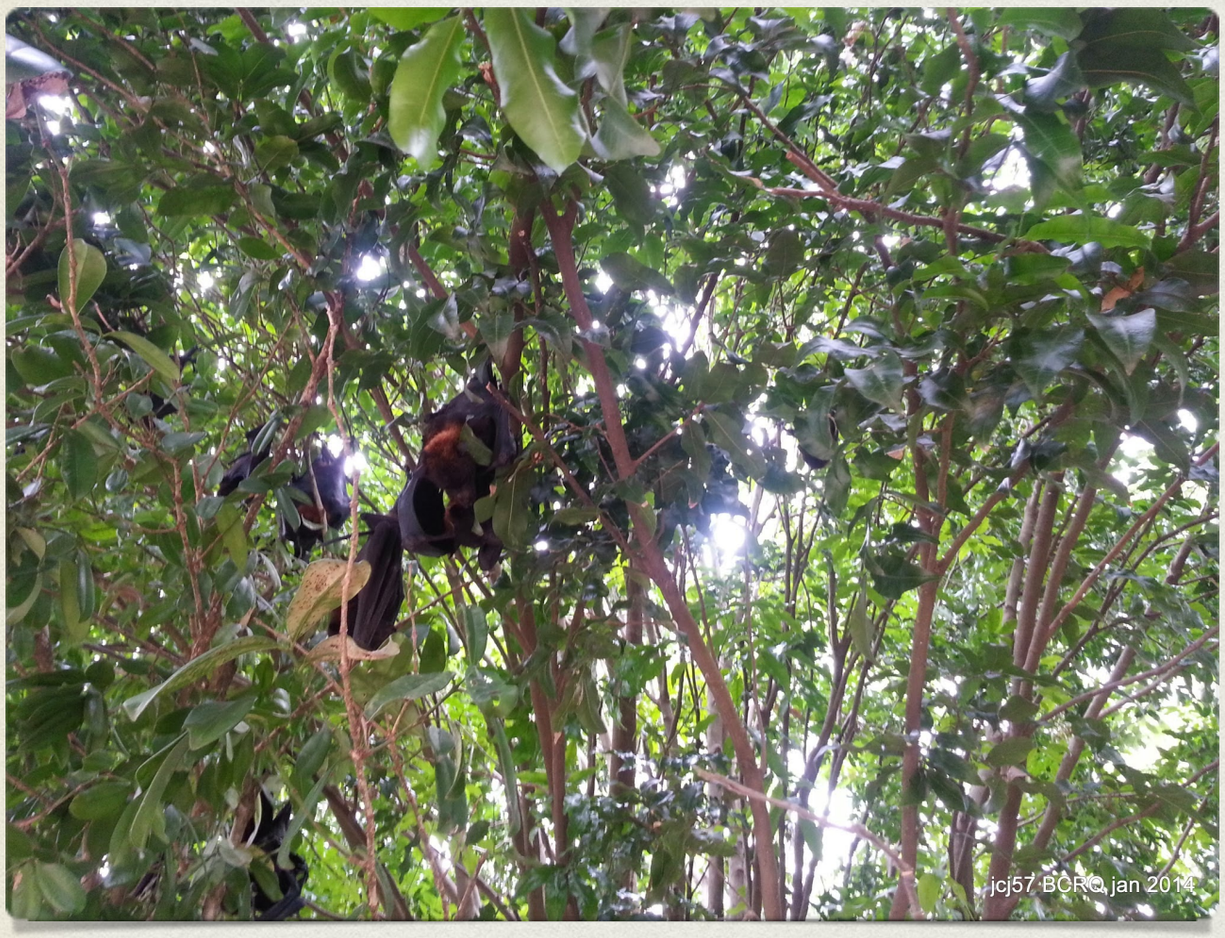
CLINICAL SIGNS OF HEAT STROKE



SIGNS OF PROGRESSION

- Signs of shock become more pronounced as the syndrome progresses.
- These animals MUST go to a wildlife hospital or require euthanasia.
- High normal or low rectal temperature
- Oral mucous membranes may vary from pale, to muddy or brick red (endotoxaemia) to cyanotic (blue / grey) if dypsnoeic from pulmonary oedema.
- prolonged OMMCRT - >3s
- lowered blood pressure
- Tachypnoea (fast breathing) and hyperventilation
- Severe dehydration - sunken eyes, wrinkling of membranes, tacky OMM and tenting of skin.

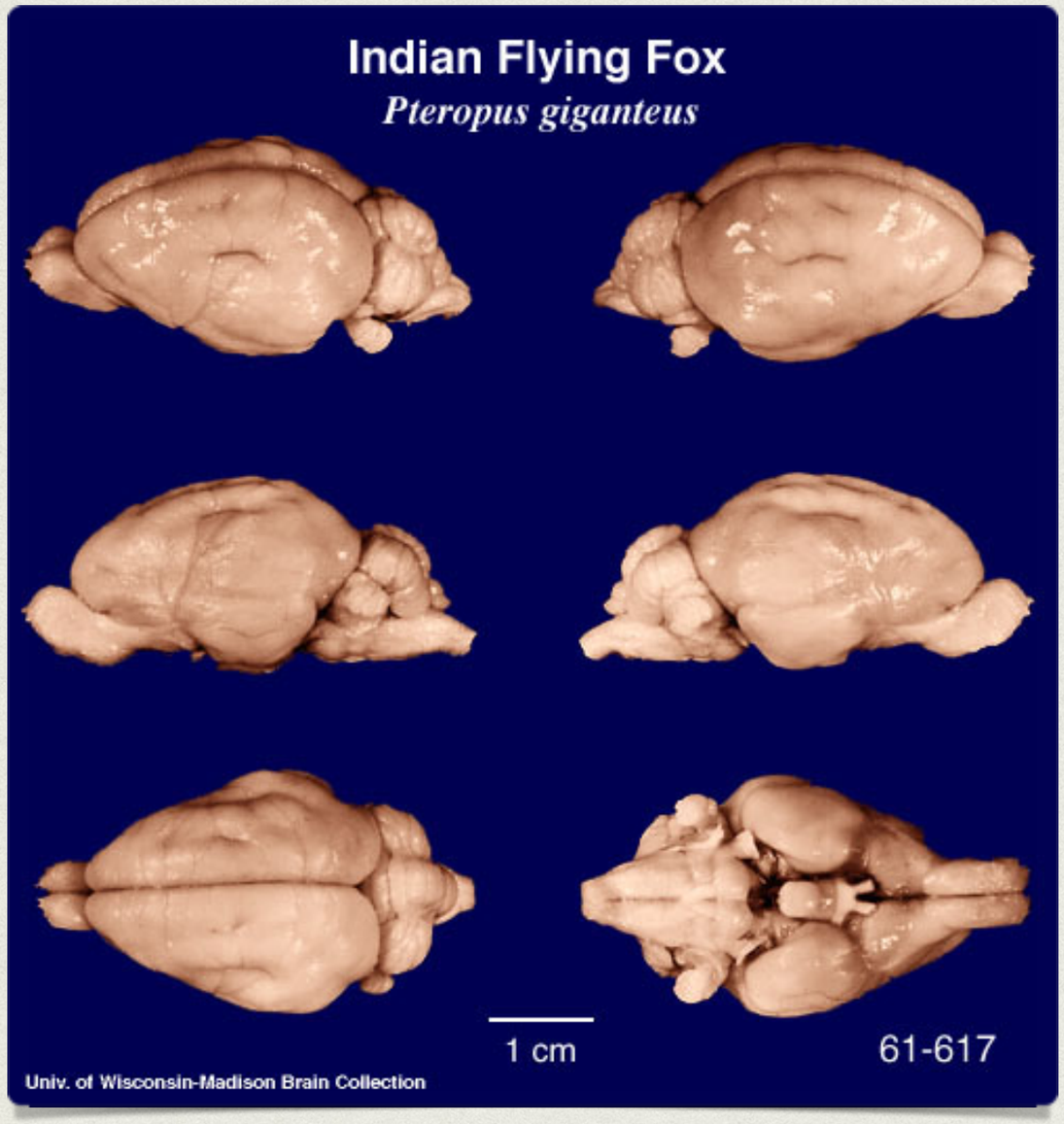
CLINICAL SIGNS OF HEAT STROKE



SIGNS OF PROGRESSION

- May be corneal ulceration due to loss of the watery component of the tear film with dehydration.
- Haematemesis - vomiting blood - may be red, brown or black depending on location of bleeding
- Haematochezia - fresh blood in faeces
- melena - digested blood in faeces leading to tarry stools
- All of the above are associate with gastrointestinal injury and sloughing of the mucosal lining - faeces are often a dark brownish red and very malodorous.

CLINICAL SIGNS OF HEAT STROKE



http://www.brainmuseum.org/Specimens/chiroptera/flyingfox/index.html

SIGNS OF PROGRESSION

- Pupils are commonly dilated (from stress) but may be constricted (miotic) if heat stroke has caused cerebral oedema or swelling of the brain.
- Altered mentation - depression / seizuring
- Cortical blindness - from direct damage to the visual cortex and CN 2.
- Respiratory distress +/- cyanosis (blue colour of OMM) later in syndrome due to pulmonary oedema.

CLINICAL SIGNS OF HEAT STROKE

SIGNS OF PROGRESSION

- DIC - Disseminate Intravascular Coagulation - Blood vessel lining damage all over the body triggers coagulation factors all over the body.
- These become depleted, resulting in random haemorrhage all over the body.
- This is seen as petechial haemorrhages visible throughout the body but visible on the oral mucous membranes.
- DIC is accompanied by evidence of random thromboembolism or clots +/- haemorrhage.
- Muscle tremors, seizures, collapse and coma often precede final cardiopulmonary arrest.

COMPLICATIONS POST HEAT STROKE

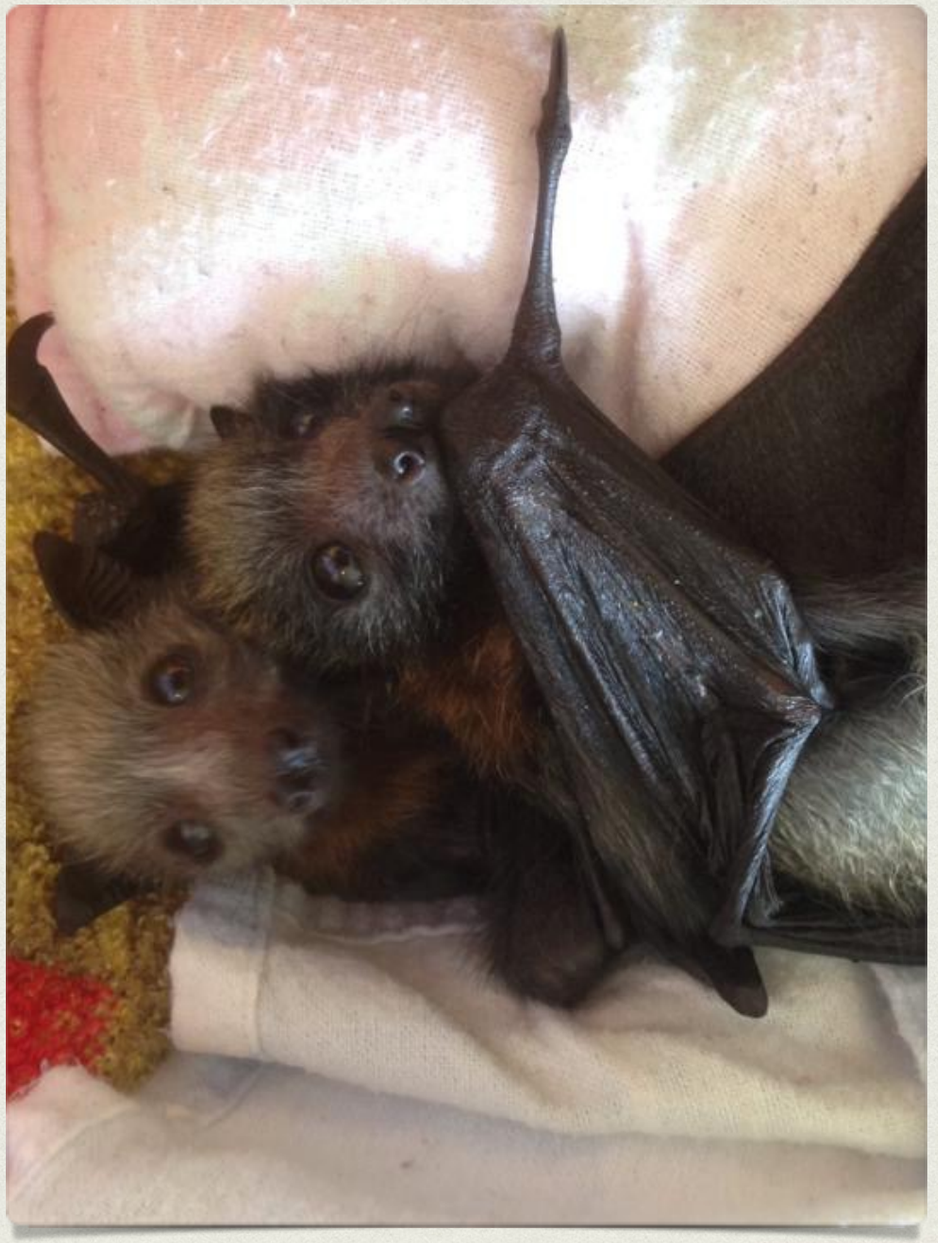
- Animals surviving the initial insults will have lost the lining of the gastrointestinal tract and therefore have minimal protection against infection entering the blood stream from the gut.

- They also have severely compromised absorption of nutrients from the gastrointestinal tract as well as significant protein and fluid losses from the gastrointestinal tract.

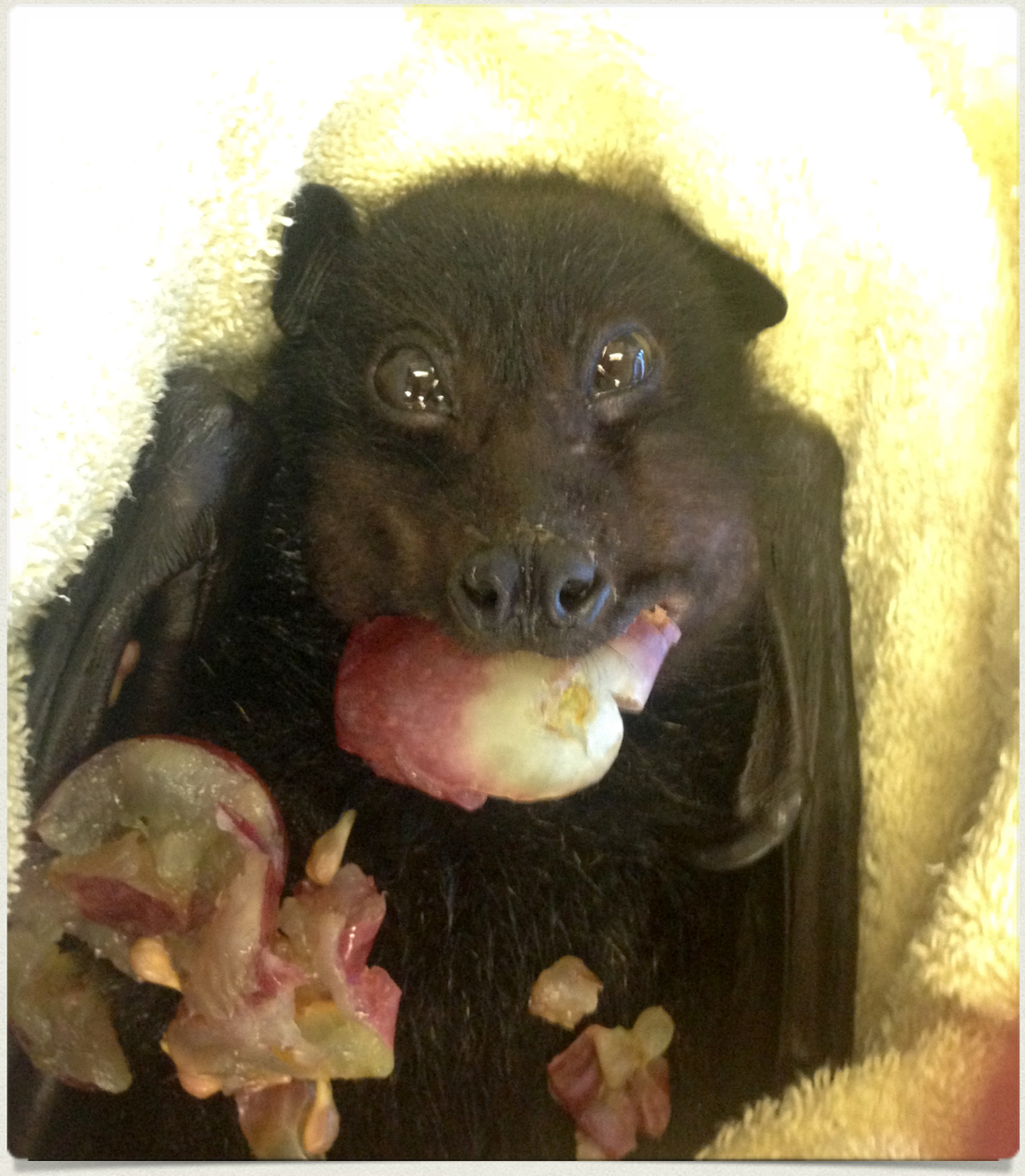
- Surviving animals also have severely compromised renal function and may develop chronic renal failure resulting in protein, glucose and electrolyte losses via the urine.

- Animals with chronic renal failure suffer from increasing ammonia circulating in the blood stream.

- This can result in some animals exhibiting ulceration of the margins of the tongue from ammonia in saliva and further blood vessel damage manifesting as a vesicular (or blistering) pattern in the vasculature of the membranes from further blood vessel damage and thrombosis.



COMPLICATIONS POST HEAT STROKE



- Maintenance of hydration and body condition, protection from infection and recovery from myopathy are the keys to getting a flying fox through heat stroke.

- Nutritional supplementation is absolutely vital at this stage to counter the loss of protein via the gastrointestinal tract and kidneys.

- Absorption of nutrition from the gastrointestinal tract will be compromised until the mucosal lining recovers and is completely replaced which can take weeks after the initial insult.

- High quality protein supplementation can be achieved via addition to smoothies and close monitoring of weight gain.

- Gentle return to exercise and movement to ensure that contraction and loss of range of motion does not result as a sequelae to muscle damage is also vitally important.

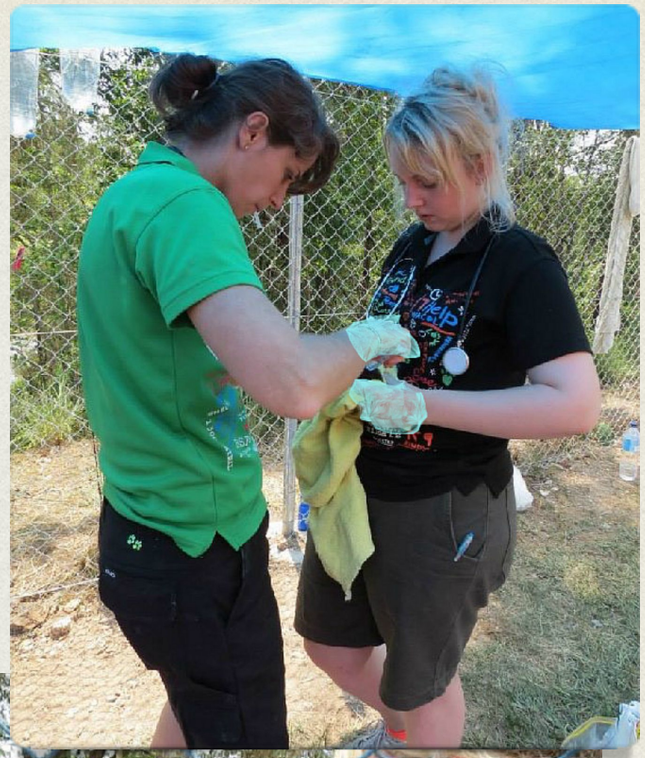
TRIAGE

- All people at site will need to be id'ed on arrival
- Sign on with a site manager
- All people need to be vaccinated
- The key is heat events is ultimately prevention of heat stroke, via actively spraying the camps with water either by large tanks mounted on utes up to water tankers.
- Divide the area up into areas actively sprayed and monitored by people
- Those animals that are low, panting and actively licking or able to be caught easily need to be collected, assessed and either taken to a wildlife hospital or immediately euthanised.
- Toe tag every animal in care to ID.
- Triage order will depend on Clinical signs seen.
- Females with dependent young, seizuring , moribund, extreme shock / dehydration top priority
- then males
- then females without young.

TRIAGE

IMMEDIATE TREATMENT ON SITE

- Rectal temperature
- Pamlin 0.5mg/kg IM if not seizuring. 1mg/kg IM or intracloacally if seizuring
- Active cooling - spraying with cool or tepid water with fanning to increase evaporative cooling - continue active cooling until temperature reaches 40 degrees.
- NB Attempting to cool faster than just spraying with cool water and fanning can potentially initiate a shivering reflex making the condition worsen.
- No PLR, no corneal reflex, completely moribund with OMMCRT >4s immediate euthanasia
- Temp of > 42degrees at temperature taking despite active cooling measures - immediate euthanasia.
- Fluids SC - 10% body weight - (1/2 x Hartmann's and 1/2 x 2.5%Glucose/Saline)
- Take all animals to a wildlife Hospital.



IN-HOSPITAL TREATMENT

COOLING

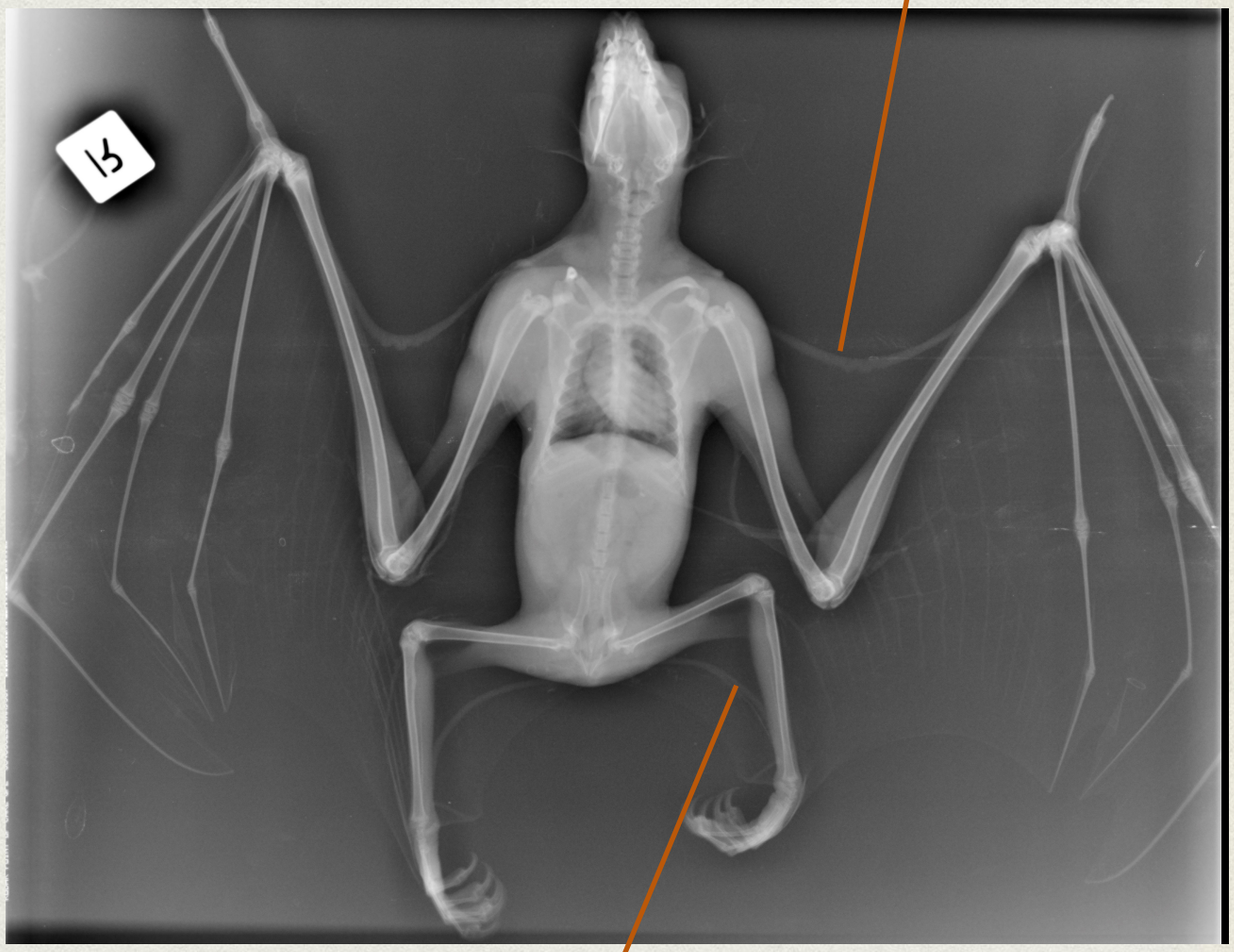
- Wet patient with cool or tepid water.
- enhance evaporation by providing air movement around patient
- Important not to use ice or cold water as cutaneous vasoconstriction can lead to a loss of heat dissipation, shivering and therefore increased heat production
- Alcohol on skin can potentially lead to cutaneous absorption and intoxication from cutaneous vasodilation and is therefore not recommended.
- Cooling measures should continue on the animal once at hospital also if the core temperature is still more than 40°C.
- If the core temperature is less than 40 degrees discontinue cooling to avoid inducing hypothermia.

IN-HOSPITAL TREATMENT

- Iso/O2 anaesthetic - via mask
- Supplemental oxygen only if moribund
- Place IV catheter
- Saphenous (interfemoral vein) -best tolerated.
- Cephalic vein - often needed temporarily in severe shock if interfemoral not available.
- a PCV, Total protein, biochemistry and a platelet count should be done to gain a base line level for treatment.
- It must be noted that nearly all drugs used on wildlife are defined as "off-label" meaning that drug companies have not specifically tested the drug for that species but scientists and vets have extrapolated their use for wildlife.

IN-HOSPITAL TREATMENT

- The chest should also be auscultated
- Oral mucous membrane refill time, colour, and moisture should also be noted
- Radiographs should be taken
- Fluorescine stain of both eyes as flying foxes are very prone to exposure ulcers from the drying out of the liquid component of the tear film.
- Conoptal eye ointment should be placed if any eye ulcers are detected twice daily until 48 hrs after the last negative fluorescine stain taken every 48 hrs.



IN-HOSPITAL TREATMENT

MODECATE (FLUPHENAZINE DECANOATE)

- 2.5mgkg IM once. (off label)
- Antipsychotic drug commonly used in macropods and other animals (off label) to prevent capture myopathy.
- In the author's opinion, modecate is also of considerable benefit in the prevention and treatment of capture myopathy in flying foxes.
- Onset of action of approximately 24hrs and lasts approximately 3 weeks.

VITAMIN E/SELENIUM

- 1ml per 30 kg IM once
- Acts to mop up free radicals released by myopathy and heat stroke

IN-HOSPITAL TREATMENT

IV FLUIDS

- In severe heat stroke, the patient will have relative and absolute fluid losses resulting in hypovolaemia and perfusion deficits requiring aggressive treatment.

- Hypoglycaemia is also a common factor in heat stroke, especially in flying foxes, so crystalloid treatment often involves the use of glucose in fluids.

NEED TO ADMINISTER RAPIDLY JUST ENOUGH FLUIDS TO IMPROVE

- oral mucous membrane colour
- OMMCRT
- Blood pressure
- Heart rate
- Pulse Quality
- Urine production

IN-HOSPITAL TREATMENT

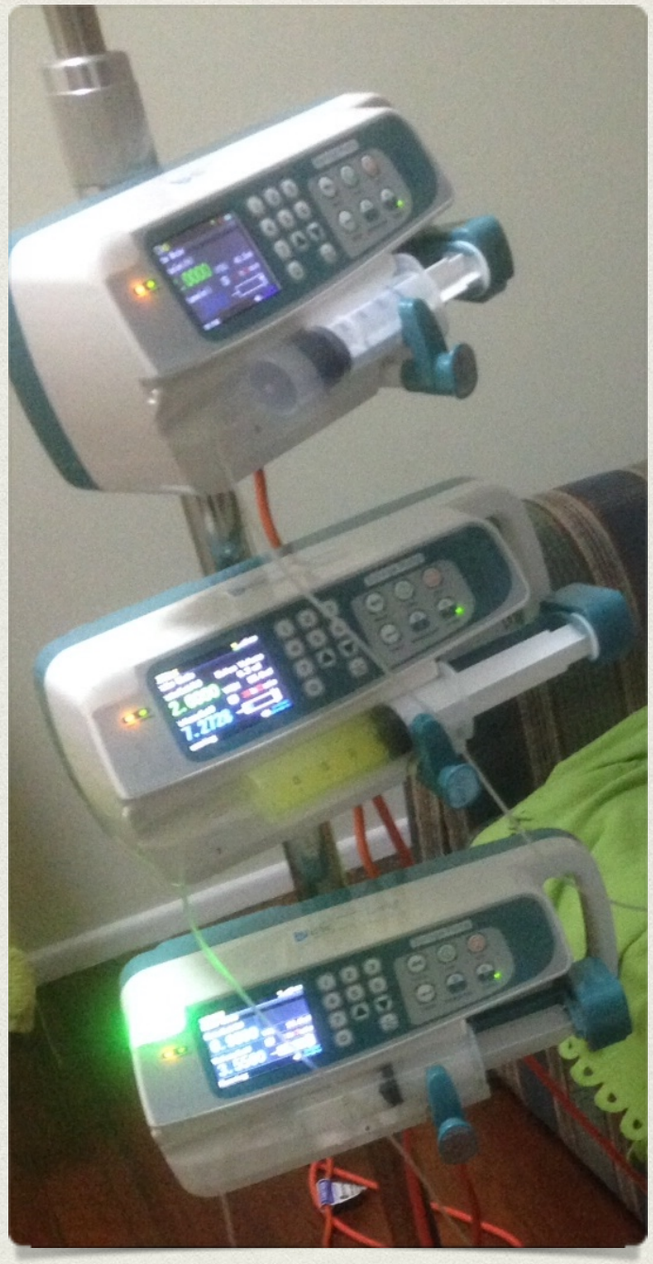
IV FLUIDS

- In cases of severe shock, a combination of crystalloids (hartmann's and 2.5%Glucose/ saline) and Colloids (eg; Dextrans. hetastarch) may be required to restore normal perfusion.

- Crystalloids - Usually 1/2 shock rates of 45ml/kg/ hr are used (Composed of 1/2 Hartmann's and 1/2 2.5%Glucose/saline) are used to start to effect then double maintenance or 6ml/kg/hr.

- Colloids expand the intravascular volume and aim to counter the fluid loss from the increased vascular permeability associated with heatstroke.

- If colloids are used, usually hetastarch, voluven or gelofusine is used at 5ml/kg slow IV as a bolus.

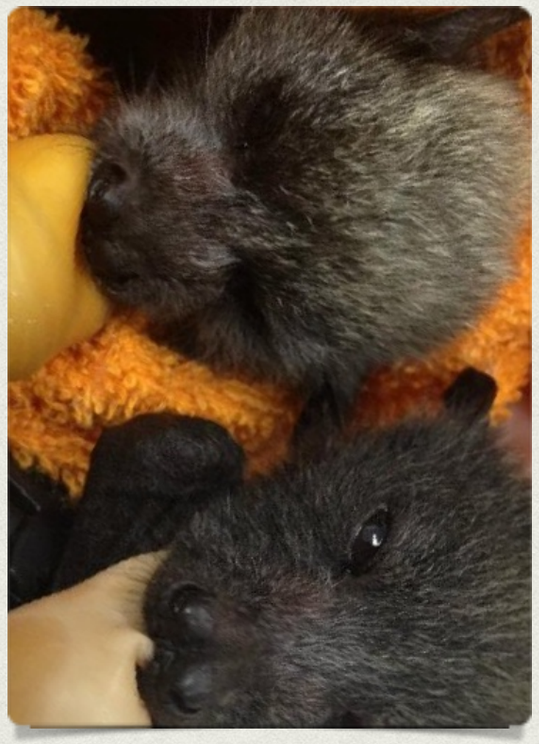


IN-HOSPITAL TREATMENT

CORTICOSTEROIDS

- The only corticosteroids used in the treatment of heat stroke at the RSPCA Wildlife hospital is soludeltacortef or methyl prenisolone sodium succinate. It is a very short acting corticosteroid.
- When using corticosteroids the advantages need to be balanced against the disadvantages.

Advantages of Corticosteroid use:



- Aids in shock reversal.
- Promotes gluconeogenesis or glucose production by the liver.
- Stabilises cell membranes
- suppress inflammation
- reduces cytokine production (products of cell damage)
- reduces endotoxaemia (common in heat stroke in flying foxes due to GI tract lining break down)
- - antipyretic properties

IN-HOSPITAL TREATMENT

CORTICOSTEROIDS

Disadvantages of corticosteroid use:

- But it can also cause worsening GI ulceration and promote ischaemic damage of the kidneys
- It is the authors opinion that the use of solu-delta-cortef in heat stroke treatment is highly advantageous and feels that the advantages highly outweigh the disadvantages.

GASTROINTESTINAL PROTECTANTS

ZANTAC (Ranitidine)

- 1mg/kg IV or SC BID
- H2 receptor antagonist
- reduces acid output in the stomach
- has prokinetic activity (helps with gut movement)

IN-HOSPITAL TREATMENT

GASTROINTESTINAL PROTECTANTS

CARAFATE (Sucralfate / carafate)

- 1 pinch added to each smoothie
- Acts as a locally acting treatment for GI ulcers

BROAD-SPECTRUM ANTIBIOTICS

- Ticarcillin (Timentin) - 40mg/kg BID
- Fortum (Ceftazidime) - 20mg/kg BID
- These 2 antibiotics are most commonly used due to their strength and speed of action against gram negative bacteria most commonly associated with gut lining slough and the enterotoxaemia that follows.

http://extraordinarylight.blogspot.com.au/2010/12/magic-in-bush.html

IN-HOSPITAL TREATMENT

ANTI-FUNGAL AGENTS

- Nystatin (nilstat) Antifungal
- 50000I.U./kg orally twice daily
- Always use when compromised animals are on antibiotics.
- The loss or disruption of normal gut flora as well as antibiotics provide conditions ideal for the growth of Candida albicans, a yeast, which can further compromise recovery of the normal gut mucosa.

IN-HOSPITAL TREATMENT

MANNITOL 20 (OSMITROL 20%)

- 1ml/kg slow IV
- Osmotic diuretic - draws fluid out of tissue into blood vessels.
- Used in cases of increased intracranial pressure

PAMLIN (DIAZEPAM)

- 1.0mg/kg IM or slow IV or intracloacally
- Anxiolytic
- muscle relaxant
- reduces myopathy
- Stops status epilepticus.

http://www.abc.net.au/radionational/programs/summerfeatures/give-a-bat-a-bad-name/4397186

MONITORING

- Chest auscultation
- light response and neurological signs
- Oral mucous membranes
- Oral mucous membrane capillary refill time.
- Urine production - volume, Urine specific gravity.
- Blood biochemistry and electrolyte monitoring.

http://www.abc.net.au/radionational/programs/summerfeatures/give-a-bat-a-bad-name/4397186

CONTRAINDICATIONS

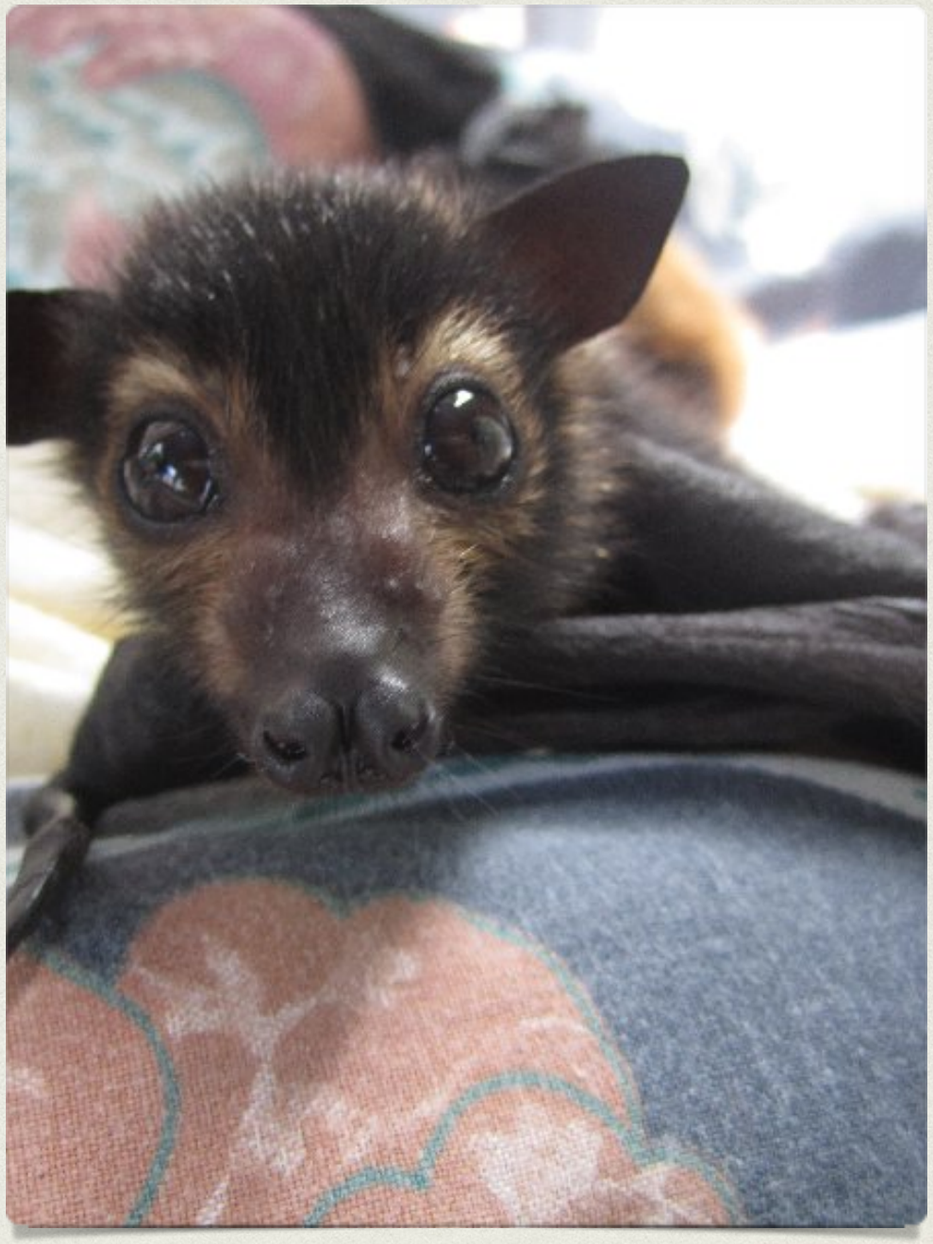
- It must be stressed that any non-steroidal anti inflammatories e.g; meloxicam(metacam), aspirin etc are highly contraindicated in the treatment of heat stroke.

- Animals are highly dehydrated and in shock with compromised renal function and an already damaged gastrointestinal tract.

- Non-steroidal anti inflammatories provide no advantageous effects and further contribute to renal and gastrointestinal damage as well as clotting issues.

CONCLUSION

- Prevention of heat stress becoming heat stroke is the key to higher survival rates.
- This will require significant cooperation between Wildlife groups, hospitals, EHP and local government.
- We are, after all, trying to achieve the same thing - better welfare for wildlife and hopefully future survival of an incredibly misunderstood and important group of beautiful animals.
- There is no doubt that this year's heat event and those to come will have significantly harmed the future survival of some of the vulnerable species of flying fox.

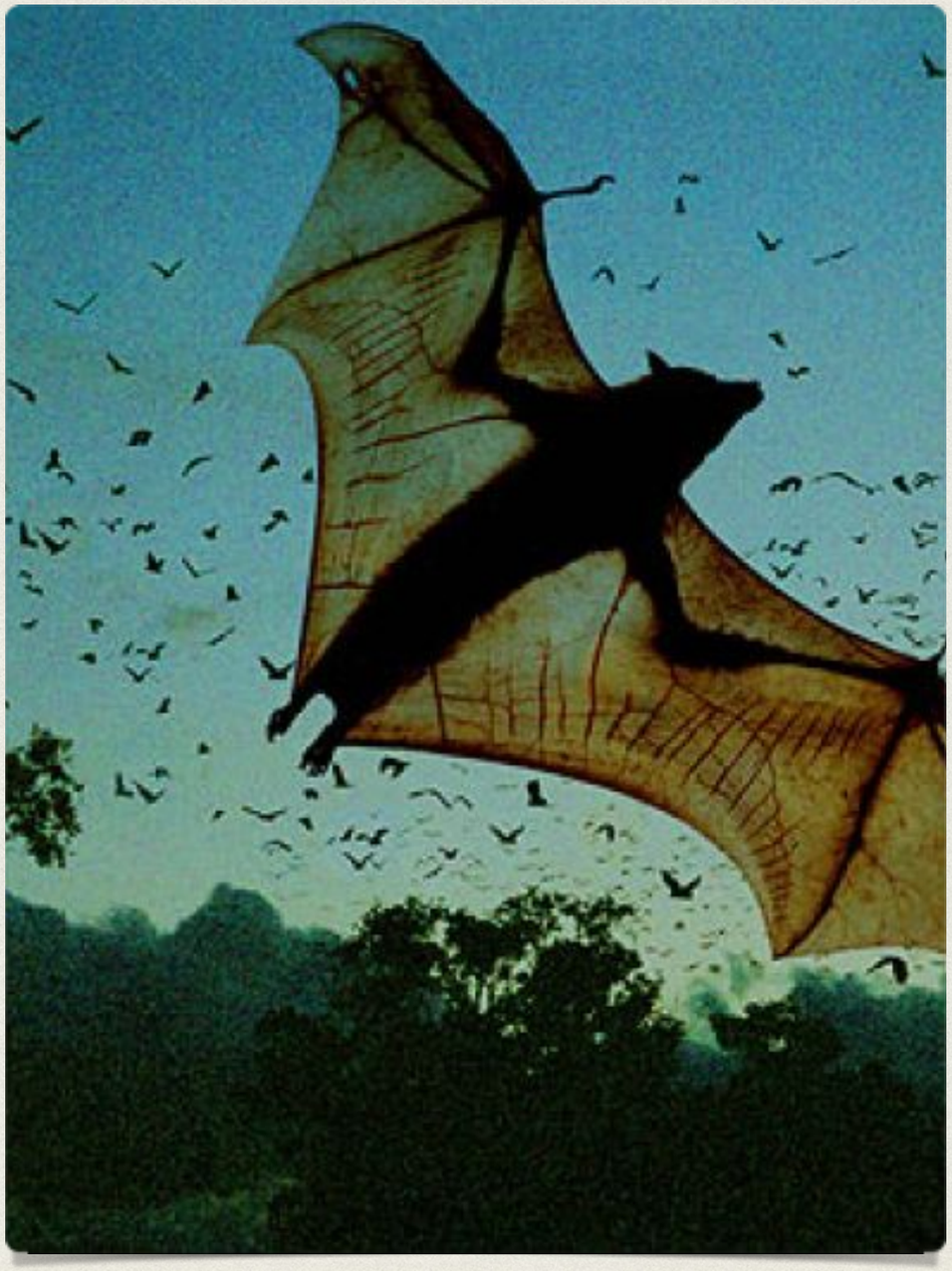


CONCLUSION

- It has been stated, that in an era where such an important species is being actively vilified and killed for what amounts to mainly political gain and media fuelled hysteria, more information needs to be dissipated about what will happen to Australia's environment if they succeed in killing every last flying fox in Australia.

- The Australian landscape, our lifestyle, agriculture and wildlife will be negatively affected and no-one will be able to say we didn't see it coming.

- Justin put it perfectly when he described the bat die-offs as the "canaries in the coal mine" - these need to be seen as our official warning and chance to not only save bats but also ourselves and our children.



"Never doubt that a small group of thoughtful, committed citizens can change the world; indeed, it's the only thing that ever has"

-Margaret Mead

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