

Mansfield Veterinary Clinic

# May News

## Survivors Party



This summer we had another bad season for snakes, with 37 pets (29 dogs, 8 cats) being brought into the clinic with suspect snakebites. Of these, 9 were proven not bitten, 2 did not make it to the clinic alive, 3 died despite treatment (1 with fluids, 2 with fluids and antivenene), and 23 survived with treatment (1 cat on fluids and pain relief alone).

As you may have read in previous newsletters (March and April) we have had some amazing stories of survival. This is due to a combination of seriously tough animals, perseverance from owners and our own increasing ability to treat snakebites successfully.

As a celebration for these tough pets, we are having a survivors party on Thursday the 17th of May at 5pm. We invite all survivors and their supporters to come along. We will be having nibbles, hosted by our own survivor, clinic cat 'Lena' (November 2004, 2 vials of antivenene.)

Hopefully, we are at the end of the season, and we all look forward to some cold (and very wet) days when all the snakes will be hibernating.

**NEXT  
PUPPY PARTY  
AND PUPPY  
PRE-SCHOOL  
STARTS ON  
WEDNESDAY,  
9TH OF MAY  
AT 4PM.  
BOOKINGS  
ESSENTIAL.**

## Vet Stat

Part of our success in snakebite treatment comes from the ability to monitor a patients progress. Recently we purchased a new piece of equipment, the Vet Stat machine, which will enhance this ability.

The Vet Stat is capable of analyzing blood samples for electrolyte and blood gas balances. We often suspect our snakebite patients of having changes in these balances, however having to send samples to Melbourne and waiting 24 hours for results, we usually rely on educated guesses and response to treatment instead. Having these answers immediately, we hope will increase survival and speed recovery.

These analyses will also be of use in our day to day treatments. They will give us more information for all sick animals and also for pre-anaesthetic blood testing, adding another element to safe anaesthesia.



## Taken in Hook, Line and Sinker

While out fishing over Easter, Elmo was a bit hungry and decided to snack on a bit of corn. Unfortunately, he did not notice the hook, line and sinker attached. So, into the vet clinic he came, with the remnants still hanging out his mouth.

The first step was to take x-rays to determine where the hook was situated, which can be anywhere from the mouth onwards. In Elmo's case, the hook had become stuck in his oesophagus about halfway between the mouth and stomach. This can be a very difficult position, because surgery would involve opening the chest to access the oesophagus (a risky procedure), and healing of the oesophagus after surgery is not very good.

Fortunately for Elmo, the clinic owns an endoscope. The endoscope is a fiberoptic instrument which can be used to pass into small passages, such as the oesophagus, to directly visualise what's inside. The fish hook was clearly visible embedded in the oesophageal wall, still with corn attached, and luckily having caused minimal damage at this stage.

*“Fish hooks can become lodged in the oesophagus, the stomach or the intestine. Sometimes abdominal surgery is required to remove them.”*

The next step was quite difficult in that the hook had to be manipulated remotely, using instruments long enough to reach the hook, while being held from outside the mouth. Particular care had to be taken not to damage the wall of the oesophagus with the hook and barb, as mentioned earlier, healing of this region is slow and can cause strictures.

After some time, the hook was extricated from the wall and removed via Elmo's mouth, with no further damage. Elmo was taken off food for 24 hours and sent home on a soft diet and antibiotics for a few days.

## Type II Ostertagiasis

The most important type of worm encountered in cattle is the brown stomach worm, *Ostertagia ostertagi*, which is found in the abomasum (one of the stomachs). The worm causes two forms of disease:

Type I is caused by large numbers adult worms in the abomasum, leading to weight loss and diarrhoea.

Type II is caused by worm larvae emerging from the abomasal wall. When the larvae are ingested by cattle from the paddock, a proportion of them will become inhibited in the wall of the abomasum. Over time, the numbers of inhibited larvae build up. At the beginning of autumn, an unknown signal from the environment (probably rain) causes synchronous emergence of these inhibited larvae, resulting in massive damage to the abomasal lining.



Cattle affected are usually 2–4 years of age, old enough to have a good build up of larvae, but too young to have developed good immunity. Stresses such as calving can also lead to emergence.

Signs include weight loss, diarrhoea, ventral oedema (bottle jaw) and death, and is commonly mistaken for liver fluke disease. Faecal egg counts will most likely be low, as the larvae are incapable of producing eggs. Blood tests for the stomach enzyme 'pepsinogen' are diagnostic.

Prevention is the key, once symptoms occur, management is far more difficult. We suggest drenching with a macrocyclic lactone (eg. Cydectin) in late summer to kill off the suppressed larvae and paddock management to prevent build up in young cattle.