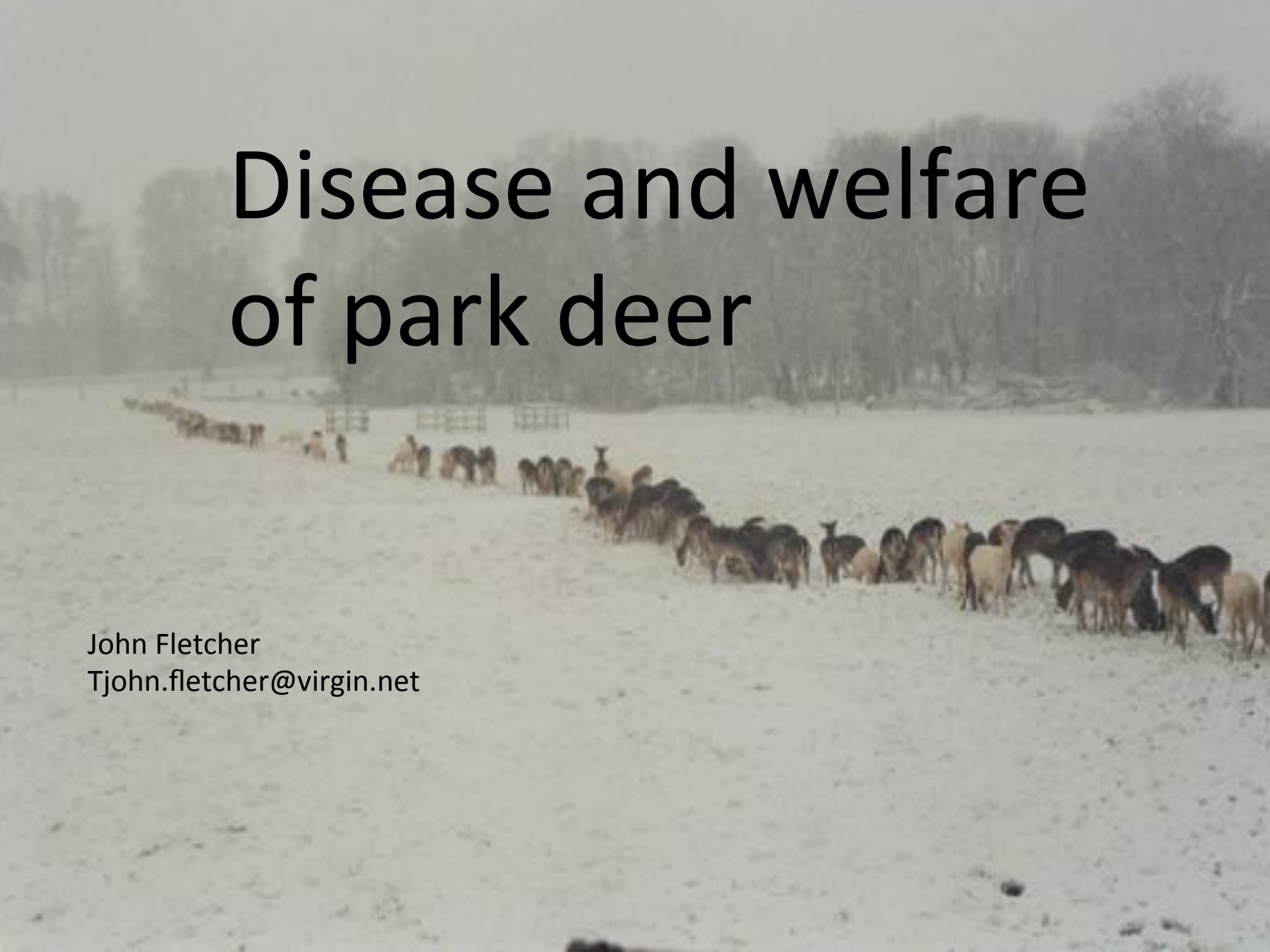


Disease and welfare of park deer

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Some diseases that might be diagnosed or suspected in park deer.

Mineral deficiencies: copper, selenium, cobalt, iodine

Parasites: external – keds, lice,
internal – lungworm, warbles, gastrointestinal nematodes, liver fluke

Bacterial: Tuberculosis, Johne's disease, Pasteurellosis, Necrobacillosis, Yersiniosis

Viral: MCF, IBR (hvc-1), parapox,

Protozoal: red water, cryptosporidiosis

Management related: Trauma, post capture myopathy, rumenal acidosis, CCN/PE
due to thiamine deficiency, water intoxication etc

Absent but relevant: Chronic wasting disease (CWD), Foot and Mouth,

Plant poisoning: foxglove, daffodil, acorn

Most traditional deer parks have no handling system .

In feed drugs are a last resort and usually ineffective – so deer parks **cannot generally treat disease (at least with drugs)** – they must manage their herds so as to **prevent** disease.

As a result management has to be by skilful adjustment of deer numbers to suit available grazing by careful, planned **culling**.

Pasture improvement will allow parks to carry more deer and for deer to perform better

In addition deer keepers will always need to **feed** their deer – even if a park had very low numbers of deer feeding would always be needed at some times of the year.

Because deer have low energy reserves, feeding must be started early – once deer are in poor condition during the winter it is very difficult to restore condition.

In some parks darting deer with a tranquilliser may be a realistic option for handling occasional animals in emergency – it should never be a routine means of handling or treating deer.



Some parks may construct temporary handling facilities - if they wish, for example, to transport a large number of deer to another property – it is not a recommended way of routinely treating park deer to prevent disease.



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They obviously are adapted to calve in the spring and lactate through the summer – so their energy demands are highly seasonal

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Fallow deer are ideal parkland species being less likely to show aggression to people than red deer



Even red deer, in this case, tame but not artificially raised, can be quiet enough to be sat upon **during the summer**



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They obviously are adapted to calve in the spring and lactate through the summer – so their energy demands are highly seasonal

Summer mortality is very unusual.....



It is not what we expect but red deer and presumably fallow, are very poorly insulated compared to sheep and cattle – they survive by moving into shelter which may be difficult when they are enclosed in a park

CALCULATED LOWER CRITICAL TEMPERATURES FOR RED DEER, SHEEP AND CATTLE (SIMPSON et al., 1978).

WIND SPEED	RED DEER	SHEEP	CATTLE
10 m./min.	+5°C	0°C	-5°C
250 m./min.	+15°C	+12°C	+6°C

Not only are deer poorly insulated but they have low energy reserves.....

CALCULATED ENERGY RESERVES FOR RED DEER, SHEEP AND CATTLE (SIMPSON et al., 1978).

WHERE HEAT LOSS EXCEEDS M.E. INTAKE BY 20%

RED DEER	SHEEP	CATTLE	
13-52	223-286	181-271	(DAYS)

Almost all mortality in deer whether wild or in parks occurs in winter and spring









Park keepers need to maintain good pasture (ca. 12 cm) during summer so that hinds can rear calves strong enough to thrive through their first winter.

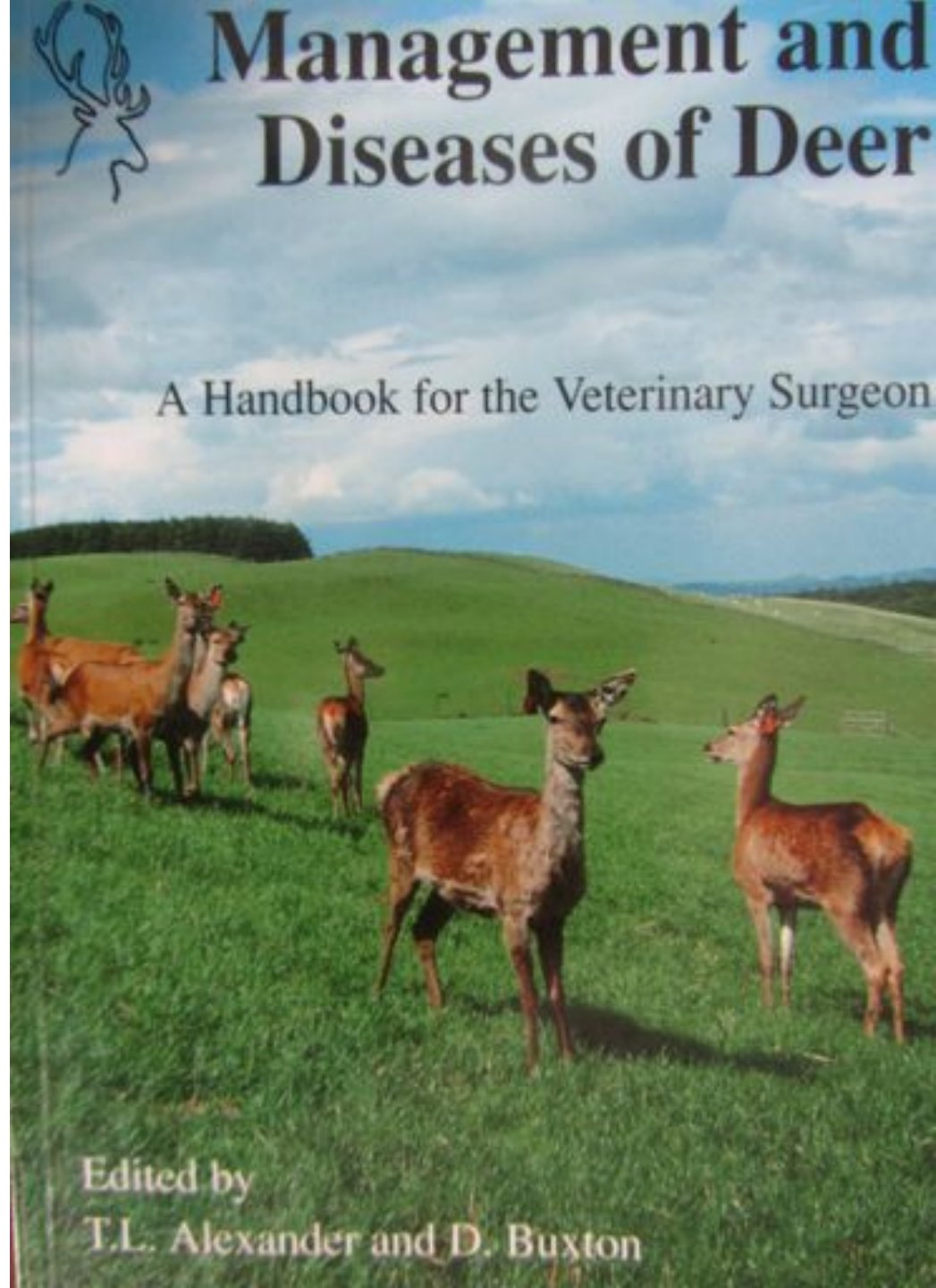


Almost all diseases in parks are the result of overstocking, poor pasture management and inadequate feeding.

Consult Veterinary Deer Society website where Management and Diseases of Deer can be downloaded free of charge. This provides nutritional requirements of red deer at different seasons in work by Clare Adam....

It also contains information on most diseases diagnosed in deer.

Now out of print but also very valuable is Jerry Haigh and Bob Hudson's 'Farming Wapiti and Red Deer'.



Copper deficiency....

Calves are born with good copper reserves but where pasture is poor and copper deficient signs may appear by late summer – presenting as swollen joints in calves. ‘swayback’ or enzootic ataxia usually appears in yearlings and adults. Liver samples from a number of deer are best to assess likelihood of deficiency.

Reduce stocking density & consider treating pasture to remedy.

Hind with enzootic ataxia due to copper deficiency (courtesy Jerry Haigh)



Fig. 15-9 Enzootic ataxia in red deer. Note poor condition. (Courtesy Colin Mackintosh.)

Selenium deficiency.

Has been reported once in SE England where deer were not thriving and also in New Zealand.

Most likely on intensively managed pasture in Se deficient areas.

Concomitant Vit E deficiency likely.

Both locations had a history of Se deficiency in local sheep flocks.

Blood sampling for Se should take into account Vit E levels

Response to single subcutaneous injection – be aware of toxicity.

Johne's disease – problems with diagnosis,
vanishes clinically if pasture well maintained and low stocking density.

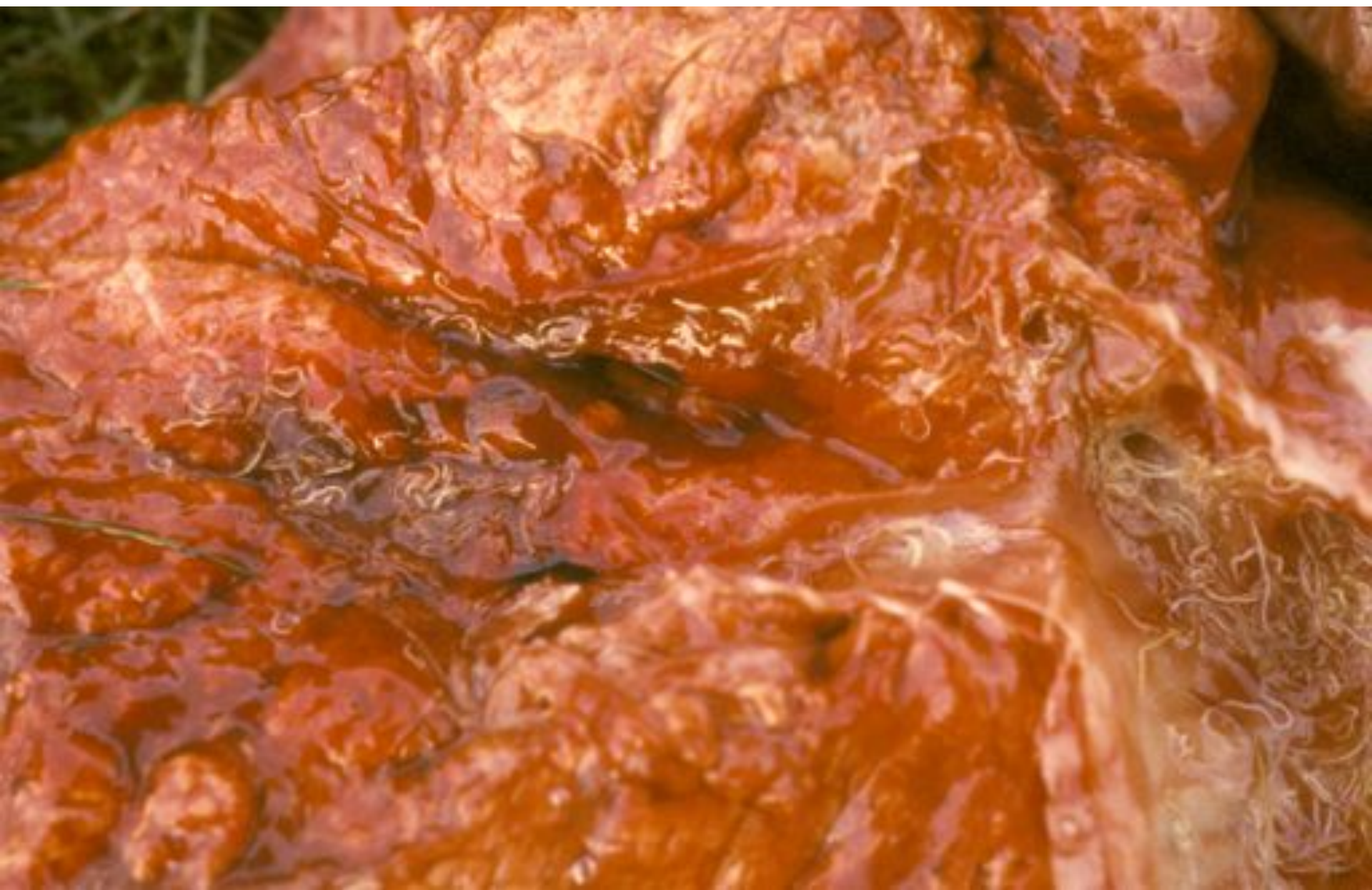




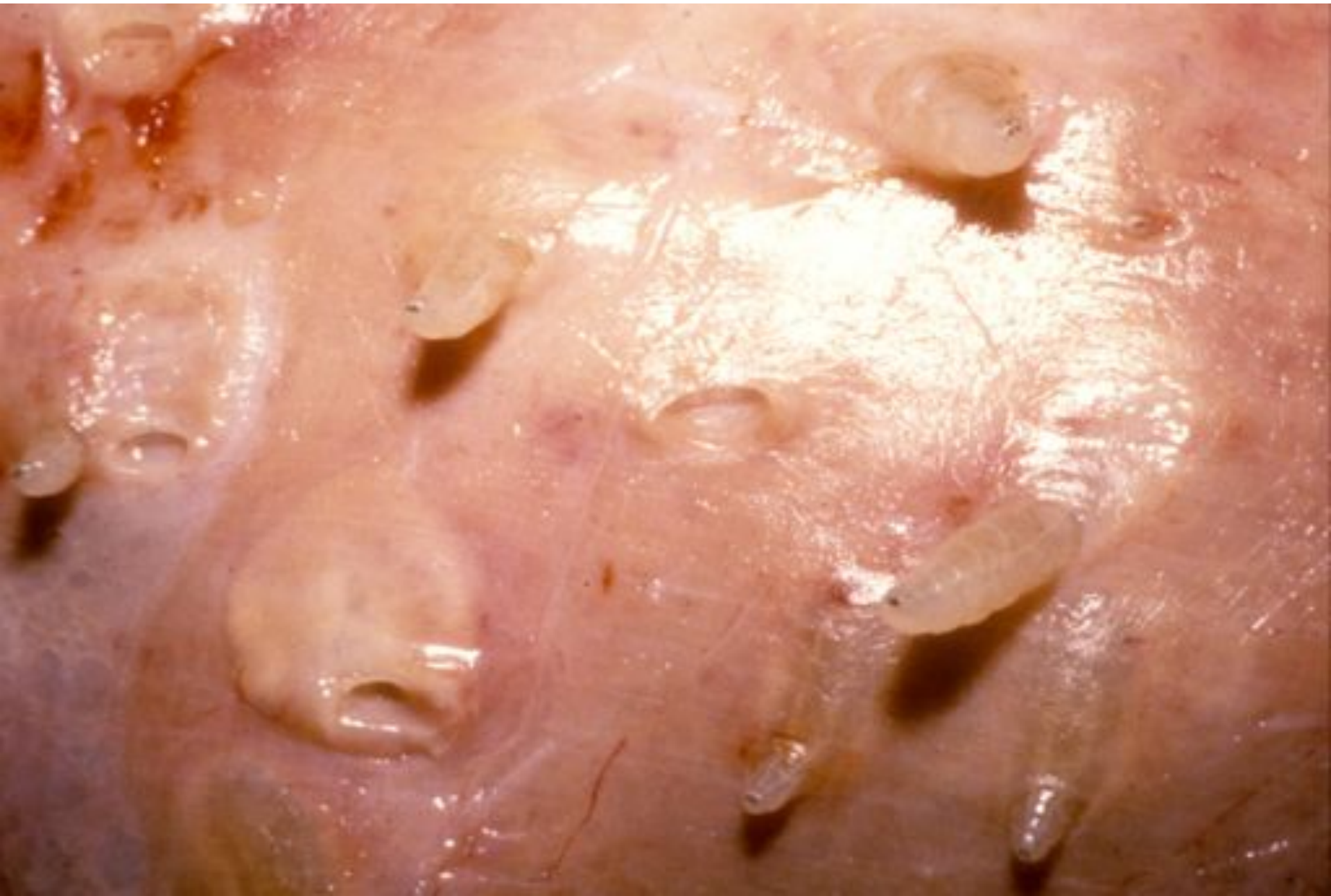






















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cm

Veterinary Laboratories Agency

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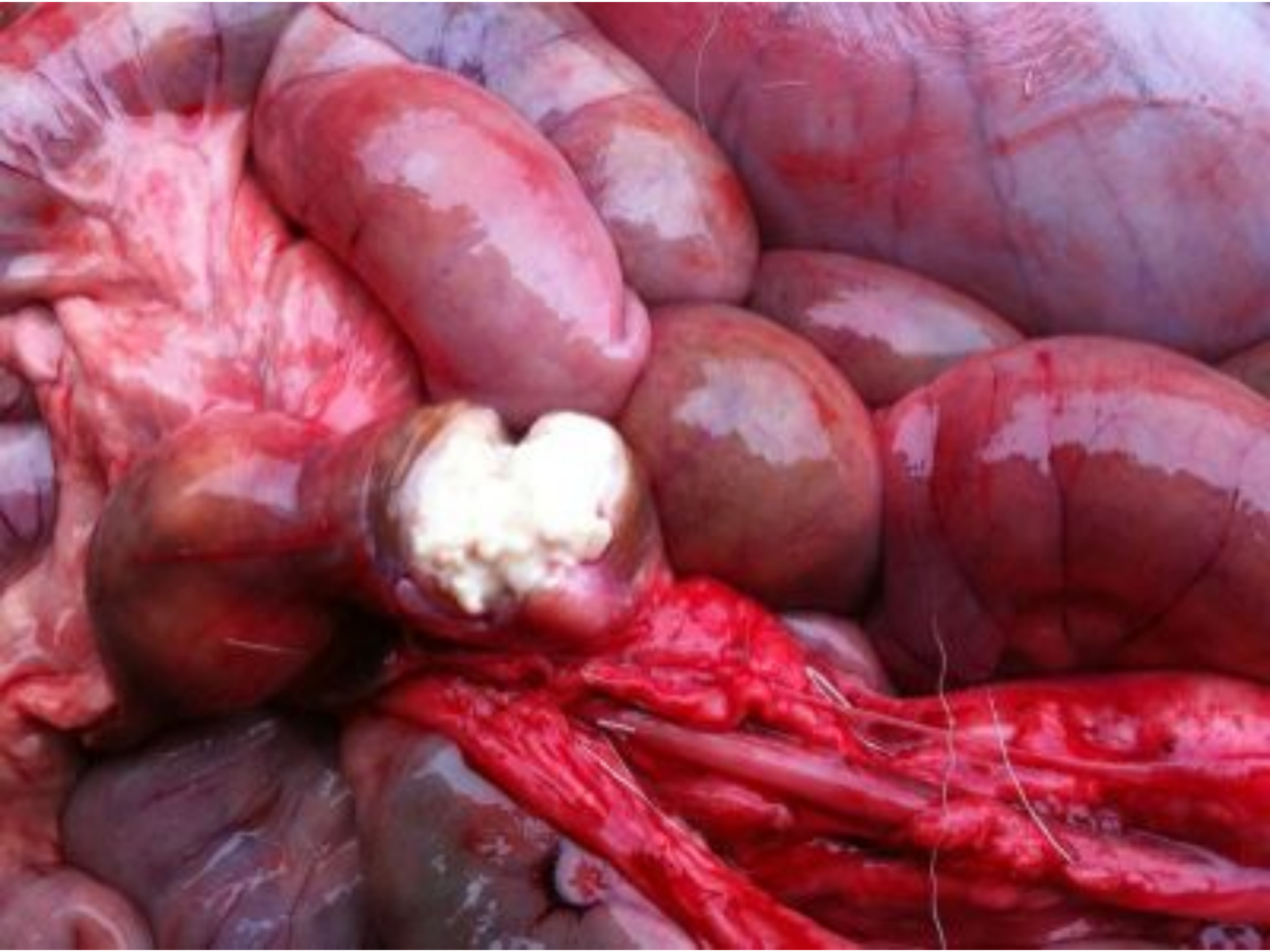
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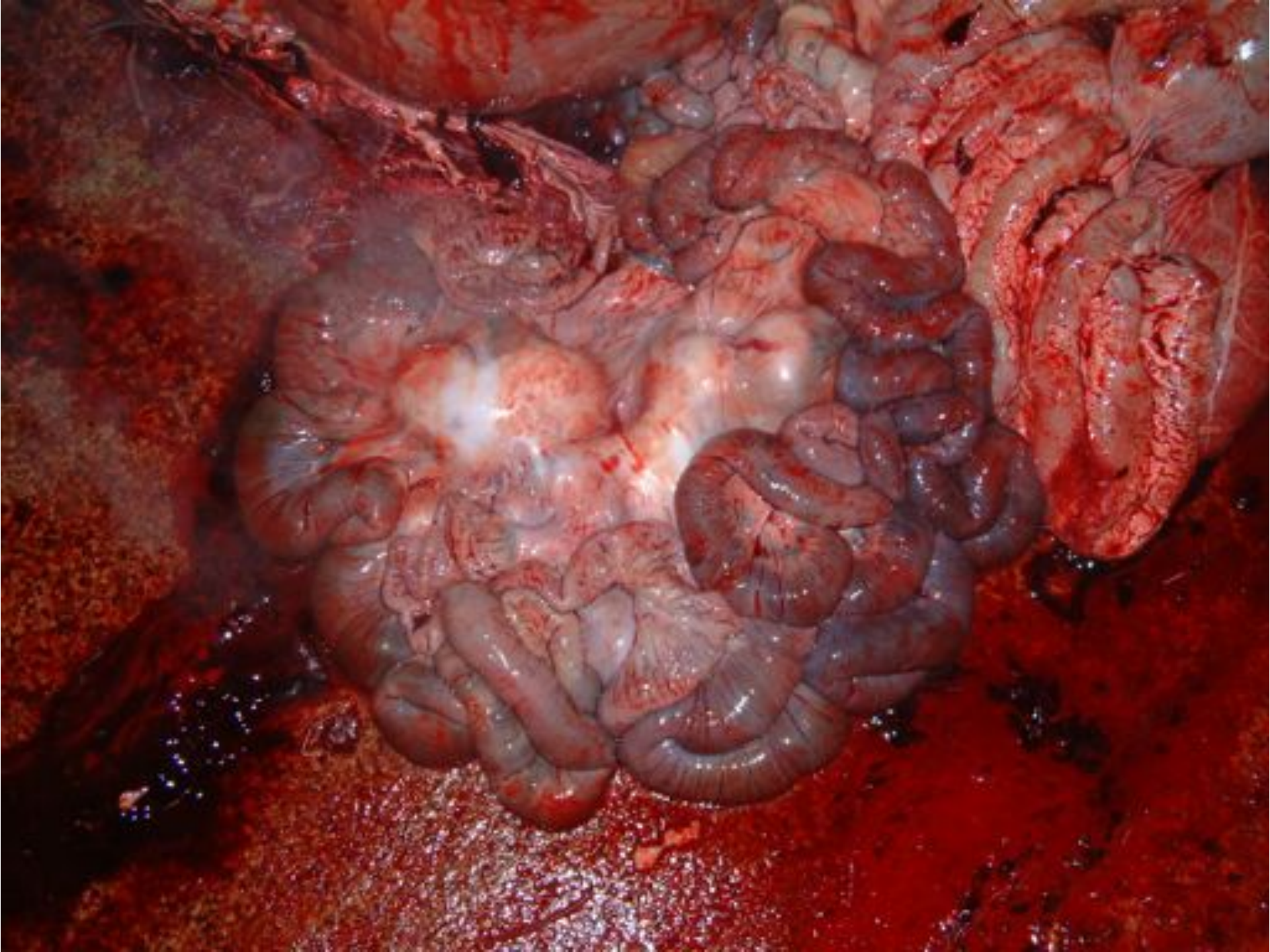
Occasional traumatic accidents such as these two stags bound together or stags with rope, wire etc wrapped round their antlers may demand darting and it might be wise to have contingency plans for rapid access to a dart gun.







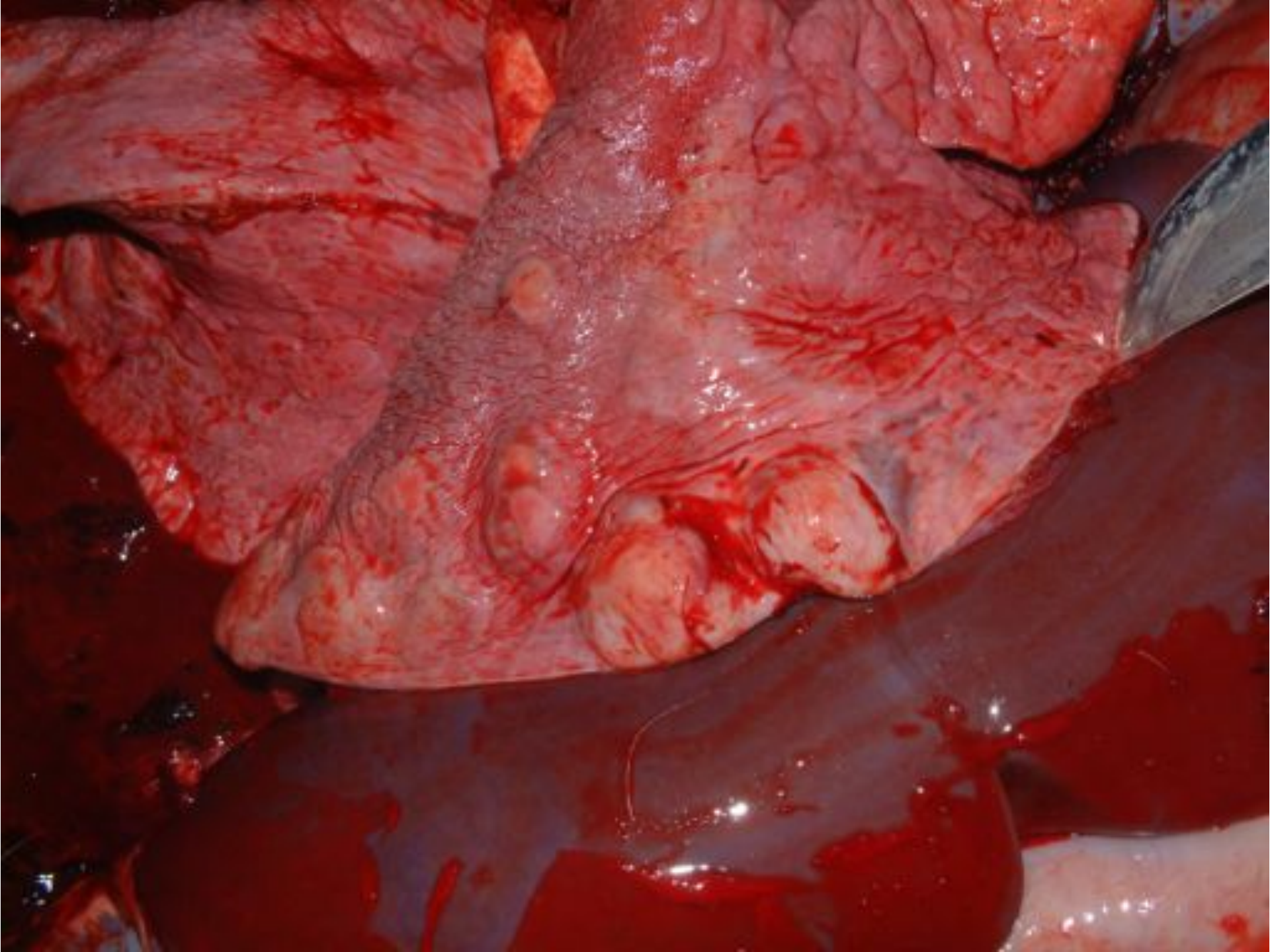


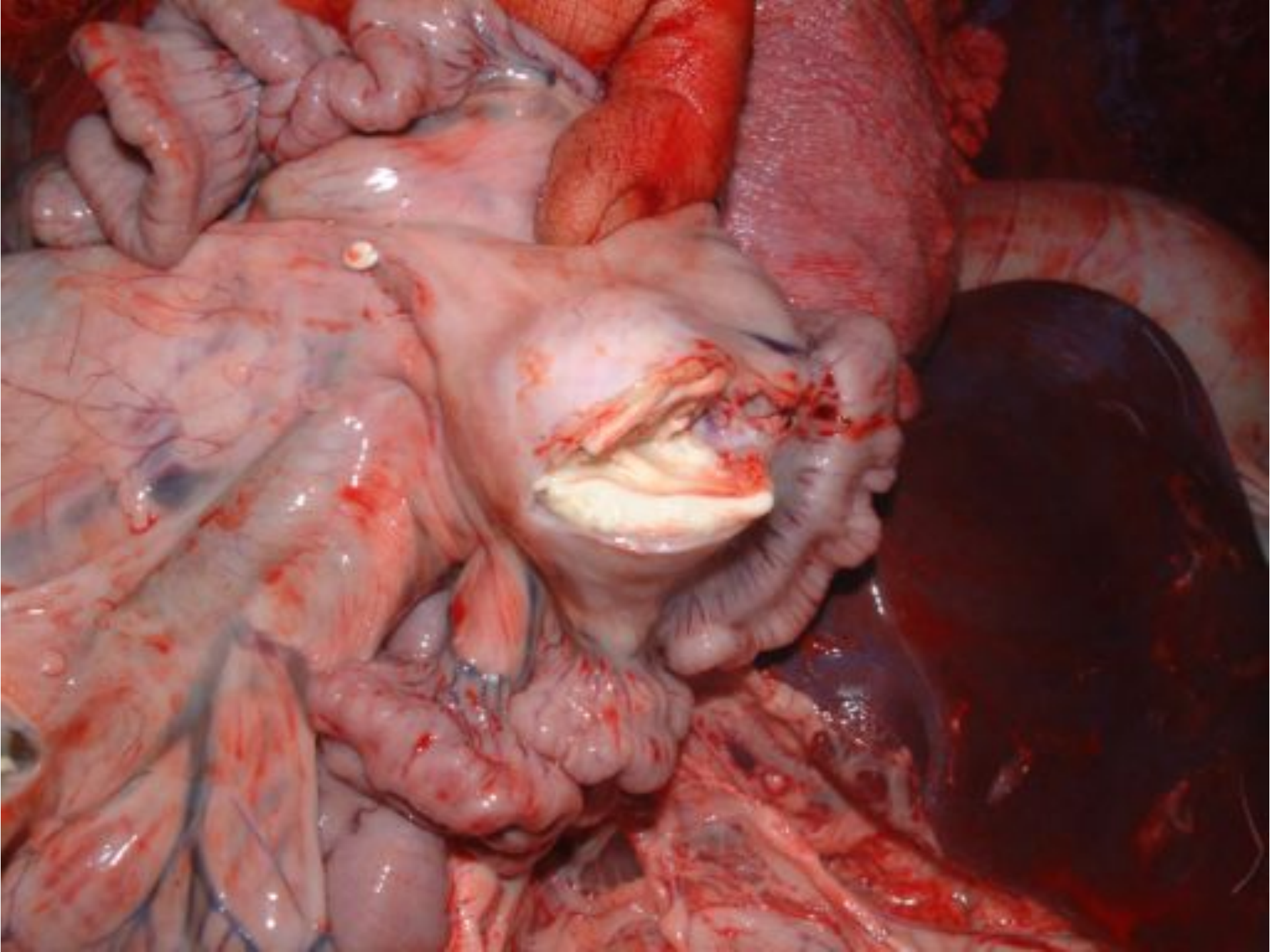




















CCN (cerebrocortical necrosis)

Presumed diagnosis in this roe deer is poisoning by oil seed rape.

This is caused by 'OO' varieties of rape which are high in 'cabbage anaemia factor'. After prolonged exposure secondary thiamine deficiency may develop and cause CCN.

In enclosed deer CCN may occur after relief of water deprivation and also where deer are fed poor roughage







Deer are not well equipped for hunting but if the opportunity arises they are surprisingly keen to eat other animals.

On the left a rabbit after half an hour and on the right after one hour.

