Case Reports

Post-Velvetting Infections

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Introduction

Velvetting has been an integral part of New Zealand deer farming since its inception 20 years ago. Infections following velvetting are rare. Until recently I was only aware of one significant outbreak involving a large South Island velvetting herd which had pedicle infections in stags after velvetting. These stags had been sedated to a state of lateral recumbency for velvetting. In our practice I know of only a handful of cases of abscesses in the previous sixteen years usually following nerve blocks of the zygomaticotemporal nerve.

Clinical Observation and Treatment

I was therefore surprised to get a call on the 20th of October 1996 from my largest deer client reporting recently velvetted stags with swollen heads. My surprise quickly turned to alarm when I viewed the 35 deer in the shed. 19 had massive oedema involving some or all of, the eyelids, face, nostrils and submandibular area. In one case the oedema extended down the neck to the front legs. 8 were less affected and the rest unaffected. 25% had been velvetted 3 days earlier the majority only the previous day. One stag had died and one was near death in the paddock just before I arrived.

I strongly suspected clostridial infection and treated all the deer in the shed with antibiotics. Those severely affected were given 20cc of Penstrep LA (100 000 IU procaine penicillin, 100 000 IU benzathine penicillin, 250mg dihydrostreptomycin/ml). Those mildly affected and those not yet showing signs were given 10cc of Penstrep LA. Four were given subcutaneous Tetracycline (Bivatop) but the response to this over the next two days was poorer than that to the penicillin and it was not used again. The affected deer were retreated every second day with Penstrep LA initially and then daily with Amoxil until the wounds were clean.

Post Mortem

Having treated the sick deer I autopsied the stag that had died in the paddock. Grossly there was severe (25-30mm thick) oedema of the head and ventral neck. The oedema was all ventral to the line of infusion of the local anaesthetic. The lungs were congested with blood and the bronchi were completely full of bloody froth.

Histology of the subcutaneous tissue anterio-medial to the left eye showed an extensive area of necrosis of the fascia and adjacent muscle. There were large numbers of bacteria and some gas distended areas throughout the necrotic area. There was also an intense narrow band of leucocytes at the margin. *Clostridium septicum* was isolated from the subcutaneous tissue and a retropharyngeal lymph node.

Epidemiology

Retrospective analysis showed 405 stags were velvetted during the outbreak (14-30 October). 7 died and 8 were euthanased. A further 33 were affected but survived. Two thirds of the survivors sloughed necrotic tissue and the prognosis for these must still be guarded. All but one of the deaths occurred 24-96 hours after velvetting, the same time frame in which the oedema developed. The overall morbidity was therefore 12% and the mortality 3.7%. Figure 1 shows the distributiion of occurrence of the condition. Data are summarised in Table 1.

The infections were highly clustered around the 17th and 19th of October. Six of the 11 stags velvetted on the 17th developed infections, all but 1 of the last 6 velvetted. Two of the 6 infected died and 1 was euthanased. Twenty three of 33 stags velvetted on the 19th developed infections, all but 4 in the first 22 velvetted. 3 of those infected died and 4 were euthanased.
For the stags velvetted on the 17th and the 19th the morbidity was 66% and the mortality was 20%.

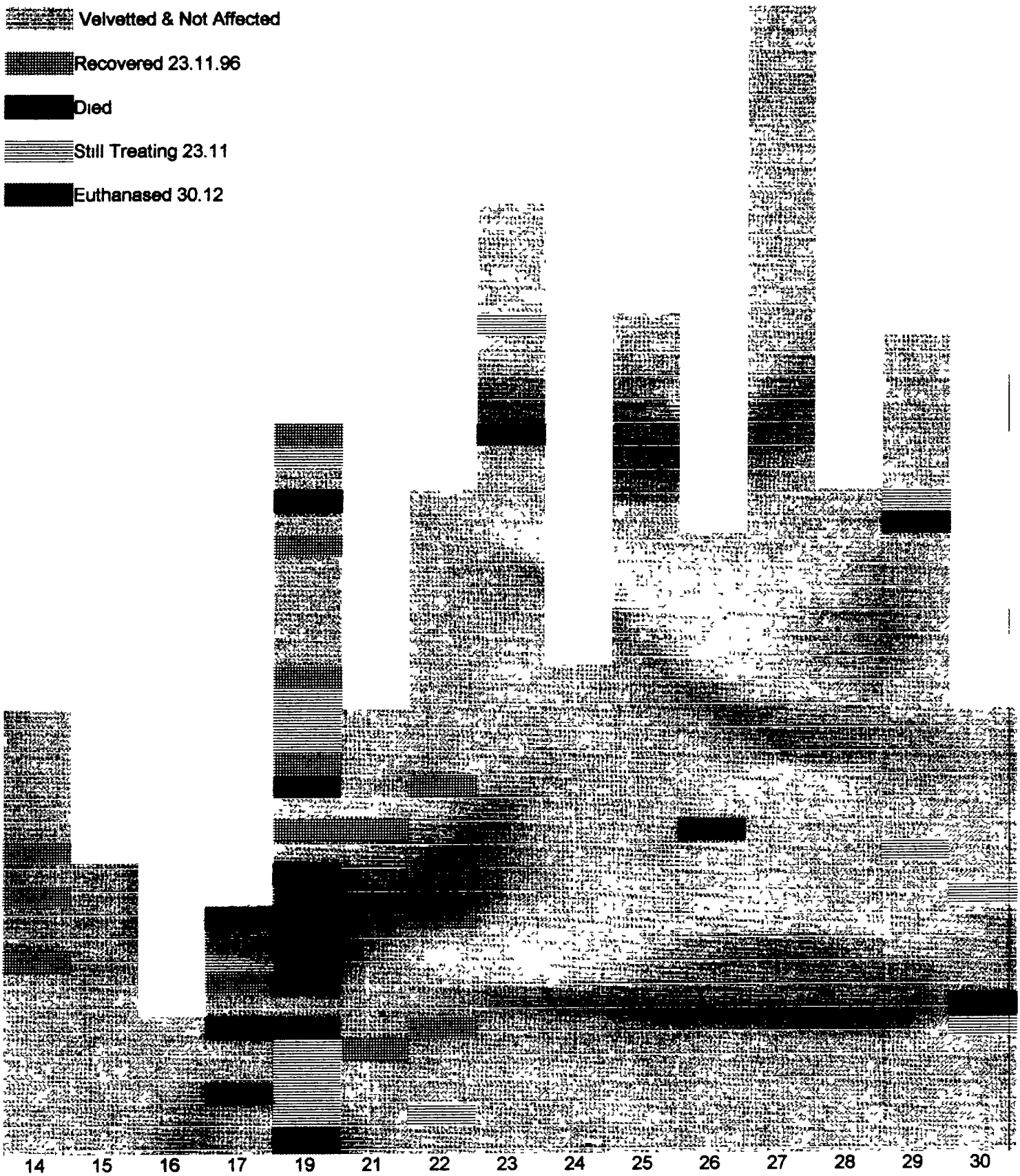
After my visit on the 20th the stags were observed very closely and treated with antibiotics at the first sign of oedema. 16 out of 320 stags velvetted from the 21st - 30th October were affected. 1 died, 3 were euthanased and 8 more developed skin necrosis. For the stags velvetted from the 21st-30th October the morbidity was 5% and the mortality was 1.25%.

Table 1. October 1996 velvetting infections - Property A

Dates	No. velvetted	No. affected	No. died	No. euthanased	Morbidity	Mortality
14-30	405	48	7	8	12%	3 70%
21-30	320	16	1	3	5%	1 25%
17 & 19	44	29	5	5	66%	20%

The highly clustered nature of this outbreak which is typical of other reports is shown in Figure 1.

Figure 1. Numbers of stags velvetted, affected and dying on each of the days of removal, in chronological sequence



On the 31st of October we changed to lidocaine without epinephrine and started penicillin injections and clostridial vaccinations. No infections occurred after this date.

On the 30th December 1996 I re-examined the group of stags with skin necrosis. Eight despite excellent pasture were in poor condition and on my advice were euthanased. The poor condition was due to a number of different lesions viz; tongue paresis, sloughed lips and dyspnoea from nasal stenosis.

Observations on other farms

Shortly after the initial outbreak my other two certified velvetters each had a single case of what appeared to be the same infection. On one property swelling around the eye of one stag was observed the day after velvetting. I treated this stag with a single dose of long acting penicillin and it recovered completely. On the other property the stag was not seen until necrosis had occurred and the owner shot it after a few days.

Relationship with anaesthetic type

All 3 of my clients velvet in drop floor crushes and were using lidocaine (20mg/ml) with epinephrine (0.036mg/ml) for the second season. The same product was used in the 1995 velvetting season by all 11 certificated velvetters supervised from our Taihape and Raetihi Clinics to velvet 3000 stags, with none of the above signs reported.

During the initial part of the 1996 season the same product was also used on 2000 stags by twelve certified velvetters supervised from our Taihape Clinic, again with none of the above signs reported. At the same time as these cases occurred I was velvetting on several properties (including a 250 and a 100 stag herd) using "Xylaket" sedation and lidocaine without epinephrine and had no cases of malignant oedema.

Discussion

The severely affected property has been farmed by the same owner as a deer farm since 1980. It is currently stocked with 1000 three year and older velvetting stags. It has a history of a high prevalence of clostridial infection in sheep despite vaccination. Because of this the owner when commencing deer farming was concerned about infections in velvetting stags. There have however been no signs of clostridial infections in the deer in the 16 years prior to this season.

An interesting observation by the owner was that when he used lidocaine with epinephrine on his stags they seemed irritated post velvetting, rubbing their heads on the fences and not grazing. On changing to plain lidocaine this behavior ceased and the normal behaviour of grazing post velvetting resumed.

The unanswered and probably unanswerable question is why did this outbreak of infections occur? Bacterial contamination of the local anesthetic is a possibility but at 5 stags per 100cc bottle that would mean 9 bottles over the worst 2 days and I think that is improbable. Contamination of the local is even more improbable for the widely spread cases.

The half full bottle remaining from the velvetting on the 19th was submitted to the lab and no anerobes or other bacteria were able to be grown. This bottle would have been used for the last 2 or 3 stags velvetted and the last 2 both had infections.

My initial and current belief was that the outbreak was related to the epinephrine in the local anaesthetic. Initially I thought the *Cl. septicum* spores may have been present in the tissues prior to velvetting as occurs in blackleg. However, then you would expect to see malignant oedema deaths during the roar. Malignant oedema infection of sheep and cattle is also usually reported as occurring through a wound. Fluorescent antibody testing of stags at slaughter from this property and others might help decide if *Cl. septicum* spores are commonly present in deer. It would not help though in answering why the bacteria were able to grow.

It seems unlikely to me that the *Cl. septicum* spores could enter via a cut antler surface because of the blood flow. In addition I saw no oedema of the pedicle or immediately adjacent tissue nor any exudation from the cut surface. Bacterial entry could have occurred through the needle punctures for local anaesthetic administration. However hundreds of thousands of stags have been velvetted using local analgesia without developing malignant oedema and I do not believe the subcutaneous passage of a needle around the pedicle could create enough anoxia for anaerobic bacterial growth.

We know from cattle research that only a small anaerobic volume is required for *Cl. septicum* spores to become vegetative (5ml of calcium chloride is used to induce muscle necrosis. In vaccinated cattle only a pea size lesion results.) We also know from sheep and cattle research that there are species differences. Unvaccinated cattle in the USA and NZ all have titres to *Cl. septicum* while unvaccinated sheep do not.

We also know that the intramuscular injectable formulation of Ivomec caused *Cl. septicum* deaths in 3 horses. Investigation of this using Guinea Pigs showed that when *Cl. septicum* and the Ivomec vehicle were injected all the Guinea Pigs died. When *Cl. septicum* and CaCl were injected they all died but when *Cl. septicum* was injected on its own only a few died.

The pattern of necrosis was similar with many of the stags having a narrow strip running from the region of the eye. There was no necrosis lateral or caudal to the pedicle. I suspect this is because the skin of the forehead anterior to the pedicle is more closely attached to the bone of the skull than the skin lateral or caudal to the pedicle. As a result of this I suspect the epinephrine comes in closer contact with the arterial blood supply while laterally and caudally there is more subcutaneous tissue for the epinephrine to diffuse through.

My hypothesis then is that the vasoconstriction of the epinephrine in some circumstances can cause sufficient anoxia for *Cl. septicum* spores to become vegetative. We will certainly never use lidocaine with epinephrine for velvetting stags again but I am left wondering what additional factor or factors initiated the problem in late October after 50 stags had already been velvetted, the last 30 of them with lidocaine containing epinephrine. Was there a higher concentration of epinephrine in the new batches used from the 14th of October? This seems unlikely considering that each batch is imported with a certificate of analysis and three batches were involved. Variation of epinephrine within batches to explain the high incidence on the 17th and 19th is an attractive explanation but would seem even more unlikely although it is not inconsistent with two anecdotal reports of failure of analgesia with 2 separate bottles of the product.

I had one bottle of local anaesthetic independently analysed for epinephrine and it was very close to the label concentration.

We had an exceptionally warm September with pasture growth double normal. Did this allow proliferation of pasture fungi with mycotoxins which constricted blood vessels or made them more sensitive to epinephrine creating a degree of anoxia sufficient for clostridial growth? Such speculation can probably never be answered.

Other occurrences

There have been a number of similar post velvetting problems predominately in the North Island in the last 6 years as the following table shows.

Table 2. Known cases of post-velvetting infections

Location	Year	Herd size	Affected & survived	Died or euthanased	Local anaesthesia L = Lidocaine; L + E = Lidocaine + Epinephrine Both = L & L + E
Hawkes Bay	1991	?	1	1	L + E
Wairarapa	1991	1			L
Waikato	1992		8		L + E
Sth Canterbury	1992	500	6	4	Both
Northland	1994	?	7?	28?	L + E
Northland	1994	160	3	0	L + E
Bay of Plenty	1995	140	0	5	Both
Horowhenua	1995			5	L + E
Rangitikei	1992	500	5	2-3?	L + E
Rangitikei	1995	80		3	L + E

Location	Year	Herd size	Affected & survived	Died or euthanased	Local anaesthesia L = Lidocaine; L + E = Lidocaine + Epinephrine Both = L & L + E
Waimarino	1996	1000	31	15	L + E
Waimarino	1996	200	0	1	L + E
Waimarino	1996	100	1	0	L + E
Sth Canterbury	1996	50?		1	L
Taihape	1996			1	L

The problem has occurred with two different local anaesthetics containing epinephrine and to a much lesser extent with lidocaine alone.

In March 1992 a Te Awamutu Veterinarian wrote to the Animal Remedies Board detailing an avascular necrosis in 8 stags out of 30 velvetted on one day. The product involved contained 3% Lidocaine and 0.02mg/ml of epinephrine. The worst affected stags sloughed skin rostral to the point of injection. All the stags had facial oedema and neurological defects with partial eye closure, slow eyelid reflexes and partial 3rd eyelid prolapse.

He questioned the use of this product for velvetting and wanted to know of any similar incidents.

A very similar incident occurred in the same season at Ashburton. The product was almost certainly the same but because the local anaesthetic was one month past expiry date the problem was not followed up and there are no written records.

In light of these two reports I was very interested to receive a letter from a retired general surgeon who distinctly remembered 50 years ago as a house surgeon being told to never use lidocaine with epinephrine to desensitise fingers or toes because of the risk of avascular necrosis. This information appears to be rela tively unknown amongst the Veterinary profession and seems relevant to small animal clinicians as well as ourselves.

In 1994 two cases occurred in Northland. Unfortunately the Veterinary surgeon involved died in an accident before I heard of the cases. I do however have the clinic records for both properties involved and have been able to discuss the cases with both farmers.

On the most severely affected farm the problem was first recorded in the clinic records on the 27.11.94. On that day there was a farm visit 5 days post velvetting. "5 deer were dead" "23 had swollen faces and lips" but "the pedicles were O.K." The farmer told me "he thought it was a faulty batch of local but the vet thought it was a bug".

Most of the stags velvetted on that day were affected and only 7 stags survived. Most deaths were "straight away but some were 3-5 months later".

The clinic records clearly show lidocaine with epinephrine was used for velvetting from October 1994 to January 1995 (along with 10% Rompun for restraint).

All the affected stags were treated with penicillin. On 10.1.95 the records show "more swollen noses". There is no mention of deaths or numbers affected but the antibiotic records indicate around 5 or 6 stags were probably affected.

The second Northland property had three deer affected. The clinic records show "one stag had face slough" like the stags on the first property and the farmer confirmed 3 stags were treated with antibiotics. The clinic staff confirmed Lidocaine with epinephrine was the only local anaesthetic purchased for velvetting in 1994.

The brief notes on the clinic records and the two farmers observations, to me strongly suggested the same infection as my clients deer suffered. The third case of post velvetting infections I am aware of occurred on a Tauranga deer farm. 5 stags were affected so severely that they were shot 2 to 5 days after velvetting. *Colostridium septicum* was isolated from one of the stags.

The velvetting was performed by the farmer (a certificated velvetter) in a crush using nerve blocks rather than subcutaneous infusion. 5cc was used per site. In this case both lidocaine with epinephrine and lidocaine were being used and it was not possible to determine which stags had which product. A lot of the stags on this property have an area of scarring approximately 2cm x 1cm where the local anaesthetic was injected. This is similar to many of my client's stags.

There has also been a case in central Hawkes Bay where lidocaine with epinephrine was used and 2 cases in the Rangitikei. In one of the Rangetikei cases the stags were velvetted by a veterinarian who told me he filed an Adverse Drugs Report with the ARB. Both the veterinarians involved have, like me, velvetted thousands of stags in the last 20 years using plain lidocaine and have never before seen cases of facial oedema and necrosis.

In 1995 a certificated velvetter in the Horowhenua had 5 stag deaths following one days velvetting. His veterinarian told me he had been prescribing the same epinephrine containing product for the past 3 seasons with no problems but now like me would not use it again. He also told me the post mortems on these stags showed gross lesions typical of clostridia and that the first stag velvetted that day had "dead velvet" (presumably damaged and full of blood).

The last three cases I have heard of are the only ones where epinephrine has definitely not been involved. A South Canterbury farmer told me his stag "looked like a cattle beast" the day after velvetting. The stag subsequently shed all the skin off his face and lost his nose.

The second case occurred 5 to 6 years ago in the Wairarapa. The stag was velvetted by a veterinarian who told me this stag, although it survived, sloughed tissue from the side of it's face. The third case was from Taihape. The stag was velvetted by a veterinary

surgeon from our practice and died 2-3 days after velvetting. When it was found "the head was as big as a football and the rest of the body was normal".

Comments and Recommendations

I have several concerns about this problem.

Firstly, I want to ensure that as far as possible no more stags suffer from the condition I have described. Please encourage your certified velvetters to report any adverse effects following velvetting to yourselves and the NVSB. Please also report these occurrences to the Animal Remedies Board.

Secondly, I believe the Animal Remedies Board should have notified the profession of the possibility of avascular necrosis from using lidocaine with epinephrine for velvetting and should have reviewed all local anaesthetics to establish consistency of label claim and species indications.

Thirdly, while Veterinarians could perhaps be regarded as being at fault for prescribing local anaesthetics not licensed for deer the product used by our practice was included in the distributors list of products for Deer Velvetting. Perhaps the distributor should have sought a licence extension for deer. But would the ARB have made the connection with the previous product?

Following the outbreak of infection in my clients deer I sent off the standard ARB form along with a letter outlining the situation and asking for information on any similar cases. The response to me from the ARB was to send me copies of letters received by them from the licensee and manufacturer of the product.

I only learned of the 1992 adverse report after the farmer whose deer were affected phoned me. Fortuitously he had read an article in the farming press about my clients problem and fortunately could remember the first part of the trade name. His Veterinary surgeon remembered the licencee's name but this was still insufficient for the ARB who told me "it was so long ago" that without the full trade name they would be unable to find anything. I had perhaps naively expected in this age of computerisation that they would have had active ingredients and species cross referenced with adverse reactions.

In contrast the help received from the pharmaceutical companies holding the licences for the two products involved has been excellent and much appreciated. Veterinarians in industry and practice have also been most helpful as have the deer farmers involved.

The occurrence of these infections over a number of years in widely separated locations, in my mind strongly indicates an association with epinephrine. Certainly clostridial vaccination can probably be recommended although two of the dead stags in my case had

received annual vaccinations on two or more occasions prior to their purchase 3 or 4 years ago. This indicates to me that immunity to clostridial vaccination is probably short lived.

I have been unable to determine any history of severe clostridial infections in sheep on any of the other properties. In Northland the property was previously stocked with cattle. Clostridial infections are regarded by most practitioners as rare in deer. Although vaccination was widely practised in the early days of deer farming when animal prices were high most commercial deer farms do not currently vaccinate. This in my view further points to epinephrine being the significant factor in *Clostridium septicum* infections of stags heads after velvetting.

I am still interested in hearing of other similar cases and especially in knowing the type of anaesthetic involved.