BVD VIRUS AND ALPACAS - THE DETECTIVE STORY

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This is the detective story about the discovery that BVD (bovine viral diarrhea) virus, originally thought to be an insignificant problem for camelids, does indeed cause illness and abortions in alpacas, and that it can also cause the persistently infected (PI) state in alpacas. This has fairly far reaching consequences for alpaca farming. For detailed information on BVD virus itself, please see the accompanying article. Like any detective story, my narrative is long and detailed. Also like a detective story, looking back, the solution looks fairly clear; working forward from the beginning it was not clear at all.

The story starts in December 2003. I have a herd of approximately 50 alpacas, and there are three barns. In the first barn there are three separate areas; the groupings at that time were: East section: moms and their cria born that year; West section: maiden females, born in 2001 and 2002; South section: young males born in 2002. There is a solid partition between the East and West sections, and only fence line contact for animals in the east and south sections. Adult males are in the second barn. The third barn is for the quarantine area (primarily for alpacas that have come for breeding) and is not used in the winter.

In December 2003 in the West section there were eleven maiden females (Elena, Mandy, Abela, Tillie, Madison, Mikayla, and Gabriella, all born in 2002, and Harley, Savanna Dawn, and Merienda all born in 2001, and Celeste who was four years old). Harley and Merienda had been bred in June, and Elena, Mandy, Tillie, Abela and Mikayla had been bred in October. In the South section there were 6 young males: Timoteo, Jupiter, Vaquero, Photon, Dirk, and Cosmo, ranging in age from 14 – 19 months.

On December 1, I noted in my farm record book that Elena in the West section, an 18 month old female who had been bred October 9, was lying around more than usual and not as interested in eating her pellet food (the supplement designed to deliver the necessary minerals and vitamins). Her temperature was normal at 38.3 C. After that, she persisted in not being as interested in her pellet food (and this could have been overlooked in a different setting, as my alpacas are fed their supplement in individual bowls and under supervision to prevent the fast eaters from stealing from the slow eaters) but she seemed normal otherwise - eating hay and normally active. On Saturday December 13 she was even less interested in her pellet food but was still eating hay; it looked as if her mouth was uncomfortable when she was eating the pellets. Her jaw felt normal and I couldn't see any abnormality in her mouth, but I started her on the antibiotic Excenel in case this was a manifestation of a dental abscess. I had the vet out on Monday morning December 15 to examine her and do blood work including liver function tests. Of course Elena looked quite perky when the vet was here, and I remember saying to the vet that she would just have to believe me that this was a profound change for this alpaca.

There were no abnormalities noted on physical exam, including her mouth. Elena was eating hay in the morning, but was lying around more than usual and did not eat hay in the evening. She had Ivomec, and was started on omeprazole to cover the possibility of an ulcer. The next day, December 16, she did not even get up at feeding time. I started syringing in pureed pellet food and liquids.

Blood test reports arrived back later that day. Unfortunately there were no normal values given with the results, and lab values in Canada are reported in S.I. units (e,g mmoles per litre) as opposed to what are called conventional units (e.g. mg. per deciliter) used in the United States. My vet faxed the lab result to me. I had to use the conversion charts in *Medicine and Surgery of South American Camelids* to convert the values to conventional units so that I could compare them with the normal values given in that book and in Dr. Norm Evans' *Veterinary Lama Field Manual*. There were some abnormal results: there were elevated liver enzymes (e.g. AST 877 with a normal up to 250), low protein, low albumin, and low platelets. This looked like possible hepatic lipidosis (also known as fatty liver). I quickly read up as much as I could on hepatic lipidosis, and then started syringing in glucose and electrolyte solution, continued the omeprazole, added Sulcrate and switched the antibiotic to Septra. Despite all that, she had a very rapid downhill course, and died later that evening with her head in my husband's lap and me by her side.

An autopsy was done the next day and it showed hepatic lipidosis. Now for a quick aside about hepatic lipidosis. The simplified explanation is that when caloric needs are not being met, either by poor quality or quantity of feed, and/or increased needs, such as lactation, or under the influence of stress, the body mobilizes fat from fat stores and it goes to the liver. In humans, the liver would convert the fat to glucose and the glucose would be used by the body as a source of energy. In some animals, such as alpacas and cats, the fat stays in the liver, and the deposition of fat in the liver cells impairs liver function and leads to liver failure. It is often a spiraling downhill course because as the liver function becomes impaired, the appetite decreases so there is even less food intake. In alpacas, stress and poor nutrition are considered the prime causes of hepatic lipidosis. It was no consolation to read that well run alpaca farms could lose animals to hepatic lipidosis. It just didn't make sense. There had been absolutely no stressors. My hay had been tested: 12% protein and 58 % TDN, and there was unlimited hay put out. All the females receive 200 grams twice a day (total of 400 grams – almost a pound) of a specially formulated pellet ration to provide their selenium and other vitamins and minerals, and the extra calories they need in our cold winters. All these alpacas were in good body condition (body scoring). None of the nursing mothers, who would have the highest caloric needs, in the East section of the barn were affected - they received identical hay and pellets. I thought it more likely that whatever had made Elena reluctant to eat her pellets had resulted in decreased feed intake and that had set off the chain of metabolic events resulting in hepatic lipidosis.

Then on December 23, two more females from the West section, Merienda (two and a half years old, five and a half months pregnant) and Mandy (eighteen months old, two months pregnant) were both not eating their pellets with much enthusiasm - it really

looked as if their mouths were uncomfortable. This was the same presenting symptom as Elena. I immediately had the vet out to do blood work. The vet had suggested that Elena's reluctance to eat food at the beginning of her illness was a manifestation of liver problems. I thought it more likely to be the other way around: that the reluctance to eat pellets came first (from some other cause) and the resulting decreased feed intake caused the hepatic lipidosis. The vet was unable to get a blood sample from Merienda. Mandy's blood tests, including liver enzymes, were all normal, other than an elevated haptoglobin value in keeping with an inflammatory (which would include infectious) process. I therefore thought this confirmed that Elena's, Mandy's and Merienda's reluctance to eat pellets was not from liver problems (early hepatic lipidosis), as the liver enzymes would have been elevated in Mandy at this stage if this were the case. I thought Elena's hepatic lipidosis was a result of her decreased feed intake from whatever had made her uncomfortable or reluctant to eat the pellets, but had no idea what was causing that. I put both Merienda and Mandy on omeprazole for a week as the vet said sometimes not eating supplements may be a manifestation of an ulcer. Neither of them ever looked or behaved unwell; they had normal activity and were eating hay, and they were eating their pellets normally in a few days.

On December 24 in the evening another alpaca in the West section, Tillie (seventeen months old, bred October 6) did not eat any pellets, stayed lying down and would not eat hay. Her temperature was 39.2 celsius (just borderline). Since I was worried that her not eating would result in hepatic lipidosis, I started glucose and electrolyte solution by syringe – by providing calories that way, it should stop the body from thinking it had to mobilize fat reserves. Early the next morning (this was one of the worst Christmases on record) I started Septra (antibiotic), omeprazole and Fastrack and continued syringing in the glucose and electrolytes. I realized my intervention with Elena had been too little and too late, and I vowed to Tillie I would not let her die. I did not see her eating any hay, and mostly she stayed lying down. The next day she started eating hay. By December 27 she was eating well and she was chewing her cud for the first time since becoming unwell. She was back to normal by December 29.

On December 27 another alpaca in the West section, Abela (seventeen months old, bred October 13) was having trouble eating her pellets and she was lying around more than usual in the morning. I started her on omeprazole. She looked a little brighter the next day but was still eating pellets slowly. By December 29 in the evening she was eating normally.

In the week after this, two more females in the West section, Gabriella (twenty months old, not pregnant) and Madison (fifteen months old, not pregnant) both had several days of not eating pellets normally and both appeared to have increased redness at the corners of their mouths – no sores, no blisters, no ulcers, nothing apparent inside their mouths, just a bit of redness at the corners of the mouth. I did not put them on any medication and they were back to normal in a few days.

At the end of December the six young males from the South section were moved to the adult male barn. On January 8, Jupiter was not eating pellets normally. The next

day he wouldn't eat any pellets and was not chewing his cud, so I put him on glucose and electrolytes, septra, omeprazole, and fastrack; he was cud chewing by January 11 but was still having difficulty eating pellets. The corners of his mouth looked red. He ate better on January 12, and on the 14th the medication was stopped and he seemed normal.

On January 9 Dirk was eating his pellets but would not eat hay; he looked somewhat unwell and was shivering (by then we were having a cold spell with temperatures at -20 Celsius). He had been chased and ridden a lot by the older boys and I think this stress may have compounded his illness. Jupiter, Dirk and two of the other young males were put in a separate area without contact with the adult males. Dirk continued to eat his pellets fairly normally but was eating very little hay, lying around more than usual, and not cud chewing. His temperature on January 9 was 38.4 C. I gave him glucose and electrolyte solution, Septra and omeprazole. By January 11 he was eating hay well; by January 13 he was cud chewing, and I stopped the medications on the 14th.

None of the alpacas had diarrhea. Elena, Gabriella, Madison, Jupiter and Dirk all had a few small patches of fleece that came out at the immediate time of illness or shortly after, and in the case of Dirk it continued for several months. There were no bald patches, but there would be very obvious tufts of fleece sticking out, or that had fallen out.

There were no other alpacas with these symptoms. I could not figure out what was causing this. All groupings received identical hay, water and pelletized supplement. I sent off their water for 'livestock suitability' and coliform / *E.coli* testing and the tests came back normal; the pellet feed was tested and the analysis confirmed the proper ingredients.

To say that this was a stressful time would be a vast understatement. I racked my brain about what was different about the West and South sections that it was only alpacas there that had been unwell and not any in the East section or from the adult males' barn. The only thing I came up with was that there were some pigeons in the overhangs off the West and South sections. I wondered if they were carrying some disease. My husband Paul and I became experts at netting pigeons; and Paul became an expert at chopping off their heads. All the pigeons were dispatched except for one pigeon that I took in to the vet's office and asked that it be sent for testing. No signs of any disease were found in it.

In late December our friend Grant, whose son has a dairy farm in the area, dropped by for a visit. When he heard of our woes he said that his son had told him of a bad outbreak of a virus causing illness and death in cattle. He found out for me this was called BVD 2. I read up on BVD, bovine viral diarrhea, but it didn't seem applicable; the illness described in cattle was fever, discharge from the nose and eyes, erosions in the mouth, and diarrhea; its major impact in cattle seemed to be the abortions it caused. What few reports there were about alpacas or llamas and BVD implied it was not much of a problem at all; one web site even said how marvelous it was that alpacas never got BVD. One study with llamas and BVD concluded that llamas could be infected with

BVD but had few or no clinical signs. Also, there seemed no way that BVD could have come to my farm. The closest cattle are a mile away. There had been no cattle on our farm for several years. Visitors are asked if they have been on other farms and if so, are given foot wear from here – the only possibility would be cattle manure on the vet's boots if the vet had not cleaned his/her boots. The vet office said they had not heard of any BVD outbreaks in their practice. It was an interesting disease that I had never heard of before, but it didn't seem to have any relation to the problems on our farm.

Jupiter and Dirk were the last two alpacas that appeared unwell. The final tally was: one death, three alpacas with severe enough symptoms that I put them on 'shotgun therapy' (cover everything) and they recovered, and five alpacas with mild symptoms (looking like they had a sore mouth) that recovered on their own. I don't think the omeprazole, a drug for ulcers, had anything to do with their rapid recovery as some got better without it, and of those on it, none relapsed when I stopped it after a short length of time. The whole picture fit with some infectious disease: primarily one age group affected, and the whole spectrum of severity from not unwell, to mildly unwell, to quite unwell, and one death. However I could not find any illness described that fit the symptoms. It was a mystery, and it had been a nightmare, but I was thankful it was finally over.

In the East section with mothers and cria were a visiting female for breeding, whom I will call VF, and her chronically unwell male cria whom I will call MC. They arrived here September 21, 2003 and were put in the quarantine area by themselves (separate barn and pasture). MC had been born on another farm, Farm B, on June 13, 2003, and had only weighed 9 pounds at full term – a very low birth weight. Records from Farm B showed he had done fairly well, with good weight gain, until early August when he developed pneumonia and diarrhea. He was treated with antibiotics, had panacur and was on amprolium after a diagnosis of coccidia. He had not had solid manure or any weight gain for the month prior to arriving here; his weight on arrival here was 25 pounds. His fecal sample still showed coccidia. In the quarantine area I use a separate pair of boots and a separate shovel for shoveling manure – these are used only in the quarantine area. Since I knew MC had coccidia I was even more obsessive – I removed his manure with disposable gloves rather than smearing it around with the shovel, and poured liquid bleach where it had been. He was treated for the coccidia; his stool became normal within a few days of arriving here and he started to gain weight. I remarked to anyone who would listen about the wonders of having lots of fresh uncontaminated, not overgrazed, pasture. VF and MC both received a course of Panacur because VF's fecal sample had shown tapeworm. Repeat fecal samples were normal. VF and MC were to stay for an extended period of time; they would have to be integrated with my herd at some point for over the winter, so they were put in the East Section with the adult females and cria on October 8. I had looked up in *Medicine and Surgery of South American Camelids* to see if there was any infectious disease that fit with MC's 'unthriftiness' (as the vets call it) but couldn't find anything, so assumed it was safe to put him in with the other alpacas. MC never looked totally healthy. He had very slow weight gain, and a runny nose off and on; he didn't often play with the other cria. I had never had a poor doing cria, but had heard of other farms that had. I still hoped that with

good care and proper nutrition he would eventually become normal. Starting in November, he had recurrent courses of antibiotics (Septra or Excenel) for pneumonia. He finished a course of Septra on January 30, but he still had some nasal discharge and was not eating normally. He was put on Baytril, another antibiotic, on February 5 for ten days and seemed somewhat better; at that time he weighed 40 pounds (many of my cria are that weight by 4 weeks of age, and he was almost 8 months old). His stool became softer; testing was normal. He developed watery diarrhea, and despite supportive treatment died suddenly on February 28. When I notified the owners of his death, which really was not unexpected, I forgot to ask if they wanted an autopsy. Since it was a warm Saturday and it seemed unlikely the owners would want to pay for an autopsy on what had been a chronically poor doing cria (that they had already spent a lot of money on) and there was the problem of what to do with the body until Monday, he was buried. I later confirmed with the owners that that was fine. His mother VF has always looked healthy. There was no direct contact between VF and MC and the alpacas in the West or South sections. None of the adult females and cria in the East section (that VF and MC were with) ever appeared unwell then or since.

No other new alpacas had joined my herd in 2002 or 2003. One female who came for breeding and her cria were in the West section in the spring and early summer of 2003, after being in the quarantine area for a week or two and having normal fecal tests. The other alpacas that came for breeding in 2003 were in the quarantine area the whole time and never with my herd. The last of these left August 21 and the quarantine area was cleaned and not used until VF and MC arrived September 21.

After MC's death I mulled over what could have been the diagnosis for this chronically unwell cria. I had 'bought into' the concept of unexplained poor doing cria; I had never had one, but I had heard of others who had. I am a family doctor; in humans you would never say "That child died because he was a poor-doer" – there is always a diagnosis. There just had to be a diagnosis for MC. Of course I do realize it's the economic considerations that preclude all the testing to come up with a diagnosis in animals. I read up on a lot of illnesses, remembered something from my reading on BVD and looked again at that information, and specifically about persistently infected (PI) calves. These are the calves that were exposed to the BVD virus as fetuses (their mother was clinically or sub-clinically infected) during the crucial stage of gestation, approximately 40 to 120 days, when they did not recognize the virus as foreign. They never mount an immune response to the virus, and if they don't abort they end up as permanent carriers. After being born, they shed huge quantities of virus in every secretion (saliva, tears, nasal discharge, urine, and feces) the rest of their lives, and are the major source of the spread of BVD in cattle. Although some PI calves are normal, many are poor-doers – low birth weight, poor weight gain, and they have repeated infections such as pneumonia; the majority are dead before one year of age. This was the exact picture of MC. There is also something called mucosal disease with BVD – this occurs only in PI animals and is the result of superinfection with an antigenically similar strain of BVD, usually from mutation of the strain the animal already has and it results in severe diarrhea, and invariably death. MC had died after a fairly sudden onset of diarrhea. There did seem to be striking similarities between MC and a typical PI calf; however I

decided it was still pretty far-fetched, especially as no alpacas in the East section of the barn with him had been unwell, and also, camelids just weren't supposed to be affected by BVD. VF had been on four different farms during her pregnancy with MC. I contacted the farm where VF had spent most of her pregnancy and asked if they knew anything about her background that would have resulted in her producing such a low birth weight and poor doing cria, They thought it was likely just stress from VF being moved around a lot during her pregnancy. I figured I'd never know what had been MC's underlying problem.

Then, on March 29, 2004, Mandy aborted at five and a half months gestation; she did not appear unwell. She was the alpaca who had had two days of not eating her pellets normally in late December and had had normal blood work. I thought about BVD again and its association with abortions, including abortions that can occur months after the initial infection, and figured I had nothing to lose by asking for the fetus to be tested for this. After all, the vet's office probably already thought I was a little nuts for bringing in that pigeon for testing. The preliminary report showed no significant lesions in the lungs, heart, liver, kidney or placenta, and came with a comment that the pathologist could not find any references on BVD or other viral abortions in alpacas, and that alpacas seem to be quite resistant to viral diseases. Immunohistochemistry testing for BVD took longer, and it was positive: it showed staining for BVD antigen in lung, kidney and heart. The comment on this report was that this was a very interesting case. Then virus isolation (the gold standard test for BVD) was done and it was positive from a skin sample.

It took a while for the whole significance of this to sink in. It now seemed very likely that the illnesses in December had been from BVD. However I still had no idea how BVD had arrived on my farm. I spoke with Grant's son, the dairy farmer who had mentioned about BVD. He told me it had only been on one farm that he knew of, and that farm was close to him; that would be approximately 20 - 25 km. from me. The vet involved was not from the vet clinic that I use. There was no connection between that farm and mine in any way. Also that was BVD2 and my aborted fetus had BVD1 – these are two quite different strains of the virus – so that case had nothing to do with the BVD on my farm. I spoke with a dairy farmer who is approximately two km. from me and he said he was not aware of any problems in the area; the last outbreak he had had was over five years ago. Although it was thinking about MC and what could have been wrong with him that had brought BVD to mind, I didn't think I could seriously consider MC as a PI animal – alpacas just weren't supposed to be affected by BVD (although it appeared I had just proved that wrong) and no alpacas in with him had been ill. I had uncharitable thoughts about whether one of my vets could have tracked the virus in on manure contaminated boots. At one point I was convinced that would have to be the only way BVD had come to my farm. The only problem was that vet visits and the start of Elena's illness didn't fit in regards to the incubation period of BVD, and the vets had not even been in the west section of the barn.

All this testing had taken quite a length of time. It was now May and I had been spit testing any of the females who had been bred in the fall and didn't look obviously

pregnant (spit testing had stopped for the winter in November). Spit testing showed that Abela and Tillie from the west section were no longer pregnant. Abela had been mildly unwell and Tillie had been quite unwell in December. It looked as if BVD had caused not only Mandy's abortion, but these early pregnancy losses as well. The only alpaca in the west section that had been at two months gestation in December and was still pregnant was Mikayla. In the east section. I already knew that Velvet wasn't pregnant after a breeding late in the fall. But Misty, who had been bred on October 10, was no longer pregnant. She had passed her spit tests up until her last one on November 10 and it was very unusual for her not to maintain a pregnancy; of her preceding six pregnancies, only once had she come open after passing the two week mark.

Shearing of all the alpacas was completed by the third week of May, 2004. Stress breaks in fleece are well known in fleece bearing animals that have been subjected to stress such as illness, high fever, or malnutrition. When pulling on a sample of fleece by holding at the cut end in one hand and the tip end in the other hand, the fleece will break at the level in its growth where the stress occurred. Severe stress breaks (fleece easily broken) were visible in Tillie, Jupiter, and Dirk – the alpacas who had been ill enough that I thought they required the whole gamut of treatment. In fact, just looking at a sample of their fleece you could see where the break would be – there was a broader wave of crimp there. Stress breaks were also evident in Abela, Gabriella, Madison, and Mandy; these four had had mild symptoms that had lasted only about two days, and Mandy was the one who had aborted. But there were also stress breaks in the fleece of Mikayla (from the West section), and Timoteo, Photon, Vaquero and Cosmo (young males who had been in the South section). None of these five had shown any signs of being unwell. Every alpaca born in 2002 had a stress break in its fleece. Merienda, born in 2001, who had had several days of not eating pellets normally in December, did not have any stress breaks in her fleece, and neither did Celeste, Harley, or Savanna Dawn who were also in the West section. VF, the mother of MC, did not have any stress breaks in her fleece, and in a cursory check of some fleeces from alpacas in the East section and the adult males no others with stress breaks were found. I have had very little illness in my herd in order to compare other fleeces in alpacas that may have been subjected to stress or illness. Savanna Dawn, born in 2001 and in the West section, had had a uterine infection in the summer of 2003; she had had several uterine lavages and courses of antibiotics. She had no stress breaks in her fleece. In April 2003, Mikayla as a 7 month old had had an episode of lethargy, decreased appetite and a fever of 40.1 Celsius. She was put on antibiotics empirically and improved quickly. She was sheared a month after that, and when I examined her fleece sample that I had kept from that shearing (of May 2003) there were no stress breaks in it. She had quite a long staple length, and I think even though there was only a month between illness and shearing, meaning that the stress break would be quite close to the cut end, if there was a stress break it would be detected. I found it amazing that BVD virus had been a severe enough assault on the alpacas to cause a stress-break in their fleece, but that in some of them they had never even appeared unwell. It was also amazing that Mikayla did not have a stress break in her 2003 fleece even though she had had an episode of being obviously unwell and febrile with some illness, and yet she had a stress break in her 2004 fleece from what must have been a subclinical BVD infection. This was quite the virus.

When the BVD virus isolation test had come back positive, the pathologist had discussed with my vet about getting a BVD antibody test done on Mandy, the alpaca who had aborted. In the spirit of scientific enquiry I decided to get antibody tests done on a number of animals. Having antibodies does not mean that the animal is unwell or contagious – it shows that the animal was exposed to the virus at some time in the past and mounted an immune response – this could be from a clinical infection (appeared unwell) or a subclinical infection (never appeared unwell) or from immunization. For example, most of us have antibodies to chickenpox, because we had that illness as children. It appeared there must have been at least some subclinical infections, based on the fleece stress breaks, but I still expected the results would show that BVD had been confined to the West and South sections of the barn. By now, I was concerned about Mikayla's fetus. She was the only alpaca in the West section that had been in early gestation at the time of the illnesses in December and that was still pregnant. Mandy, Tillie and Abela had aborted or had early pregnancy losses; Harley and Merienda were much further along in their pregnancies. Mikayla had been bred on October 14, 2003, so if she had been infected with BVD in December there would be real risk of having a PI cria, if that could happen in alpacas. The International Camelid Institute had no information on BVD and camelids. I contemplated aborting Mikayla. I had met Dr. Patrick Long, co-author of *Llama and Alpaca Neonatal Care*, and contacted him for advice on what drug and dose to use; he kindly put me in touch with Dr. Mattson, a BVD expert at Oregon State University. Dr. Mattson said although it was theoretically possible for there to be such a thing as a PI camelid, he assured me he had tested hundreds of samples from llamas and alpacas and had never found a PI animal, and advised me not to abort her.

The antibody tests came back. The lab had first run an ELISA test on all the samples checking for the virus itself – these were all negative, meaning there were no acute or PI infections (not that I had expected there would be). From the West section, I had tested Tillie, Abela, Mandy, Merienda, Savanna Dawn, Harley, Celeste and Mikayla; all of them had antibodies to BVD1. From the east section I had tested VF (I was starting to be more suspicious that her son MC might have been PI), Velvet, and Shawnee – VF and Velvet both had antibodies; there had not been enough blood from Shawnee's sample to do the test. From the young males who had been in the South section I had tested Dirk, who had been unwell, and Cosmo who had not been unwell. Surprisingly, Dirk did not have a detectable antibody level, but Cosmo did. I do believe that Dirk, who had been quite ill in early January, must have had a BVD infection. He was the young male who had been very stressed by the older males after being moved to their barn, before I separated him and some of the other young males out again. It certainly made me wonder about the effects of stress and the ability to mount an effective immune response. From the adult males in their separate barn, I tested Dano, and as expected he did not have antibodies. Merienda, Cosmo, and VF tied for having the highest titre of antibodies (1:1536).

So, this meant that the females in the West without symptoms and without stress breaks in their fleece had also been infected with BVD; in fact it seemed likely that all

the alpacas in the West and South sections had been infected (and of course, other than Elena, had recovered). I was still concerned that Mikayla could have a PI cria, even though there was not supposed to be such a thing. She had definitely had a subclinical infection as confirmed by the antibody testing, and also by now she was due in a little more than three months, and her pregnant abdomen did not look as big as it should for her gestation, suggesting an abnormally small fetus. MC, whom I was suspecting more and more may have been PI, had been quite a low birth weight. Only when I found out there were some risks to causing an abortion did I decide not to go through with it.

However, even more unsettling than finding out that all the animals in the west and south had been infected was finding that alpacas in the East section also had antibodies, despite no signs of illness there. VF could have been positive as the mother of MC if he was PI, but Velvet was positive also. I got out all the fleeces from shearing from all the alpacas from the East section, and checked every one for stress breaks. I actually hoped that I would find some breaks, so that by measuring where the break was I might have an idea of when BVD was in the East section. There were no stress breaks in the fleeces from the adult females. There were also none in the adult males from the other barn. The ten cria fleeces were harder to assess – the wispy friable tips would break off; I checked cria fleece samples from other years and this was the same case. However, there were three that had an additional break further down from the tips. The crias' dates of birth ranged from June 1 to October 28, so if some of the younger ones also had stress breaks they may have coincided with the friable tip breaks. There was no way of figuring out from these cria fleeces, from cria with a wide range of birth dates, when they had had their subclinical infection with BVD. Since it appeared that alpacas in the east had been subclinically infected with BVD, I now had to worry about all the pregnant females who had been in that section and whether they would have PI cria, even though there was not supposed to be such a thing. I decided to have more antibody testing done on alpacas that had been in the East section. I tested Tulia and Nevada, two of the cria who had been born in 2003 and therefore would have been in very close contact with MC, while eating their pellets together, and two more adult females, Arani and Snow White. All of them had antibodies to BVD. All the cria born so far in 2004 were checked for the virus (a different test – the PCR test) to make sure they were not PI – all of them were negative.

Danae and another female, Teaya, and their cria had left my farm for their new home on November 22, 2003, at least a week before Elena had shown the first sign of being unwell. Danae had delivered on October 9, 2003, been bred on October 23, and aborted at her new home on May 16, 2004, a day after shearing. The fetus was not sent for testing. At that point the owners were unsure exactly which alpaca had aborted, and even if it was known for sure it was Danae, there was no reason at that point to suspect she had been exposed to BVD – the antibody tests showing that BVD had been in the East section of my barn, where Danae had been before going to her new home, had not yet been done. Danae had returned to my farm for re-breeding after her abortion. I had her antibody level done and it was positive - the highest titre (1:3072) of any done. Danae had left the farm November 22, and Elena's first sign of illness was noted down on December 1, so Danae's positive antibody test showed that BVD had been active in the east section (subclinically) prior to Elena's first symptoms. I asked Teaya's and Danae's

new owners to test Teaya's 2004 cria, born at the end of May, for the virus to make sure he was not PI; he was negative. They also had antibody tests done on three alpacas that had been in close contact with Danae, Teaya and their 2003 cria soon after their arrival there in November 2003, to make sure the new arrivals were not acutely infected with BVD at the time of their arrival and infecting any alpacas at their new home – those tests were negative. Two other females and their cria had left my farm for their new home in early September, prior to VF and MC arriving; I had one of those females tested for BVD antibodies and that test was negative. I took that as evidence that BVD had not appeared on my farm until after their departure

I now had enough evidence to start to suspect MC as a PI alpaca since it appeared likely all alpacas in the East section had been subclinically infected. The background on VF was that she was bred on July 4, 2002. She was still at her farm of origin, Farm E, in Alberta, until some time early in August 2002. She was then at Farm D in Alberta for a few weeks as a drop off and pick up spot, and went to her new home, Farm C, in Alberta at the end of August 2002. She stayed there until late March 2003 when she traveled to Ontario to Farm B where she was consigned to an on-farm auction. The new owners, who bought her at the auction, decided to have her stay at Farm B. MC was born on June 13, 2003; he and VF stayed at Farm B until they came to my farm in September 2003 for VF to be bred. If he had been PI then I expected some of the alpacas at Farm B may have been unwell after his birth, or there may have been abortions, or at least there would be some alpacas with antibodies to BVD. I emailed Farm B, explained the whole situation, asked if they had had any problems, and asked if they would agree to have some antibody tests done if I paid for them. The only two pregnant alpacas that may have been in the same enclosure as MC, or may have been moved out prior to his birth, had delivered in October 2003; MC had been there from his birth in June until September, so those two would have been fairly far along in their pregnancies during that time. Remember that VF is not in any way contagious; she is not PI, and had had the ELISA test that proved that – it would only be after MC was born that there would be the possibility of BVD infections if he were PI. Farm B reported that one alpaca who had been in with VF and MC had been lying around more than usual at the end of June, about two weeks after MC's birth, but that was just noted for one day. They thought they should pay for the antibody tests; I thought I should pay (this is Canada for you!) I suggested that if the tests were negative, I should definitely pay for the testing as it would confirm this was a hare brained idea of mine. Farm B had antibody testing done on three alpacas, two of whom had been in with MC, and one who had not. The two who had been in with MC had antibodies to BVD and the one who had not been in with him was negative. One of the ones who had antibodies was the alpaca that had been lying around more than usual at the end of June – that was likely her manifestation of BVD infection.

Since VF and MC were in my quarantine area here from their arrival on September 21 until being put in the East section on October 8, it was not possible that one or both of them had spread BVD to my herd as acutely infected animals (having just contracted it at Farm B) - they wouldn't be shedding the virus that long as acutely infected animals. It was really starting to look as if MC could have been a PI alpaca.

Of course now Farm B had to do testing to make sure none of their cria born in 2004 were PI, and to notify customers who had bought alpacas or were about to buy alpacas. They lost sales because of their integrity. On August 3, 2004 Farm B phoned to say they had been faxed the results of the PCR tests that had been done on all their cria born so far that year, and that one of them was positive. This would mean he could be PI. This cria had been born to a female who had been bred July 28, 2003 and had never been in the same enclosure as MC. To prove the PI status the test would have to be repeated after three weeks. Then ensued a very upsetting three weeks for Farm B as they instituted quarantine procedures and contemplated that if the second test were positive, the cria would have to be euthanized. With repeat testing, the PCR test was equivocal, but the virus isolation test was negative. His mother's antibodies were checked and they were negative (meaning it was unlikely she had had a BVD infection) so it appeared this cria was not PI, and that the first PCR test was a false positive. Farm B breathed a sigh of relief.

I was still trying to figure out why there was the cluster of infections in the West and South sections of my barn in December 2003, starting with Elena's symptoms first noted down on December 1. When I looked at MC's records again, the answer was obvious. MC had been on injectable Excenel (an antibiotic) for pneumonia from November 11 to November 17, with improvement in his symptoms. When he relapsed soon after, on November 22, he was put on oral Septra and the vet suggested it would be worth keeping him on this for three weeks; he was on this until December 14. I administered this twice a day – the fingers of my left hand would be in his mouth to open it up while my right hand squirted in the syringe measured amount of antibiotic. Although most of the morning doses were given on an early morning barn check, after which I returned to the house and washed my hands before breakfast, many of the evening doses were given right after MC had finished eating his pellets. He and all the other cria ate their pellets together in an enclosure separated from the adults. I would either have given MC his medicine as one of the last things I did before moving to the West section for feeding, or I would come back after putting out the food in the West, give him his medicine once all the cria had finished eating, and go back to the West to supervise. Elena (the first alpaca ill) was the fastest eater in the West section. I routinely fed her a few pellets or some grain out of my hand after she had finished her bowl to prevent her from stealing the pellets from other alpacas. In fact most of the alpacas in that section would have eaten pellets out of my hand at some time or another over the space of a few feedings. My fingers would have been contaminated with MC's saliva. The incubation period of BVD is five to seven days. I started oral antibiotics for MC on November 22 and I noted down Elena's reluctance to eat her pellets on December 1; it is likely that it had started a day before that, as I would not have noted down just one feeding that wasn't normal. Why would a doctor who routinely washes her hands between every human patient, and who washes her hands as soon as she comes in from the barn, not wash her hands after treating MC? Sheer stupidity of course comes to mind. Since this was not a conscious decision not to wash my hands, I think in my subconscious I knew I was treating a recurrent bacterial pneumonia in a compromised cria (which I was) and did not think that he had anything particularly contagious to the other alpacas. After 48 hours on the antibiotic he shouldn't have been contagious for bacteria anyway -

antibiotics are of course not effective against viruses. All the other alpacas in the East had been with him since October 8 and none of them were ill; that included all the other cria – they had the closest contact with him, as they all ate their pellets together. I never considered the possibility of an underlying viral illness transmissible by his saliva. The symptoms of alpacas in the West just didn't seem to have anything to do with MC's recurrent pneumonia. Some may have been infected from each other after the initial cases, but I'm sure I infected a lot of them with my virus contaminated hands – a PI animal sheds huge amounts of virus in every secretion, including saliva. I also started MC on a vitamin B pill in early December that he was on until his death – my fingers had even more contact with his saliva when I gave him that. I did start washing my hands after examining the unwell alpacas in the west, but I would have had MC's saliva on them prior to that. The phrase killing with kindness seems particularly apt. This was yet further evidence that MC was a PI cria.

And then there was still the question of why none of the alpacas in the East section had appeared unwell – it seemed likely they had all been subclinically infected. There are several possible explanations. One is that the age group of the alpacas in the West and South sections (most of them between one and two years old) may be more susceptible to manifestation of illness by BVD. Another possibility is the effect of 'viral load' – the amount of virus the animal ingests is correlated with the severity of the illness; it's quite likely the alpacas in the West and South received a higher viral load off my saliva contaminated hands than the alpacas in the East received from indirect contact with MC and his secretions. The third explanation was presented by a bright vet tech student at the local community college when I did a presentation on alpacas. All the adult females in the East section had been alpacas that I had bought; they had all come from other farms – perhaps some of them already had protective antibodies from being exposed to BVD in the past on the farms they had come from.

Meanwhile, I had contacted Farms C and D in Alberta by email. If MC were PI then his mother would have been in contact with BVD during her early pregnancy and that would have been on one of those three farms she had been on. She had been bred July 4 at Farm E, went to Farm D sometime in early August, and then went to Farm C around the end of August. If she were a cow it would have to be after approximately 40 days gestation (and before 120 days) that she would have come in contact with BVD in order to produce a PI offspring, so it would likely be on Farm C or D that VF had come in contact with the virus, and not Farm E. None of the farms have any cattle. Farm C said that they had had many abortions in the spring (mostly April) of 2003. They had a lot of tests done at the time of the abortions, and the abortions were attributed to a toxic mould in the hay. I told them I suspected that the mould was an incidental finding and that it was much more likely that the abortions had been caused by BVD. Farm C had their vet out to discuss the situation. Farm C emailed me with what their vet told them: "He said that he had the labs check out our abortions last year for BVD by doing a Complement Fixation Test on the brains of the fetuses. He used three different labs for the tests, and no lesions were observed, so he feels that BVD was not the cause of the abortions here. He feels they were related to feed, which contained toxic moulds." The only problem with that statement was that complement fixation is just not a test that is

ever used to detect BVD. Several months later I asked Farm C to double check with their vet about this and it turned out there was no record of any complement fixation test, or any test for BVD. In August, Farm C went ahead and had antibody testing done on four of the females who had aborted in 2003 and were flabbergasted when the tests came back positive. They then had testing for the virus on all the cria born in 2003 and 2004 to check for any PI animals, and those tests were negative. They checked a recent arrival on their farm and she was negative for antibodies; this would be good evidence that BVD was no longer active on their farm.

Farm D in Alberta had had, in 2003, two stillborn cria, one full term, and one two weeks early, and a cria that died at about 36 hours of age. In September 2004 they had BVD antibody tests done on some alpacas (dam of one of the stillbirths and dam of the cria that died; the vet was unable to get blood on the dam of the other stillbirth) and they were also positive. VF is not PI; if she were the vector between Farms D and C (meaning if she had spread it from Farm D to C), she would have to have made the move (at the end of August 2002) while acutely infected and during the short time she would have been shedding the virus. In cattle it is the PI animals that are the major vectors as they continually shed huge quantities of virus (as opposed to not as much virus and for a very short length of time, in an acutely infected animal). I thought it more likely that Farm B and C had had different exposures to BVD (probably from two different PI animals), and that I would never know with certainty on which farm VF had had her BVD infection that resulted in MC being (I thought) a PI cria.

By August, after finding out Farm B's positive antibody results from the alpacas which had been in contact with MC, and Farm C's positive BVD antibody levels in the alpacas who had been pregnant at the same time as FC, but had aborted, I was convinced that MC must have been PI. I was also starting to worry not only about Mikayla's fetus being PI, but also about the cria still to be born to the females who had been in the East section. With my pregnant females in the West section I was sure they had been exposed to BVD in mid to late December, so it was easy to know their stage of gestation at that time. With the pregnant females in the East section, there was no way of knowing how soon after MC joined them on October 8, 2003 that each was exposed to the virus and therefore at what stage of gestation the fetus was exposed. In early August, the breeding and birthing dates up to that point were:

West Section	**Breeding Date**	**Gestation mid Dec/03**	**DOB of Cria**
Harley	June 17 /03	6 months	June 4/04
Merienda	July 2 / 03	5.5 months	June 16/04
Mikayla	Oct.14 / 03	2 months	
Mandy	Oct. 13 / 03	2 months	aborted March 29/04
Abela	Oct. 13 /03	2 months	early pregnancy loss
Tillie	Oct. 6 / 03	2.25 months	early pregnancy loss

East Section	Breeding Date	Gestation Oct. 8 / 03	DOB of Cria
Annicka	June 20/ 03	3.5 months	May 30
Lambada	July 9/ 03	3 months	June 10
Cindy	July 17/ 03	2.75 months	June 13
Arani	July 18/ 03	2.75 months	June 22
Talara	July 27/ 03	2.5 months	June 22
Jasmine	Aug. 13 and 16/ 03	7 - 8 weeks	July 8
Murragamba	Aug. 25 and 29/ 03	almost 7 weeks	
VF	Sept. 22 and 26	no way she would have a second PI cria	
Misty	Oct. 6 and 10 / 03	just pregnant	early pregnancy loss
Mackenzie	Oct. 19 and 22/ 03		
Snow White	Oct. 27/ 03		
Velvet	Nov. 26/ 03	not pregnant when checked late Dec.	
Shawnee	not pregnant		

As of early August, the cria born so far had normal birth weights, were healthy and had tested negative by PCR test for BVD virus – every time there was a birth I had the vet out to do the test. MC had only weighed 9 pounds at birth, and Mikayla definitely had a smaller pregnant abdomen than she should have; I really suspected she was carrying a PI fetus. With the way my barns, fences, gates and record keeping are, there was no chance that she had been bred on any day other than the one I had recorded. All the females in the East still yet to deliver had big pregnant abdomens, so I was not as concerned they were carrying PI fetuses. Still I found it amazing that Jasmine's cria was normal – the only explanation I have is that she did not get exposed to the virus until later in October or November, or that she had pre-existing protective antibodies from a previous exposure to the virus on her farm of origin. I had not done antibody testing on every female (no one was footing the bill for all this except for me) but I assumed they would all be positive. I also assumed that because Danae, who had left the East section and the farm November 22, had antibodies, that probably the whole East section would have been exposed to the virus by then, but I will never know. It is possible that Danae was exposed on the day she left because it coincided with the day of MC's relapse of his pneumonia, and the start of his oral antibiotics (again with my hands as being the vector, as I may well have offered her a treat from my hand to get her in the trailer). In October and early November, the alpacas were still spending most of their time grazing, and I have large pastures. Dung areas in the barn are cleaned up twice a day, and in the pasture every few days, alpacas are protective of their personal space, and they do not lick things, so I'm assuming it's possible that the alpacas in the East did not get exposed to the virus immediately after MC joined the group. Perhaps many of them were not infected until after November 22.

Murragamba delivered an 18 pound healthy cria on August 7 and the PCR test was negative. I had antibody levels done on Murragamba at the same time – they were positive, but on the low side (1:32), perhaps (but certainly not definitely) indicative of exposure in the past before she arrived on my farm in 2001. VF delivered a 15.4 pound cria on August 26, and as expected, the PCR test was negative – her antibodies would

have protected her from becoming infected again. This cria continued to gain weight really well and was healthy. I was assuming that Mackenzie and Snow White, both with big pregnant abdomens, would have been exposed to the virus either pre-conception, or so early in their pregnancy, that it was unlikely they were carrying PI fetuses, even though they had been bred around the same time as Danae.

By now, Dr. Carman, the virologist at the Animal Health Lab at Guelph University (home of Ontario's only veterinary college) had been fielding many phone calls from me and Farm B as we struggled with all the issues of BVD and the different tests. I phoned her up at the end of August and explained that my plan was to euthanize Mikayla's cria at birth if it was low birth weight because that would make me sure it was PI; I did not want to have poor Mikayla cope with a cria who disappeared at the age of three or four weeks old when it was proven to be PI, and then euthanized (which is of course what must be done with a PI animal) and I did not want to cope with all the biosecurity issues of having a PI cria on the farm. I asked her if the euthanized cria tested positive for BVD virus, would that prove that there was such a thing as a PI cria (she had already told me she had not yet seen any evidence to make her think there was such a thing, despite the 'trail of antibodies' found at Farms B and C). She said that to prove the PI state there must always be two positive tests for the virus taken at least three weeks apart, in case the first test was positive from an acute infection. I knew a first positive test would not be from an acute infection acquired just before birth – there would be no source of infection, and anyway, Mikayla already had antibodies. However the scientific community would not accept anything for proof except the two positive tests taken three weeks apart. At first I didn't think I was prepared to put Mikayla and myself through this just to prove a point, but then I decided for the sake of scientific knowledge it would be the best course of action.

Farm B probably believed there was such a thing as a PI alpaca, but other than me they were the only ones. Farms C and D had pointed out the studies saying that BVD didn't cause illness in camelids or affect the fetus. No one seemed to remember that I had definitely had BVD on my farm and that it had caused illness and an aborted fetus, and there had to be an explanation for how BVD had been brought to my farm. Obviously they had not read their Sherlock Holmes stories: "When you have eliminated the impossible, whatever remains, however improbable, must be the truth". It was impossible that BVD had been brought to my farm by cattle, or deer, or manure contaminated boots – all that was left was the improbable - a PI cria – made less improbable by the 'trail' of antibodies. I have been a doctor long enough to have seen what is considered the absolute truth in regards to research findings or treatment at one point in time to be proven false some years later. It still amazes me that many people (including many doctors) do not see the logical corollary to that, which is that some of what is considered correct today will be proven to be wrong in the future. I had already proven wrong the concepts that camelids do not get seriously ill with BVD and that BVD does not cause abortions in camelids. I saw no reason not to think that the concept of no such thing as a PI alpaca might also be wrong. I had some inkling of how the first researchers felt who were treated with disbelief or derision for proclaiming that smoking was bad for you.

Snow White and Mackenzie both delivered before their 11 month mark, as they usually did, and had good sized healthy cria that tested negative for BVD. As Mikayla's due date approached I made my preparations. In the quarantine area there were still two females from another farm – one who had come for breeding and was now about 2 months pregnant, and her companion. The owners wondered if they could stay longer until it was more convenient for them to pick them up. I explained why I wanted them off the farm before Mikayla delivered, and my husband drove them part way of the way home to meet their owners. I had never moved Mikayla to the East section where all the females go at least a couple of months prior to delivery, because I did not want her there if she had a PI cria. I had kept her with her cohort of two year olds (now all bred) and the one year old girls. I went shopping and bought an extra pair of boots, lots of disposable gloves, disposable plastic boot covers, liquid bleach, hand disinfectant, and more pails, and had my plan in place about re-groupings. Mikayla's 11 months was up on September 14. For the preceding months she had had what I can only describe as an extremely care–worn expression on her face – quite unlike her usual demeanor, On September 15 there was a noticeable lightening in her expression, and I saw her looking at the boys slightly coquettishly. It was time to make the final moves. I put Mikayla and the one year old girls, all of whom I was sure would have antibodies to BVD (I had actually only tested two of them) because of their close association with MC as cria together, and none of whom were bred, in one grouping in the West section so that there was no fence line or barn contact with any other alpacas. I moved the other two year olds to the South, and moved the juvenile boys to the quarantine area. I decided to hold off on erecting solid partitions over the open slats between the West and South sections in the barn until the cria was born and see what its weight was.

On Friday September 17 Mikayla went into labor; I was there for her labor and her delivery, which was normal. The cria, a cute little white male, stood and nursed in the normal length of time; however, 'little' was the operative word – he weighed only twelve pounds and was able to walk easily under his mother. The 63 births I had had on my farm prior to this had weights ranging from 15 pounds to 24 pounds, and the average was 18.1 pounds. Mikayla is a big girl in good body condition; when I weighed her several months after this, she weighed 174 pounds. Her mother's cria have ranged from 18 – 24 pounds, and Mikayla herself weighed 20 pounds as a newborn. This was truly an abnormally low birth weight for my farm, and for an alpaca the size of Mikayla. Originally I was going to go with my sister's suggestion of naming the cria Magnum PI, but decided instead on Gabriel, as I thought he would either be joining the angels soon, or it would be a small miracle if he were not PI. I decided to assume he was PI and institute full 'biosecurity' measures. Of course the reality was that probably all the females on the farm already had protective antibodies and were at no risk at all, but I had not tested all of them. The cria born earlier that year would have ingested antibodies in their mother's colostrum, but perhaps in the older ones those levels would be waning by now. I had a pair of boots that I kept only in the west section of the barn and stepped into as I went through the door to that area, and stepped out of when I left. Everyone else donned plastic boot covers while in that area and removed them as they were leaving. I always had a supply of disposable gloves in my pockets and in a container in the West section – if I had any contact with Gabriel I donned the gloves in order to open doors and gates to

get back to the house where I immediately washed my hands and changed clothing. The solid barricade went up over the slats between the West and South sections. The self-filling water tank that straddled the South and West sections was closed on the South side and I had to start watering that group by hose and bucket.

The only unusual thing about Gabriel's birth was that he passed a really large amount of meconium starting about an hour after he was born. There was so much that I had to clean off his rear end and hind legs the next day. I had the vet out on Monday for the blood tests. I had phoned Dr. Carman after Gabriel was born on the Friday to tell her he was really low birth weight and to discuss the blood tests. If the PCR test was positive it would be confirmed with virus isolation (another blood test) and then both repeated in three weeks. I decided to get blood drawn for both the PCR and the virus isolation on the Monday to save time, as I was so sure the PCR would be positive. The PCR test is run once a week at the Animal Health Lab in Guelph, and virus isolation is set up once a week and then it takes two weeks after that for the result to be read. Gabriel's PCR test for BVD virus was repeated three times as the first reading was 'suspicious' – however the subsequent two were negative, and it was reported as such. I found that hard to believe – so, another phone call to poor Dr. Carman, who did not say 'I told you so' but did say that no test is 100% accurate. The virus isolation test result would not be available for another two weeks, and I was not prepared to stop all my biosecurity measures until I was positive Gabriel was not PI. I had frozen the placenta, so I decided to send off some of that for virus isolation too.

For the first few days after his birth, Gabriel looked a little fragile; he sometimes looked as if he had trouble figuring out how to negotiate the step up into the barn, even though it was only a couple of inches. But after that he behaved quite normally. He gained weight well; he was up to 20 pounds by 2 weeks of age. However he had persistent diarrhea - runny and yellow at first, then brown and more pudding-like in consistency; I cleaned off his rear end several times. He was normally active, but just didn't look quite the same as the other cria – his fleece looked somewhat 'scruffy'. He also had weepy eyes – not pus, but some clear tears that caused dirt staining down from the corners of his eyes. He also had a large umbilical hernia (as had MC). It was a good grouping he was in with – the one year old girls were quite tolerant of his cria behavior and were like big sisters. At one point Gabriel had them all running and pronking around the field with him. I think he would have been overwhelmed in the larger grouping of older mature (i.e. occasionally snarky) moms in the East section.

The virus isolation test (the 'gold standard' test for BVD) came back when Gabriel was almost three weeks old, and it was positive. I was not surprised, but I think Dr. Carman was. It certainly seemed Gabriel would prove to be PI. Dr. Carman was in touch with Dr. Deregt, a BVD expert in Lethbridge, Alberta, who originally wondered if perhaps I would send Gabriel to live in his lab – that was easy to answer no to. I had already decided to have the second set of tests done three weeks after the first, as that would be the only way anyone would believe there was such a thing as a PI alpaca. Now Dr. Carman asked if I would wait a further three weeks after those tests, and have a third set of tests done, before Gabriel was euthanized, so that no one would cast doubt on the

diagnosis. In return, she and Dr. Deregt would arrange to cover the costs of all the final tests, the euthanization, and all the postmortem testing – this was a relief as so far I had been funding all the research into BVD and alpacas myself with all my blood tests. The placenta tested positive on virus isolation. The PCR and virus isolation done three weeks after the first tests were also positive, confirming that Gabriel was PI. It was of course not reassuring that the first PCR test on Gabriel had been a false negative, as it cast doubt on the validity of all the PCR tests done on all the other cria. I had to have the vet out to draw blood on all the other cria again, and the Animal Health Lab repeated the tests at no charge – they were all negative.

Gabriel continued to gain weight well and was perky and active. He weighed 32 pounds at 6 weeks of age – quite a good gain from a birth weight of 12 pounds. His diarrhea persisted – not very much of it and not frequent, just not formed; but he was not unwell with it. It was of course poignant to watch this cute little cria doing all the normal cria things, and know what was in store for him. He was one of those naturally gregarious cria who always came over to see what you were doing, and always wanted to nibble on your clothing – the absolute last thing I wanted. I was continually evading him when I was in the West section. It was even more amazing that he was so friendly considering the only times I touched him were for unpleasant things – weighing, holding for blood tests, and cleaning off his rear end. All I could do was assure that his life was pleasant and carefree while he was here.

I made plans for the euthanization, which was to be several days after he was 6 weeks old. He was to have a lot of blood tests just prior to this and I certainly did not want his last minutes to be a time of fear and pain. I also did not want Mikayla to see me taking him away, never to be seen again. I decided to ask the vet for the cocktail of drugs used for general anesthesia (butorphanol, ketamine and xylazine) to be given prior to the blood tests. The vet waited in the garage. I had the syringe with the drugs. Luckily Gabriel usually stayed outside to play while his mother came into the barn for her pellet feeding. I gave him the injection after she was tucked into her food and within a minute or two he was quite sedated. I handed him over to Paul waiting on the other side of the gate and he took him down to the garage; Gabriel was unconscious before arriving there. The blood tests were done, and then he was euthanized with another injection while he was still unconscious. His mother did not know anything had happened. The amazing thing was that Mikayla did not seem to miss Gabriel – I only saw her having a quick look inside the barn once and heard hardly any hums. The care-worn expression she had had during most of her pregnancy that had lessened, but not gone, just before delivery seemed to disappear a week or so after Gabriel's death. Though Mikayla was a good mother to Gabriel (he could nurse whenever he wanted) she never seemed to dote on him the way some mothers do. I do wonder if in some way she knew things were not as they should be.

The tests done at the time of euthanization were of course also positive for BVD virus. Virus isolation tests done on most tissues from autopsy (for example, kidney, spleen, brain) were also positive for BVD virus. Fecal testing did not show any parasites to account for the diarrhea. Gabriel had looked essentially normal and at this point had

not been unwell; yet if he had been with alpacas that did not already have protective antibodies he would have infected them all with BVD. It was easy to see how BVD could spread between farms with a cria like this accompanying his mom when she went to another farm for breeding. And of course MC had shown how BVD could travel across the country from Alberta to Ontario in an unborn fetus.

I know I can never state with scientific certainty that MC was PI as he was never tested, but I think there is overwhelming circumstantial evidence that he was. I also cannot state with scientific certainty that the abortions at Farm C and the stillbirths at Farm D were due to BVD, as the fetuses were not tested for that, but the presence of antibodies in the dams would certainly be extremely strong evidence that was the case. I was very fortunate that Farms B, C, and D were willing to have antibody tests for BVD done; other farms might have preferred to ignore my request. This detective story would not have been solved without their co-operation. Dr. Carman was also very helpful and unfailingly continued to answer my phone calls, emails and questions.

It's interesting to think how things could have turned out quite differently. If our friend Grant had not mentioned about the illness in cattle that his son had told him about I really doubt I would have stumbled upon BVD as an explanation for MC being such a 'poor-doer' and as the possible cause of the illness and subsequent abortions in my alpacas. And yet that case of BVD2 twenty km. away had absolutely no connection with the BVD1 on my farm. The BVD here had come from Alberta. I usually don't breed my females until they are 18 months old, but in the fall of 2003, Mandy, Abela, Elena, Tillie and Mikayla were such big well-grown girls that I decided to try one breeding in October, and if they didn't get pregnant, to wait until the next spring. Mikayla was only thirteen months old. I know other people breed that young, but this was the first time I had, and it was only because she was as big as some of the adults and had a mature personality, and because on the day I bred her she had been looking with interest at the boys. All these girls got pregnant right off the bat. If I hadn't bred them, there would have been no aborted fetus from Mandy to test positive for BVD, and no PI cria, the first verified PI alpaca in the world, from Mikayla. (Of course I believe there have been many PI alpacas before this, but Gabriel was the first one tested). The illnesses in December and the underlying diagnosis for MC would have remained a mystery.

There are still lots of unanswered questions. What is the crucial time during gestation that alpacas will produce a PI cria if they don't abort? In cows, it usually has to be after 40 days of gestation in order for the virus to cross from the dam to the fetus. Danae had been bred October 23 and left the farm November 22 – only 30 days pregnant. She aborted after shearing at her new home the following May. She had gone through three previous pregnancies and had been sheared during each of those without aborting. There is no way of knowing whether Danae's fetus was infected with BVD as it was not tested, but her high titre of antibodies to BVD in June (1:3072) could be taken as evidence, but not proof, that it was. If so, that would mean that the virus can cross over to the fetus earlier than in cattle, and also that abortions may occur not just a few months after infection of the dam (as with Mandy), but after many months. The alpacas at Farm C aborted in April 2003, and the stillbirths at Farm D were also in 2003. VF likely had

her acute infection with BVD in August or September of 2002 at one of those farms, and if the other alpacas were infected at the same time, there was also quite a delay before they aborted or had the stillbirths. Up to what point in gestation can infection with BVD produce a PI cria? Can an alpaca be infected with BVD during the crucial phase of gestation and still produce a normal cria? Can BVD cause congenital abnormalities in cria if exposure is later in pregnancy, as it does in cattle? All of my cria that were in later gestation when their moms were exposed to BVD virus turned out normally. Can some PI alpacas stay appearing healthy for an extended length of time? At this point the only clinical information is on Gabriel and MC. Both had very low birth weights; both had large umbilical hernias. Other than unformed stool, Gabriel was fine, had gained weight well, and had had no illnesses at the time of his euthanization. MC did well for the first 6 weeks of his life, and then had pneumonia; his next episode of pneumonia was not until he was 5 months old.

Only time and testing will tell just how common BVD is in alpacas. I believe it is much more prevalent than any one has thought. Alpacas certainly have a reputation for being easy aborters and it is not unusual to hear of poor doing cria – both of these could be from BVD. The article *Communicable Disease Risks to Wildlife from Camelids in British Columbia* by Dr. Schwantje and Dr. Stephen, cited in the previous article on BVD, shows 6% of llamas sampled had antibodies to BVD, that camelid owners reported the most common cause of death being neonatal failure to thrive or stillbirths, and that 9% of camelid submissions to the provincial lab had the diagnosis of idiopathic (no cause found) abortion. No one has ever seen a connection between these. There were no pathological findings to suggest BVD in Mandy's aborted fetus or in Gabriel – only testing specifically for the virus showed it to be present. It is quite possible that many of the alpaca abortions sent for testing where no cause has been found could be from BVD – it has never been considered one of the routine tests. Poor doing cria who died would have autopsy findings in keeping with their final illness such as pneumonia, with no indication that their underlying problem may have been that they were PI. I think it likely that BVD has been around in herds for quite a while, and that it has been spread by unrecognized PI animals. Many females with cria at side go to other farms for breeding; if it was a PI cria it would be infecting all the alpacas at that farm. If there was a PI alpaca on a farm, a female going there for breeding could return carrying a PI fetus. The first BVD cases may have been contracted from cattle, or, considering that a study from Peru showed an 11.5% incidence of antibodies to BVD, some imported alpacas could have been carrying PI fetuses. The experiences at Farm C, with many abortions, are probably the exception. Just having a few abortions, or the experience at Farm D, with a couple of stillbirths, or Farm B, with only one mild illness, may be more typical.

The whole concept of persistent infection will be hard for some people to grasp. I expect that many people will not want to know about or deal with this. Alpaca owners have a reputation for being secretive about any illnesses or deaths in heir herd; I doubt that many will broadcast that they have discovered BVD cases in their herd. I also know that some people will not have enough scientific understanding to realize that I do not have an infected herd, that I have no contagious animals now, and that those alpacas that were infected and recovered are absolutely normal and have no long term consequences

from their exposure to BVD virus – in fact they are now protected from any BVD infection in the future. All the cria have been tested to prove they are not PI. I hope that by reading my experiences, alpaca owners will start to request BVD testing for aborted fetuses and any poor doing cria, and that someone will be inspired to continue research into BVD and alpacas. And of course I hope I will prevent other farms from going through the stress, worry and grief that we went through.

Appendix of Antibody Titres: Date of birth of the alpaca is in brackets after the name

Tests done June 1/ 2004:

	BVD2-VN (type 2, NVSL 125c strain)	**BVDN-VN (type 1a NADL strain)**
Females from the West:		
Tillie (July 23/02)	1:48	1:128
Mikayla (Sept. 14/02)	1:192	1:768
Celeste (Oct. 25/99)	1:64	1:256
Abela (July 13/02)	1:64	1:768
Harley (Oct.23/01)	1:8	1:192
Mandy (June 29/02)	1:24	1:512
Savanna Dawn (Nov.14/01)	1:24	1:128
Merienda (July 22/01)	1:96	1:1536
Females from the East:		
VF (May, 2001)	1:384	1:1536
Velvet (July 31/95)	1:16	1:64
Young Males who had been in the South:		
Cosmo (Aug.22/02)	1:128	1:1536
Dirk (June 23/02)	<1:2	<1:2
Adult Male from the Male Barn:		
Dano (June 9/01)	<1:2	<1:2

All of the above animals, with the addition of Shawnee from the East, had negative ELISA test for BVDV antigen at the same time. There was not enough blood left to do antibody testing on Shawnee

Tests done June 29/04:

From the East Section	**BVD2-VN**	**BVDN-VN (type 1a)**
Tulia (July 12/03)	1:48	1:1024
Nevada (Oct. 12/03)	1:96	1:2048
Snow White (Jan. 7/98)	1:64	1:192
Arani (Jan. 2/97)	1:24	1:192
Danae (May 25/99)	1:192	1:3072
Test done July 14/04		
Carmella – had left my farm for her new home in early September prior to the arrival of VF and MC on my farm	<1:2	<1:2
Test done Aug. 9/04		
Murragamba (Sept. 8/98)	1:8	1:32

The higher titres to the type 1 strain showed that that was the strain the alpacas had been infected with. The lower titres to the type 2 strain were from cross-reactivity in testing. A titre of <1:2 is negative.

Nancy Carr MD is the owner of Silver Cloud Alpacas, near Elginburg in eastern Ontario, Canada. She would like to assure readers that her herd is now completely healthy and not contagious, and in fact is one of the very few herds in North America where all the cria have been tested to make sure they are not PI. She can be reached at carralpacas@sympatico.ca or (613) 376-3389 or through her web site www.silvercloudalpacas.com