THE FOLLOWING POSTS have been extracted from public forums with no changes in spelling, grammer or typographical errors. All posts were made in english, however for some contributors this meant writing in a foreign language.

We appreciate everyone's efforts to support further research and education on Cerebellar Abiotrophy by sharing their experiences.

By Abigail Ligthert, Arabian Fantasie, the Netherlands. (posted May 2010)

Why testing is so important …how our surprise became a nightmare.

A.F.Vesjnica CA AFFECTED fillyfoal (Vestival x Adina by Andoy), born: 25 April 2010

Last year we decided to cover our grand old dam Adina one last time with our stallion Vestival (Karnaval x Verdjina s. Balaton). She came in heat after the covering so we assumed she did not get in foal.
When she became rounder than she normally is at the beginning of the new year, with some activity at the udder, we were sure she had to be carrying a foal. Since the CA testing frenzy only started after the 2009 breeding season, we found out that the sire of Adina's foal is a CA carrier in the late fall, and we immediately tested her as well, although it would not change the status quo. She never had unhealthy foals before, even from this sire, so what would be the odds ? Twiddling our thumbs we awaited foaling time.

On the 25th of april Adina gave us a tall filly. Joyous with her arrival we called her A.F. Vesjnica. Very lively, she tried to stand, what obviously never succeeds at the first try. When she still did not stand after a few hours, we started to worry.
First we gave her a bottle with the mares' first milk. Soon after that, she started to tremor with her head. We could not believe it at first. After 6 hours she finally stood on all fours, wide stanced and wobbly. On the 6th of May we got confirmed what we of course already expected: Adina was tested a CA carrier.

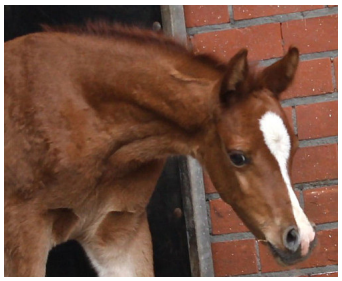
After 20 years of breeding and although we just recently had taken rigourous measures to

guarantee a CA free breeding program by eliminating all carriers from our herd, the pre-testing era caught up with us and so the very emotional decision to euthanise this filly has become a harsh reality for us after all.

This is one of the greatest heartaches a breeder should never face. Please, test before you breed and follow the CA breedingchart so you never have to experience this emotional feat.

As a breeding program, Arabian Fantasie had already decided to breed solely with CA clear horses. That does not mean that we object to careful breeding with CA carriers, but since we are blessed with a CA free herd now, we will make it an effort to keep it this way.
We hope that Vesjnica's short life will serve as an example to breeders who still have their doubts about CA testing and breeding according to the CA breeding chart.

Click HERE for the video A.F.Vesjnica



.....A.F. Vesjnica; no chance to live a life of equine dignity.....

By Barbara Schwarz, CAFRA Arabians, Germany. (posted Jan. 2010)
Jhuliaa CF CA/CA Affected @ 2 years old: http://www.youtube.com/watch?v=ipnpQonib94
She was born on May 21st 2007. She was (and is) very beautyfull, correct and ist definitly better than her dam is. When she was some weeks old, i realised, that she had a very long, perfect shoulder-forehand confirmation and i expected to have bred a filly with fantastic movements. But her movements allways were poor, like she had a blokage in her shoulder. I felt sad about it, but was not wondering too much. Even though here movements were not as i expected them to be, as a yearling i gave her in show training, she competed in three national shows and her best result was a Classwinner. When she was two years old (2009 may) i realised a head tremor for the first time when she was stressed. Talking with people here at my farm, they told me, they had seen the tremor before, when she was stressed (i.e. seperated from other horses). In summer i got the information, that her dam is CA carrier and i decided to test Jhuliaa one day. On September 21st i got the information, that her sire is a carrier too. The same evening i found the (new) german side on wikipedia about CA and there i learned for the first time, that CA may

appear also at an older age. Till this evening i only knew that CA affected foals die early, at the age of 2 to 4 months. Also on this page i read for the first time the possible symptoms in german and understood them all. In this moment i realised, that Jhuliaa is not only a little bit of "seldom" or "crazy" but obviously sincerly ill i.e. genetic ill. That was a harsh experience. In the meantime i know that she was falling sometimes in summer, she was running against a pushcart some weeks ago and she did not see a latern standing in her way about two weeks ago. For the rest, she is o.k., if you don't know where to look for, you will not realise her problem. She is alive and does well, she is on the pastures with my other youngsters.

Posted on Global Jan. 8, 2010, See also El Acrisi: (Switzerland) Story posted below.
In 1991 I used a new stallion and got 1992 4 foals 2 of them I had to put down and the pathology diagnoses was CA. Those days from clinical signs CA wasn't known. I have a VHS video that I'm able to digitalize now.I put on some extra explanations for better understanding. It's the first time after almost 20 years that I watch this video and I had to cry again because all what happend to me in l992 due to this circumstances and what happend with my life came up again. But I think it's important that all the Arabian horse breeders, owners but also my veterinarian colleagues know about this disease and this video shows a lot of the typical signs. I would like to ad that these foals are the most frienly foals you can imagine due to their luck of opposition reflexes - you just have to love their sweet temper. I think everybody should download this video to not forget about testing breeding stock to avoid affected foals in the future. In case you would like to have a DVD with better resolution - you can PM me.
Video CA affected filly - http://www.youtube.com/watch?v=C_jh06UVi2c

Christina

GS ARROW RANCHES STORY (Feb. 2009): This story is to inform everyone that Cerebellar Abiotrophy (CA) is true, real, and heartbreaking and can happen to everyone breeding Arabians…This is our story. We are very small breeding operation. We have 3 mares and one stallion. We have only had 3 purebred foals ourselves. Our stallion (PS Final Edition) does have 5 total purebred foals. We had a chance to use a nice mare (SPA MZ Katrina) with comparable lines to our stallion so we did. We get her in foal waahoo. Wait a whole year yes I mean a whole year as the mare went over. We get a nice filly (GS Yesha Mishkin). We just love her and her pedigree is nice older lines. So, we breed the mare back. Next year a very nice filly nicer than her older sis at this age (GS Aesha Zaphina). Luckily, it was late in the year so we decided to leave the mare open for 09. We wean the filly at 4 months. Right up till weaning she was fine. About a week into weaning I noticed she was having small head tremors. I figured she was just having a hard time with weaning, as she was younger than I liked for weaning but mare was getting thin and baby was very healthy. She finely gets over “Where’s the Momma?” So I put her in with her older sister for the winter. All this time I keep thinking she is not getting better but worse. Now, her symptoms are front legs out, head below

withers, her head and upper part of her neck shake all the time and when she moves she is very stiff in the upper part of her front legs. Plus when she gets up from laying down she gets up with her hind first pushes front legs out stands at a bow stance, shaking then slowly brings her front legs one by one into position. Her tremors are so noticeable that non-horse people notice. About this time I see a small article on CA. So I start researching. It all comes back to CA. I contact Cecilia Penedo At UC Davis. She informs me that it is very likely she has CA. In go the tests. Back come the tests. Zaphina is an AFFECTED CA filly. Yesha is a Carrier. Of course our stallion and the mare are carriers. For us it is what to do now? We will have Zaphina put down After I get good pics and videos of her so people can learn this is a very real thing. As per our breeding well I am not sure cause on Jan. 27, 2009 we lost our ½ Arab pinto stallion to colic. I can say this we will test from now on. I am giving a general background about myself just to inform people that I am not new to horses. I have had horses for over 30 years. We have been breeding for 13 years mostly 1/2s. We show and trail ride. I was in 4-H and have worked on several breeding farms in my years. I also work part time at a vet clinic. I am not looking for sympathy I am just informing people that Cerebellar Abiotrophy (CA) is true and real. Do yourself a favor and your future foals test before you bred this spring. Written by Trina Gifford 2/2/2009

From Bazy Tankersley, "And Ride Away Singing"; pub. 1998, pg 259 In the 1990s section: "We're not sure where it came from, but we know that our herd (probably like most herds of large size) carries a cerebellar ataxia factor. If there's anything strange with a horse, everybody immediately decides it is hereditary. However, we have learned through breeding any number of affected animals that it is not a simple dominant or recessive gene. None have produced a cerebellar ataxia foal. In other words, mares that have shown signs of ataxia as foals have never produced foals with ataxia - only normal foals. In an experiment at a veterinary college, one of our affected stallions was bred to five of our affected mares resulting in only normal foals. Obviously we never use an affected stallion here, but we have used affected mares, as indicated."

The above excerpt refers to the 1965-66 foal crops which instigated the work done by Dr. Max Sponseller, published in 1967. By the mid-60s, no etiology had been firmly established; this study presented a strong case for a genetic mode of inheritance. We know now that If an affected stallion (with expressed clinical signs) bred 5 affected mares (with expressed clinical signs), all 5 foals would have had to be affected with expressed clinical signs. As Bazy points out "all 5 foals were normal looking"; the foals had to be carriers and the parents carriers or a combination of carrier and affected with clinical signs. ~ LG-C

On pg 199-200, Bazy wrote this comment on SCID:
" I have been terrified of it, but we have never, in breeding nearly 2500 foals, had a known SCID foal "

From HorseTrace.com Forum: Lorraine Patrick, (Australia) - Older Horses Thread.
This is Rocky, the foal out of the CA mare, no signs at all 'yet' but unfortunately the mare is getting worse, Rocky is now a gelding and he's just over 4 months old .His Mum is very wobbly, clumsy, falls over and bumps into things, she'd dropped a lot of condition but we've got it back now, big problem is she can't 'use' herself properly so no real muscle tone, she's such a pretty mare too and still only 4.

- Roscoe: http://www.allbreedpedigree.com/crystearos
- Monty: http://www.allbreedpedigree.com/crystearmont
- Filly: http://www.allbreedpedigree.com/rosea+beneta

From Arabian Lines Forum: Montana: (USA) I currently own a filly affected with CA. Her case is mild enough that I have not chosen to put her down, even though she will never be ridable. But I want to encourage everyone to learn more about this genetic disease and to do what you can to help. It is appalling to watch a young horse repeatedly fall down, run into fences, run over people and have trouble with tasks as simple as eating a treat from your hand. ...Also, it's an odd disease because it varies in severity and it not usually present at birth. My filly did not exhibit symptoms anyone noticed until she was a yearling, and this was also true of another filly I know of.

Justine (England): Some years ago I bred a filly foal with CA. The first thing I noticed, that when the mare was foaling, it was a very slow process although a normal birth. These small things I always take particular notice and make a note for future reference. The filly was born, all looked ok. Two hours later the foal was still on the floor with no attempt to move, I started to have suspicions this foal was not normal. We decided to help the foal up, from this time took 7 hours to find the milk (which can be normal). Suck reflex was normal. The following morning I noticed the foal had a very slight wobble of the head when focusing on something. I called the vet, I think he thought I was just being paranoid at this early stage; it was probably the fact that the foals eyesight was not yet perfect. I watched carefully over the next hours. Upon waking from a very deep sleep, the foal got up, not front end first but back end first! Out comes my veterinary book. It looked very much like CA. 3 vet opinions later, it was diagnosed. Dr. Knottenbelt at Liverpool University (GB) was very interested in this case. We made videos and experimented with small tests, for example - throwing a carrot into the paddock to see what the response would be. In the beginning the response was more or less normal, looks surprised, and investigate. As weeks went on, the front leg action was becoming very exaggerated and balance was becoming more and more difficult. We already knew the fate of the foal and decided as long as she was sucking and not in danger of hurting herself we would keep her alive for the mother's sake. At three months old she began to fall into fences, the carrot test was still a surprised look but she could not make her way to investigate, she wanted to but her legs would not go in the direction she wanted. This was sad; we named the day for her to go to sleep. Upon the request of Dr Knottenbelt we traveled the foal to the university in a trailer deep in straw so that she couldn't hurt herself; she stood all the way and loved her back scratching. She was not at all stressed about leaving her dam. Did she know? We can't tell. On arrival at

the university, she happily walked out of the trailer with my arms around her body guiding her direction. We put her in a recovery stable with cushioned floor and walls, where vets watched her eat hay, drink water and studied her reactions. Again she was under no stress; she was a happy little soul. A few hours later she was put to sleep. The post-mortem showed the cerebrum had not grown, the rest of the brain had. CA. Forgot to say, her growth rate was normal. Her name was `poppy`, bless her.

Vygoda: (United Arab Emirates) Once you have seen a CA foal, and its progress, you can never mistake it for anything else. Over many years but not recently, I have seen 5 foals with CA, the first one that I bred of all English lines that was diagnosed as a wobbler. This foal was perfect when born, and by 4 months old had a nodding head and leg dysfunction that became increasingly worse rapidly leading it to have no control and banging into things as described. The diagnosis was queried with the vets and on autopsy was found to have CH, now known as CA. The second one was of Russian lines and the colt evidenced it when it was 2 years old – not bred by me for what difference it makes – but nowhere as bad as the foal mentioned above. The other 3 that I have seen were abroad and of English or American or French lines or combinations, all foals. How do you tell an owner what you feel is wrong with the foal - that was very difficult to do as they had not noticed it.

www.straightegyptians.com Forum:
Nancy Bourque/Ibriz Arabians (Canada) We also had a cerebellar foal. It was when we were first raising Arabians. We bought a western pleasure champion who was very heavy in Raffles bloodlines and bred her to a Padron/ Fadur stallion. The foal was fine at birth but we noticed a week or so later on that she seemed disoriented and if you touched her she would startle and run into the wall...The symptoms started appearing at about two days of age and got progressively worse... Outside, she would just wander around the mare and act like she was unaware of what was going on around her. We had the vet for her, took her to a clinic etc and after checking out things they said it was cerebellar hypoplasia. My vet brought me a lot of literature to read on it. At three months we put the foal down. She had gone as far as she could. It seemed such a shame because she ate well and looked like a normal foal but it was obvious she couldn't continue on the way she was going. The mare had had about 8 foals and was 18 when she foaled this filly. She had never had another cerebellar foal. I understand from the reading that I did that the trait is recessive and has to be present in both sire and dam to produce an affected foal. One in four, approximately, will have the disease, two will be carriers and one will be disease free. After all of this I checked up on what the other daughters of this mare produced and heard some stories about foals born who died in a strange manner so I assume that some of the other daughters were carriers as well. The stallion owner is aware of what happened to the foal but she denies that her horse is a carrier. The vet told me not to stop breeding the mare but to use wisdom in choosing a stallion for her. Of course, not knowing who was a carrier was a problem. And, people don't always want to tell you if these foals have been produced... I rebred the mare to another stallion and have a daughter, now 13. She appears normal but I have never bred her. The mare had several other daughters before I got her and I have heard through the grapevine that one of them has also produced at least one of these foals. In the hope of finding a cure or preventative for this horrible disease I would be pleased to help in any way I can.

Cherie: I have a foal I believe is affected also. Like you she appeared normal at birth but after a couple of weeks I noticed she was a bit wobbly. I initially put this down to size (She was extremely tall). But as the weeks have progressed it is becoming more apparent. This mare is Egyptian/Spanish though and she has had 9 other perfectly normal healthy foals.It is so sad as the baby is the friendliest little soul.

HRH Alia Al Hussein (Jordan) I am happy to say we have had such a foal but understand that it is like SCID in that both parents have to carriers of the gene and the odds for a foal to inherit it fatally are the same as for SCID, 25%,if both parents ARE carriers.

B. ter Laan: (Germany) I remember a breeding program in NL, about 20 years ago, that was plagued by what we called "head-shaking" (schuddebollen). The owners thought they could trace it to a certain line on which they had based their program. They stopped with the line, had the stallions gelded and kept the mares (I think) to prevent further breeding and started a completely new program based on other lines. I call that very courageous and have great respect for that decision.

Emma Maxwell: (England & France) I haven't seen a suspect in over ten years although I have seen it four or five times in several of the major blood lines. All the foal cases I saw followed a distinctive pattern of behavior which I will try to describe. The very first time it became apparent was between 3 and 4 months and was always a response to a startle. I still watch very carefully when I disturb a sleeping foal of that age into leaping to its feet, because the affected foals just show a little tremor or wobble of the head as they achieve their standing balance. This becomes undoubtedly more obvious over the next couple of months and while the foal may move perfectly OK when it is in control, it shows an increasingly more marked head wag, developing into a stagger when it is surprised. Apart from waking them up, the other time to watch is when they are have just been put in the field and their mother gallops off and they have to decide to follow at top speed, when the same tremors and staggers appear. I have seen one suspected adult case (in Brazil 1986) in a large herd of horses which was very disturbing as the poor mare tried to gallop up an imaginary hill when her field mates ran away across the flat field. She ended up going over backwards and was completely ‘drunken’ in all her movements.

El Acrisi: (Switzerland) HERE NOW MY STORY: When it happened in 92 I had 4 foals they had been very healthy and very well done. All have been from a new stallion I used. From the mother side I used different stallions before, in first to 2nd generation and have never had a problem before. My foundation mare is 50% Spanish, 25% Sahmeth/Marbach and 25% Karmin (polish). The way my foundation mare was bred I used all this different bloodlines by different stallions to find out what kind of bloodlines will fit best in this female line. So I did with her daughters. As I mentioned never problems before using my foundation mare and 2 of her daughters and a granddaughter for these four l992 foals. One night, around midnight, full moon, I went to the pastern to check and to sit beside the horses - it was around June, July and very warm. The horses had been on a pasture day and night and drinking water out of pond where the water flow was not very high - just by a little river came fresh water in when it was raining. Later the farmers around told me they let never let drink their cows out of this pond - the water quality should not be a good one - I lived rather new in this area. All 4 foals (4 weeks up to 2 month) had been laying on they hay and what I felt was that they had been all 4 very

cold bodies. This was just a feeling but as a vet all my instincts had been activated. So I stood on the pasture for more than an hour and observed them. After this hour I felt I have to bring the foals to the stable because I felt something is wrong with them. They came in and I felt they are a kind of somnolent. They had been drinking milk but all 4 foals' temperature was around 35 degrees, so way much too high for a warm summer night. I was electrified [petrified]. The next day the same but all blood samples I checked had been normal, also liver enzymes. Also the one year old filly was a kind of somnolent but was eating and also here the blood was normal. I started to phone around the world because I had no idea about what kind of disease it could be (U Newmarket, U Davis, U Aukland and all horse vets and breeders I knew). As I lived in a kind of protected area, a lot of spectators walked by each nice weekend - so I never knew if maybe someone gave the horses something to eat. I called a professional for botanics to check the environment for dangerous plants, We found horsetail and treated the horses but nothing changed. After a couple of days the liver enzymes raised up but not extremely but significant enough. 2 of the foals and the yearling filly had been ok after a couple of days the oldest filly and the last born stood ok by blood but not by physics. The filly got this soldier type high legged walk and a little tremor, also the youngest which had more problems with the balance. I went to the University hospital in Zurich but they couldn't help and had also no idea. After that I called the University of Berne and they want me to bring my horses but how could I bring 4 mares and 4 foals because they also had no idea about the symptoms I explained. But technically it wasn't possible so one of their chief assistants came by to see the horses and he also had no idea about what it could be. So we decided that I bring my oldest and worst filly so they can check her at U Bern. I did. A week later they told me they cannot find something and for all my other horses and I should give my ok to put her down. I'm a vet too, but also a breeder, so I'm interested in what can be the source of this illness: genetic and then from where is it coming and how can we try to breed it out or can it have also other origins than genetics that causes in the end result the same disease. can it be a combination of some circumstances they come together. What we know as CA in foals there is [in human medicine] known as sub acute cerebellar degeneration with Purkinje cell loss (exactly the same histopathology like our CA!) Autoimmune antibodies are doing an autoimmune reaction against the 34-KD-Proteins of Purkinje cells which they kill and it comes to the Cerebellar Abiotrophy.
In human medicine studies they had in some cases the possibility to make visible the auto reactive Immunoglobulin on the surface of the Purkinje cells.
Often it is also in connection with tumor activity somewhere. Isn't this an interesting new link to think about?

Szedlisa: (Spain & USA) In my own experience having bred a mare which showed obvious neurological symptoms as an 18 month old filly, it was diagnosed at that time (late 70s) as either a grass sickness or a vitamin E deficiency. And as the filly had been one of the first imports from Spain, who knows what she could have been exposed to, or what might have happened during travel. Spain's borders had been closed for so long that really little was known about their horses and even less about how they were raised. Leased out, then sold to the lessee the filly grew up and was used as a broodmare. She produced two extremely athletic high quality foals which went onwards to win Regional and National honors under saddle in the USA. She further produced several very good daughters some of which were used for breeding. Absolutely no symptoms present. Then BINGO, one severely affected foal which was put down within weeks of birth. By this time Cerebellar Hypoplasia had been differentiated from Cerebellar Abiotrophy

and the vets were called in again. With the suggestion of Michael Bowling to consider CA, the vets and the then owners considered this new approach. The original imported filly/mare was to be considered a CA mare and the new born foal an affected foal. Still CA had not been proven as an autosomal recessive. Not all but a few of the mare's daughters were used for breeding both purebreds and half-breds, producing stunning foals in both divisions with equally impressive show records. We all sighed with relief. One of these producing daughters, years later at the age of 13, showed symptoms including the head intention tremors. She was promptly euthanized. A few years later, a son of one of the daughters, sired a CA affected foal out of a Spanish / Crabbet cross mare. His only affected foal out of several sired but he was gelded. Was this turning out to be a recessive unexpressed gene? Information on CA was extremely limited. Yes there had been studies done and the results published. Nonetheless the every day vet either had forgotten the brief description during vet school or simply was not considering CA. By this time Ann Bowling had well proven that this condition was a genetic autosomal mode of inheritance with her test herd of CA arabians at UC Davis.[1985] Her studies, unfortunately were not published prior to her sudden unexpected death.; however an autosomal recessive was highly suspected and a sex-linked (X) mode of inheritance was ruled out. It was about this time that one of the daughters of the original mare, produced a foal herself with CA symptoms, unfortunately in an area where the vets did not consider CA as a possibility and treated it for everything under the sun at great expense. To no avail. This is when I was contacted as the breeder of the original mare and actually when I became aware of the existence of cerebellar abiotrophy...a short 7 years ago. Apparently the foal showed a distinct lack of balance, and fell over backwards frequently. At first its owner thought it was just a question of getting its very long legs properly organized. The foal was nursing well; started to tuck into its grain with no problem; ran and played with the other foals; all in all behaving just like any other foal . It was big and did not seem to have a very good "stop" on itself, as it often did a one point landing into fences and any other solid object. Big eyed, gorgeous to look at, and really tall, it started to have these little accidents more and more frequently around 4 months of age. Then the head intention tremors started...at first with only an occasional slight shaking which got more pronounced as it got older. The owner went through a heart wrenching time as the foal worsened and finally the decision was made to euthanize it. Those little accidents had become serious and involved multiple stitching, antibiotics and 7/24 care. The whole procedure was heart breaking. Anyone who has experienced a CA foal will 1) recognize the symptoms in the future and 2) will never want to go through it again. The foal described above was just starting its hypermetric mode of action...an exaggerated way of walking and running. It was as if it were galloping up a hill (only on a flat area) and then reach a point where it would fall over to the side or backwards. At a walk when under stress, each step would get higher and higher as if it were climbing up stairs. Again on a flat piece of ground. The leg shoots out from the elbow very similar to the goose-step marching stance of some military forces. In fact the CA foals walk like a goose, a rather stiff-legged gait with the heel of the hoof slamming into the ground first as it comes down. Lets get some more descriptions posted, those samples sent and enough information into the hands of the researchers so that a test can be developed and none of us have to go through this with our horses. I know I never want to again!

From A White Horse Forum:
Trailsend Arabians: (USA) I have a 12 month old filly that started showing signs of a wobbler in

the last month. I had put her into halter training in March, and the trainer noticed a few things mid-May. I'm a veterinarian, and we had another vet come out, and we both concurred that she looked like a wobbler, so we did some neck films. We thought we could see something at C5-6, which was consistent with the clinical signs at that time, so we thought we had the diagnosis in the can. Just to make sure I was giving her every chance, as some wobblers will improve with dietary changes or need surgery, I made an appointment for her to see one of my favorite instructors at my alma mater for evaluation and discussion on when we might need to consider surgery. My trainer was keeping her for me meanwhile, as it was a couple weeks before I could get her in. I picked her up from the trainer's two mornings ago, and the trainer mentioned she was seeing her head shake. My heart dropped to my shoes when I saw my filly, as that indicates the lesion is much higher than we had thought. I thought and made mental lists of diseases through the entire 350 mile trip in the pouring rain. It was a very short list. Upon arrival, my instructor looked, we chatted, and he went to find another instructor (whom I also knew), a resident, and a neurologist, who is one of the best in the field and whom I also knew. We all -- that makes FIVE veterinarians, three of whom are boarded specialists in three different areas -- are 99% sure she has CA. There is no ante mortem diagnosis available. I am getting into contact with UCD; I am on the east coast and have to wait until they wake up. An EPM test on spinal fluid is pending -- that is our last hope that she has a weird presentation of EPM -- but the spinal fluid is normal otherwise. It could be acquired and not genetic, but I'm hoping UCD can help me find out more there. It's also a little unusual in that clinical signs did not start until she was 11+ months old, as usually they appear around 6 months, but they may know more on that too. My girl is home for good now. She can get around and doesn't fall, but she trips if the ground is uneven. Her intention tremors are mostly noticeable when she is focusing on something. We have no idea if her clinical signs will stabilize, if she will continue to deteriorate at this rate, or what. I have no idea of how long I have with her, but every day is a gift. I know eventually I will have to put this filly down. What I am looking for is information on the day-to-day dealings with a CA horse; my glib advice to owners is now ringing hollow. I'm also flat-out scared that this is going to destroy my fledgling breeding program, and that of my filly's sire's owner, if it does turn out to be a genetic form and not acquired. This filly's pedigree reads like a National Champion's Who's Who with no mention of one horse I had previously heard of being a possible problem. I thought I was safe.

From A Black Horse Forum:
Lovemypinto: (USA) About 5 years ago, a gal at the barn had a mare that foaled a filly. These same symptoms appeared just about the time of the weaning. The head had a nodding like shake....that is the only way I can describe it. The filly was diagnosed by a local vet, he put the filly thru some tests, one as I remember that was explained to me they blindfolded the filly and she couldn't maintain her balance. Another test was turning her (I believe still blindfolded) and the filly couldn't stay standing up. I wasn't there...this part was told to me and it was 5 years ago this happened. The vet felt strongly that it may be CA...Filly was taken to Oregon State Vet school. They confirmed the diagnosis, filly put down. A post was done...all the tests done...confirmed. It was a very sad affair, owner devastated needless to say. The worst part about it was this was a really nice filly, seriously nice filly.

Joni Hyrick: (USA) This has opened up heartbreak for me almost 1/2 of my lifetime ago. My first Arabian horse was purchased as a weany. When she was 5, I bred her to a local western NY

stallion (both pedigrees attached). I was so very "horse poor" at that age, and put everything into a $300.00 stud fee, all the best feed and care, and when her foal was born on Mother's day in 1984, we waited anxiously for the foal to get up. He couldn't. He'd try, but all he could do was spin himself around in circles. I took emergency time off from work and slept at the barn. I got up every 2 hours, milked the mare, and fed the foal. I paid for several different vet opinions, and no one knew what the problem was. We could pick him up and sometimes he could balance. He would always need to have his head down for balance, and his front legs would lift up in a very strange way. We brought the mare and foal to Cornell University where the head veterinarian at that time felt the foal had Cerebellar Abiotrophy. He asked permission to call all the vet students to observe the movements of the foal as we held him and let him try and walk. He'd occasionally flip over backwards if he got going too fast. I had no choice but to euthanize the foal. They told me that as sad as it was, could they perform an autopsy and perhaps use some autopsy photos in future textbooks if the diagnosis was confirmed. The letter from Dr. de Lahunta is attached as well as a photo of "the little gipper", and the related pedigrees. Perhaps you can start a database reference which can assist someone else in the future. We kept the mare and foal in the arena to keep the foal from injuring himself in the stall. He was only a few weeks old when I lost him. Additionally, after I received the letter, I forwarded it to Jack Bridgman (the owner of the stallion). He refused to believe his stallion had anything to do with the foal's death (though I learned later they lost another foal under similar circumstances, but never had an autopsy done). He refused to refund me the breeding money, but said I could have a re-breeding! I couldn't believe anyone would be that way. I had extensive veterinary bills, AND now I couldn't even get the stud fee back. I hope this information helps someone else.
Joni (Sire:BelNegor - Dam:Velzaddad - Foal:TheLittleGipper)

Donna B (USA): I have a friend whose stallion produced two CA foals. As soon as they figured out what the problem was they gelded the stallion and have turned him into a wonderful performance gelding. I am sure they would be happy to get hair samples from him and the mares and send them in.

MM Arabians (USA) As a breeder of ONE CA foal, I would agree with staying focused with both a & b. It is a heartbreaking disease. Been there, experienced that.
a) Become more aware of the causes and symptoms of CA so we can all help spot animals who might be affected and help them be accurately diagnosed.
b) Help support efforts to increase funding for DNA research so that we get a test for this thing ASAP.]
HOWEVER, I have bred 100+ foals, using the same bloodlines and have NEVER produced another foal with the same problem. And, with the affected foal, it was a complete outcross stallion, from the mare lines. But, I only look at 8 generations back. The only solution is to develop a CARRIER test. My one experience was devastating to me, with 10 broodmares. The other two posters, with 1 broodmare, have stopped them in their tracks! Thank heavens I am not in their shoes. I am concerned that Resolutions proposed, will BURY this lethal disease. A very large % of the affected foals may be hidden. Develop the carrier test, educate and take away the fear, like we did with SCID.