

n a pleasant day in 2004, I met a new client at a dilapidated old barn just north of I-90 in Gilberts. Kelli was a bright young lady who, along with her mother, was there to purchase a 4 year old Morgan named Be-Bop. He wasn't tall but he was muscular, well put together and had a wonderful temperament. He was quite a contrast to the old farmer that owned him and the falling down barn, but he passed his prepurchase exam and Kelli began a relationship with him that lasted a quarter of a century.

Fast forward 20 years and Kelli has married, has growing kids and a host of other interests, but Be-Bop has maintained a central place in their family. We have been monitoring him carefully because Morgans have a naturally increased risk of laminitis which increases with age. Laminitis or Founder is the result of 3 main inciting causes. It can be structural stress, such as

a horse standing on one front foot primarily because of a severe injury to the opposite foot. More often it is the result of an acute onset caused by grain overload, colitis, uterine infection/retained after birth, or other types of acute metabolic stress. And finally, what we see most in our practice is a chronic laminitis caused by metabolic changes in glucose metabolism as the result of insulin resistance (usually younger, overweight horses) or equine Cushing's/PPID Disease (older horses) that have a pituitary adenoma. In rare cases, administered cortisone can cause the same problem. Be-Bop had complications from the first and last forms.

It is worth a brief detour here to discuss the acute metabolic form since the understanding of it has evolved over the last 20 years. Anatomically a horse supports 90 percent of his weight through the attachment of the external hoof wall to the internal bone, the coffin bone, in the foot. Microscopically these interlocking fingers are called laminae and inflammation of these laminae is called laminitis. We used to believe that laminitis was caused by abnormalities in the blood flow to the foot. Dr. Pollitt from Australia proved that it is the enzymes MMP's that cause the damage. Normally these enzymes are activated in small sections allowing the hoof wall to shimmy or slide down the foot as it grows out. If these enzymes are activated too broadly then the interlocking laminae separate and the wall pulls away from the coffin bone. This separation usually occurs in the front feet at the toes. It is rather like pulling your fingernails halfway off and then making you bear a thousand pounds on them. Understanding this is important because forcing a horse to walk or giving them medication to increase circulation at this stage

actually makes the damage worse and causes unnecessary pain. We approach this acute form as a genuine emergency to be treated as quickly as possible medically and with complete rest in a heavily bedded stall.

Getting back to Be-Bop, we were regularly checking his lab results in 2014 because of concerns about equine Cushing's Disease. He was on Prascend (Pergolide 1mg) because it is the only pharmaceutical known to be consistently effective in treating the pituitary gland abnormality responsible for the disease. Other drugs and supplements can also be of some benefit when used along with diet, exercise and proper hoof care. The approach seemed to be controlling his issues and radiographs taken in August of his front feet showed no rotation/ founder on his left fore and just a

small dished appearance to his right fore.

Over the course of the next year he became more stiff on all four legs. His Prascend dose was increased from 1mg to 2mg and eventually to 3mg. Radiographs in September of 2015 indicated that he had now rotated about 12 degrees in both from feet. At this point, we felt like we had stabilized his Cushing's with the 3mg dose and, in fact, for the rest of his life his lab values and pituitary laminitis were well controlled. This was vital because no amount of work on his feet would help Be-Bop if the underlying cause had not been addressed. Plans were made to return with Be-Bop's farrier, Rob Grevengoed, to put reverse shoes and "hoof cinches" on him. Once the hoof wall at the toe has become separated in these cases then the toe works like a wedge to

continue to pull the wall away from the coffin bone as the foot grows out. Trimming and shoeing in the chronic stage should be designed to move loading back to the heels and unload the toes. The wall is st trying to grow out together and parallel to the coffin bone at the to of the foot. Chuck Potter, a farrier from Minnesota has developed an elegantly simple system to try to keep that relationship intact. It's called the Hoof Cinch and I have used his device to de-rotate a serie of cases of founder. This system can be used to treat cases at any stage from acute to very long term and mild to quite serious. It can be applied with or without shoes and is painless and simple to put on. It usually reduces the pain leve helps, maintain proper circulation and it improves sole growth which protects the rotated tip of the coffi bone. Six weeks after his shoeing change and hoof cinch application our patient was walking much more comfortably. However x-ray revealed a new problem.

Unfortunately for Be-Bop, the separation allowed for a white line infection to develop in his right front foot. This caused further damage to the laminar bonds in the hoof wall and his founder became worse. This is the type of condition that causes the structural weaknes that can result in rotation. It can occur with no other complicating factors but of course Be-Bop already had his original issues. The organisms that cause white line disease can't tolerate oxygen so the best treatment is to open up the affected hoof wall to let air and medication get to it. Since it is no longer attached to the underlying

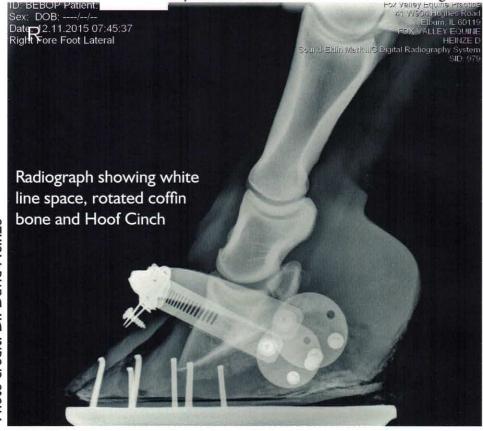
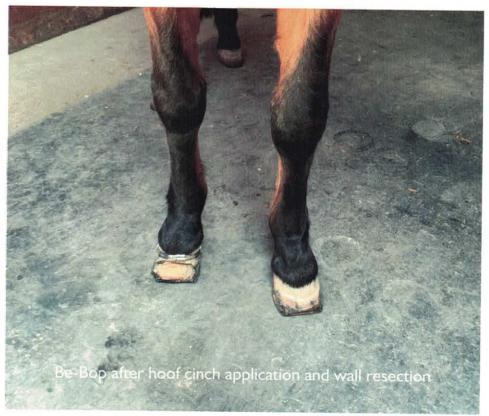


Photo credit: Dr. David Heinze



bone it isn't supporting any weight. Rob and I worked at resecting the wall and Kelli treated it diligently. At this point, we were not trying to get him back to work, we were just trying to save his life. After colic, laminitis is the second greatest cause of death and disability in horses. Besides removing the diseased hoof wall the principals for treatment are very similar for white line disease and founder, so we persisted in our approach.

Follow up x-rays in
December showed that the hoof
wall deviation was much better
on his new growth above the hoof
cinches and Be-Bop was moving
better still by February of 2016.
We had a set-back in June when
his white line infection on his right
front became worse and he was
much less comfortable. Aggressive
resection of the diseased wall and a
new Hoof Cinch had him walking
sound in 2 weeks. By August, he

was walking quite well and willing

to trot and canter.
His hoof wall had
grown down with no
new infection and
we returned him to
a normal exercise
program.

Be-Bop required monitoring of his lab values but they continued to be in a wellcontrolled range. He also maintained a tendency to develop minor white line infections but they only involved smaller areas and didn't affect the integrity of his foot. By February of 2017, the rotation in his left front was back to zero

degrees and his right front had gone from 27 degrees in December of 2015 to 9 degrees. He was comfortable and willing to work under saddle. Follow up films a year later were similar and stable. Be-Bop remained a wonderfully kind and cooperative patient through this entire experience. He is an example of both the patience and commitment that someone like Kelli has to have to persevere through a severe founder case. He is also an example of how much a situation like his can improve over time with that perseverance.

We lost Be-Bop this past summer to an episode of colic. It was sad to lose an old friend but I was glad we gave him an extra four years. It was a gift to know him and Kelli and to see the bond that they shared.

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