



The Equine Heart

by Bill Thomas, DVM

A horse gallops with his lungs, preveres with his heart, and wins with his character. —Federico Tesio (1869–1954)

A horse's heart must supply blood to all parts of one of the largest domesticated land mammals (1,000 pounds or more), not only at rest and during routine activities, but also during periods of extreme physical stress such as that encountered during strenuous training or racing. That it is able to do so attests to the system's remarkable efficiency and adaptability.

The cardiovascular system of a horse consists of a pump (the heart), a distribution system (arteries), exchange areas (capillary beds), and a collection and return system (veins). The heart of a 1,000-pound adult horse is about the size of a large melon and weighs about 10 pounds. As in all mammals, it consists of the left and right atrial receiving chambers, left and right atrioventricular inflow valves, left and right pumping ventricles, and aortic and pulmonic semilunar outflow valves, plus attached inflow veins and outflow arteries (see photos on page 3). The left heart collects blood returning from the lungs via the pulmonary veins and pumps it to the body

via the aorta. The right heart collects blood returning from the body via the large veins and directs it to the lungs via the pulmonary artery. The output of each ventricle in an adult horse at rest is about 25 to 40 liters/minute, compared with 4 to 5 liters/minute in an average adult human (1 liter ≈ 1.05 quart).

The cardiac cycle consists of a contraction/ejection phase (systole) and a relaxation/filling phase (diastole). The output of the heart is the product of the heart rate times the output of each individual systole (stroke volume). The stroke volume in turn is influenced by the structural integrity of the various anatomic components of the heart (valves, septa between chambers, and so forth), the strength of muscular contraction of the ventricles, the resistance to flow faced by the ventricles during systolic ejection, and the ability of the ventricles to adequately fill during diastolic relaxation. Structural or functional abnormalities of any of



these factors can adversely affect the overall functioning of the system, resulting in an inability to produce adequate cardiac output either during periods of increased demand (exercise) or, in severe cases, even at rest.

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DIRECTOR'S MESSAGE

Matters of the Heart



Dr. Gregory L. Ferraro

For as long as horses have been bred, raised and used by humans, their owners and caretakers have praised their "heart". While this reputation for quality or quantity of "heart" is obviously based on a horse's psychological rather than anatomical characteristics, it is also prophetic of the medical truth.

The "heart" spoken of in horsemen's terms reflects the attributes of stoicism, bravery, dedication and nobility of character. In fact, these attributes of character also reflect the typically robust character of the horse's anatomical heart and cardiovascular system. For, while horses are frequently called upon to push their anatomical hearts to the limit in athletic competition, the occurrence of heart failure or disease is strikingly rare.

Heart attacks rarely occur in horses. Degenerative heart

disease, vascular infarct, and cardiac myopathies—so common in the human world—almost never occur in horses and when they do they are generally secondary to some easily preventable cause, such as damage from migrating parasites. There are, however, certain anomalies and diseases of the heart that are seen in horses and are important for horse owners to understand. For that reason, we have dedicated this issue of our *Horse Report* to describing some conditions most often encountered by equine cardiologists.

We will describe some common congenital defects, some not too uncommon causes of heart problems, and the diagnostic methods that are currently in use. While not every disease or issue is covered, we hope to provide you with a general understanding of the horse's heart and of some of the problems your horses might experience. During the course of your reading, you may want to contemplate a somewhat larger truth that is also reflected in these pages.

While humans and their beloved dogs and cats often experience the maladies of heart disease and cardiac failure, the horse remains one of the few creatures with generally excellent

cardiac health. Since most of our heart disease is secondary to such changes as hardening of the arteries and subsequent vascular degeneration, perhaps our horse's proclivity for the spartan life holds some lessons for us. Horses exercise regularly. They tend toward the vegetarian lifestyle. They rarely imbibe in alcohol, and they are loathe to smoke! Wisdom resides not only in those with the voice to pronounce it, but also in those with the character to live it. ✱

Lost in the Fog 2/4/02 – 9/17/06



Charismatic champion
with the crooked blaze.

Farewell to a noble horse.

The Equine Heart

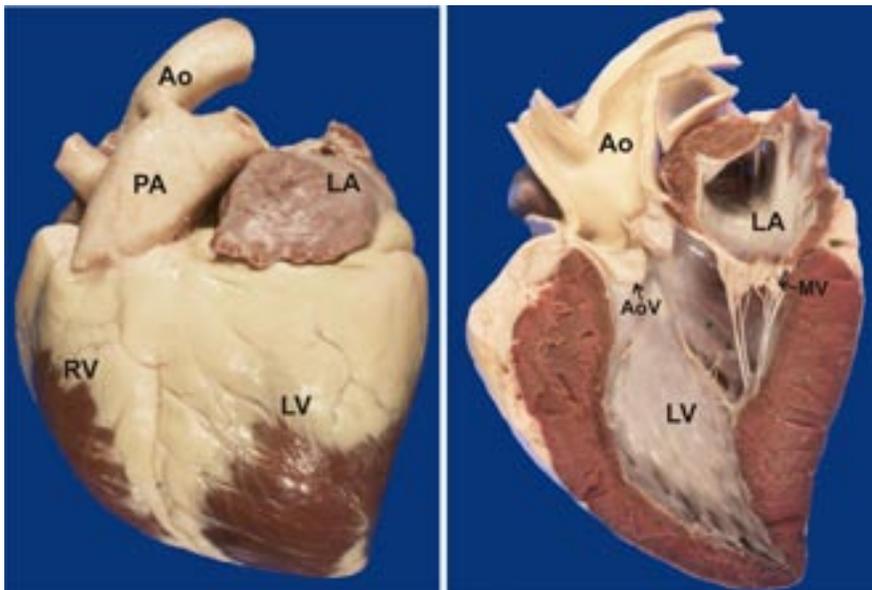
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Cardiac Disorders

The principle observable signs of heart problems in horses include:

- loss of condition
- increased fatigue during exertion
- shortness of breath
- increased rate or effort of breathing
- weakness occasionally resulting in collapse or fainting
- signs of fluid accumulation in the abdomen or beneath the skin of the lower thorax

Depending on the severity of the problem, these signs may initially appear only when the horse is subjected to moderate or strenuous exercise. With a severe cardiac disorder, these signs may appear during normal, nonstrenuous activities or even at rest.



Left: Photo shows the outside of a horse heart. **Right:** Photo shows the inside of the left heart. Ao = aorta; PA = pulmonary artery; LA = left atrium; LV = left ventricle; RV = right ventricle; MV = mitral valve; AoV = aortic valve.

A physical examination in a horse with cardiac disease will usually reveal an abnormality in the audible heart sounds or in the heart rate and rhythm. When an abnormality is suspected, further evaluations by electrocardiogram and echocardiogram (ultrasound imaging) may be performed to more precisely identify the nature and severity of the abnormality. Chest radiographs may also be used to help determine heart size and abnormalities in the lungs and chest cavity, but these are often difficult to obtain with conventional equipment in adult horses because of their large body size.

Fortunately for the species, cardiovascular disease accounts for a relatively small percentage of all illness and death in horses, compared with humans, dogs and cats. However, because most horses are not kept as companion pets but are expected to perform work or athletic feats with a rider, the consequence of cardiovascular disease in an individual horse may be greater than for dogs and cats, which are not routinely expected to “earn their keep.” In addition to the potential effect of a cardiac problem on performance, the safety of the rider must also be considered in any discussion of the prognosis and future use of the horse.

Heart Murmurs

Initial evaluation of a horse's chest and heart involves examination with a stethoscope. One of the signs of an abnormality is the detection of a heart murmur during this examination. What is a heart murmur? It is the sound of turbulent blood flow, usually caused by an abrupt increase in flow velocity. When blood moves smoothly through the heart and blood vessels, very little sound is produced (like water flowing smoothly through a hose). Most heart murmurs are caused by blood flow that becomes turbulent because of increased velocity due to a leak or obstruction in one of the heart valves or because of abnormal communication between different parts of the heart (like the increased velocity and spraying sound you get when you put your thumb across the end of the hose). However, there are

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some soft, short, variable heart murmurs that may be heard with no other detectable evidence of heart disease. Such murmurs are referred to as normal or “innocent”. If there is uncertainty about the origin or significance of any heart murmur, further evaluation is usually performed by echocardiography, which provides detailed images of the inside of the heart and can detect abnormal blood flow patterns.

Congenital Heart Defects

Congenital heart defects are abnormalities that are present at birth. They occur much more commonly in humans and dogs than in horses. These abnormalities are often discovered within the first few weeks to months of life when a heart murmur is heard during stethoscopic examination of the chest and heart.

A wide variety of simple or complex congenital heart defects may occur, but only a few have been recognized often enough to be reported in more than a few individual horses. The most accurate technique for identifying specific defects and evaluating their severity is two-dimensional echocardiography (ultrasound imaging) supplemented by Doppler echocardiography (imaging of blood flow within the heart and associated blood vessels).

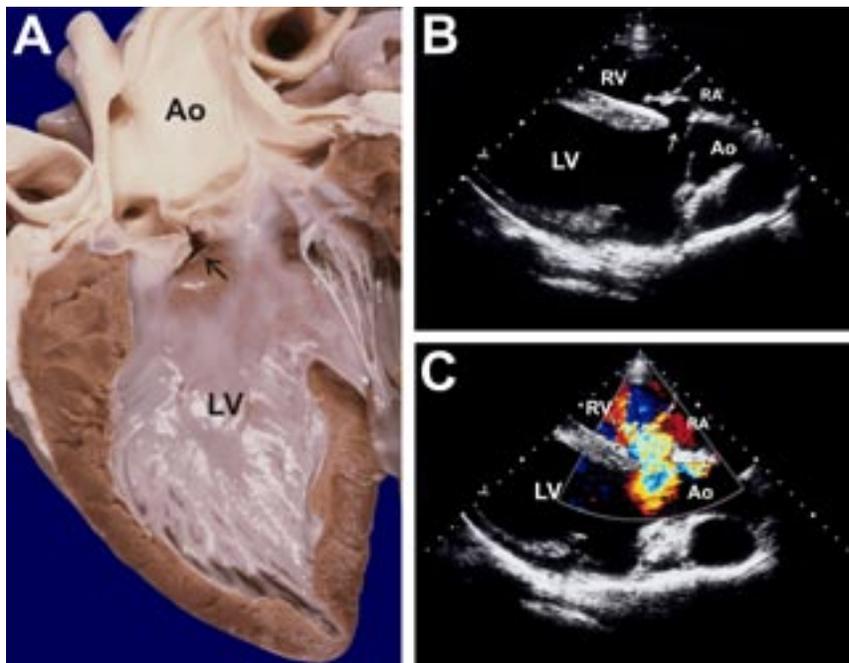
The most commonly reported congenital heart defect in horses is ventricular septal defect, described below. Another defect, patent ductus arteriosus, which is common in humans and dogs, is relatively rare in horses beyond 1 to 2 weeks of age.

The ductus arteriosus—a large blood vessel—closes more slowly after birth in horses than in humans and dogs, so that a soft, continuous murmur can be heard in newborn foals up to about a week of age.

Ventricular Septal Defect (VSD). Ventricular septal defect consists of a hole in the muscular wall between the two ventricles and is the most commonly recognized congenital heart defect in horses. It also occurs as one part of more complex defects. In simple cases, the hole results in the passage of oxygen-rich blood from the higher pressure left ventricle to the lower pressure right ventricle and pulmonary artery, primarily during ventricular systole. Because some of this blood bypasses the lungs, it is not fully oxygenated. A systolic heart murmur is usually heard on the

right side of the chest over the cranial part of the heart.

Depending on the size of the hole and the amount of blood passing through it, the pulmonary arteries and veins and the left atrium and ventricle are subjected to an increased workload because of this extra volume of blood. If the hole and the resulting shunt are small, the adverse effect on cardiac function may be minimal, and the horse may be fully capable of engaging safely in moderate physical activities without evidence of fatigue or shortness of breath. If the hole is larger and the shunt is greater, there may be signs of cardiac insufficiency with minimal exertion, and the horse may be very limited in its athletic ability. The nature of the defect can usually be confirmed using two-dimensional and Doppler echocardiography.



Ventricular septal defect (VSD). (A) shows a small VSD in the upper ventricular septum just below the aortic valve (arrow). (B) shows an echocardiogram with the VSD opening between the left ventricle and aorta (arrow). (C) shows same view but with multicolored, disorganized turbulent flow through the VSD into the right ventricle during ventricular systole. RA = right atrium, RV = right ventricle, LV = left ventricle, Ao = aorta.

Patent Ductus Arteriosus (PDA). The ductus arteriosus is a large blood vessel connecting the fetal pulmonary artery to the descending aorta, allowing blood from the right ventricle to bypass the nonfunctioning lungs and be directed toward the abdomen and placenta. In all mammals, the ductus constricts at or shortly after birth, eliminating this fetal connection and allowing for the normal development of the blood vessels in the lungs. Unlike most other domestic animal species, persistent slight opening of the ductus arteriosus is quite common in newborn foals. Because the pressure in the aorta is higher than that in the pulmonary artery throughout the cardiac cycle, blood flows through the ductus from the aorta to the pulmonary artery, and a “continuous” murmur can be heard over the pulmonary artery on the left side of the chest.

Closure of the ductus usually occurs within the first week of life, and the murmur disappears. If the ductus remains open beyond the first week, it is called a **persistent** or **patent ductus arteriosus**. The resulting shunt may cause blood volume overload in the pulmonary arteries and veins and the left atrium and ventricle. Although slight patency (an open state) in the first week is very common in foals, patency beyond the first week is rare.

Complex Defects. Congenital heart defects are uncommon in horses, but when they occur, multiple or complex defects appear to be more common than in other species such as dogs and cats. These may occur as combinations of embryologically unrelated defects, or as

recognized combinations such as **tetralogy of Fallot** (consisting of a ventricular septal defect, pulmonic stenosis, rightward malpositioning of the origin of the aorta, and right ventricular hypertrophy/thickening) or **truncus arteriosus** (consisting of a ventricular septal defect and a single large arterial trunk exiting both ventricles). In the most severe cases, there may be shunting of oxygen-poor, darker venous blood from the right heart chambers to the left heart, bypassing the lungs and causing cyanosis (a bluish color to the membranes of the mouth and eyes) at rest or during exertion. These defects can be diagnosed accurately only by X-ray angiography or, more recently, two-dimensional and Doppler echocardiography.

Heart surgery is rarely performed in horses. Congenital heart defects can now be accurately diagnosed using sophisticated ultrasound imaging, but treatment options are very limited for the types of defects horses tend to get.

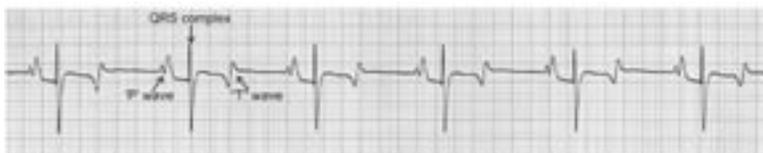
Acquired Heart Disease

Generally speaking, acquired heart disease is relatively uncommon in horses, although it is encountered slightly more frequently than congenital heart defects. It occurs most often in horses older than 5 years and only occasionally in younger horses. Degenerative changes affecting the heart valves, myocardium and lungs are associated with aging, increasing in frequency with age. The most commonly diagnosed conditions are heart rhythm irregularities and leaks in one or more heart valves. The most common signs associated with heart disease include a reduction in exercise capacity (exertional fatigue), shortness of breath especially following exertion, or the detection of a heart murmur, irregular heart beat, or other audible abnormality in a horse without other signs of illness.

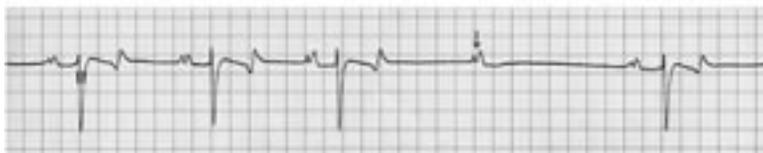
Identification of the electrical rhythm of the heart requires recording of an **electrocardiogram**, shown below. The normal resting heart rhythm of horses is usually slow (35 to 50 beats per minute) and regular (called sinus rhythm). Many horses also have short pauses in their resting heart rhythm caused by “dropped beats” (called second degree AV block). These are considered to be normal if they disappear during exercise.

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Normal sinus rhythm



Second degree AV block



Atrial fibrillation



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Atrial Fibrillation. Atrial fibrillation is an electrical disorder of the heart rhythm—also known as an *arrhythmia*. There are different kinds of arrhythmias, but the most commonly recognized one associated with diminished athletic performance or more serious signs of cardiac insufficiency is atrial fibrillation. With this arrhythmia, the normally regular, organized atrial electrical waves become irregular, disorganized and chaotic, and the atria fail to contract normally. This results in a very unpredictable, irregular heartbeat. Although atrial fibrillation often develops in horses with advanced structural heart disease and atrial dilation, horses most often develop this arrhythmia with minimal or no detectable additional signs of heart disease. In such cases, signs of cardiac insufficiency are usually not recognized at rest or with mild to moderate exertion, but become apparent at more strenuous levels of exercise.

Accurate diagnosis of arrhythmias requires evaluation of an electrocardiogram, where the lack of normal atrial waves and the very irregular ventricular waves can be readily identified. Further evaluation of the structure and mechanical function of the heart by echocardiography is also recommended, because the prognosis for treatment, recovery and return to previous activity levels is directly related to the presence or absence of underlying mechanical cardiac dysfunction.

If there is little or no evidence of underlying cardiac

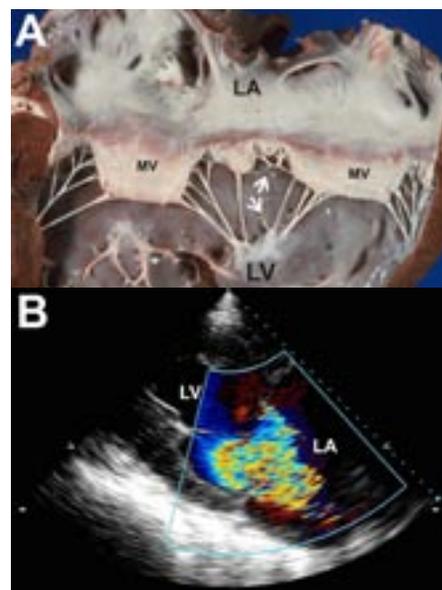
dysfunction, administration of oral or injectable drugs, especially quinidine, is often successful at converting the arrhythmia to a normal rhythm. Most of these horses are able to return to their previous levels of activity and performance, although some experience one or more recurrences of the arrhythmia, necessitating retreatment or retirement from strenuous activity.

If serious cardiac disease with atrial dilation are present, the prognosis for functional recovery is poor and conversion of the arrhythmia is usually unsuccessful or temporary. Treatment of the signs of cardiac insufficiency with drugs such as digitalis and diuretics may be considered in selected cases where little physical activity is expected.

Valvular Heart Disease.

The most commonly recognized acquired structural heart disorders in horses are degenerative valvular deformities. The process causes thickening and deformity of valve leaflets. These defects result in incompetence and insufficiency of one or more heart valves, associated heart murmurs, and dilation of the chambers that must handle the extra regurgitated blood on either side of the incompetent valve. If the valve leak is severe enough, the pressure in the veins leading to the affected side of the heart increases to the point where fluid accumulation (edema) occurs.

Valvular disease is initially diagnosed by the detection of a heart murmur during a physical examination. It is very important, however, to understand that soft, "innocent" murmurs are



Top photo (A) - Photomicrograph showing the pathology of acquired mitral valve disease. Valve leaflets are mildly thickened and there has been rupture of a chorda tendinea (arrows).

Bottom photo (B) - Ultrasound image showing multicolored, disorganized turbulent flow through the mitral valve into the left atrium during ventricular systole. LA = left atrium, LV = left ventricle, MV = mitral valve.

often heard in normal foals and adult horses. In order to advise an owner or rider about the significance of any heart murmur, it is critical to distinguish between a normal murmur and a pathologic murmur of valvular regurgitation, and to assess the severity of any suspected valve leaks. Two-dimensional and Doppler echocardiography are the most accurate and least invasive methods to help make such determinations.

In general, mild to moderate valvular insufficiency in a horse without reported signs of illness is compatible with continued use for mild to moderate physical activity. More severe valvular disease, especially when it is accompanied by obvious signs of cardiac insufficiency, atrial fibrillation, or severe enlargement

of the heart, is cause for a poor prognosis and a strong recommendation against any riding or forced physical activity.

Myocardial Disease. Although autopsy studies have shown that small areas of muscle death and fibrous scarring are commonly found in the atrial and ventricular muscle of horses, most of these lesions are considered to be incidental findings. Such lesions appear unlikely to affect the overall function of the heart muscle, but they may contribute to the tendency for some horses to develop abnormal electrical activity (arrhythmias). Myocarditis is occasionally suspected in a horse that develops an arrhythmia or other electrical disorder following an infectious disease such as strangles, influenza or an internal abscess. Toxic damage to the heart muscle may also rarely occur as a result of severe dietary deficiency of vitamin E and selenium, or as a result of ingesting the chemical monensin (usually from cattle feed).

Vascular Disease. Horses are known to develop several types of disorders that affect primarily their blood vessels. However, atherosclerosis—vascular disease associated with high blood pressure, high cholesterol and fats in humans—is exceptionally rare in domestic animals, including horses. Therefore, the consequences of this condition in humans, including heart attack, stroke and other peripheral arterial disease, are also rare in horses.

The only common condition affecting the veins of horses is **thrombophlebitis** of the jugular vein(s) caused by

repeated jugular vein puncture, injection of material outside the vein, or use of a jugular vein catheter. The resulting chemical or physical irritation or infection in or around the vein causes inflammation, swelling and tenderness, followed by formation of a firm clot in a small portion or long segment of the vein. Treatment involves removing the cause and applying symptomatic treatment for discomfort and any associated infection.

Several conditions may affect the systemic arteries in horses. The most common condition is parasitic arteritis due to the vascular migration of the larval forms of the intestinal parasite *Strongylus vulgaris*. The resulting dilation and thrombosis (and potential obstruction) usually occur at the origin of the large arteries to the intestines, although other arteries may be affected. Fortunately, this condition can usually be treated or prevented by an appropriate antiparasitic drug treatment program.

The other most commonly recognized arterial disorder is called **aorto-iliac thrombosis**. In this condition, a clot develops at the point where the abdominal aorta branches toward the hind legs. The resulting restriction of blood flow to the hind limbs can cause signs of lameness, stiffness, weakness and abnormal gait that develops during exercise and usually disappears at rest. Unfortunately, this condition is often progressive and rarely reversible, markedly limiting the athletic uses of an affected horse. Finally, degenerative changes in the wall of large arteries may weaken a vessel and predispose to rupture and bleeding. The most commonly reported sites of

such rupture are the root of the aorta in stallions and the uterine artery in mares.

Summary

The causes of heart disease in horses are often multiple and difficult to determine in individual cases. There is increasing evidence that genetic background may play a major role in a horse's susceptibility to developing disease. Some heart muscle disorders have clearly been shown to have a major genetic component, and familial or breed tendencies in some conditions strongly suggest that genetics play a role in these conditions. Unlike in humans, diet and exercise have not been shown to be factors in heart disease in horses, since horses almost never develop atherosclerotic vascular disease leading to stroke or heart attack. Almost all heart diseases except the congenital defects tend to increase with age in horses, just as they do in humans, dogs and cats.

There are no known preventive strategies to reduce the likelihood of heart disease in horses. However, owners of all animals are counseled to avoid in-breeding, which may increase the risk of congenital heart defects. We strongly urge owners not to breed any animals with known congenital defects of any kind, including heart defects. It is probably wise to also avoid breeding horses that have developed an acquired heart disorder relatively early in life, as this may indicate an increased susceptibility for that condition in offspring. *

Little Horse with a Big Hole in His Heart

Thanks to Dr. Mary Beth Whitcomb of the UC Davis School of Veterinary Medicine for this story and the accompanying ultrasound images.

Among the permanent residents of the Center for Equine Health is a little horse named Cash who was born with a ventricular septal defect, or a hole in his heart. He has been living here since he was 2 years old and is now a thriving 7-year-old performing a valuable service as a model for veterinary students. A ventricular septal defect means that Cash has an opening between the right and left ventricles of his heart, which causes blood to flow from the left to the right ventricle when the heart contracts. Similar to other species, VSDs are the most common congenital heart defect in horses. It is not known whether these types of congenital defects are hereditary.

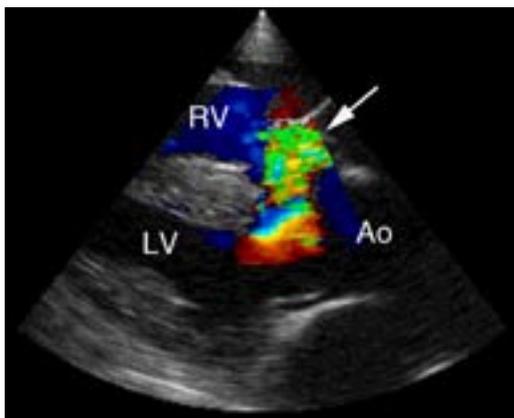
While the condition may sound severe, it is important to realize that many horses live full lives with a normal or near-normal life expectancy as long as the hole is smaller than 2.5 cm (about 1 inch) in diameter. Horses with a VSD larger than that often live a shorter life but may not show any symptoms until later in life. While Cash did have exercise intolerance when put into training as a 2-year-old, he is otherwise healthy and shows no outward symptoms in his less demanding model career at the Center for Equine Health.



Cash today at age 7, thriving at the Center for Equine Health with his herd mates.

Cash's loud heart murmurs have helped teach numerous veterinary students the art of critical auscultation—listening to the heart with a stethoscope.

He also helps teach students and specialists-in-training in equine internal medicine how to perform an ultrasound of the heart so that more veterinarians will be skilled and familiar with this important condition. ❁



These are ultrasound images of Cash's heart. The **left image** shows a defect (VSD, shown by arrow) in the muscle between the left ventricle (LV) and right ventricle (RV). The defect—ventricular septal defect or VSD—allows blood to flow through it into the right ventricle when the heart contracts. In a normal heart, there should be no direct connection between the left and right ventricles. In the **right image**, color flow Doppler was used to show the blood that is being diverted through the VSD from the left ventricle to the right ventricle. The green signal (arrow) represents abnormal blood flow.

Atrial Fibrillation: A Common Problem in Horses

Thanks to Dr. Gary Magdesian of the UC Davis School of Veterinary Medicine for providing information on these clinical case studies.

Atrial fibrillation is an electrical disorder of the heart rhythm, also known as an arrhythmia (an irregular heartbeat). It is the most commonly recognized form of an arrhythmia in horses except for sinus arrhythmia, which is common in fit, athletic horses. With this arrhythmia, the normally regular, organized atrial electrical waves become irregular, disorganized and chaotic, and the atria fail to contract normally. This results in a very unpredictable, irregular heartbeat.

Although atrial fibrillation often develops in horses with advanced structural heart disease and atrial dilation, horses often develop this arrhythmia with minimal or no detectable additional signs of heart disease. In such cases, signs of cardiac insufficiency are usually not recognized at rest or with mild to moderate exertion but become apparent at more strenuous levels of exercise or not at all. Accurate diagnosis of this arrhythmia requires evaluation of an electrocardiogram (ECG), where the lack of normal atrial ('p') waves and the very irregular ventricular waves are readily identified. Further evaluation of the structure and mechanical function of the heart by echocardiography is also recommended, because the prognosis for treatment, recovery and return to previous activity levels is directly related to the presence or absence of underlying mechanical cardiac dysfunction.

If there is cardiac disease with

atrial dilation, the prognosis for functional recovery is poor and conversion (to make it regular) of the arrhythmia is usually unsuccessful or temporary. Treatment of the signs of cardiac insufficiency with drugs such as digitalis and diuretics may be considered in selected cases where little physical activity is expected. If there is little or no evidence of underlying cardiac dysfunction (which is termed **benign atrial fibrillation**), administration of oral or injectable drugs, especially quinidine, is often successful at converting the arrhythmia to a normal rhythm. Recently, electroconversion of atrial fibrillation without the use of any drugs has been reported in horses. Most of these horses are able to return to their previous levels of activity and performance, although some experience one or more recurrences of the arrhythmia, necessitating retreatment or retirement from strenuous activity. Many cases of benign atrial fibrillation can continue to perform at a submaximal level of performance without ill effects, even if the arrhythmia fails to convert.

Case Study

Several years ago, an 11-year-old Thoroughbred gelding event horse was brought to the UC Davis Veterinary Medical Teaching Hospital for a leg-related problem. Upon examination, the attending veterinarian noticed an irregular heartbeat. In fact, the heartbeat

was irregularly irregular. The owner stated that the irregularity had been noticed three years prior and had been treated, but that the horse had been otherwise normal and performing well. Atrial fibrillation was suspected and was subsequently confirmed with an electrocardiogram.

To evaluate the cause of the atrial fibrillation and determine whether it was benign—that is, not associated with any underlying heart failure, defect or disease—the horse was referred for a cardiology consultation. This exam showed that it was a case of **benign atrial fibrillation** because there was no structural heart disease. As a result, this horse had a good prognosis. It was treated with the drug quinidine to normalize—or convert—the irregular heartbeat. The horse was sent home with a recommendation to rest for 6 to 8 weeks and then to work back slowly into his prior eventing activities.

For other horses, the prognosis is not as good. Another form of atrial fibrillation is known as **pathologic atrial fibrillation** and is the result of some form of heart failure. An ultrasound of the heart in such a case would reveal an enlarged heart. Other signs might include a high heart rate (exceeding 60 bpm), jugular pulsation, ventral (abdomen and legs) edema, and a cough. This form of atrial fibrillation can be secondary to a viral infection, to selenium deficiency, or to a torn valve that may occur as part of aging. *

Oleander Poisoning: The Preventable Illness

Oleander is an evergreen shrub that seems to grow everywhere in California—in yards, parks and along freeways. It is often grown as a hedge that can reach up to 20 feet tall. The leaves are thick, leathery and dark green. White, pink or yellow flowers that are sweetly scented grow in clusters at the end of each branch.



The culprit: oleander

Oleander is one of the most poisonous plants and contains numerous toxic compounds, many of which can be deadly to people, especially young children. The toxicity of oleander is considered extremely high; a very small amount can have a lethal or near-lethal effect. A single leaf can kill a child. In animals, around 0.5 mg/kg of body weight can be lethal.

The primary toxins in oleander are cardiac glycosides, which affect the heart. Cardiac reactions consist of an irregular heart rate, sometimes characterized by a racing heart that subsequently

slows to below normal further along in the reaction. The heart may also beat erratically with no sign of a specific rhythm. Other toxic effects include nausea, excess salivation, abdominal pain, diarrhea (sometimes with blood), kidney failure and colic in horses. Poisonings from this plant can also affect the central nervous system and cause drowsiness, tremors, seizures, collapse, and even coma that can lead to death. Oleander sap can cause skin irritations, severe eye irritation, and allergic reactions characterized by dermatitis.

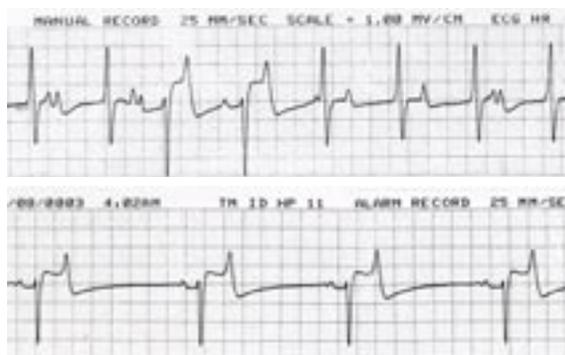
Drying of plant materials does not eliminate the toxins, and poisonings have been reported from the smoke of the burning plant or use of the branches as skewers for food.

Oleander is especially dangerous to horses, as it is sweet. Symptoms of a poisoned horse include severe diarrhea, colic and abnormal heartbeat. The following case from the Veterinary Medical Teaching Hospital illustrates the high toxicity of oleander and the complexity of physical effects it causes as well as treatment for what is a preventable illness.

Case Study

Several years ago, a sick four-year-old Standardbred racehorse was brought to the UC Davis Veterinary Medical Teaching Hospital. The owner reported that it had stopped eating the day before and was clearly unwell. The horse appeared to be in shock, judging from the color of its mucous membranes, a heartbeat racing at 160 beats per minute (bpm), and a slightly elevated temperature. (Normal heartbeat for a horse is 28 to 44 bpm.) It also had significant discomfort from ileus—a condition in which the bowel does not move the contents at normal rates of flow because of lack of neuromuscular control. The ileus had caused a backward flow of fluid and intestinal contents back into the stomach. Since horses cannot vomit, this poses a serious problem. To treat this condition, a nasogastric tube was inserted into the horse to drain the accumulating fluid while tests were performed to determine the underlying problem.

An electrocardiogram (ECG) revealed that the horse had ventricular tachycardia, which is an irregular and overly rapid heartbeat. Pleural effusion—fluid in the chest around the lungs—and pericardial effusion—fluid around the heart—were signs that the horse's heart was failing.



Top: ECG showing ventricular tachycardia in horse with oleander toxicity. Note the irregular heart rhythm caused by oleander.

Bottom: ECG in same horse as above, showing return to a more normal sinus rhythm after treatment (conversion) with lidocaine.

This condition was treated as an emergency with lidocaine administered intravenously to slow the heart rate. Eventually the heart rate was brought down to 60 bpm, substantially closer to the normal rate of 40 bpm than before, and the arrhythmia was converted to a normal sinus rhythm.

Blood work and urinalysis results then indicated a build-up of toxins and renal failure. By now, the horse was quite weak and was staggering. It was immediately put on intravenous fluids to flush out the toxins and eventually was stabilized. Meanwhile, testing continued to determine the exact cause of illness.

During the course of this treatment, the horse was unable to eat and received total intravenous parenteral nutrition.

Toxins had also caused clotting problems in the blood, and the horse began to bleed from the nose. As a result, he received a blood transfusion, as well as 3 liters of platelet-rich plasma.

Finally, the diagnosis of toxicity was confirmed by laboratory tests, which showed the presence of oleander in the blood, feces and stomach fluid. The owners of the horse had not realized that the pasture the horse had been turned out in days before was surrounded by oleander. Fortunately, this story has a happy ending. With continued intensive treatment and supportive care, the horse began to recover and was eventually released from the VMTH. Three months after this incident, the owner reported that the horse was doing very well and was back in training.

Not every case ends this well, so remember that preventing exposure to oleander is by far the best course of action for your horses and other animals. ✱

Dr. Jeffrey Norris Wins the 2006 Wilson Award

This year's James M. Wilson Award was presented to Dr. Jeffrey Norris for his work in characterizing a bleeding disorder in a Thoroughbred mare, providing a diagnostic tool for identifying it, and demonstrating that the dysfunction is heritable.



Dr. Jeffrey Norris and Treasure Dance

The Wilson Award is given each year to an outstanding equine research publication authored by a graduate student or resident in the UC Davis School of Veterinary Medicine. Dr. Norris's research culminated in the manuscript, *Investigation of a Novel, Heritable Bleeding Diathesis of Thoroughbred Horses and Development of a Screening Assay*, and was honored with the Wilson Award.

Bleeding in racing horses associated with exercise appears to involve several different physiological factors, yet few severe cases are clinically investigated. Blood clots are required for stopping bleeding. The clots form when activated blood platelets produce a fibrin net that covers a wound in the blood vessel wall and traps blood cells to keep them from flowing through the hole in the damaged vessel. The objective of Dr. Norris's research was to identify the precise location in the clotting process that prevented a blood clot from forming in a Thoroughbred mare with an uncontrollable bleeding disorder. This mare, named Treasure Dance, had unusual bleeding episodes that evidenced during routine dentistry, hypodermic injections, or even after playing in pasture. In his research, Dr. Norris discovered that while Treasure Dance's platelets could alter their surface to initiate clotting, they were not able to take the next step and turn on and organize the machinery that actually makes the clot. He also tested two of Treasure Dance's offspring (produced by embryo transfer and carried to term by two surrogate mares) to determine the heritability of the problem and discovered that one of the foals indeed had the same problem. In subsequent research, Dr. Norris will be conducting DNA studies with the ultimate goal of developing a test that could help avoid perpetuating this genetic defect.

Dr. Norris received his PhD in Biophysics from the University of California, Davis, and is now a Postdoctoral Fellow here in the laboratory of Dr. Fern Tablin.

Cardiology Service at the UC Davis Veterinary Medical Teaching Hospital (VMTH)

The Cardiology Service of the VMTH examines and consults on all species of animals with known or suspected heart disease. Routine evaluations include the history and review of prior records, physical examination, an electrocardiogram, and an echocardiogram (ultrasound imaging). Additional examinations are available if needed. The service consists of Dr. William Thomas, Chief, and Dr. Mark Kittleson, three residents and a technician.

Appointments may be made by calling the Large Animal Clinic at (530)752-0290. Appointments are usually for Tuesday afternoons, but other arrangements may be made on an individual basis. Referring veterinarians are encouraged to discuss a case with one of the cardiology faculty or residents on duty prior to the appointment if possible. Owners are asked to bring any available records or results of prior examinations for review and comparison.



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