Echocardiography for the Assessment of Congenital Heart Defects in Calves

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KEYWORDS

• Cardiac • Malformation • Bovine • Imaging • Ultrasonography

KEY POINTS

- Congenital heart disease in calves commonly presents as chronic respiratory disease, failure to thrive, or poor growth.
- The most common congenital heart disease in calves is a ventricular septal defect, either alone or in combination with more complex abnormalities.
- The prognosis for survival varies from guarded to poor and depends on the severity and hemodynamic relevance of the defects, but there is no specific prospective study in calves.

Videos showing echocardiographic examples of congenital heart defects in calves accompany this article at http://www.vetfood.theclinics.com/

OVERVIEW

Congenital heart disease (CHD) in calves is uncommon, being observed in less than 0.2% of all bovine hearts inspected in 2 large necropsy studies.[1,2] A diagnosis of CHD is suspected following a history of ill thrift, poor growth, respiratory disease that fails to respond to appropriate therapy, and/or if a heart murmur is detected on physical examination.[3,4] Echocardiography is the most useful diagnostic test to confirm or rule out the presence of CHD. The detection of the common simple congenital abnormalities (eg, ventricular septal defects [VSDs]) is straightforward, but complex congenital abnormalities can prove more difficult to evaluate and interpretation of the images takes some experience and skill. Familiarity with the normal cardiac anatomy and a logical and standardized approach to the echocardiographic assessment are crucial to confirming a diagnosis of CHD. The authors recommend the

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systematic approach of sequential segmental analysis (SSA) when evaluating calves for CHD.[5]

EQUIPMENT AND SETTINGS

Echocardiography can be performed easily in calves in the field setting as well as the hospital. Despite the cranial location of the heart and the narrow intercostal spaces, echocardiography in calves is more rewarding than with adult cows.

Most calves can be evaluated using a medium-frequency probe (3.4–5 MHz) with a small footprint. A small, phased array probe is preferred, but microconvex, curvilinear, or linear probes can be used as well.

For two-dimensional echocardiography (2DE), the image should be optimized for a frame rate of at least 25 frames per second and typically 30 to 60 frames per second to fully appreciate cardiac motion. Higher frame rates can be achieved by reducing the sector width and imaging depth if necessary. An imaging depth of 10 to 15 cm is adequate for most young calves, whereas a depth of 15 to 20 cm may be necessary in older calves. Only 1 focal zone should be used, which is set to the far field. Tissue harmonics imaging results in a more favorable signal/noise ratio, increases depth of penetration, and improves endocardial border definition and visualization of cardiac structures, but echoes of fine structures such as valves and chordae appear thicker in harmonic imaging.

The 2DE-guided M mode uses a very high frame rate and therefore is capable of recording high-frequency motion (eg, a fluttering valve), which might be missed by the slower sampling rate of a 2DE study. Some echocardiography machines offer anatomic M mode, which can be used to derive M-mode tracings offline from 2DE cine-loop recordings and allows positioning of the M-mode cursor freely on the two-dimensional (2D) image, independent of the sector apex. However, this advantage can only be achieved at the expense of a lower temporal resolution, related to the low recording frame rate of 2DE recordings.[6]

In color Doppler imaging mode, a high frame rate (eg, achieved by narrowing the sector with and imaging depth), a slight reduction of tissue priority settings (favoring color priority), and selection of color maps with variance coding (eg, green coding of turbulent flow) facilitates recognition of intracardiac blood flow patterns. The velocity range is usually set near the maximum possible limits.

In spectral Doppler imaging mode (ie, pulsed-wave and continuous wave Doppler), the power can be reduced by 1 to 2 steps to increase clarity of the Doppler tracing, whereas specific filter settings allow the elimination of low-velocity noise. The velocity scale should be adjusted depending on the expected blood flow velocities to be recorded. More details on equipment and machine settings can be found elsewhere.[7]

A surface electrocardiogram (ECG) should be recorded simultaneously with all echocardiographic recordings for timing of cardiac events. If possible, cine loops containing at least 3 cardiac cycles should be recorded and stored. This method allows for offline measurements at several time points during the cardiac cycle and further evaluation of complex defects using slow motion playback. Still images are less optimal because subtle abnormalities may be difficult to detect.

PATIENT PREPARATION AND RESTRAINT

Young calves can be easily restrained in sternal or lateral recumbency, whereas older calves should be gently restrained standing. Most dairy breeds tolerate this procedure without sedation; however, light sedation may be necessary in older beef calves.

Clipping a small area (rectangle 8–10 cm) between the fourth and fifth intercostal spaces (immediately behind the elbow) on both sides of the chest improves the image quality. The skin can be gently cleaned with alcohol and ultrasonography coupling gel applied. If necessary, the thoracic limbs can be moved cranially to allow access to the relevant intercostal spaces.

IMAGING APPROACH/PROTOCOL

The technique for routine echocardiography in cattle is well described.[7–9] Familiarization with the normal anatomy and standard imaging planes for echocardiography is essential when attempting to diagnose CHD. In general, the imaging planes for calf echocardiography are similar to those required for equine and small animal echocardiography (**Fig. 1**).[7]

Right Parasternal Views

In a normal calf, most echocardiographic views are taken from the right parasternal imaging window. Long-axis 4-chamber, right ventricular outflow tract (RVOT) and left ventricular outflow tract (LVOT) imaging planes provide the basis for capture of the main cardiovascular structures (atria, ventricles, great vessels, atrioventricular [AV] and arterial valves) and subjective assessment of cardiovascular function. Short-axis 2D and M-mode images of the ventricles obtained at the level of the papillary muscle and chordae tendineae, mitral valve, left atrium (LA), and aorta (Ao)/pulmonary artery (PA) provide additional morphologic and functional information. From there, color flow mapping and spectral Doppler analysis can help identify the presence of intracardiac shunting, valvular regurgitation, or any inflow or outflow tract obstruction.

Left Parasternal Views

The left parasternal imaging window can be useful for detecting left heart disease.[8] Long-axis views of the atria, ventricles, Ao, and in particular the main PA can be obtained. In the authors' experience, in some cases because of the positioning of the heart or concurrent lung disorder, the left parasternal window may provide better access to some or all of the cardiac structures.

Apical 4-Chamber/5-Chamber Views

Depending on the calf's size, an apical 4-chamber or 5-chamber view, similar to that used in human and small animal echocardiography, can provide superior alignment for color and spectral Doppler studies. These views are obtained by sliding the transducer near the apex close to the sternum and scanning dorsally and cranially to view all chambers. The 4-chamber view includes the LA, left ventricle (LV), right atrium (RA), and right ventricle (RV), whereas the 5-chamber view also includes the Ao.[7]

SEQUENTIAL SEGMENTAL ANALYSIS

The anatomy and spatial orientation of the cardiac chambers and great vessels can be markedly abnormal in many cases of complex congenital malformation. This abnormality makes interpretation of echocardiographic recordings difficult and often necessitates the use of unconventional imaging planes to display all the relevant cardiac structures.

Therefore, for echocardiographic assessment and diagnosis of CHD, the recommended approach in both human and veterinary echocardiography is SSA. All

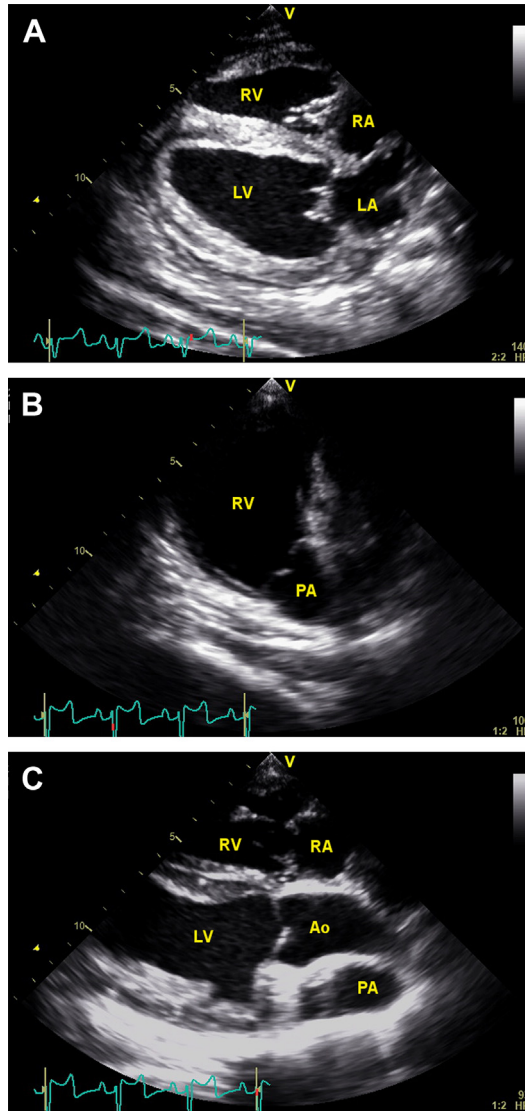

Fig. 1. 2D B-mode recordings in (*A*) right parasternal long-axis 4-chamber view, (*B*) right ventricular outflow tract (RVOT) view, (*C*) left ventricular outflow tract (LVOT) view, (*D*) right parasterna short-axis view at the level of the papillary muscles, and (*E*) left parasternal long-axis view. The base of the heart is to the right, the apex to the left of the screen in the long-axis images. An ECG is superimposed for timing. The red marker on the ECG tracing indicates the timing of the frame within the cardiac cycle. Ao, aorta; LA, left atrium; LV, left ventricle; PA, pulmonary artery; RA, right atrium; RV, right ventricle.

hearts, no matter whether normal or abnormal, are made up of 3 segments: atria, ventricles, and the great vessels (Ao and PAs). Using SSA, the orientation and relationship between cardiac segments are investigated in a stepwise fashion. The cardiac segments are identified based on their anatomic features and not their just spatial orientation.[5,10]

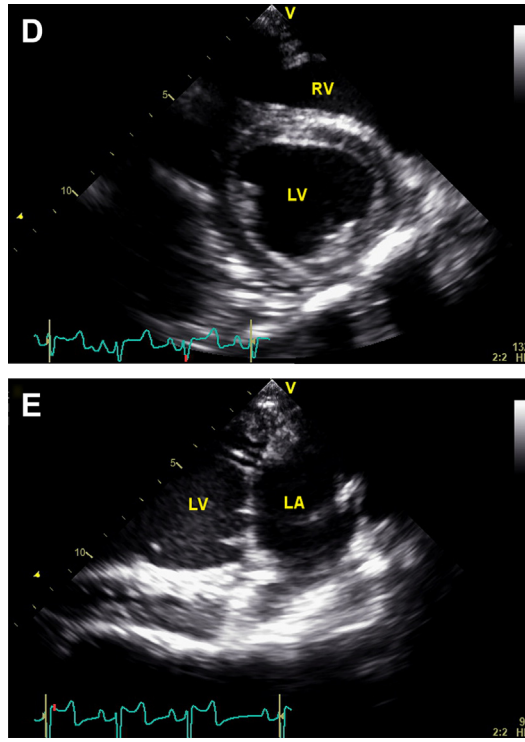

Fig. 1. (*continued*).

Step 1: Atrial Arrangement

Determining the presence and arrangement of the atria is the first step in SSA. The atria are best identified based on the morphology of their appendages. The morphologic RA is characterized by a broad-based, triangular appendage with the terminal crest and extensive pectinate muscles. The morphologic LA has a narrow-based, tubular appendage with no obvious terminal crest and less obvious pectinate muscles.

The arrangement of the atria can be described as usual (situs solitus), mirror imaged (situs inversus, reported in 5 cases in the literature[11]), right isomerism (morphologically bilateral right atria), or left isomerism (morphologically bilateral left atria). Partitioning of the atrial chambers can occur (cor triatriatum dexter or sinister) but is extremely rare with no cases reported in the literature. The authors have recognized one 2-month-old brown Swiss calf with cor triatriatum sinister (**Fig. 2**).

Step 2: Ventricular Arrangement

Assessment of the ventricular arrangement is the second step of SSA. The morphologic RV possesses coarse apical and septomarginal trabeculations, the leaflet of the atrioventricular (AV) valve attaches directly to the septum, and there is an obvious moderator band. The morphologic LV has fine apical trabeculations and a smooth upper part of the septum without attachments to the AV valve. Absence of a ventricular septum can result in a solitary, morphologically indeterminate ventricle.[11,12] The ventricles can be hypoplastic but complete (with developed inlet, trabecular, and outlet portions) or they can be hypoplastic and incomplete (rudimentary, often lacking the

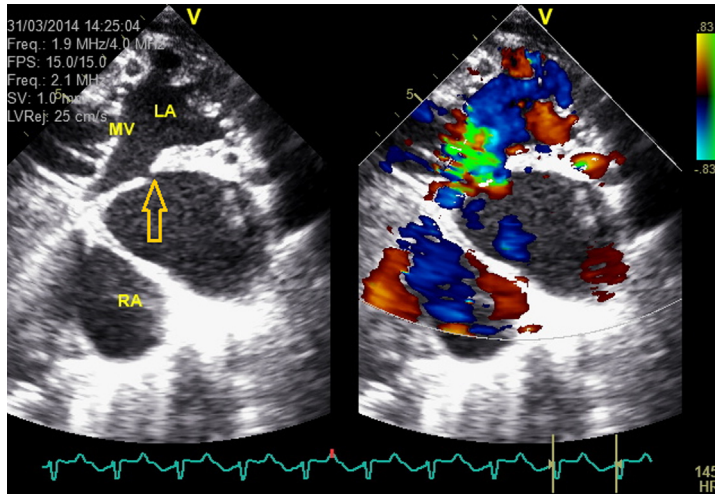

Fig. 2. 2D B-mode (*left*) and simultaneous color Doppler recording (*right*) of the left parasternal long-axis, LA view. This calf was diagnosed with cor triatriatum sinister. The LA is divided into 2 compartments with a narrow communication (*arrow*) from the upper compartment (with pulmonary veins draining into it) to the lower one (communicating with the LV and mitral valve (MV) and the RA via a large atrial septal defect [ASD]). Color flow mapping indicates turbulent flow (color coded in *green*) through the communication between compartments. Additional turbulent flow is seen in the lower LA compartment as a result of right-to-left shunting of blood across a large ASD.

inlet portion). There are several reports of calves with abnormal ventricular shape and arrangement in the literature.[11,13,14]

In cases with severe congenital malformations, it may be difficult to differentiate the LV and RV. Hypoplastic ventricles are often more easily detected. Failure to identify a rudimentary chamber is most indicative of a solitary, indeterminate ventricle.

Step 3: Atrioventricular Connections

The third step of evaluation is to describe the AV connections, by establishing how the atrial segments are connected with the ventricular mass. There are 3 main groups of AV connections.

Biatrial, biventricular connections may be considered concordant (atria connected to the appropriate ventricle), discordant (atria connected to the inappropriate ventricle), or ambiguous (eg, with atrial isomerism).

Biatrial, univentricular connections are present if the atria only connect to 1 ventricle, either as a double inlet AV connection (both atria connect with the same dominant ventricle), an absent right AV connection (only the LA is connected with the ventricular mass), or an absent left AV connection (only the RA is connected with the ventricular mass). These last 2 scenarios occur when 1 atrium ends blindly in a muscular floor at an AV junction (AV valve atresia). Usually, the connected ventricle is dominant and the nonconnected ventricle is hypoplastic and rudimentary, or a solitary ventricle exists.

Uniatrial, biventricular connections occur when a solitary AV connection straddles and overrides the ventricular septum, connecting the atrial mass to both ventricles.

A double inlet LV, an absent left AV connection, and 2 atria with a single ventricle have been reported in calves.[11,13,14]

Step 4: Morphology of the Atrioventricular Valves

The fourth step is to determine the morphology of the AV valves, independent of the AV junctional connections. There may be 2 AV valves, or just 1 common valve. The valves can be straddling and overriding, dysplastic (malformed), or partially or completely imperforate (atretic). The leaflets of the AV valves can have an abnormal length or shape, or they can be thickened, fenestrated, or fused. The papillary muscles and chordae tendineae might be altered with respect to shape, size, length, position, or orientation.

When investigating AV valve morphology, standard 2D images are most useful. The use of M mode can provide additional information to assess valve motion. Doppler studies can assist to identify regurgitation (turbulent systolic blood flow in the atria) or stenosis (high-velocity transvalvular diastolic blood flow, V_{max} >2 m/s). This combined information can help assess the type and severity of the malformation. A variety of AV valve abnormalities, usually in combination with other complex congenital defects, have been described in calves; however, tricuspid valve atresia has not been reported.[11]

Step 5: Ventriculoarterial Connections

This fifth step is extremely important in diagnosing complex malformations in calves because abnormal ventriculoarterial connections are frequently described in the literature.[12,13] When 2 great arteries are present (either normally developed or hypoplastic), the junction is termed concordant (arteries connected to the appropriate ventricles), discordant (arteries connected to the inappropriate ventricles; eg, transposition of the great vessels), or double outlet (both arteries arising from either the left, the right, or an indeterminate ventricle; eg, double-outlet RV [DORV]). These variations are shown in **Fig. 3**. An echocardiographic example of a calf with a DORV is shown in **Fig. 4** and Video 1.

The Ao and PA can be differentiated by identifying the coronary arteries, which always arise from the Ao, and the origin of the branching vessels (brachiocephalic trunk originating from the Ao, left and right PAs arising from the main PA).

If there is atresia of either the Ao or the main PA, this is termed single outlet, as is the presence of a single common or solitary arterial trunk. A common arterial trunk is characterized by a single arterial vessel arising from the base of the heart that gives rise to the systemic, pulmonary, and coronary circulation. In a solitary arterial trunk, the Ao and coronary arteries are identified but the PAs are absent and the pulmonary circulation is provided by systemic-pulmonary collaterals arising from the descending Ao.[12]

Visualization of the great vessels and their branches using echocardiography is crucial for the correct antemortem classification of the malformation, but this can be extremely challenging. If an atretic vessel is strandlike, it may be impossible to detect using echocardiography. A careful postmortem dissection is required to confirm the diagnosis in these cases.

Step 6: Morphology of the Arterial Valves

The morphology of the arterial valves is assessed in the sixth step. Overriding valves are assigned to the ventricle supporting more than 50% of their circumference. The valves can be normal (tricuspid) or they may contain an abnormal number of cusps (eg, bicuspid, quadricuspid). They can be dysplastic, hypoplastic, or atretic. The cusps may be thickened, fenestrated, or fused.

Similarly to the AV valves, the morphology and function of the arterial valves can be assessed using 2D and M-mode echocardiography and the presence of regurgitation and/or stenosis can be evaluated using Doppler studies.

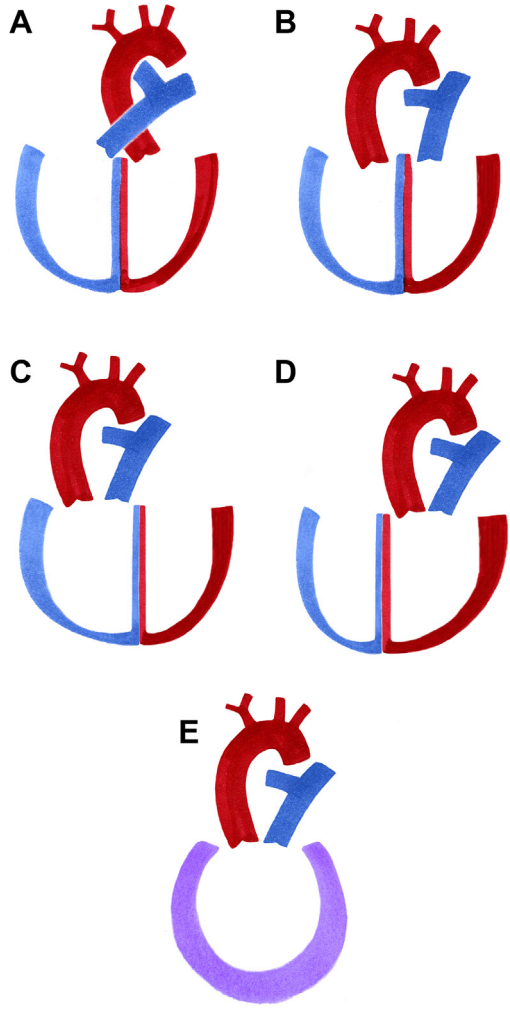

Fig. 3. (*A*) Concordant (normal) and (*B*) discordant (transposition) ventriculoarterial connections. The Ao is colored in red, the PA in blue. Double outlet can be from the (*C*) morphologically RV (*blue*), (*D*) morphologically LV (*red*), or (*E*) indeterminate ventricle (*purple*).

Step 7: Associated Malformations

In addition, the septal structures, outflow tracts, great arteries (including the arterial duct and the aortic arch), the coronary arteries, and the system and pulmonary venous connections should be assessed for malformation. Most of the described complex CHDs in calves have 1 or more of these abnormalities.[3,11]

SPECIFIC CONGENITAL MALFORMATIONS

Abnormal Communications (Shunts)

Atrial, atrioventricular, and ventricular septal defects

A thorough interrogation of all septal structures (atrial, AV, and ventricular) should be performed. The most common congenital defects reported in calves are ventricular

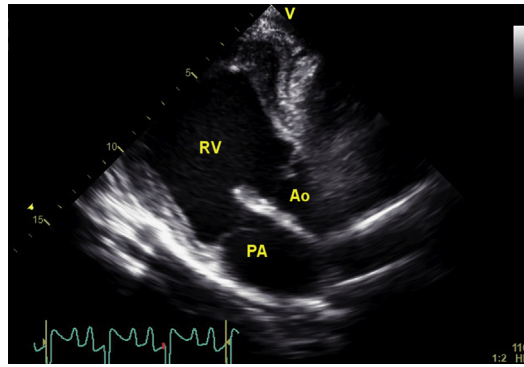

Fig. 4. 2D B-mode recording, right parasternal long-axis, RVOT view. This calf has a DORV; with both the Ao and PA visualized next to each other and connected with the RV.

septal defects (VSD), either alone or in combination with other complex malformations.[1,3,11] Abnormalities of the atrial and AV septa are also well documented, although much less common than VSDs.[11,15]

Interatrial communications are most commonly detected in combination with complex malformations but they also infrequently occur as isolated defects. They are divided into defects of the oval fossa (secundum-type atrial septal defects [ASDs]), cranial and caudal sinus venosus defects, and coronary sinus defects. Differentiation (with echocardiography) between a secundum-type ASD and a patent foramen ovale (PFO) can be difficult, especially with smaller defects. In the case of a PFO, the septal structures are fully developed but, because of high RA pressures (ie, pulmonary vascular disease), the flap of tissue covering the foramen ovale remains unfused and right-to-left shunting of blood can occur. Care should be taken not to overinterpret the echocardiography findings, because the oval fossa within the interatrial septum can appear anechoic in normal animals and Doppler studies can be confusing because of streaming of normal flow patterns (ie, caudal vena cava flow) overlying the interatrial septum. Saline contrast (bubble) studies can provide additional information and confirm right-to-left shunting of bubble-containing blood into the LA (**Box 1**). Even with this method, streaming of normal blood from the caudal vena cava may produce negative contrast in the RA, falsely suggesting left-to-right shunting of blood.[5,16] The common locations for interatrial communications are shown in **Fig. 5**.

Atrioventricular septal defects can be grouped into those that affect the interatrial component of the AV septum (ostium primum defects) and those that have a common AV canal with a common AV junction (endocardial cushion defects). The latter are usually large and easily detected with 2D echocardiography. AV septal defects have been infrequently detected in calves.[11]

Communications between the ventricles or VSDs can occur as isolated abnormalities or as part of a complex malformation. In general, VSDs are characterized according to the location and size. The defects can be located in the area of the membranous septum, adjacent to the right/noncoronary cusps of the aortic valve and the septal leaflet of the tricuspid valve (the most common type, termed perimembranous or paramembranous), immediately below the aortic and pulmonic valves (the less common type, termed subpulmonic, supracristal, subarterial, or doubly committed), or within the muscular part of the interventricular septum (rare type, termed muscular). The common anatomic locations for VSDs are identified in **Fig. 5**. Detection of VSDs using

Box 1
Saline contrast (bubble) study protocol

Materials

- Intravenous (IV) catheter placed in the right jugular vein
- IV extension set
- Three-way stopcock
- Use 20-mL of normal saline in a 20-mL syringe, connected to the 3-way stopcock
- Empty 20-mL or 30-mL syringe, connected to the 3-way stopcock

Procedure

- Use either the left or right parasternal long-axis view to image the region of interest (eg, interatrial or IVS with adjacent chambers). Agitate the saline for 30 seconds immediately before injection by moving it rapidly back and forth between the 2 syringes that are attached to the 3-way stopcock. Obtain a cine-loop recording immediately before, during, and for 10 to 15 seconds after injection of the agitated saline into the intravenous catheter. The agitated saline containing small air bubbles provides positive echo contrast and, if no cardiac shunts are present, the bubbles will only be present in the right heart (Fig. 10). If bubbles appear in the left heart shortly after injection, there must be some form of rightto-left shunting of blood, most commonly a VSD, ASD, or both (Fig. 11). If transpulmonary shunting through arteriovenous connections is present, a small number of bubbles might appear in the LA and LV only after a few heart beats (but not immediately after injection).

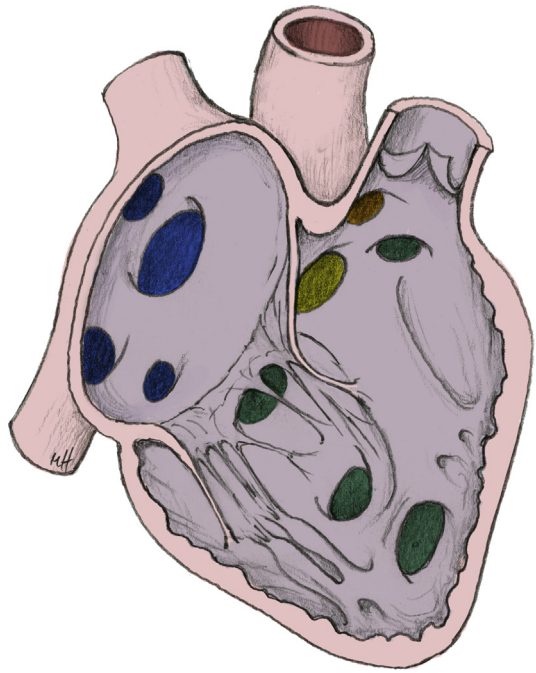

Fig. 5. The common locations of ASDs and VSDs. The largest blue area is the oval fossa, where true ASDs occur. The other, smaller blue areas are locations of other possible interatrial communications. The yellow area is the location of perimembranous VSDs. The orange area shows the location of subpulmonic VSDs. The green areas are possible locations for muscular VSDs in the inlet, apical, and outlet portions of the septum.

echocardiography is straightforward, although nonconventional views may be required in addition to the standard imaging planes. Color flow mapping may help detect small VSDs that are not clearly observed on the 2D images and care must be taken to screen the entire IVS from apex to base in long-axis and short-axis imaging planes for the presence of small muscular VSDs. Spectral Doppler recordings can assess peak shunt flow velocity, allowing estimation of the pressure gradient across the defect (**Box 2**).[5] Examples of perimembranous and subpulmonic VSDs are shown in **Figs. 6–8** and Videos 2–5. An example of an ASD is shown in **Fig. 9**.

Patent ductus arteriosus

In calves, the ductus arteriosus can remain patent following birth, as an isolated abnormality or in association with other malformations. In most normal calves, the ductus arteriosus should be functionally closed within the first few days of life.[11] It can be challenging to identify the arterial duct and ductal flow with 2D and color Doppler echocardiography, but assessment should include interrogation of several views of the main PA from both the right and left imaging windows. Spectral Doppler imaging may indicate disturbance of diastolic flow in the main PA in cases with significant left-to-right shunting of blood.

Aortopulmonary window

This term describes a communication between the great arteries in the setting of separate pulmonic and aortic valves. Differentiation should be made between an aortopulmonary window and a common arterial trunk, with a common trunk connected to the base of the ventricle via a single valve and a VSD present.[12] This defect has been reported once in calves.[11]

Box 2
Prognostic evaluation of VSDs

Estimation of the size of the VSD from 2D and color Doppler images:

- VSD to aortic root diameter ratio less than or equal to 0.3: small, restrictive VSD
- VSD to aortic root diameter ratio greater than or equal to 0.6: large, unrestrictive VSD

Estimation of the pressure gradient between RV and LV based on spectral Doppler velocity measurements across the VSD:

- Velocity greater than 4.5 m/s indicates a normal LV-to-RV pressure gradient of greater than 80 mm Hg
- Velocity less than 3.0 m/s indicates a decreased LV-to-RV pressure gradient of less than 36 mm Hg (caused by RV hypertension and/or LV hypotension)

Assess for evidence of pulmonary hypertension:

- Enlarged PA compared with Ao (PA/Ao ratio >1.0)
- High-velocity pulmonic insufficiency (>2.5 m/s if present)
- High-velocity tricuspid regurgitation (>3.2 m/s if present)

Assess for evidence of left-sided volume overload

- LA and LV enlargement

Note that these key points are based on current practice in equine cardiology and should provide a rough point of reference when applied to calves; published data to support prognostic factors associated with VSDs in calves are lacking.

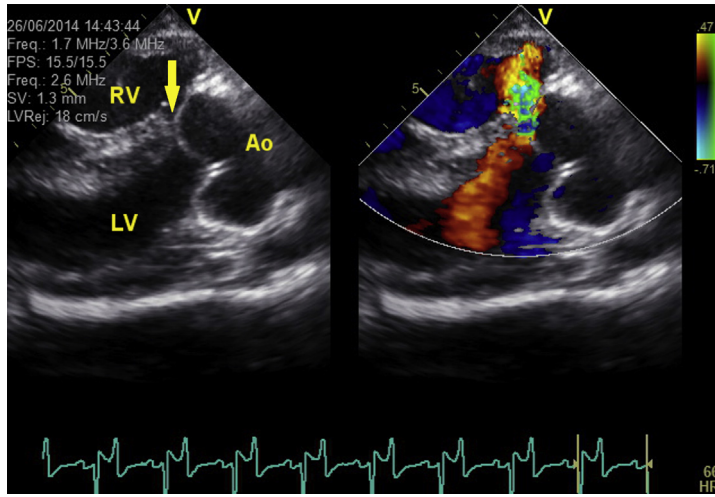

Fig. 6. 2D B-mode (*left*) and simultaneous color Doppler recording (*right*) of the right parasternal long-axis, modified LVOT view. A restrictive perimembranous VSD (*arrow*) is present. Color flow mapping indicates turbulent shunt flow (color coded in *green*) through the defect during diastole.

Outflow Tract Obstructions

Even in the absence of abnormal ventriculoarterial connections, attention should be paid to the outflow tracts of each ventricle. Stenosis of the outflow tract can be located in the subvalvular, valvular, or supravalvular regions. Subvalvular stenosis can result from shelves of fibrous tissue, muscular (septal) hypertrophy, or anomalous

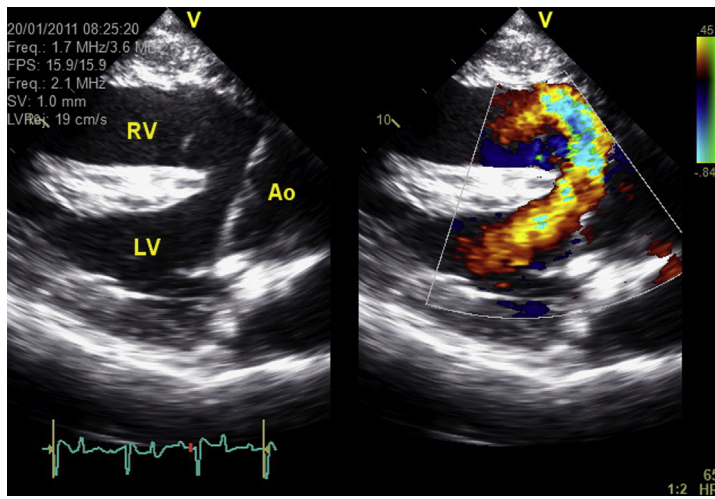

Fig. 7. 2D B-mode (*left*) and simultaneous color Doppler recording (*right*) of the right parasternal long-axis, modified LVOT view. A large perimembranous VSD is present concurrently with an enlarged overriding Ao. Color flow mapping indicates end-diastolic flow across the defect. This calf was diagnosed with a tetralogy of Fallot.

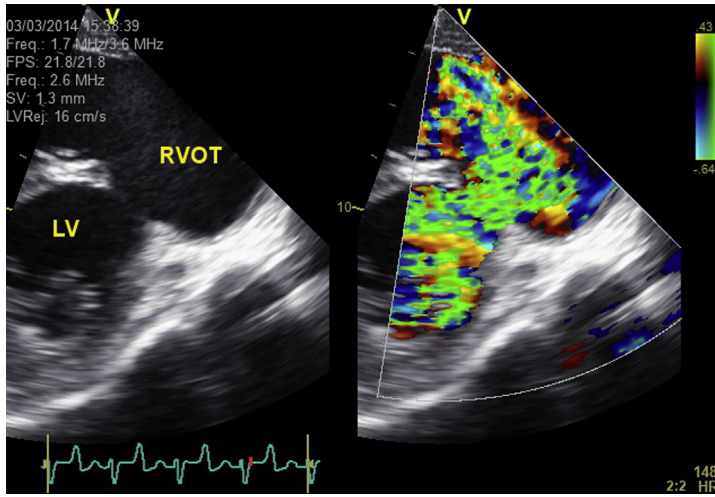

Fig. 8. 2D B-mode (*left*) and simultaneous color Doppler recording (*right*) of the right parasternal short-axis view at the level of the mitral valve. A large subpulmonic VSD is present, connecting the LV with the RVOT. Color flow mapping indicates turbulent shunt flow (color coded in *green*) through the defect during systole.

muscular bundles (hypertrophied septomarginal trabeculations). Valvular stenosis can result from hypoplasia or dysplasia of the valves with fusion, tethering, or thickening of the leaflets. Supravalvular stenosis can occur because of constriction of the sinotubular junction or diffuse hypoplasia of the great artery. Dynamic outflow obstruction may contribute to subvalvular stenosis as a result of muscular hypertrophy, accessory AV valve tissue prolapsing into the outflow tract, or anterior motion of the septal mitral leaflet during systole. Standard 2D and M-mode echocardiographic assessment allow evaluation of the ventricular outflow tracts, valve morphology, and motion. Spectral Doppler recordings can quantify the resulting hemodynamic consequences and allow estimation of the degree of stenosis (aortic and pulmonic outflow tract velocities >2 m/s [pressure gradients >16 mm Hg] can indicate stenosis).[5,7] To the authors' knowledge, isolated pulmonic or aortic stenosis has not been reported in

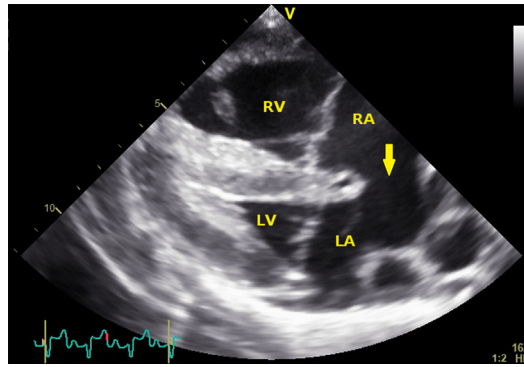

Fig. 9. 2D B-mode recording in a right parasternal long-axis, 4-chamber view focused on the atria. A large ASD is present (*arrow*), connecting the RA and the LA atrium.

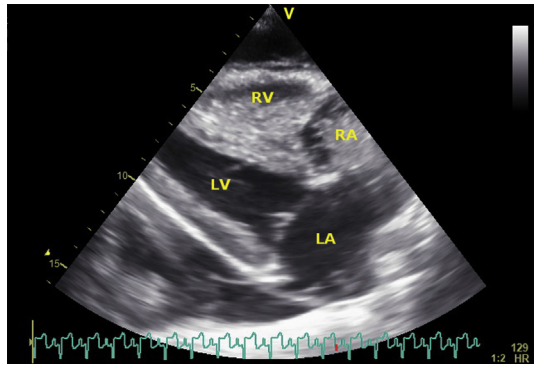

Fig. 10. 2D B-mode recording in a right parasternal long-axis, 4-chamber view. The image was captured while a saline contrast (bubble) study was being performed. The contrast is visible as hyperechoic content in the RA and the RV but not in the LA and LV. There is no evidence of an intracardiac shunt.

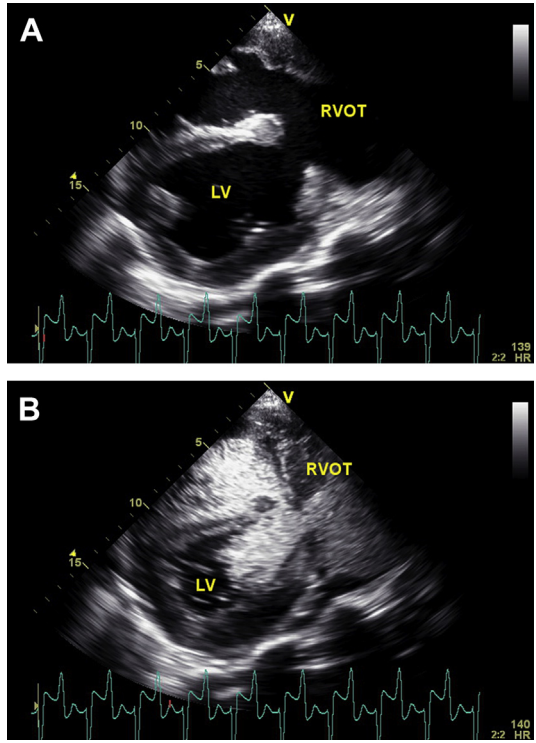

Fig. 11. 2D B-mode recording in a right parasternal short-axis view, below the level of the mitral valve. (*A*) A large subpulmonic VSD in its typical location, connecting the LV with the RVOT. (*B*) Image recorded in the same imaging plane, captured during a saline contrast (bubble) study. There is intense saline contrast present in the RV and the RVOT, extending through the defect into the LV, indicating diastolic right-to-left shunting across this large defect.

calves but may be detected in combination with other complex CHD abnormalities (eg, pulmonic stenosis with a tetralogy of Fallot).[11]

Tetralogy/Pentalogy of Fallot

A combination of pulmonic stenosis, right ventricular hypertrophy, and a VSD with an overriding Ao forms the complex known as tetralogy of Fallot. This form of CHD is well documented in calves, with more than 42 cases in the literature.[11,17,18] A severe form of this abnormality can be associated with pulmonic atresia.[19] If combined with an ASD, these abnormalities become a pentalogy of Fallot.

Coronary Abnormalities

Coronary artery anomalies can occur with variations in the site of origin, aberrant course, abnormal number, and fistulous communications between branches of the coronary arteries and a cardiac chamber or vessel. A large number of coronary artery variations have been described in calves. In contrast with humans, calves more often have an anomalous origin of the right coronary artery (originating from the pulmonary trunk).[11] Coronary artery–ventricular fistulas also dominated among other coronary artery abnormalities.[11] Echocardiographic detection of coronary abnormalities can be extremely difficult and even a postmortem diagnosis can be challenging in many cases.

Anomalies of Systemic and Pulmonary Venous Connections

These defects are considered rare in calves.[11] Examples include total or partial anomalous pulmonary venous return, persistent left cranial vena cava, or aberrant caudal venous return via the azygous vein. Echocardiographic assessment is difficult in most cases. Careful dissection in situ during a postmortem examination is required for a correct diagnosis.

Abnormalities of the Aorta and Aortic Arch

These defects are considered extremely rare in calves.[11] The most common is aortic tubular hypoplasia (described by some investigators as aortic coarctation or obstruction of the aortic arch usually at the level of the ductus arteriosus). An in vivo diagnosis via transthoracic echocardiography is often not possible.

Abnormal Heart Location

Ectopia cordis, mainly cervical, in which the heart is not appropriately contained within the thorax, is a frequently described condition in calves with more than 165 cases reported in the literature.[11] The clinical diagnosis is generally obvious in these cases. Occasionally, additional cardiac malformations are reported in these ectopic hearts.

HEMODYNAMIC CONSEQUENCES OF CONGENITAL HEART DISEASE

Following the echocardiographic description and diagnosis of the morphologic abnormalities, the hemodynamic consequences of the malformations need to be investigated in order to assess their clinical relevance and prognosis. With careful consideration, the blood flow patterns through the heart, including intracardiac shunting, and the pressure conditions in the cardiac chambers can be predicted based on the sequential segmental analysis and theoretic hemodynamic scenarios.

Real-time, 2D echocardiographic evaluation of the cardiac chamber dimensions, wall thickness, septal motion, and ventricular function are all important parts of the

overall assessment of cardiac function and hemodynamic consequences. Saline contrast (bubble) studies (for more information see **Box 1**), color flow mapping, and spectral Doppler assessment allow further identification of blood flow streaming patterns and can be used to quantify intracardiac shunts, or evaluate inflow or outflow tract stenosis or valvular regurgitations.

Assessment of the Hemodynamic Relevance of Ventricular Septal Defects

At present, published data to support prognostic factors associated with VSDs in calves are lacking. However, information is available for horses and small animals and may be useful as a rough point of reference when assessing the hemodynamic relevance of a VSD in calves. Both the size of the defect and estimation of the pressure gradient across the defect have been used to determine whether the VSD should be considered restrictive or nonrestrictive.

Based on the equine literature, VSDs smaller than one-third of the aortic root diameter and with a left-to-right shunt velocity of greater than 4.5 m/s indicate restrictive physiology and are associated with normal performance and favorable prognosis.[20,21] In practice, calves with restrictive VSDs do not show clinical signs associated with CHD and are therefore often not recognized as such, unless a heart murmur is heard as an incidental finding. In contrast, defects with a VSD to aortic root diameter ratio of 0.6 or greater and with a left-to-right shunt velocity of less than 3.0 m/s, evidence of marked pulmonary overcirculation, and left-sided cardiac enlargement are hemodynamically relevant and associated with clinical signs of heart disease and a guarded to poor prognosis (see **Box 2**). Pulmonary hypertension may develop secondary to chronic pulmonary overcirculation as a result of the left-to-right shunting across the VSD. In advanced cases, increasing RV pressures can lead to reversal of the shunt flow, now being right-to-left across the VSD. This condition, termed Eisenmenger complex, is infrequently reported in calves with severe, hemodynamically relevant VSDs (see **Box 2**).[3]

PROGNOSIS AND OUTCOMES OF CONGENITAL HEART DISEASE

Clinically, patients with CHD present with one of 4 common groups of history or physical findings.

1. Failure to thrive/poor weight gain/chronic respiratory disease
2. Signs of cyanosis
3. Signs of congestive heart failure (right, left, or biventricular)
4. Heart murmur heard incidentally on physical examination

The patient's clinical presentation can also provide evidence for the hemodynamic consequences of the malformations that may be present. Typically, animals with severe, complex abnormalities are younger and often show signs of intracardiac shunting with cyanosis and respiratory distress, weakness, or failure to nurse. There are some lesions (eg, hemodynamically relevant VSDs) that take some time before clinical signs of disease are detected, because the heart's compensatory mechanisms play a delaying role in the onset of clinical signs. Cattle show a remarkable tolerance for severe congenital malformations, with some cases only detected in older calves, heifers, or adult cows. Note that cyanosis is only present in CHD leading to right-to-left shunting (ie, tetralogy of Fallot, transposition of the great arteries, truncus arteriosus, tricuspid atresia, and total anomalous pulmonary venous connection), but not in the most commonly seen VSDs with left-to-right shunting. Clinically, cyanosis may be difficult to detect in milk-fed calves with a low-iron diet. These animals are

frequently anemic with resultantly low concentrations of desaturated hemoglobin, making the visual observation of cyanosis harder.

In most cases of CHD affecting calves, the advanced diagnostic work-up of complex congenital cardiac disease is largely of academic interest, because there is limited practicality to treatment or management of heart disease in cattle. An accurate diagnosis and assessment of the severity of disease may be important in individual cases with isolated defects (ie, restrictive VSDs), which can be associated with a favorable prognosis. However, severe and complex malformations are often associated with a hopeless prognosis for long-term productivity and survival.[3,22] If detailed information is required about the prognosis in a calf with CHD, referral to a veterinary cardiologist for a specialist evaluation is recommended. Note also that the hemodynamic consequences of less severe defects may take months to years to develop, and follow-up evaluations may be necessary to determine the presence and rate of disease progression.

SUPPLEMENTARY DATA

Supplementary data related to this article can be found online at http://dx.doi.org/10.1016/j.cvfa.2015.09.002.

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