Cardiology Case #5 – PDA (55 images, 38 pages)

Dog Nelly, Spanish Cross Breed, age: 4 months, 4.4kg

ultrasound examination 22nd of January in 2021
case submission on 24th of May 2021

History

Nelly, a 4 months old female Spanish cross breed, had just arrived in the Netherlands the day before. During the first examination at the vet a 6/6 heart murmur was detected and an echocardiogram was advised to determine the cause of the heart murmur.

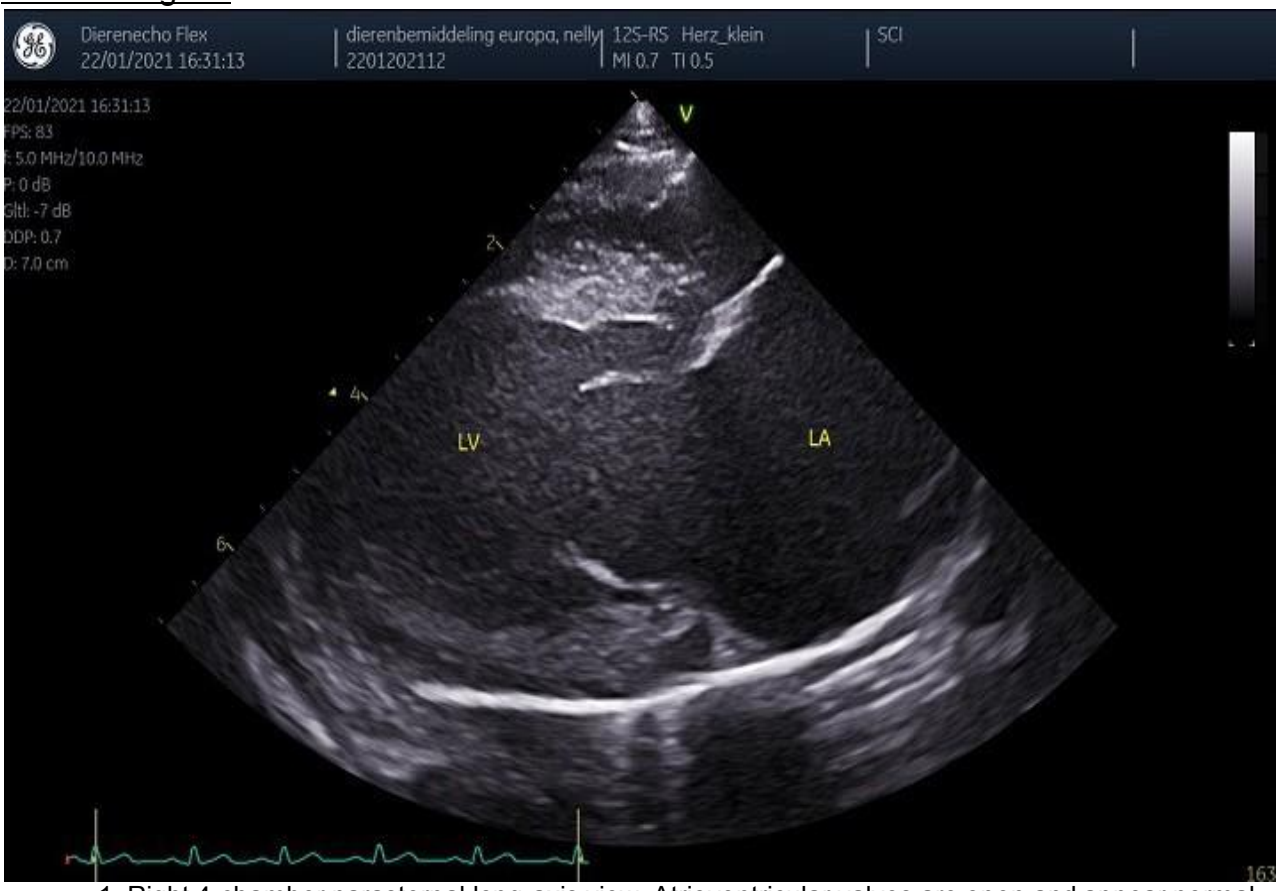
Physical examination

Nelly's temperature was 38.4 C, her heart rate was 166/min with a grade 6/6 continous murmur with punctum maximum on the left side in the 3rd intercostal space. No cardiac arrhythmias were present, her chest was clear and the respiratory rate was 26/min. Her capillary refill time was lower than 2 seconds.

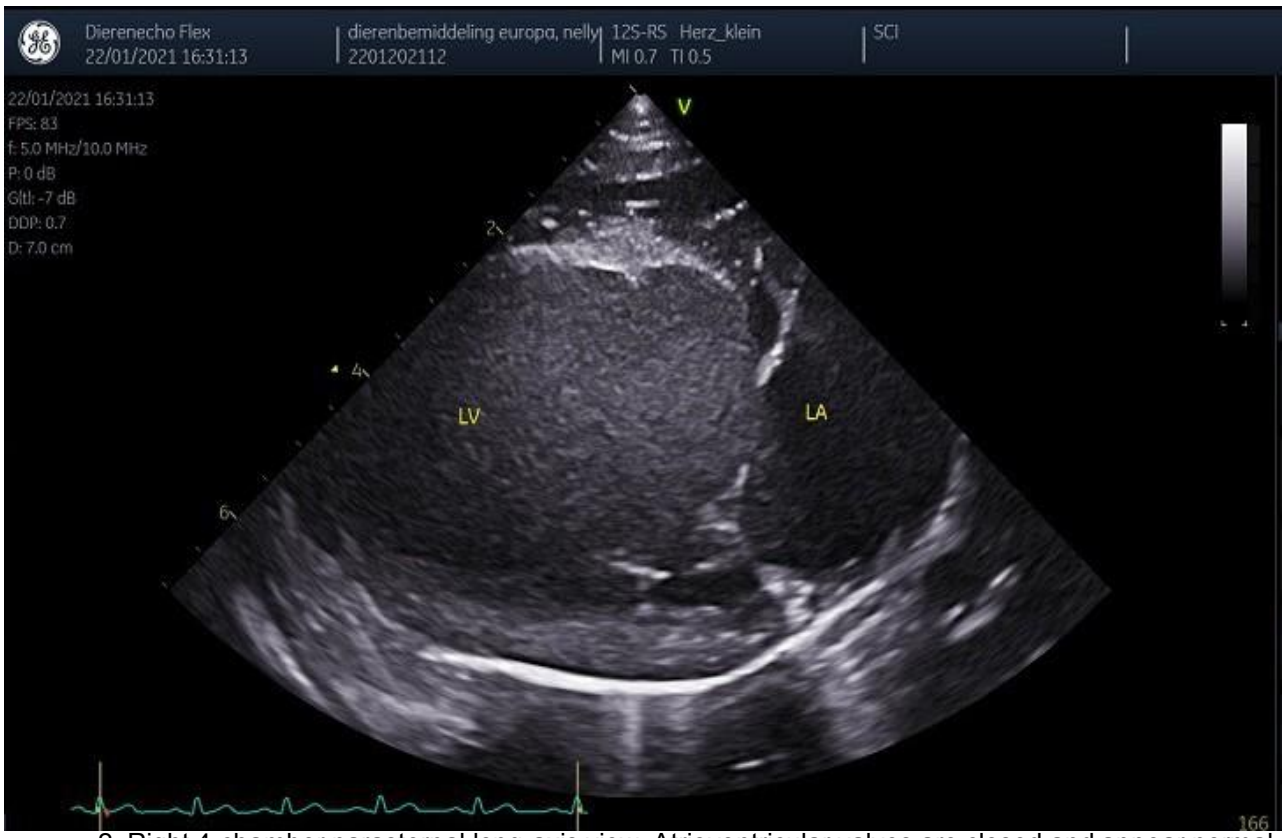
Her body condition score was 2/5.

Nelly had no dental tartar, her eyes, ears and skin were normal. All peripheral lymph nodes were normal and there were no abnormalities found during abdominal palpation. There were also no signs of dehydration.

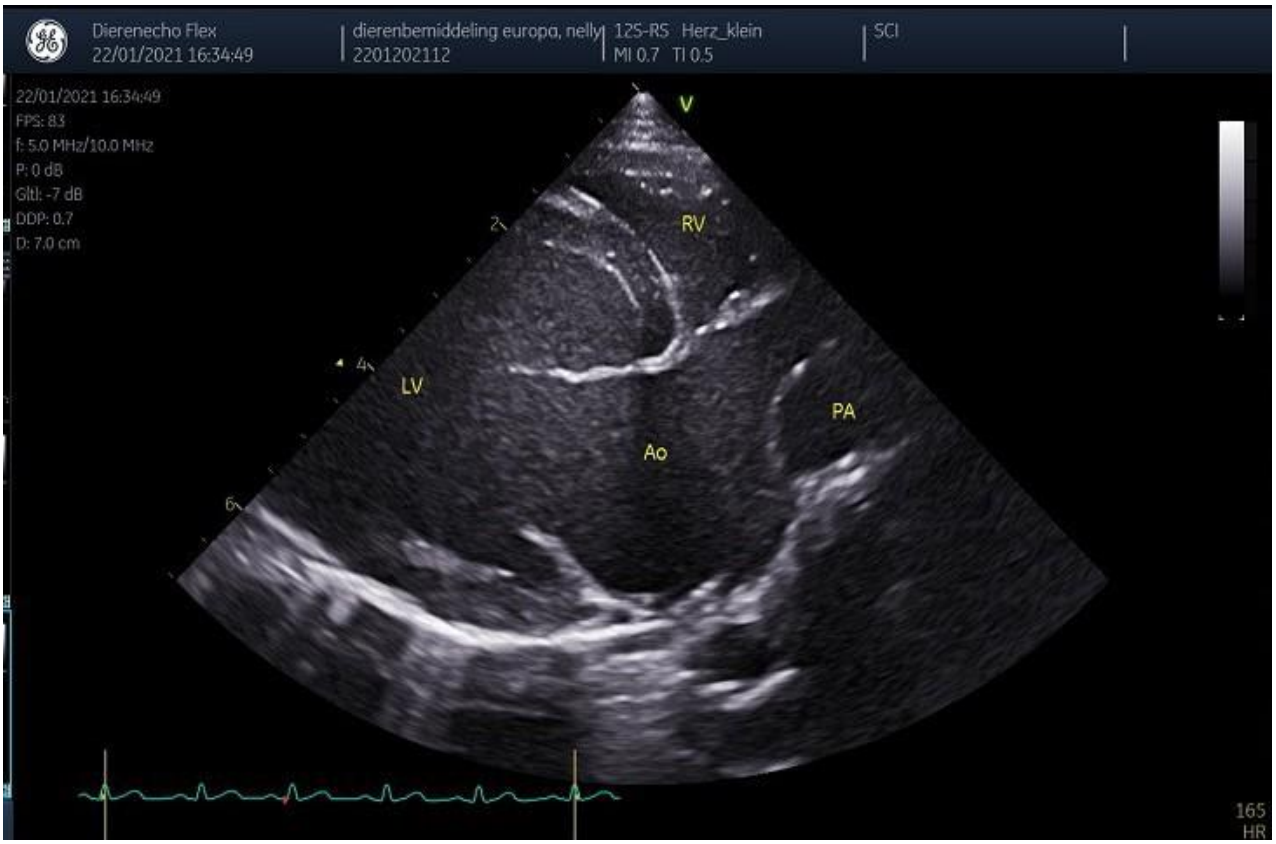
Echocardiogram



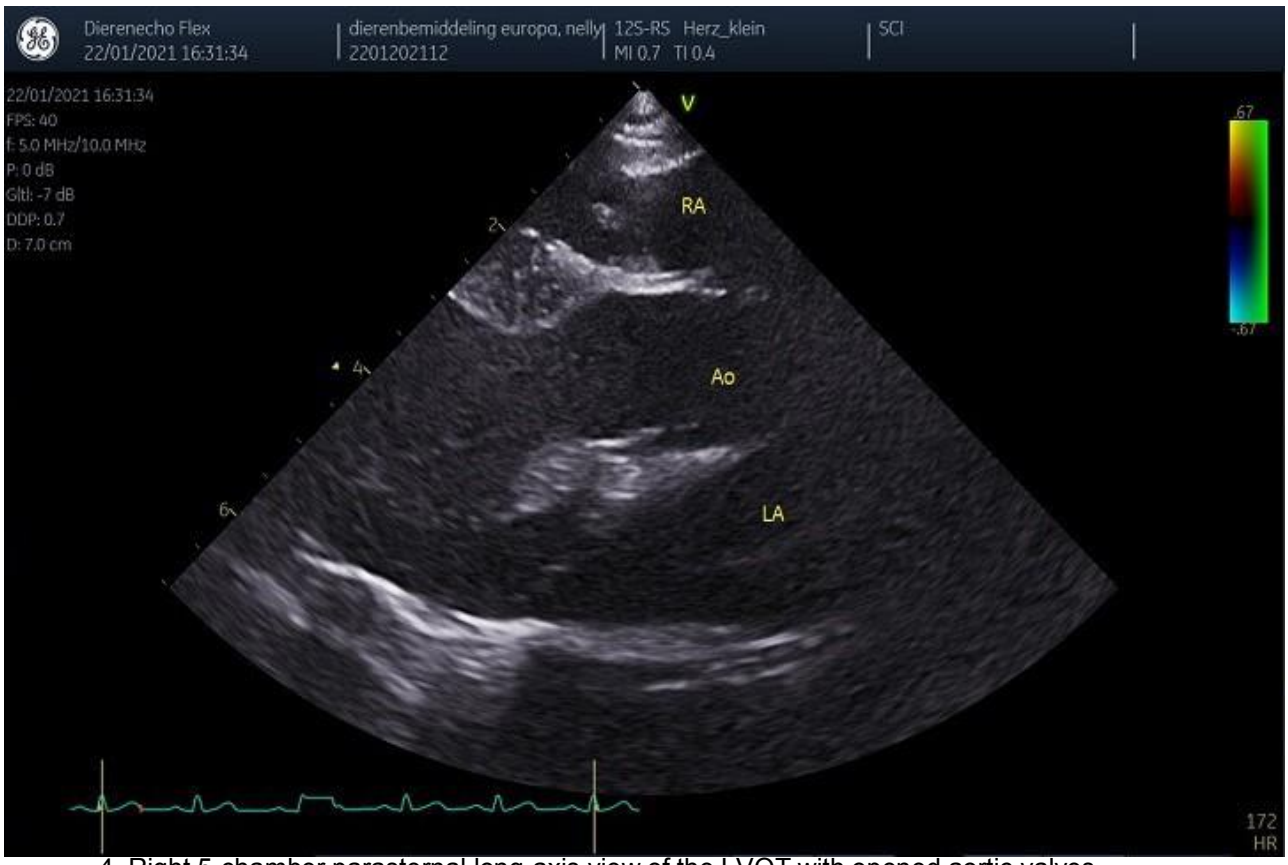
1. Right 4-chamber parasternal long-axis view. Atrioventricular valves are open and appear normal. The left ventricle and left atrium appear volume overloaded (eccentric hypertrophy).



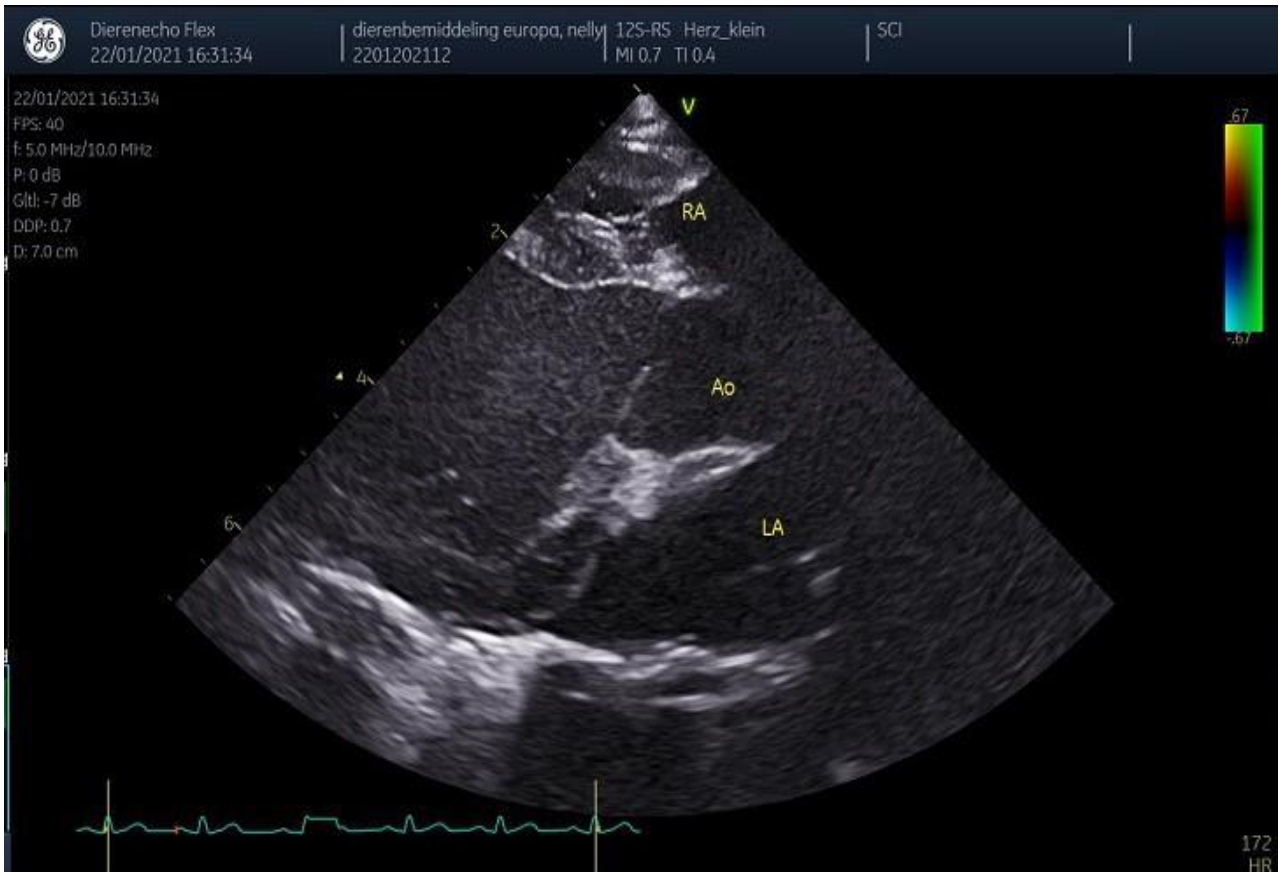
2. Right 4-chamber parasternal long-axis view. Atrioventricular valves are closed and appear normal. The left ventricle appears volume overloaded (eccentric hypertrophy). In this view the left atrium appears widened towards the right side but not very large in comparison to the left ventricle.



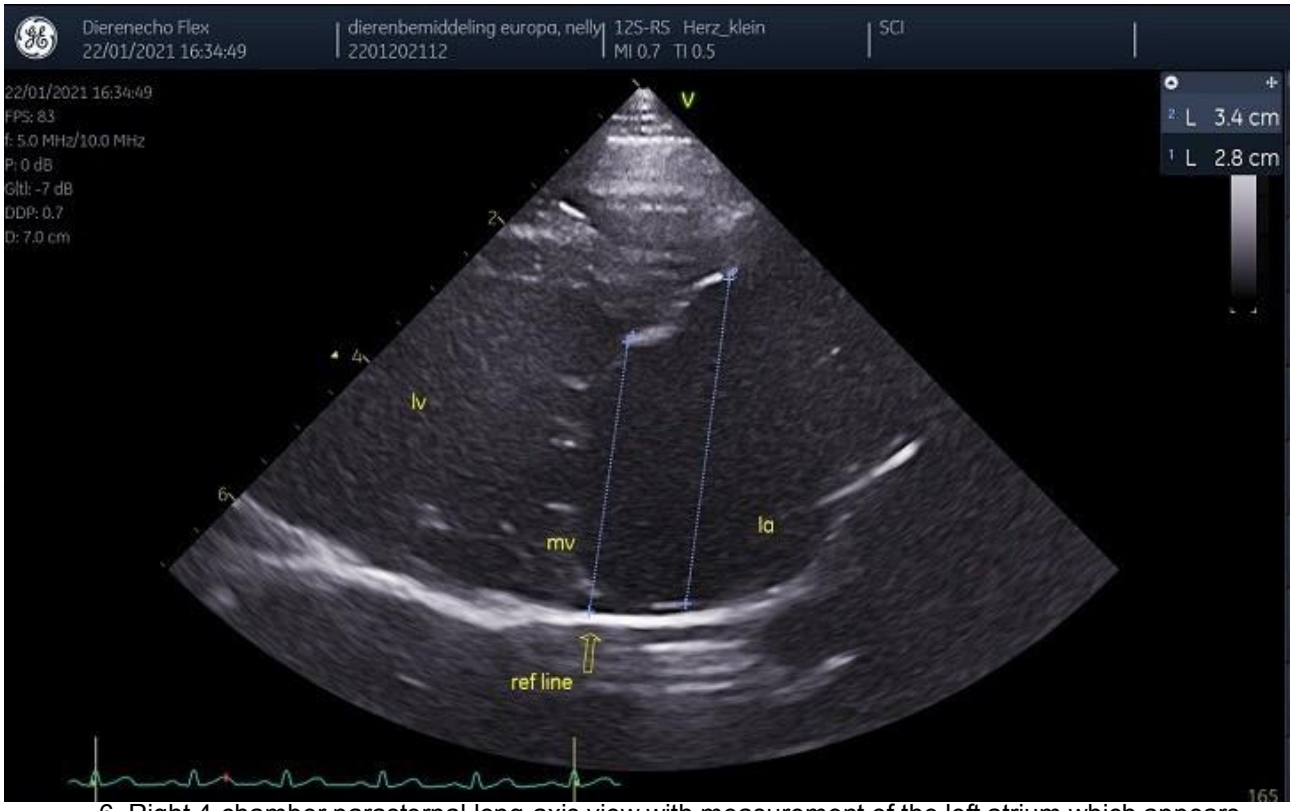
3. Right 4-chamber parasternal long-axis in mild oblique view with opened AV-valves. Valves appear normal.The left ventricle appears volume overloaded (eccentric hypertrophy). Notice the prominent pulmonary artery (PA) and enlargement of the left atrium towards the right side.



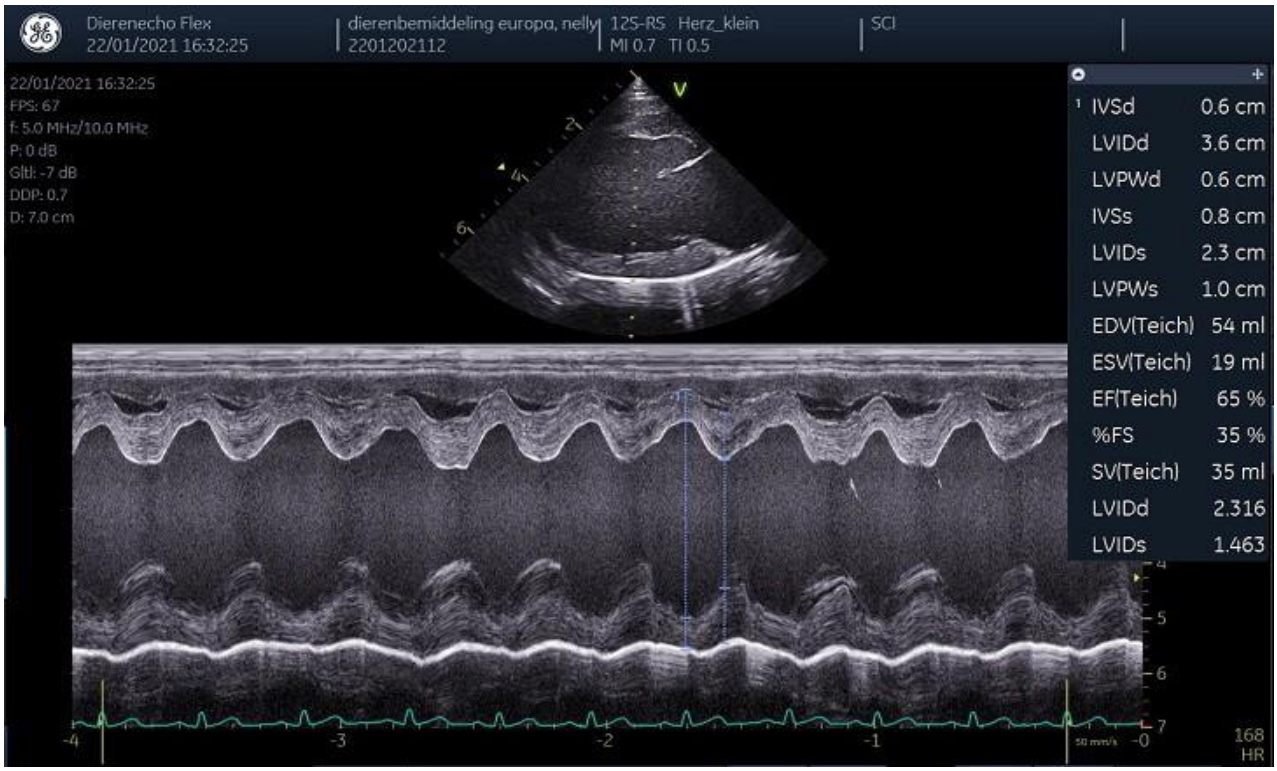
4. Right 5-chamber parasternal long-axis view of the LVOT with opened aortic valves.



5. Right 5-chamber parasternal long-axis view of the LVOT with closed aortic valves.

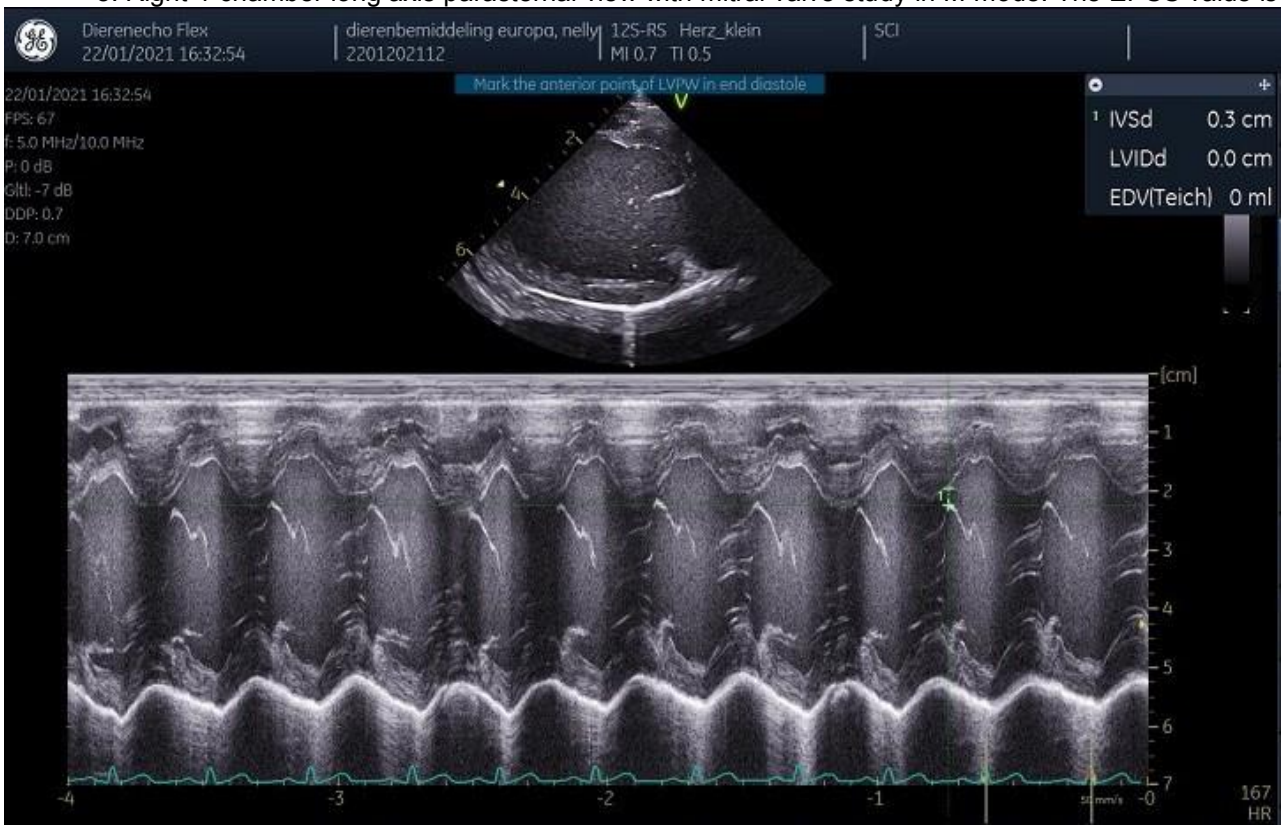


6. Right 4-chamber parasternal long-axis view with measurement of the left atrium which appears widened (reference range up to 2,65cm).

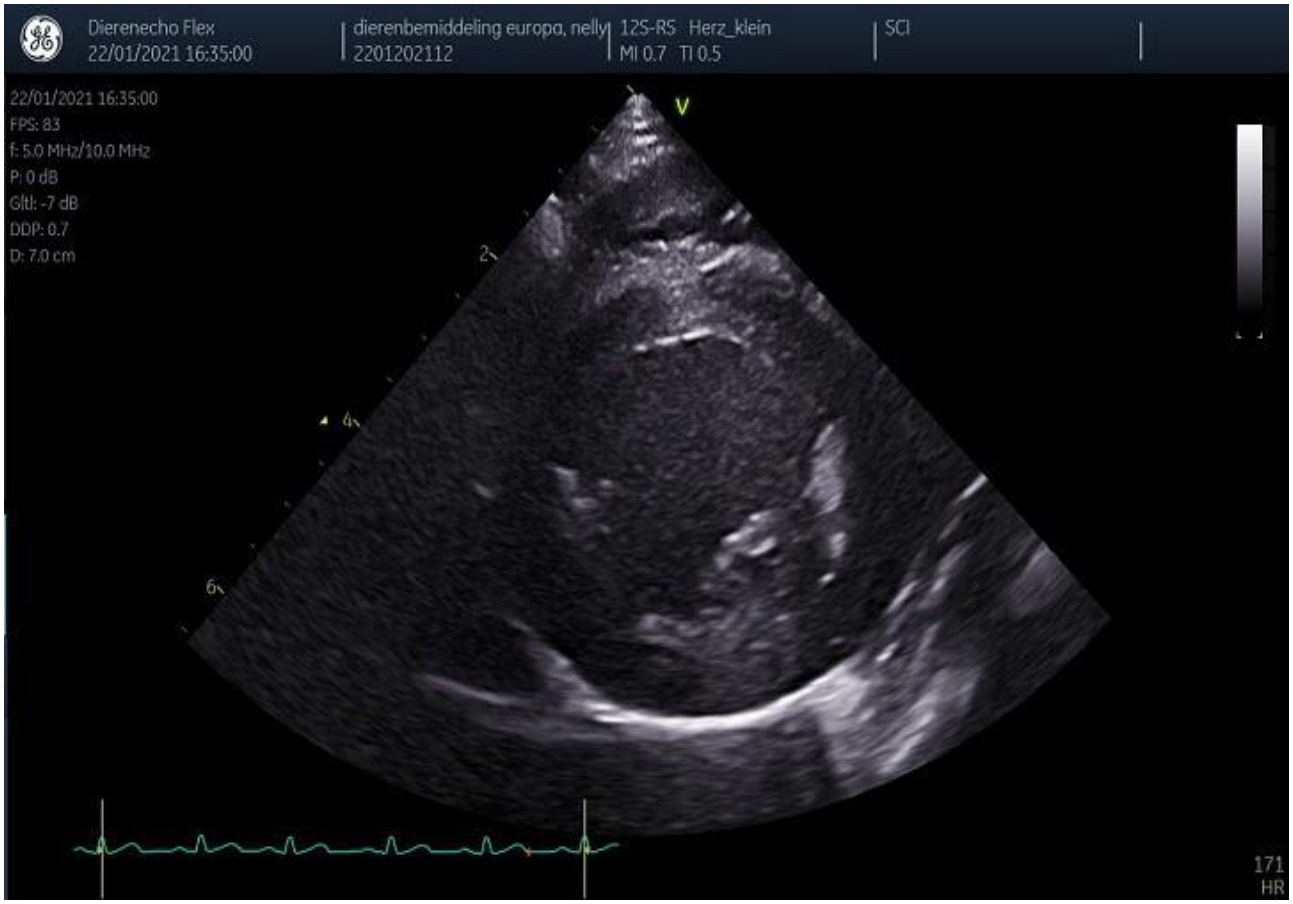


7. Right 4-chamber long axis parasternal view with left ventricular study in M-mode. Volume overload can be appreciated.

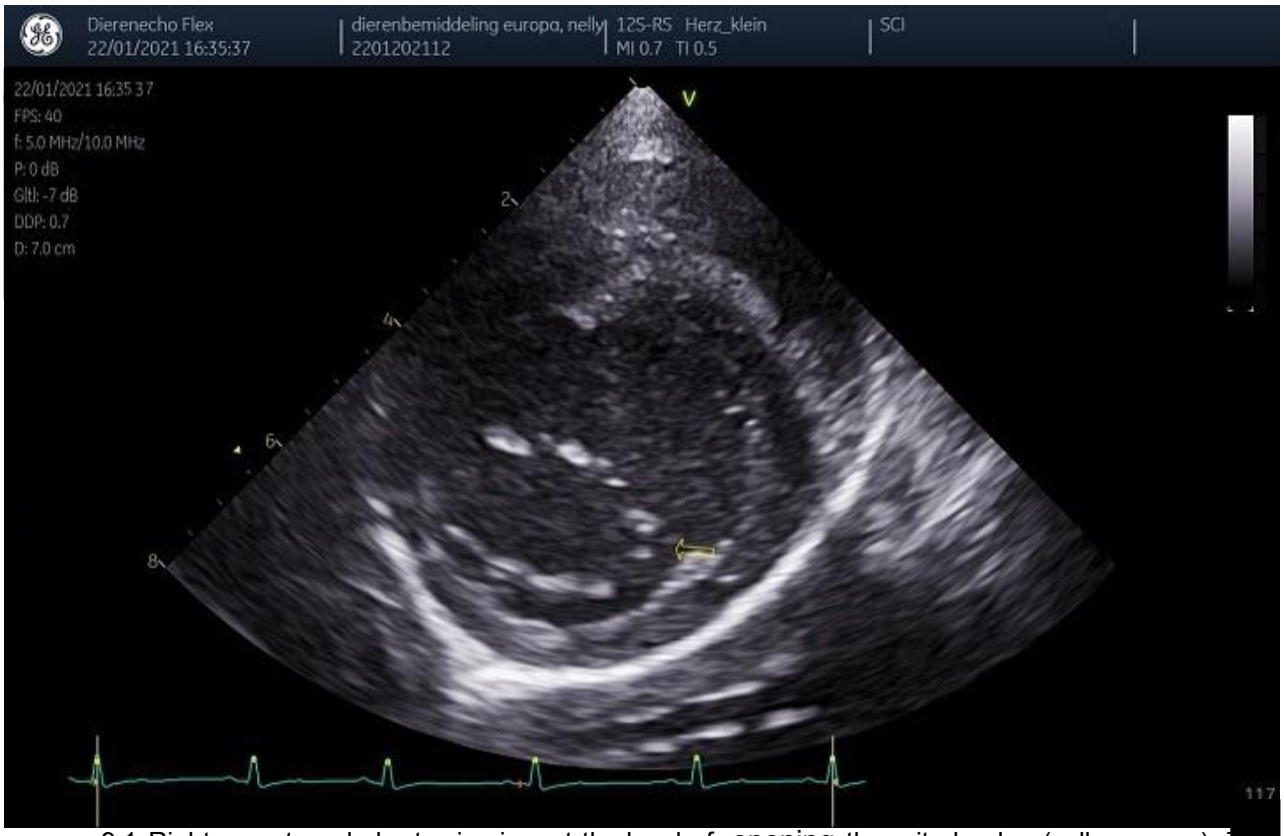
8. Right 4-chamber long axis parasternal view with mitral valve study in M-mode. The EPSS value is



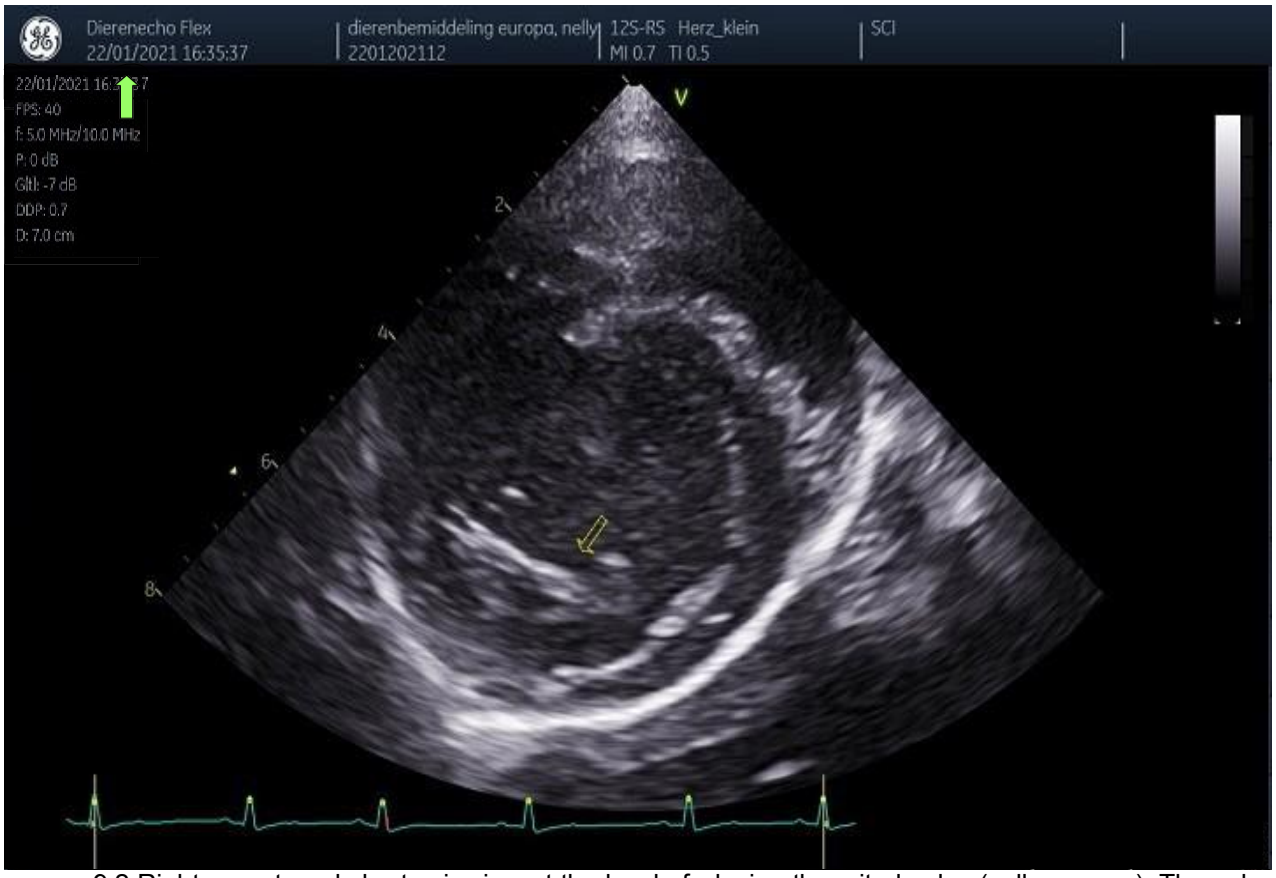
normal.



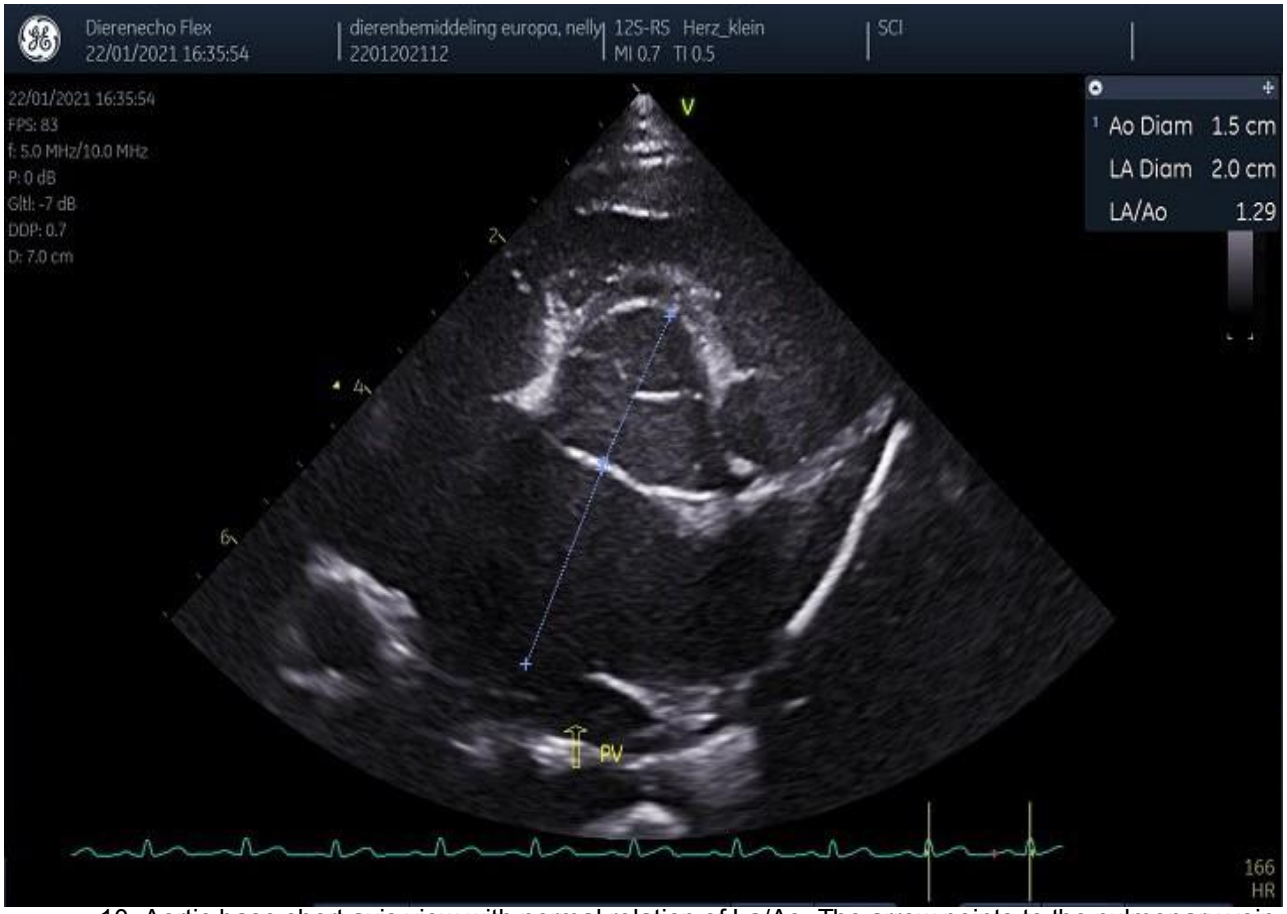
9. Right parasternal short axis view at the level of the papillary muscle in diastole.



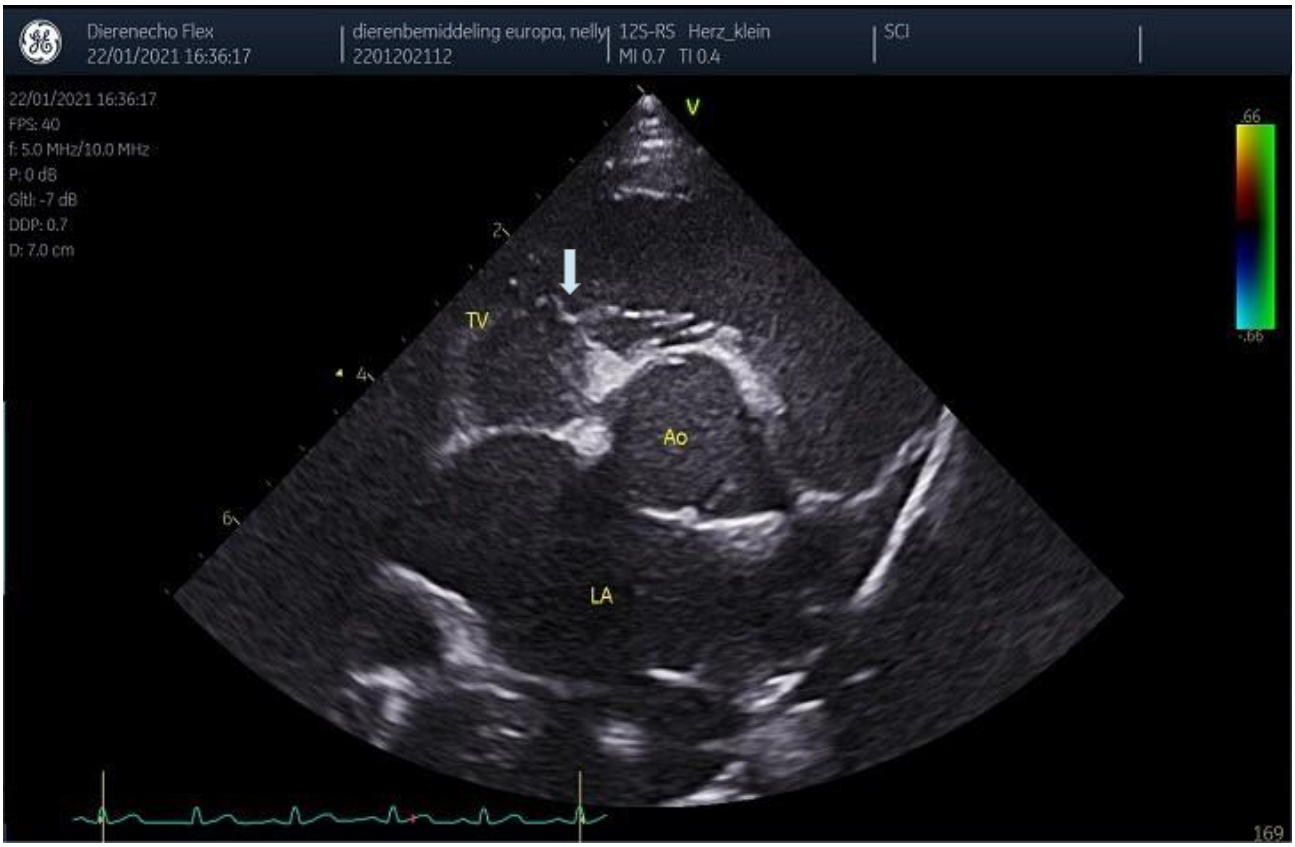
9.1 Right parasternal short axis view at the level of opening the mitral valve (yellow arrow). The valve appears normal.



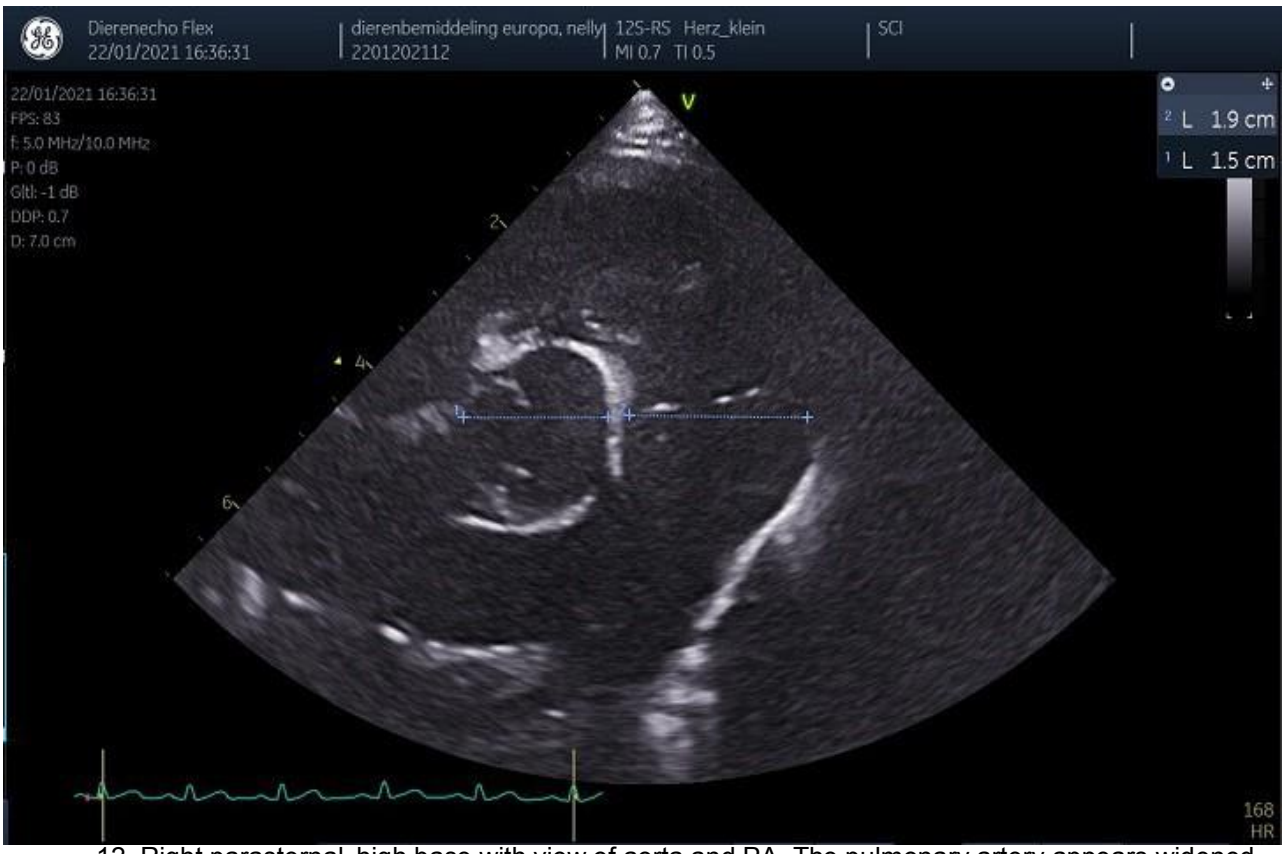
9.2 Right parasternal short axis view at the level of closing the mitral valve (yellow arrow). The valve appears normal.



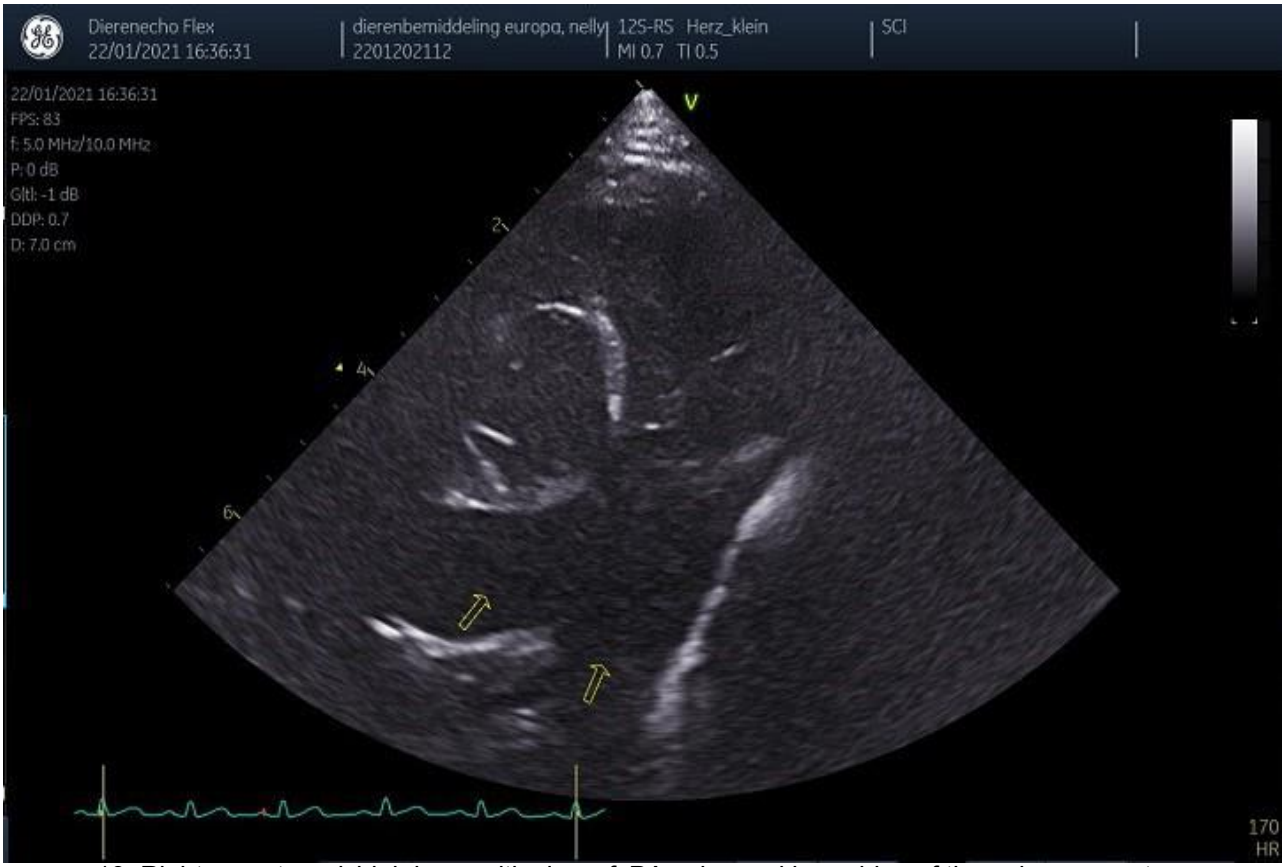
10. Aortic base short axis view with normal relation of La/Ao. The arrow points to the pulmonary vein, which makes the left atrium appear larger than it really is.



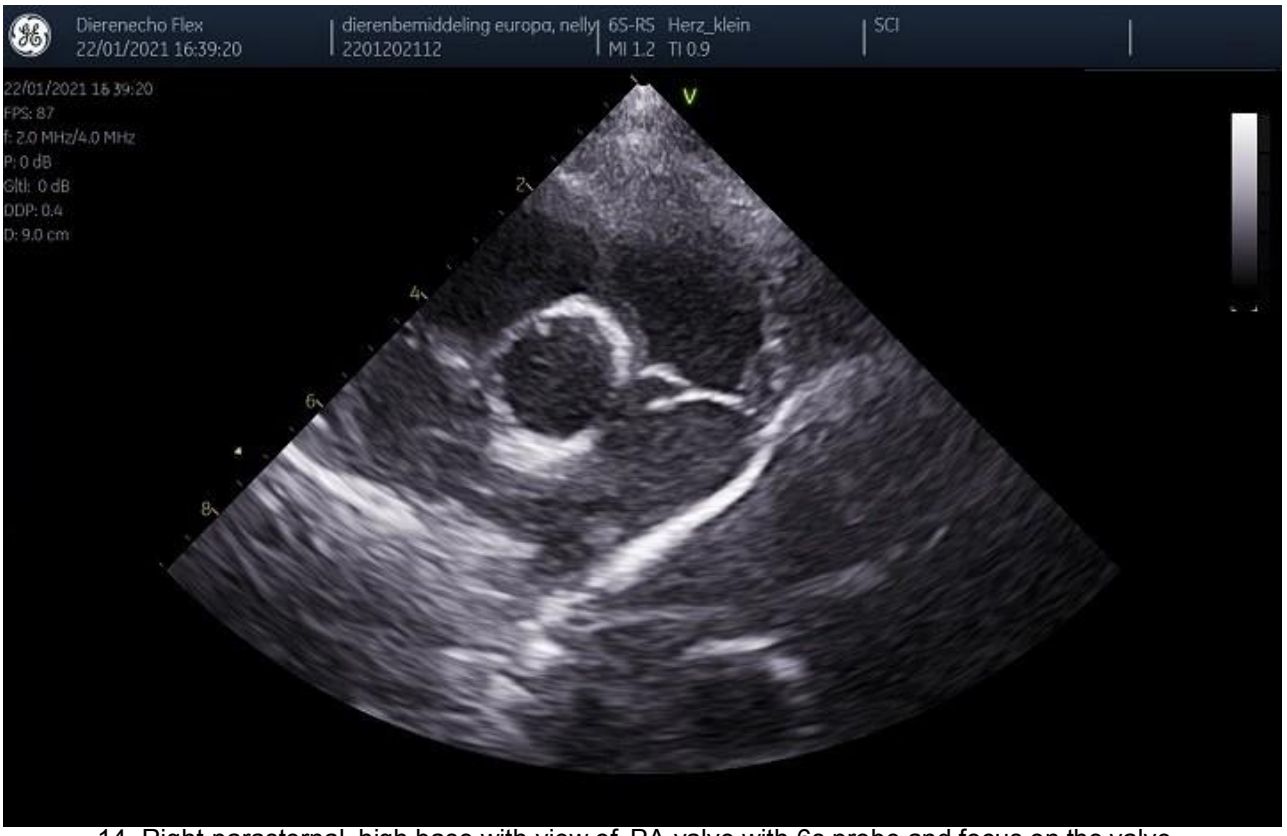
11. Right parasternal high base with view of aorta and normal tricuspid valve (arrow).



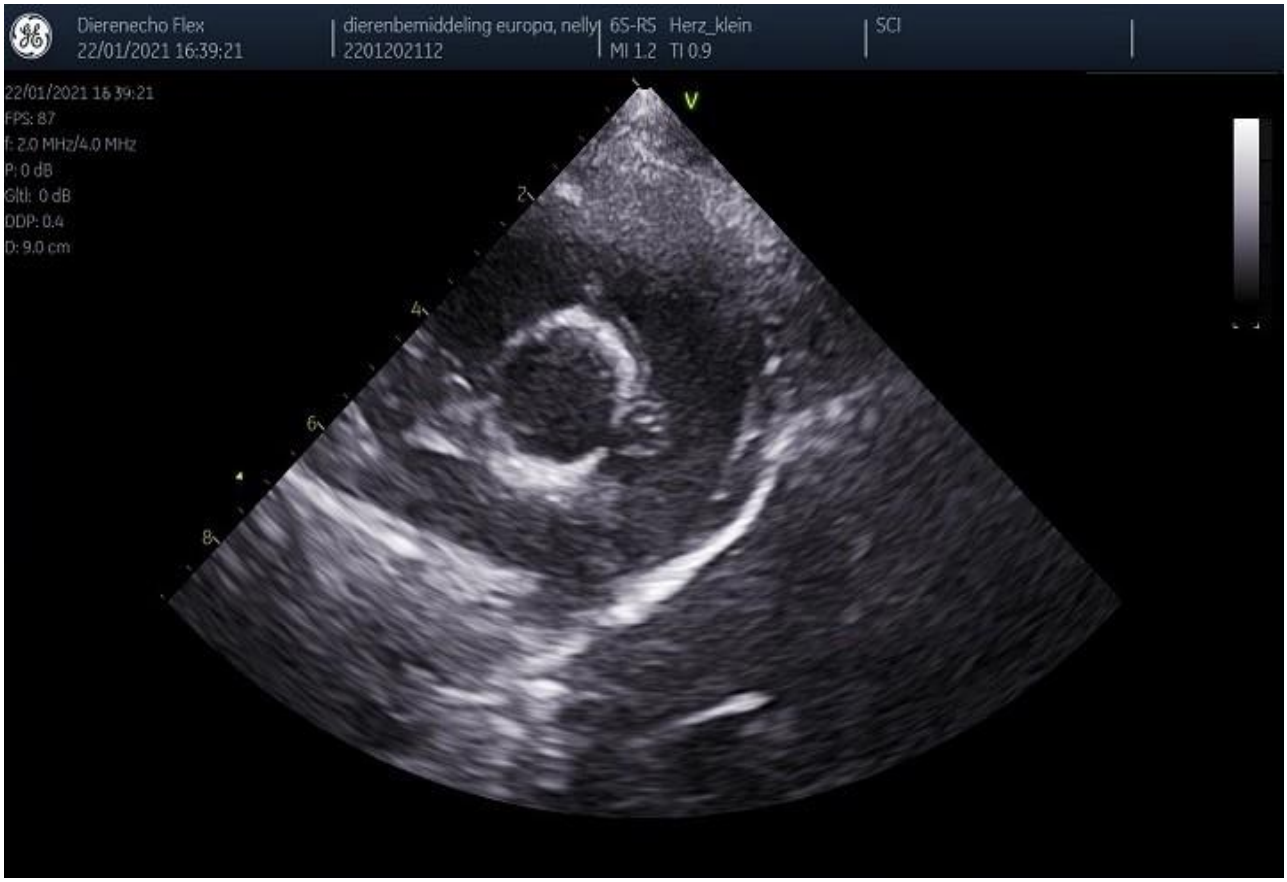
12. Right parasternal high base with view of aorta and PA. The pulmonary artery appears widened (Mpa:Ao 1,26).



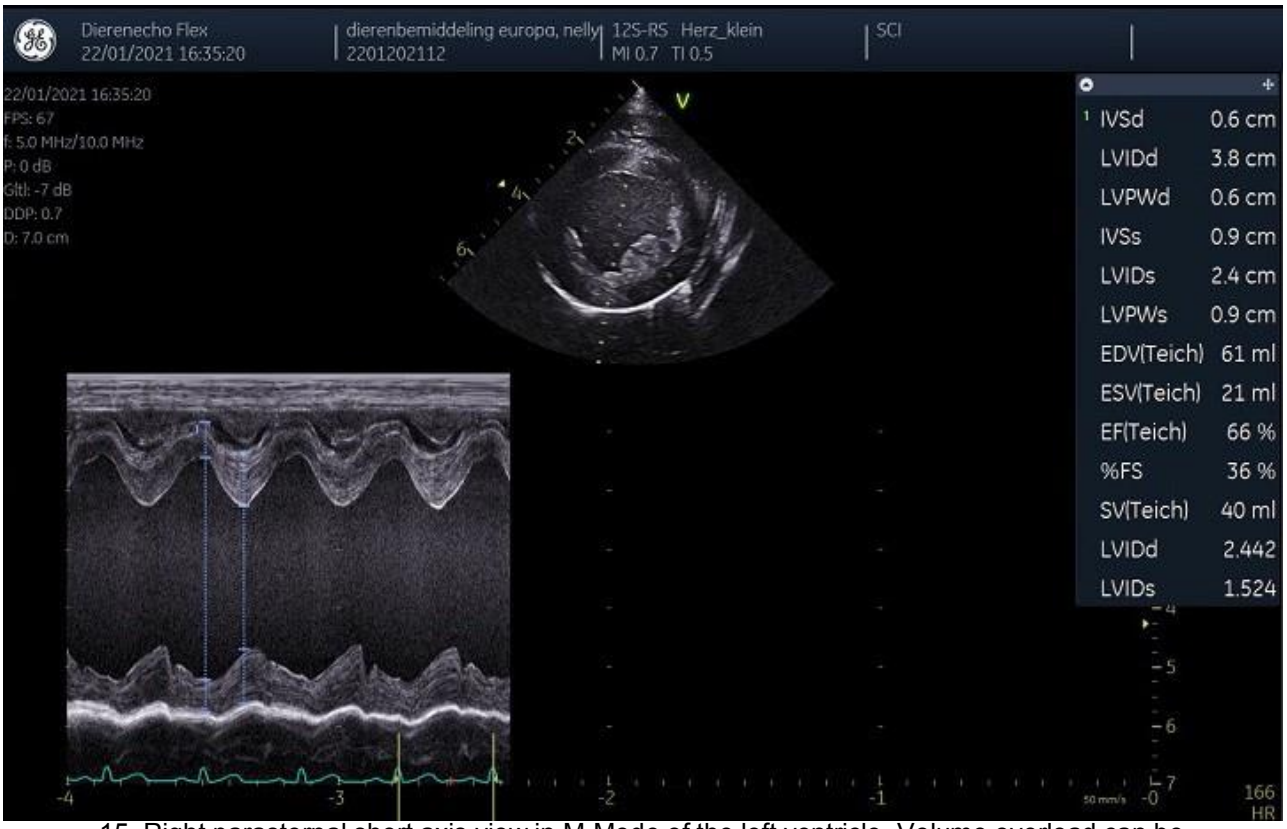
13. Right parasternal high base with view of PA valve and branching of the pulmonary artery. Notice the widening of the pulmonary artery and its branches (yellow arrows).



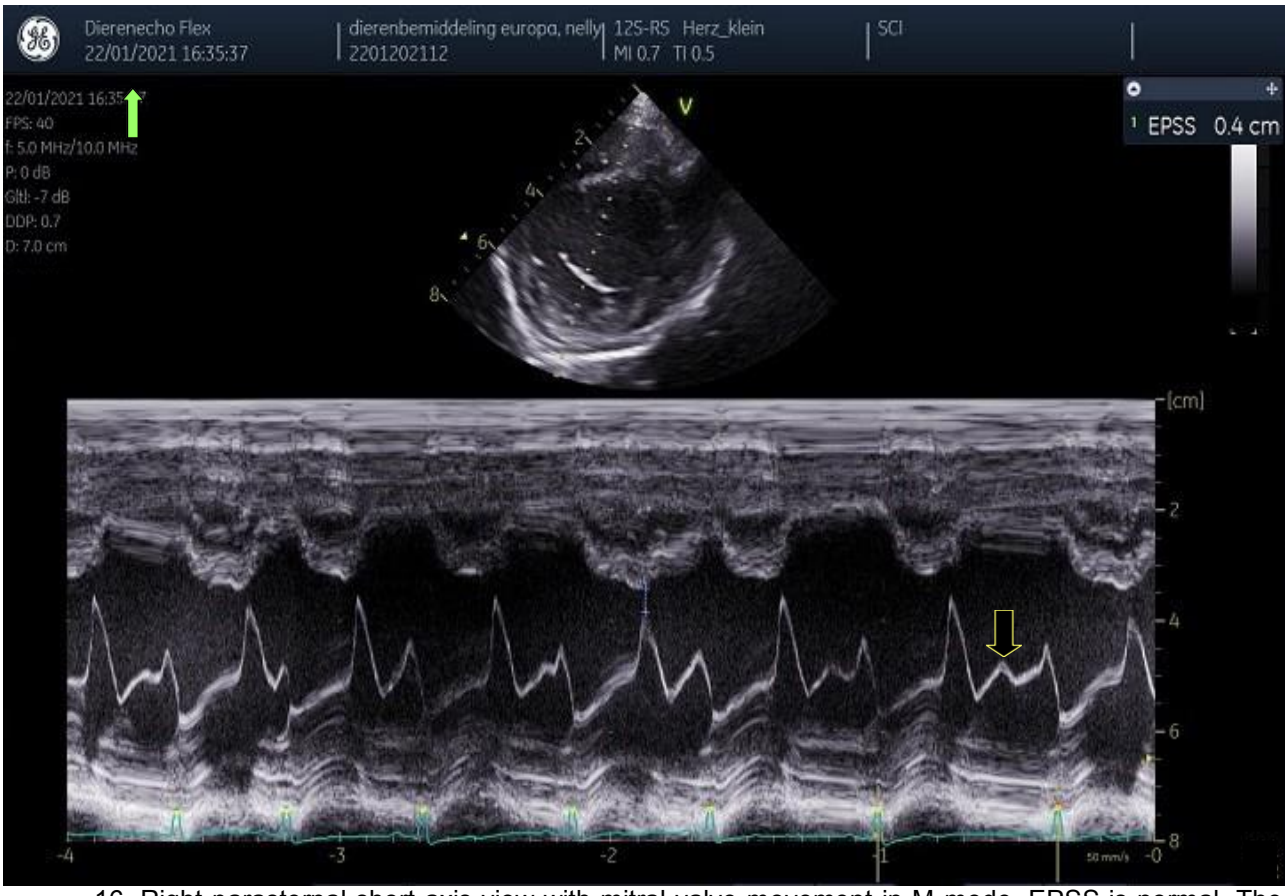
14. Right parasternal high base with view of PA valve with 6s probe and focus on the valve (closed). The valve is of normal morphology.



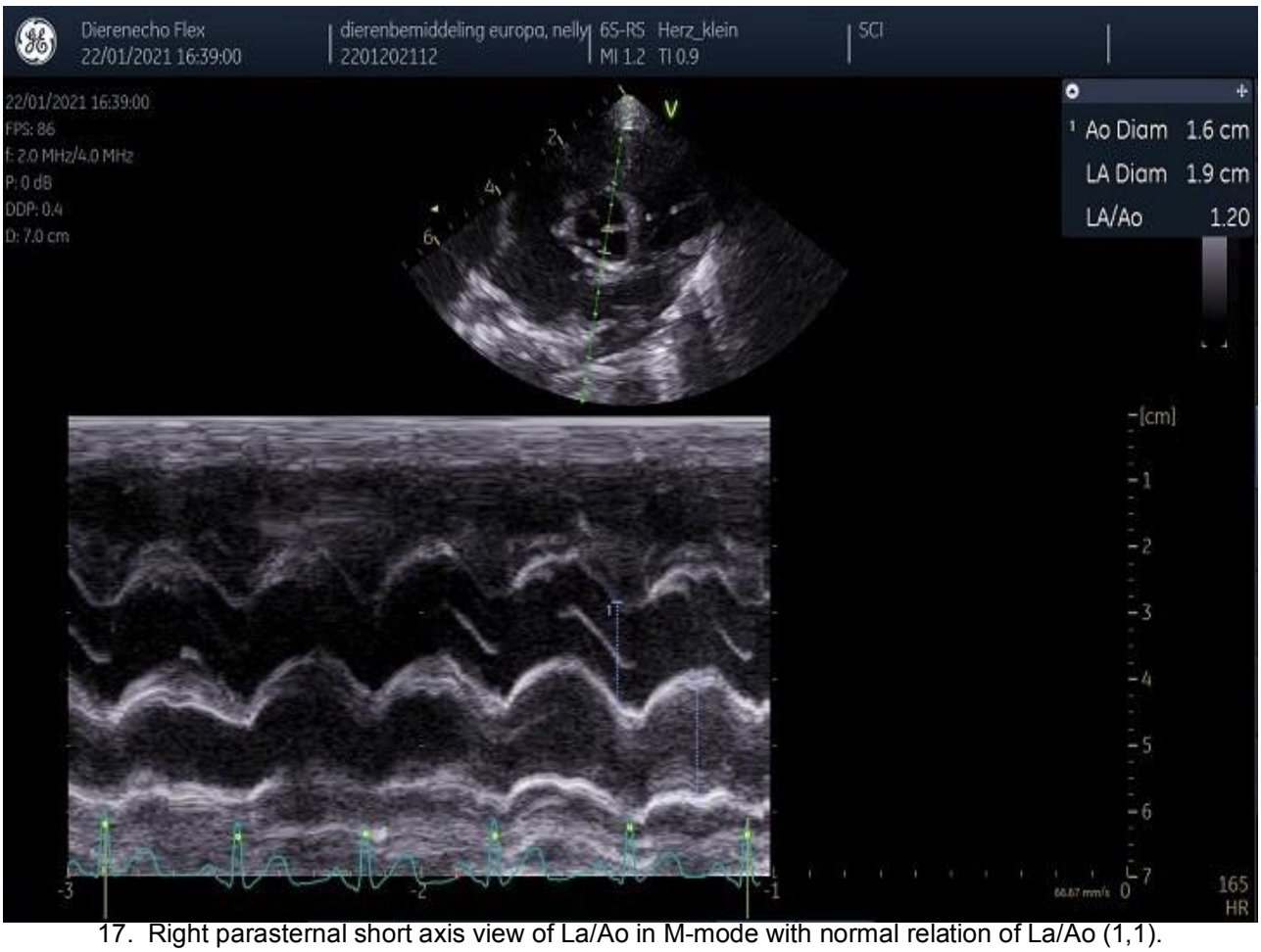
14.2 Right parasternal high base with view of PA valve with 6s probe and focus on the valve (open). The valve is of normal morphology.



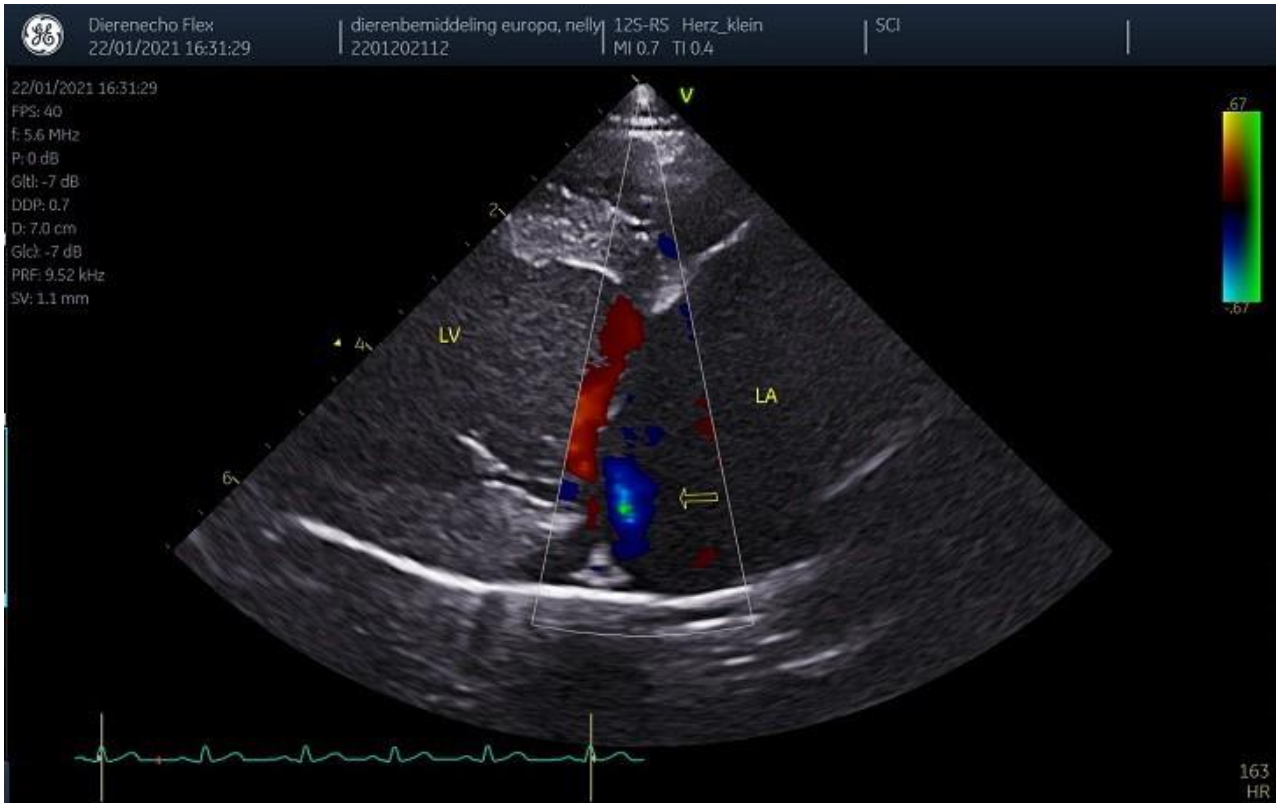
15. Right parasternal short axis view in M-Mode of the left ventricle. Volume overload can be appreciated.



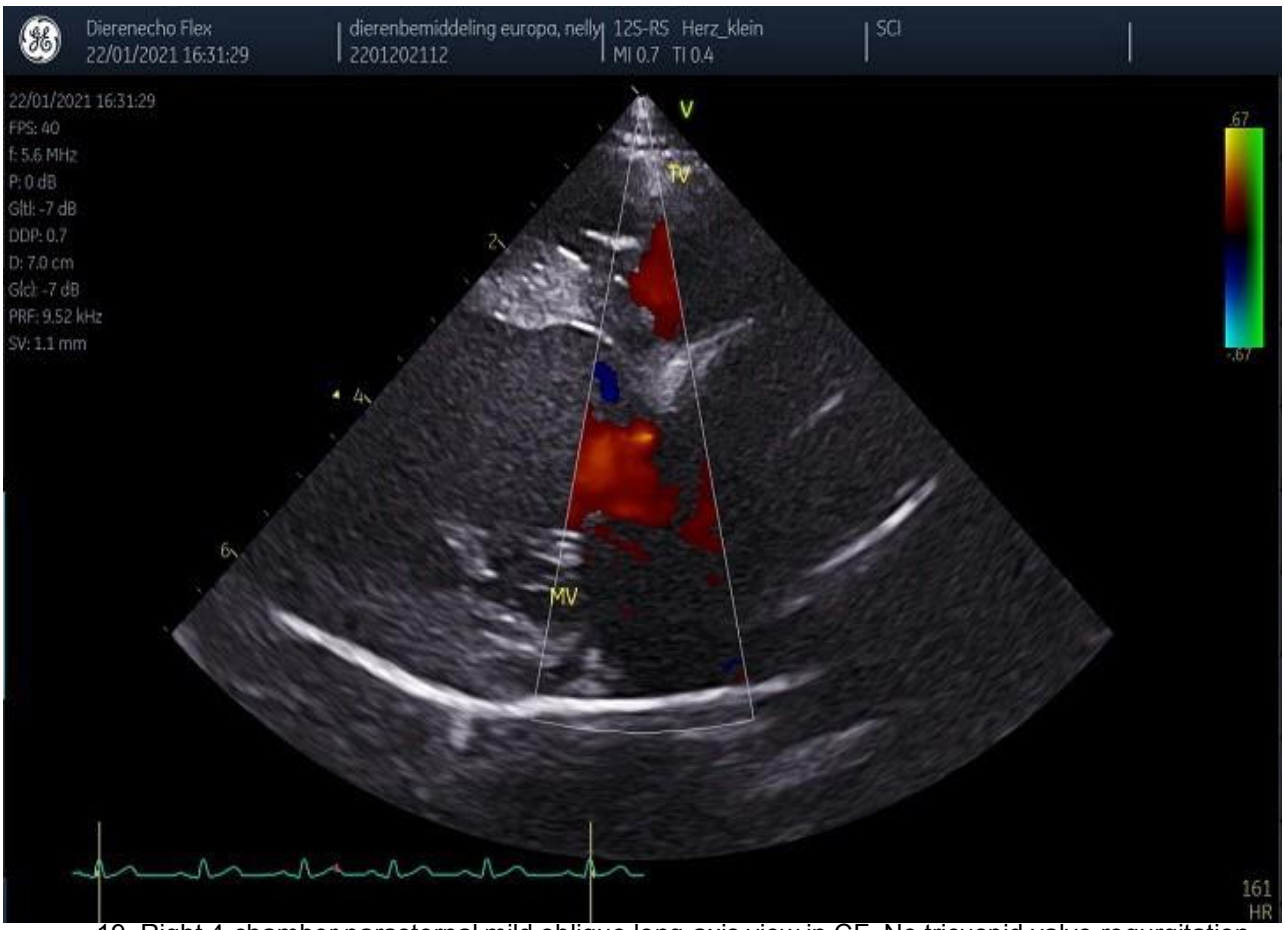
16. Right parasternal short axis view with mitral valve movement in M-mode. EPSS is normal. The little bump (yellow arrow) seen between the E- and A-wave is an unspecific finding and can most likely be attributed to continued pulmonary vein flow through the left atrium (20).



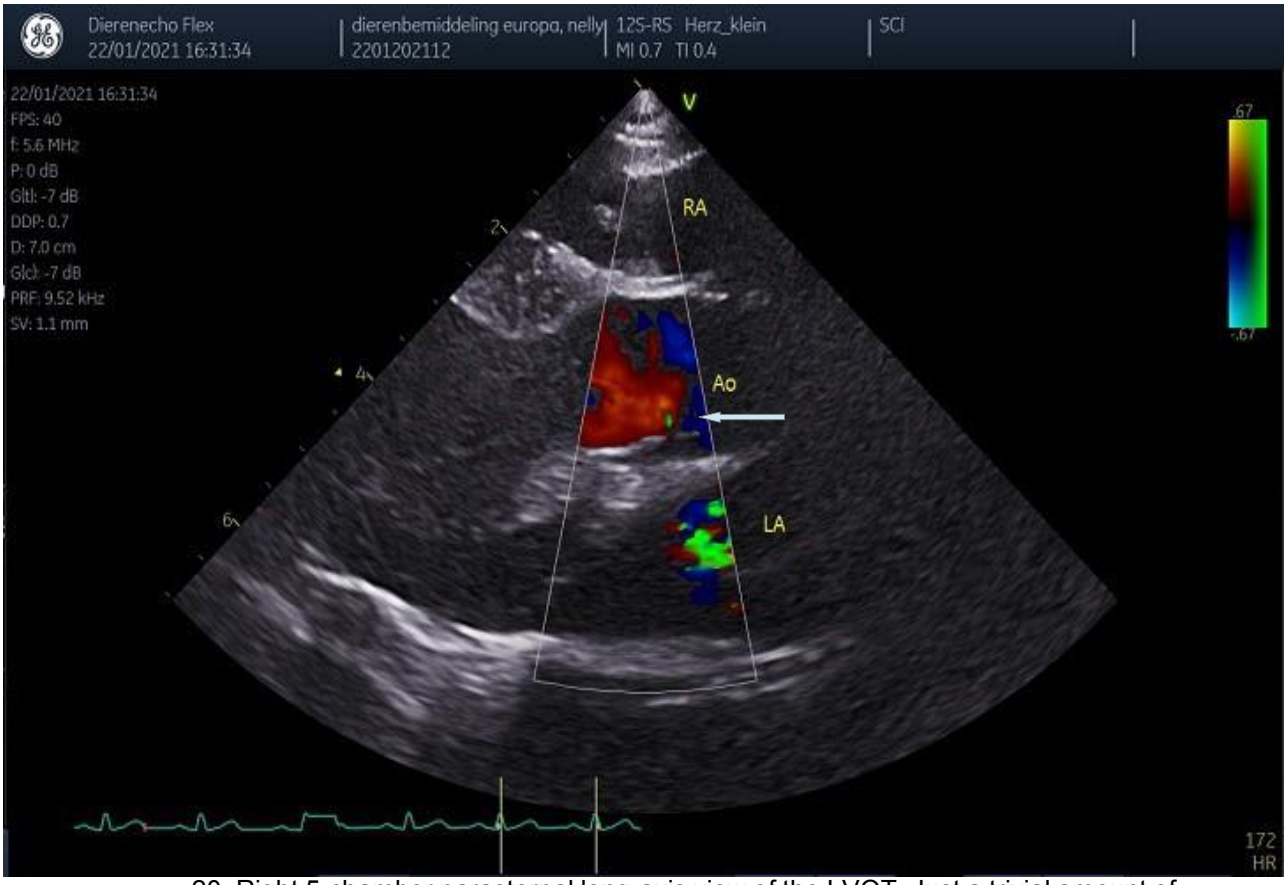
17. Right parasternal short axis view of La/Ao in M-mode with normal relation of La/Ao (1,1).



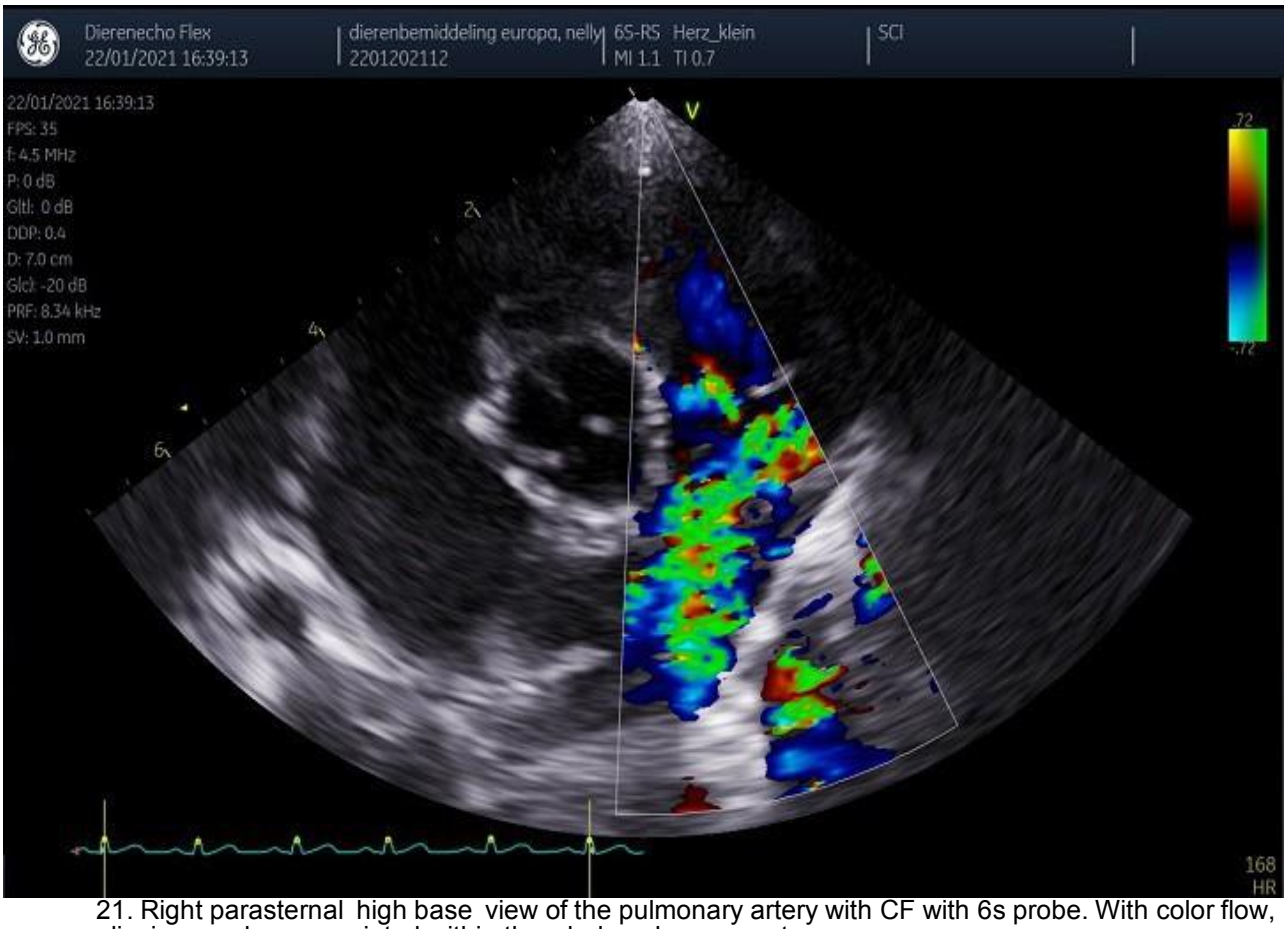
18. Right 4-chamber parasternal mild oblique long-axis view of the mitral valve in CF. Trivial mitral valve regurgitation can be detected (arrow). No sign of VSD present.



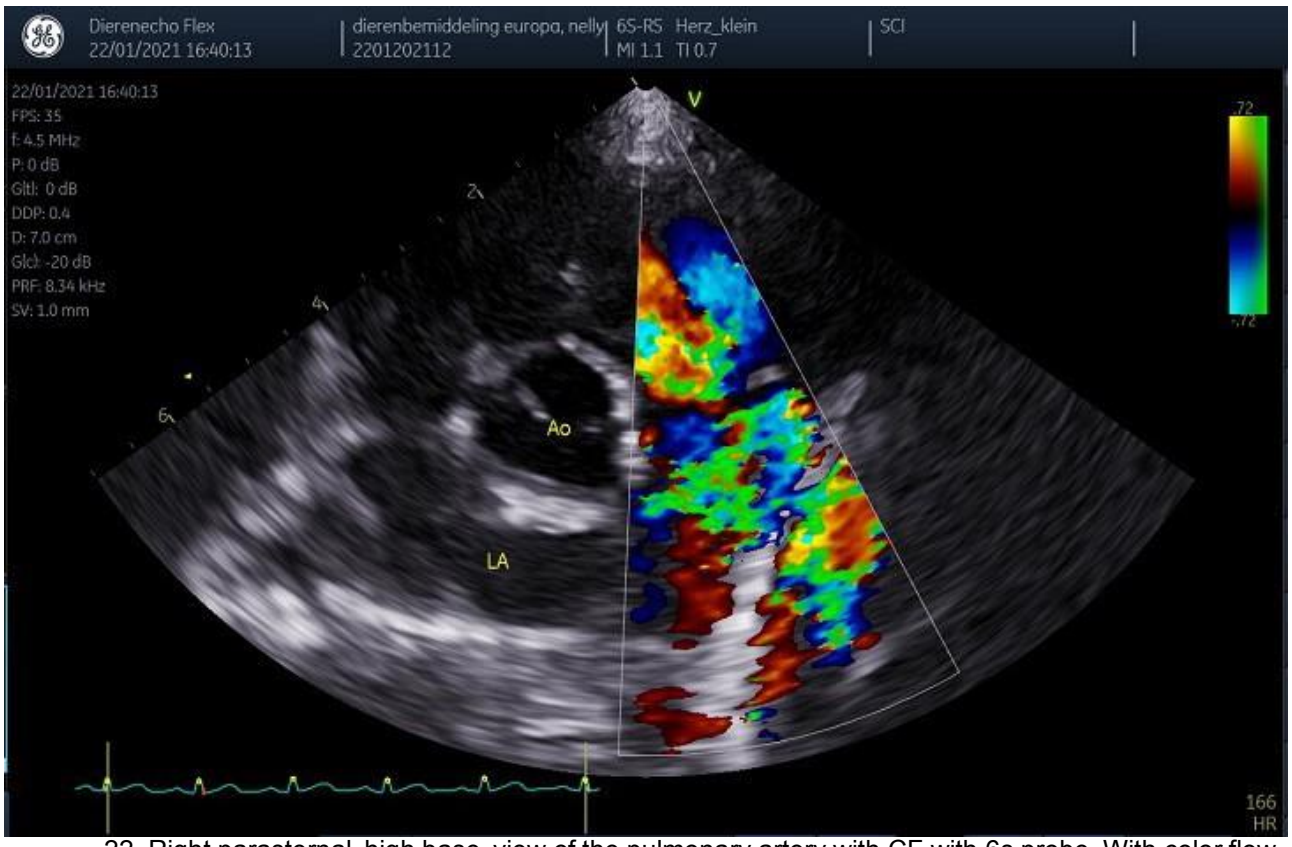
19. Right 4-chamber parasternal mild oblique long-axis view in CF. No tricuspid valve regurgitation can be detected . No sign of VSD present.



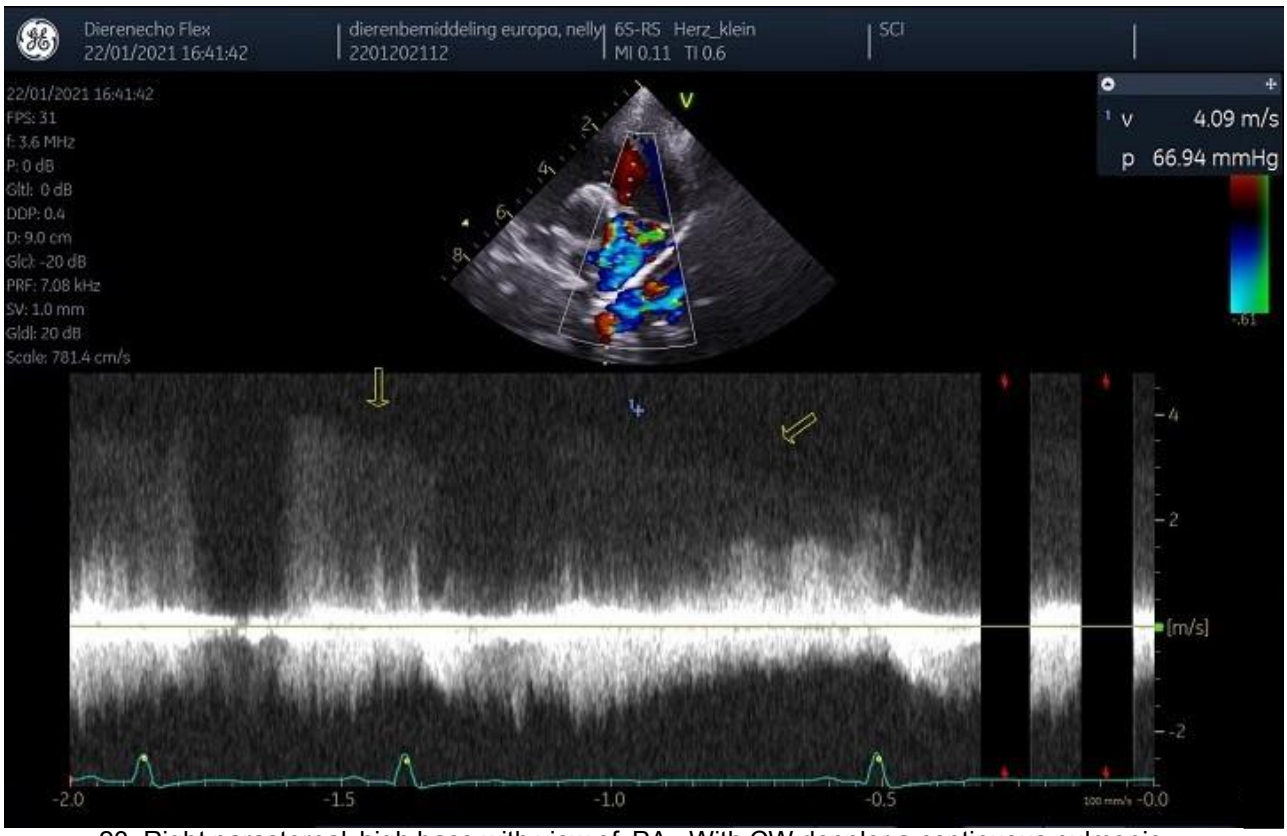
20. Right 5-chamber parasternal long-axis view of the LVOT. Just a trivial amount of regurgitation can be detected at the level of the aortic valve (arrow).



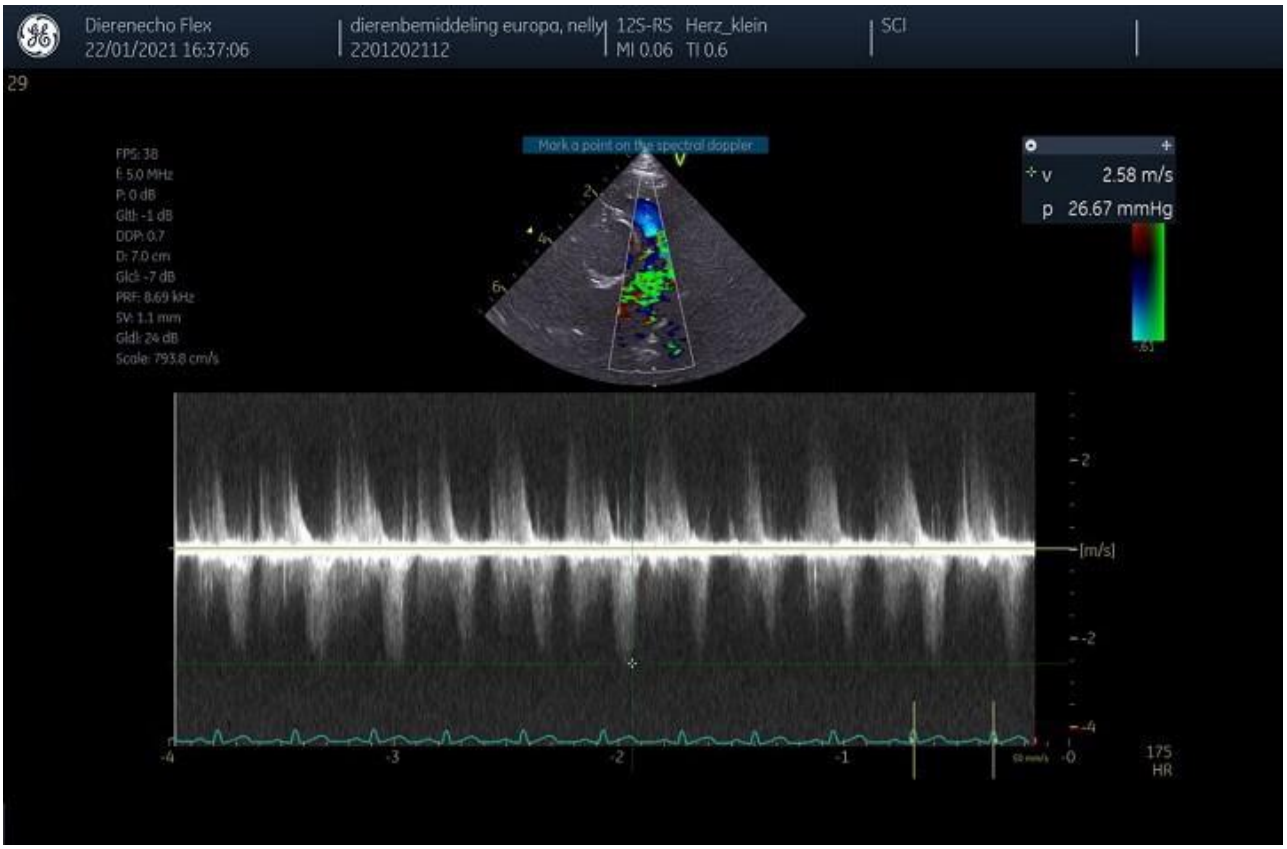
21. Right parasternal high base view of the pulmonary artery with CF with 6s probe. With color flow, aliasing can be appreciated within the whole pulmonary artery.



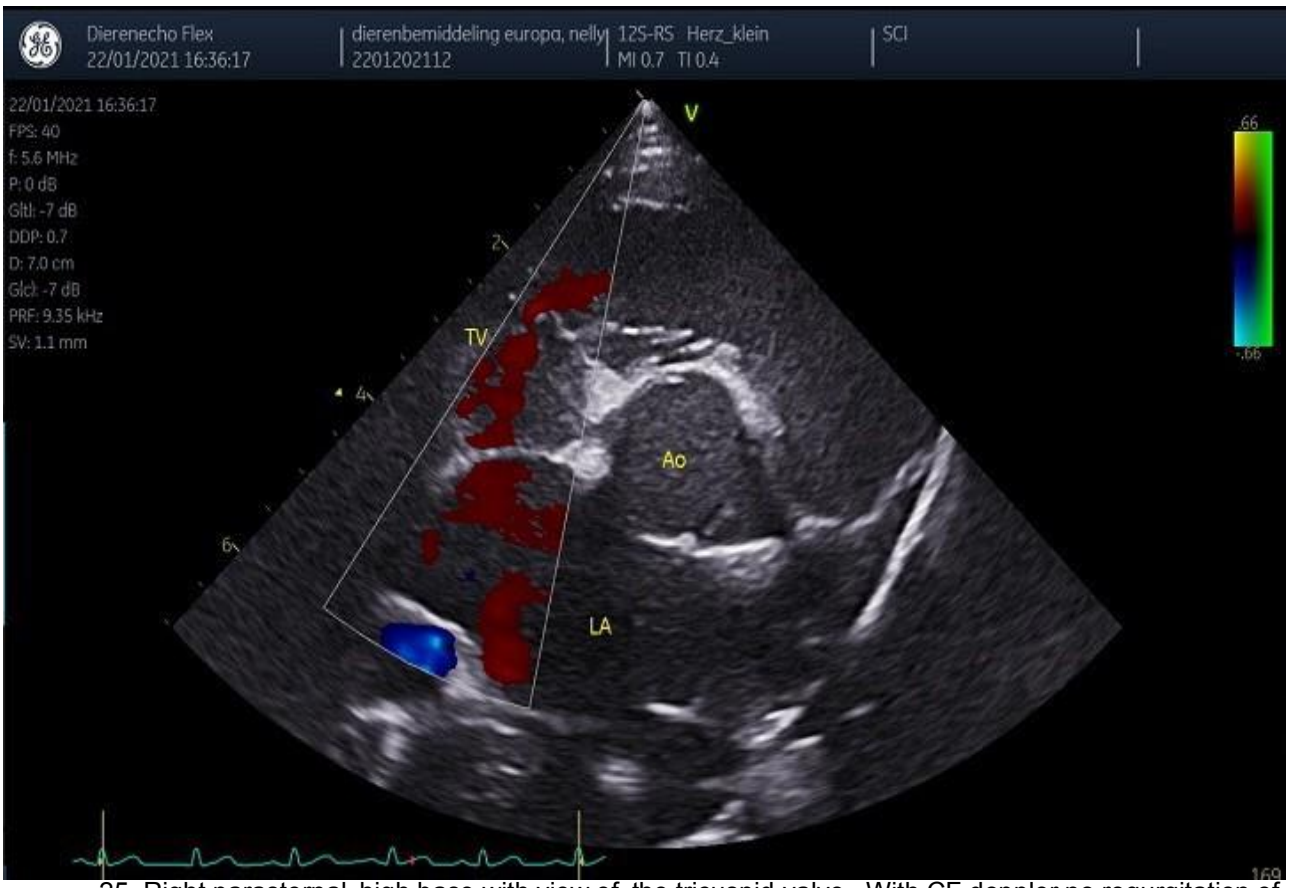
22. Right parasternal high base view of the pulmonary artery with CF with 6s probe. With color flow aliasing can be appreciated within the whole pulmonary artery. Severe pulmonic regurgitation is present.



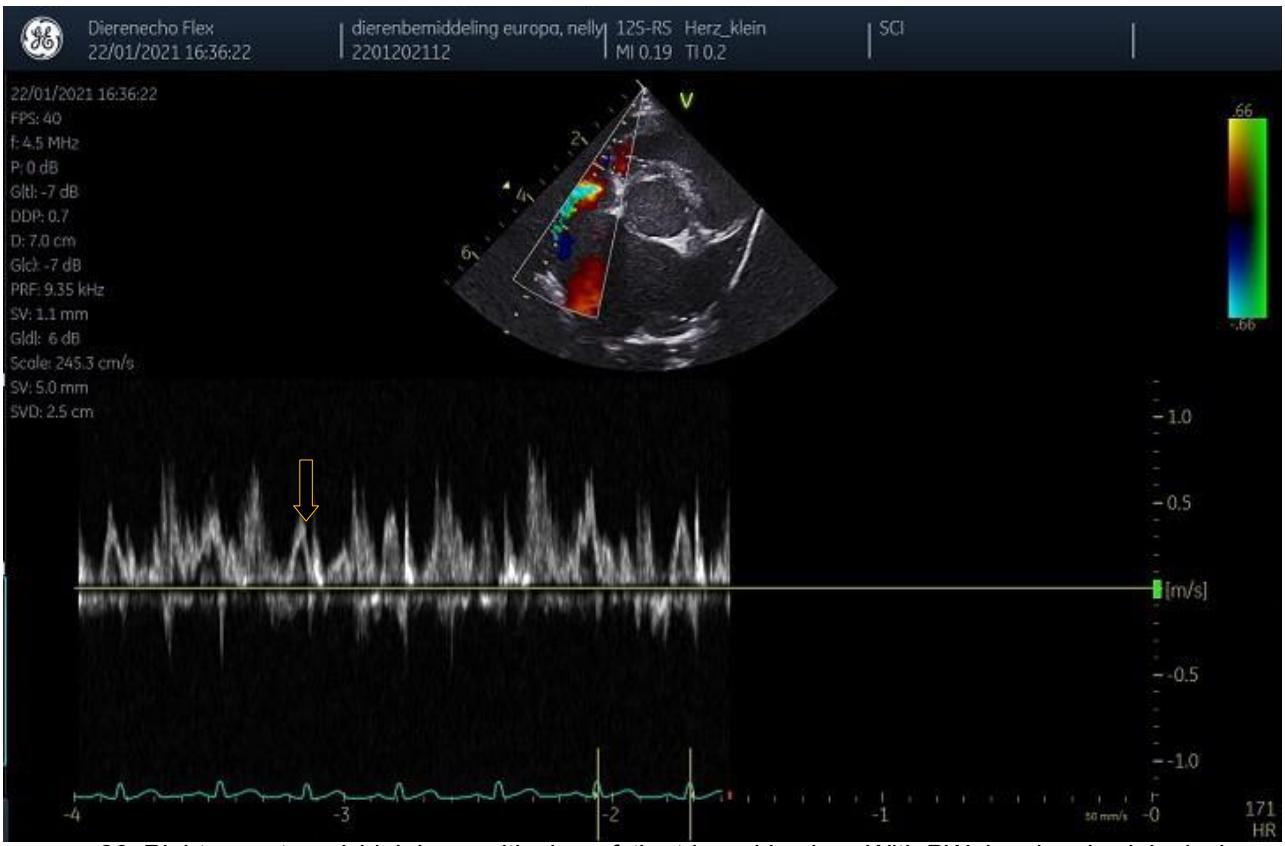
23. Right parasternal high base with view of PA . With CW doppler a continuous pulmonic regurgitation jet with high flow velocity of 4,09m/sec can be measured.



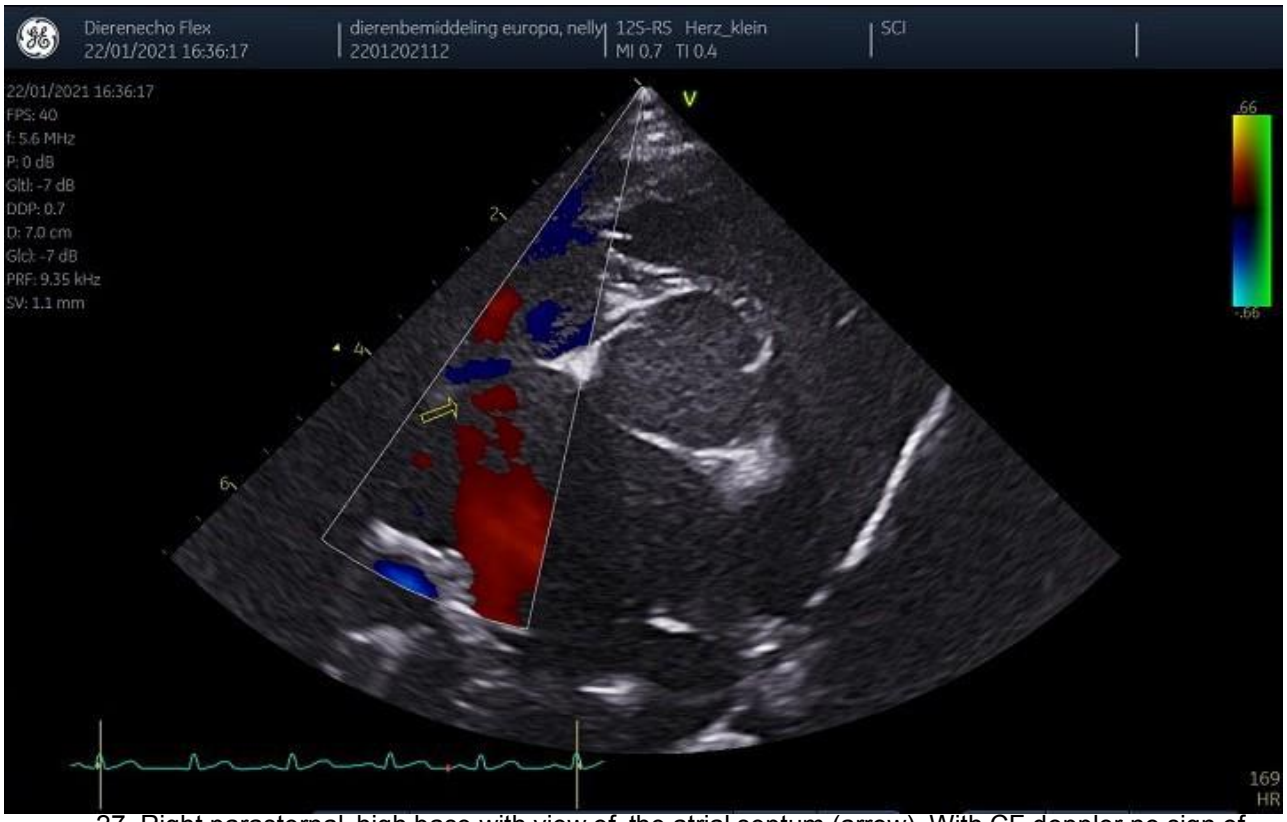
24. Right parasternal high base with view of PA . With CW doppler a maximal pulmonic velocity of 2,58m/sec can be measured.



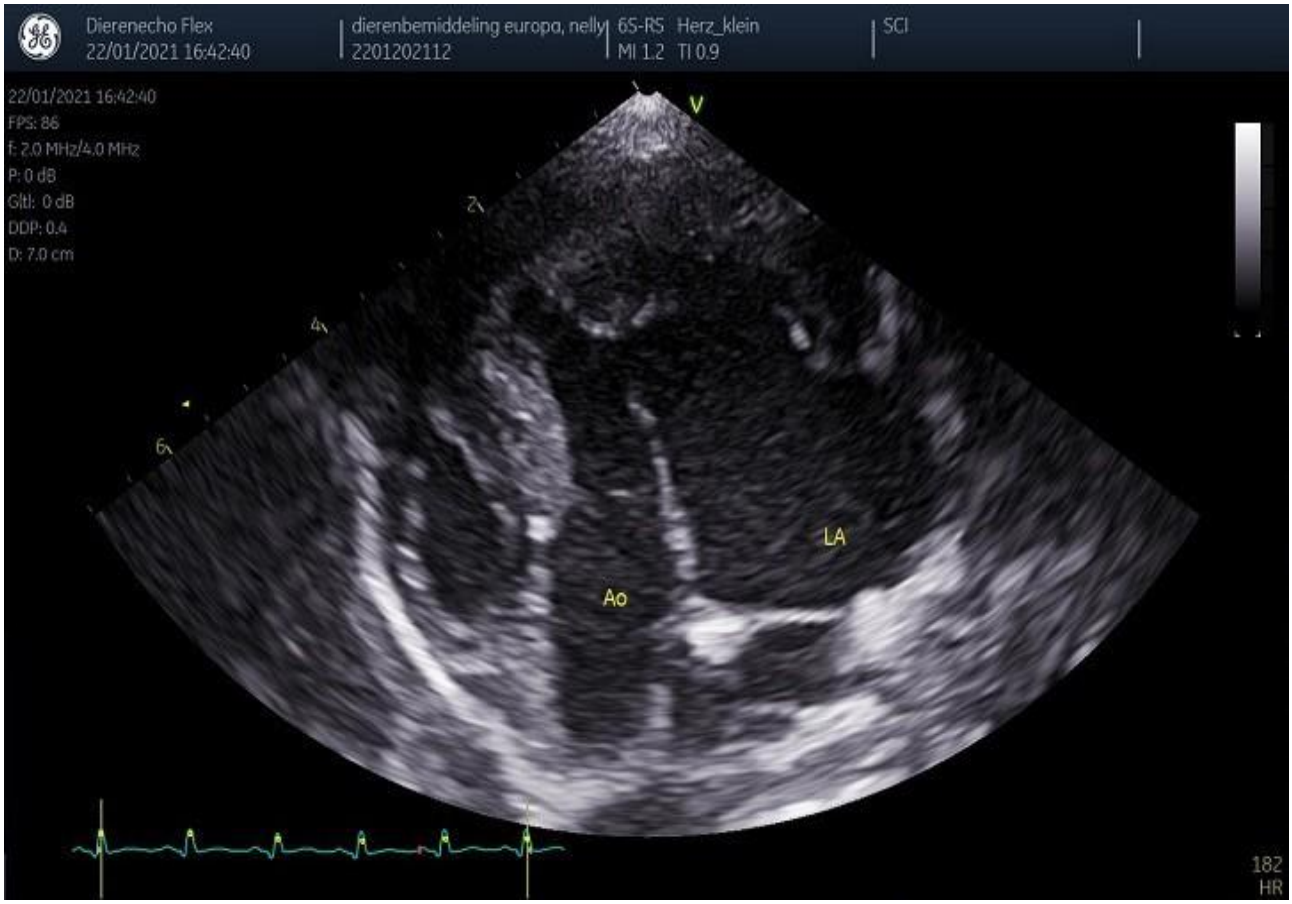
25. Right parasternal high base with view of the tricuspid valve . With CF doppler no regurgitation of the tricuspid valve can be detected.



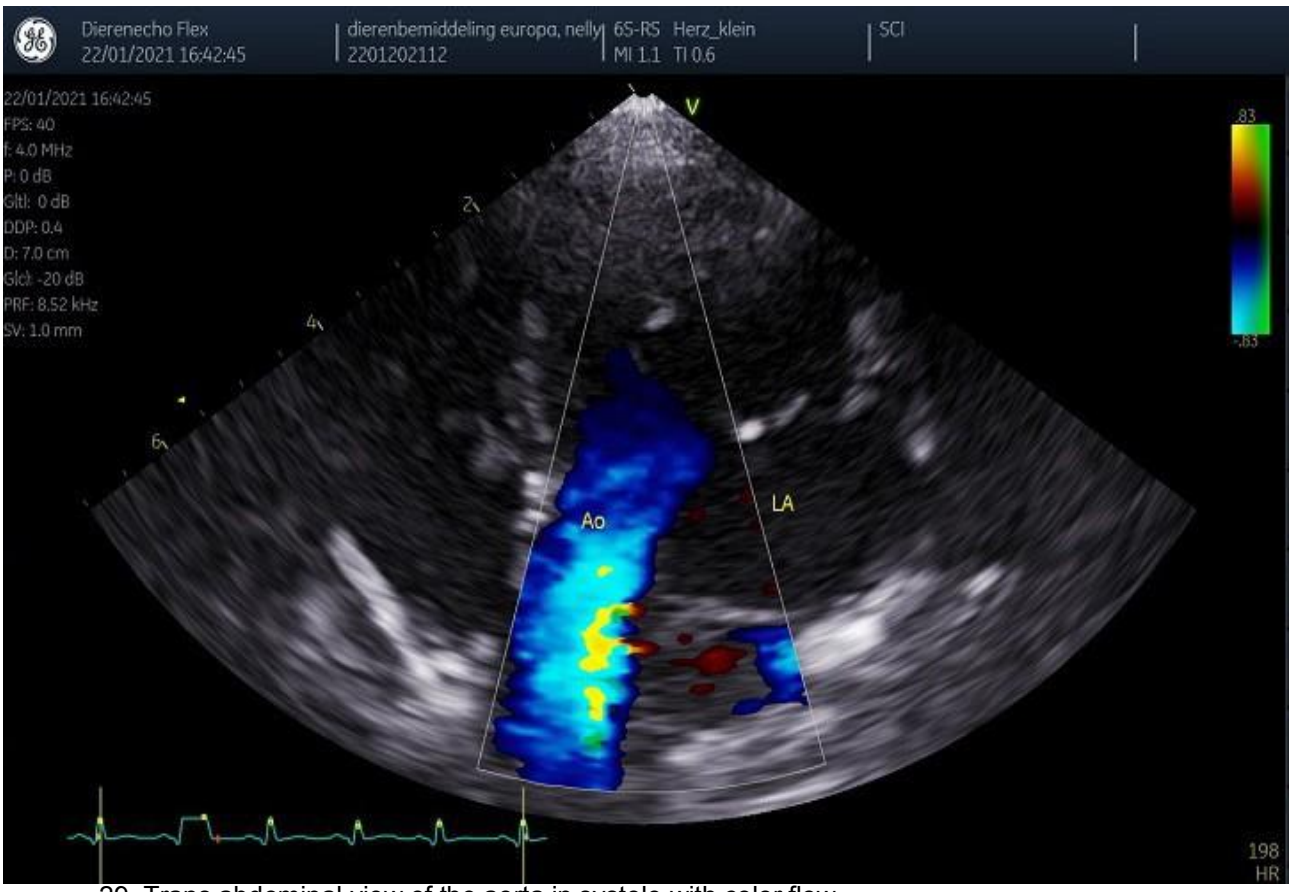
26. Right parasternal high base with view of the tricuspid valve . With PW doppler physiological profile and maximum velocity can be appreciated.



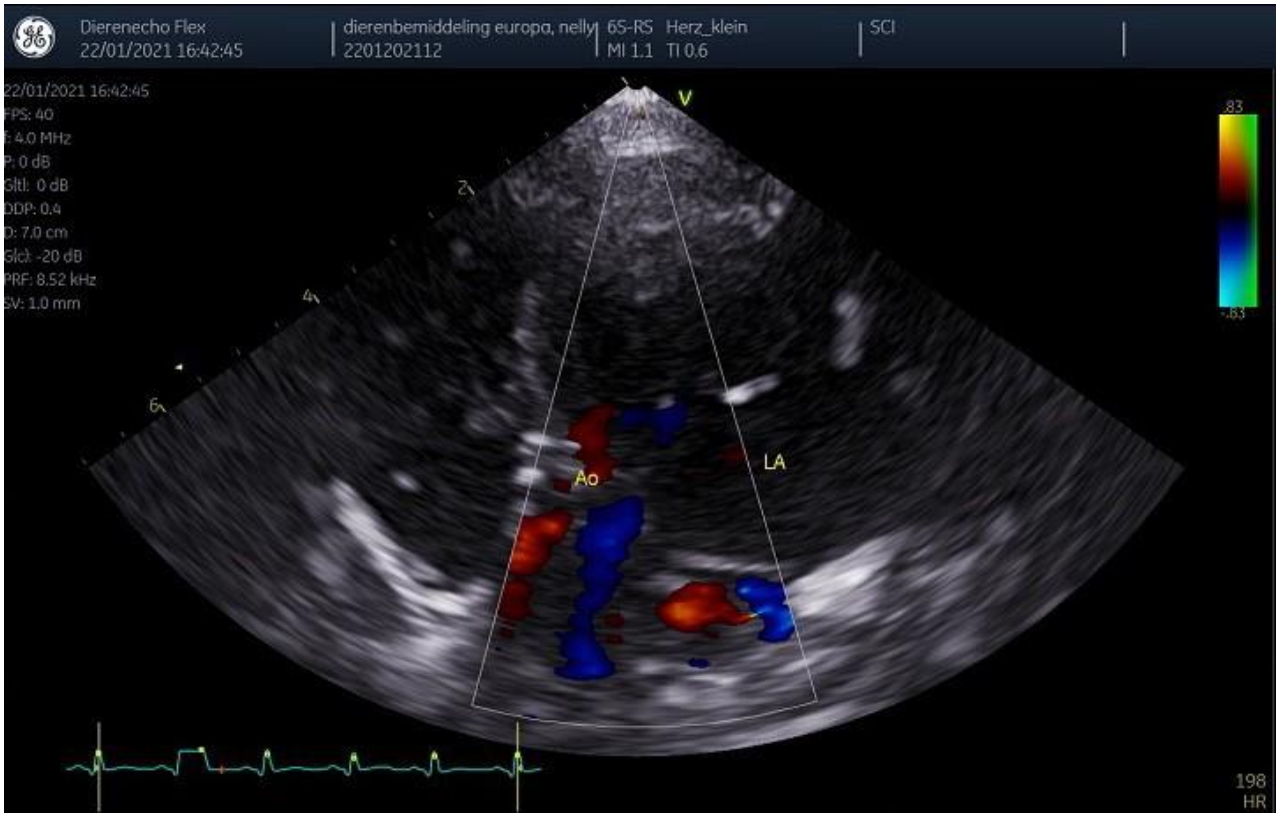
27. Right parasternal high base with view of the atrial septum (arrow). With CF doppler no sign of atrial septal shunting can be detected.



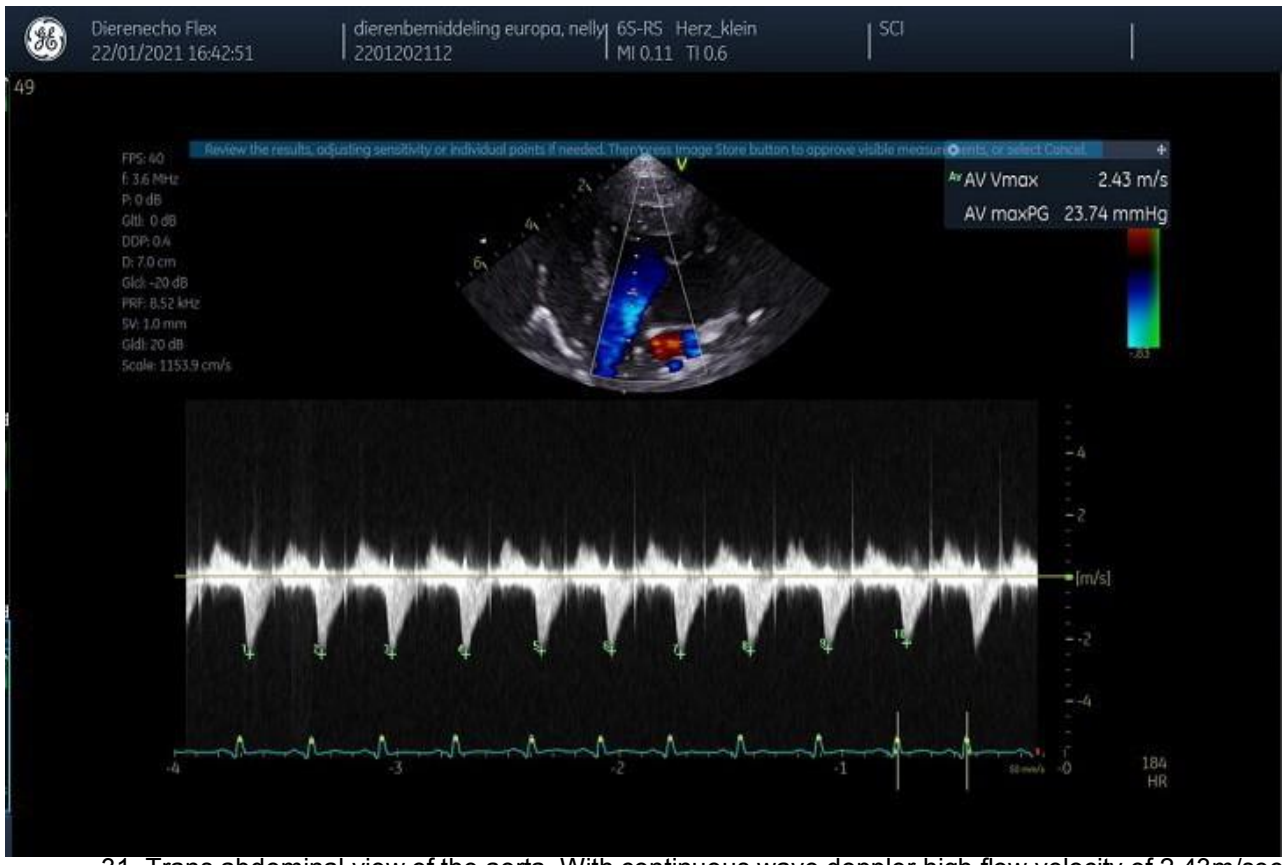
28. Trans abdominal view of the aorta with closed valve. The valve appears normal.



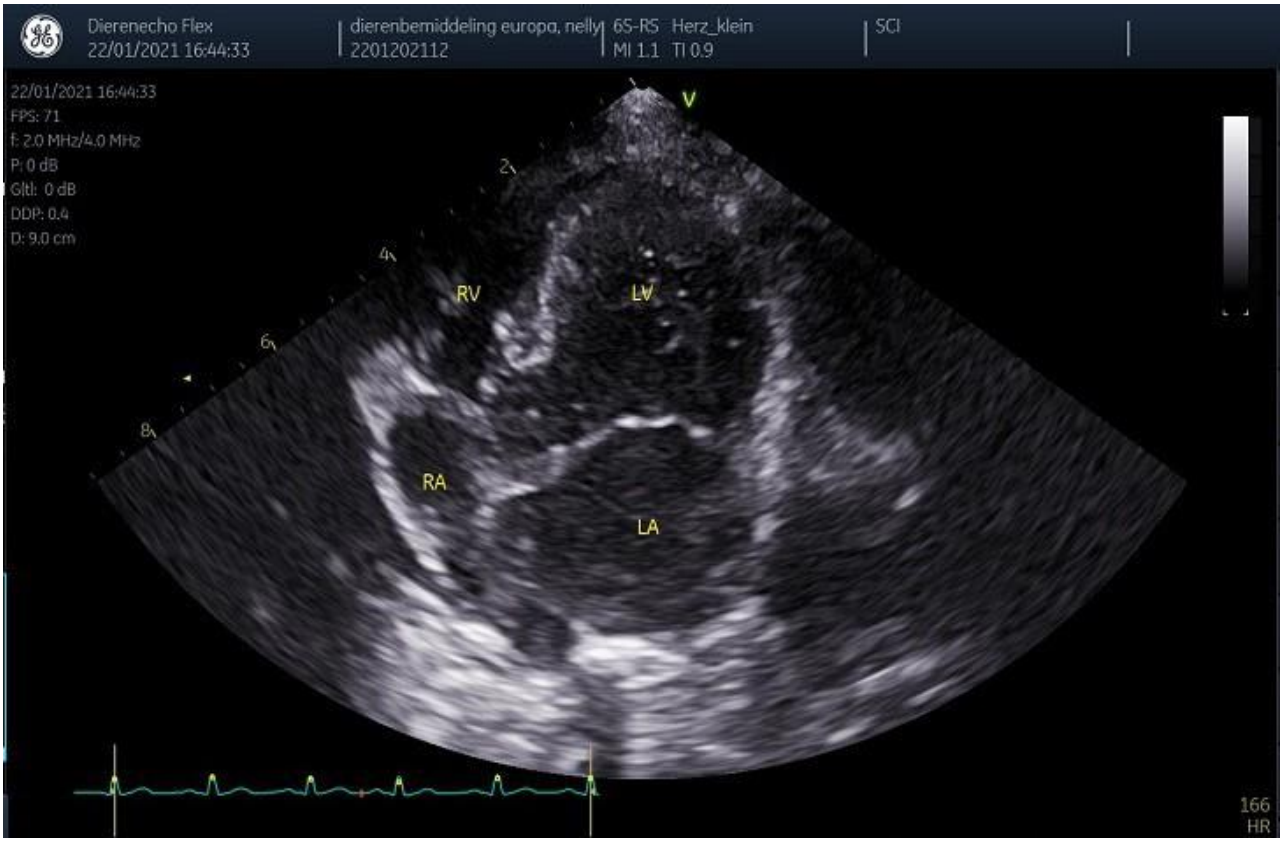
29. Trans abdominal view of the aorta in systole with color flow.



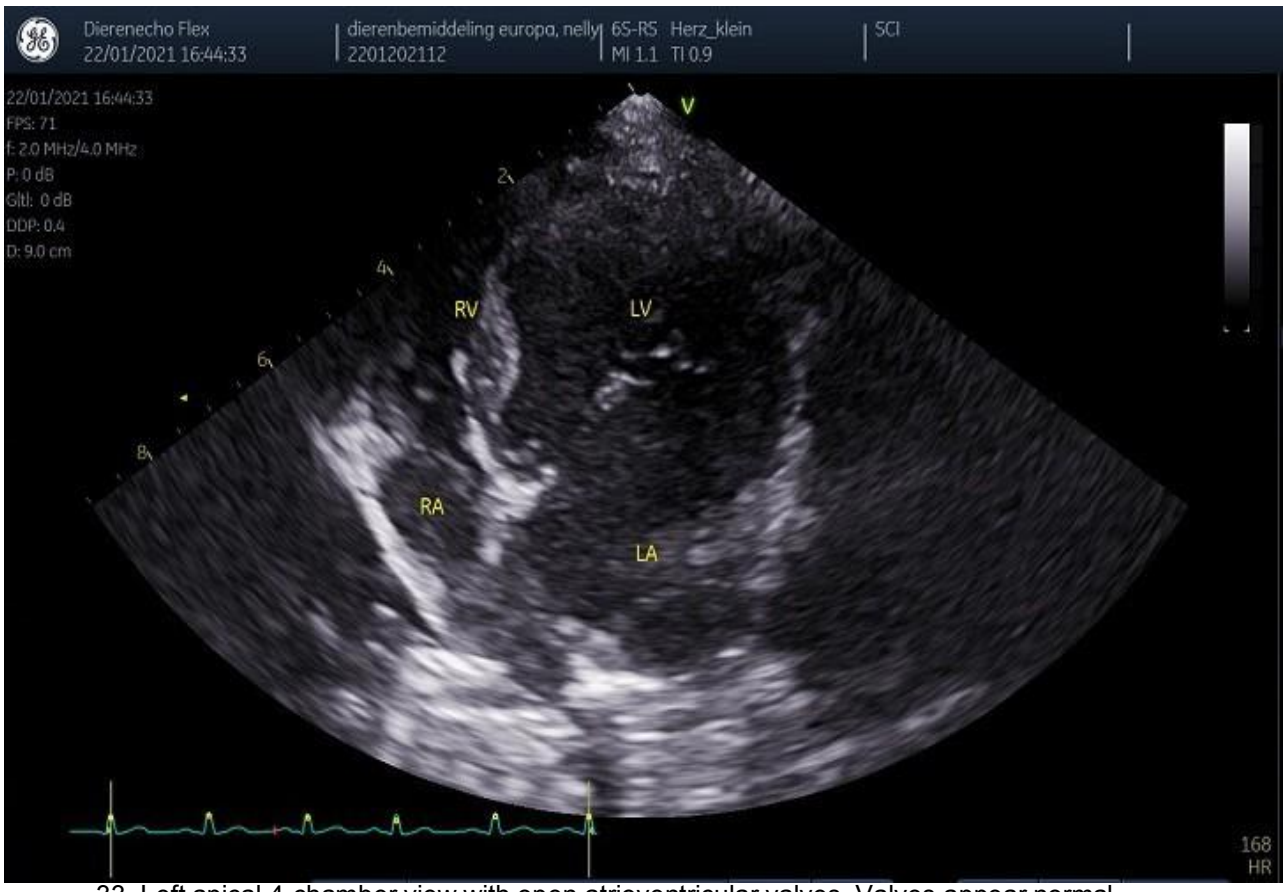
30. Trans abdominal view of the aorta in diastole with color flow. With color flow no regurgitation of the aortic valve can be detected in this view.



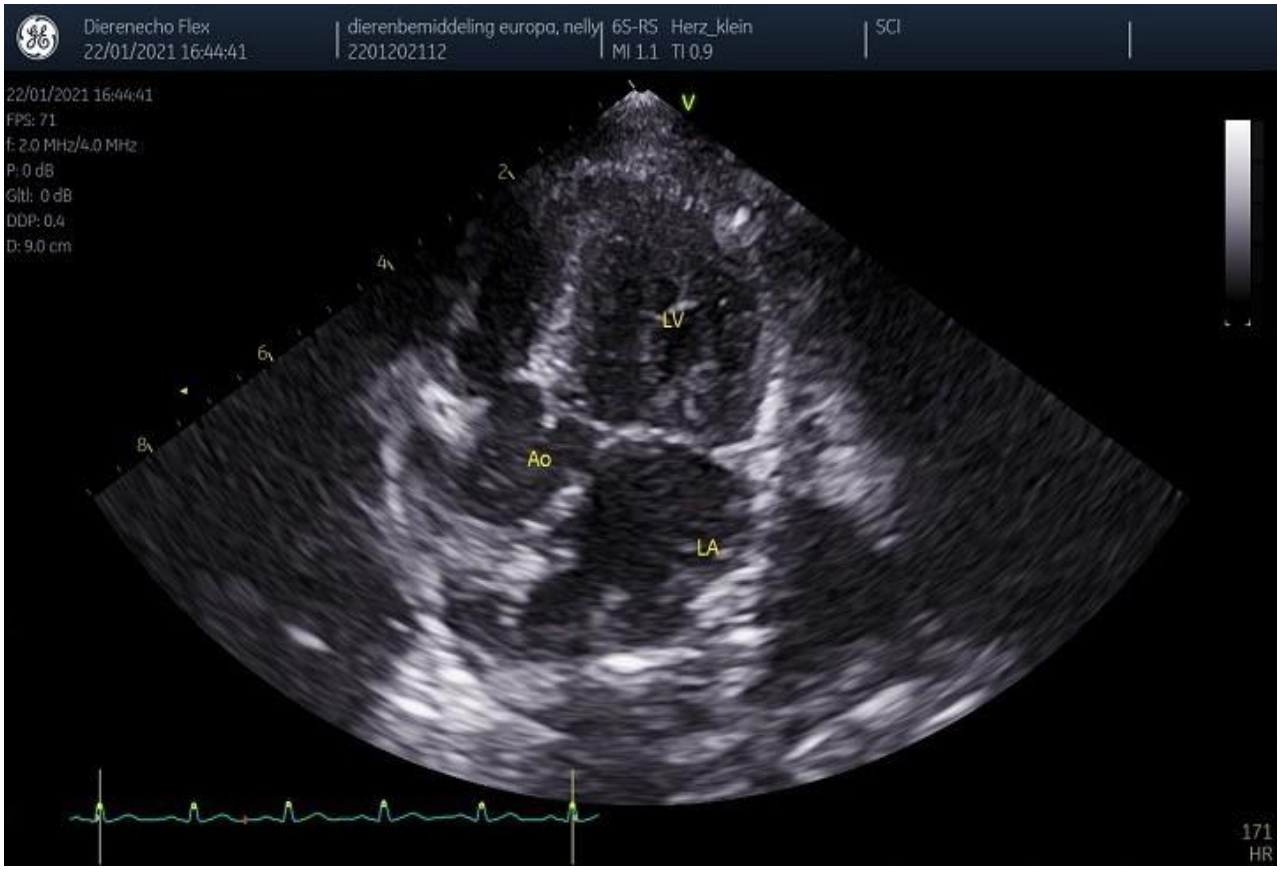
31. Trans abdominal view of the aorta. With continuous wave doppler high flow velocity of 2,43m/sec across the aortic valve can be measured.



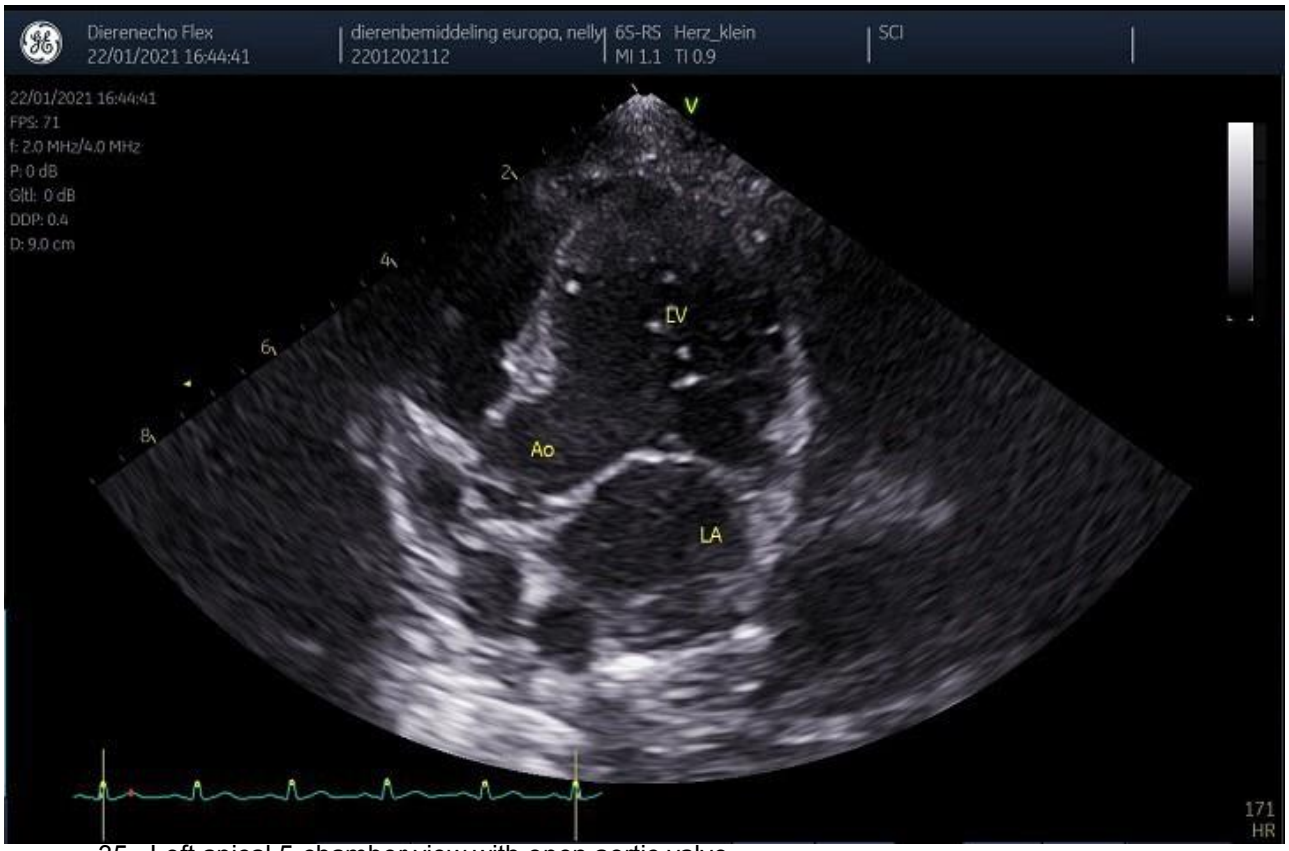
32. Left apical 4-chamber view with closed atrioventricular valves. Valves appear normal.



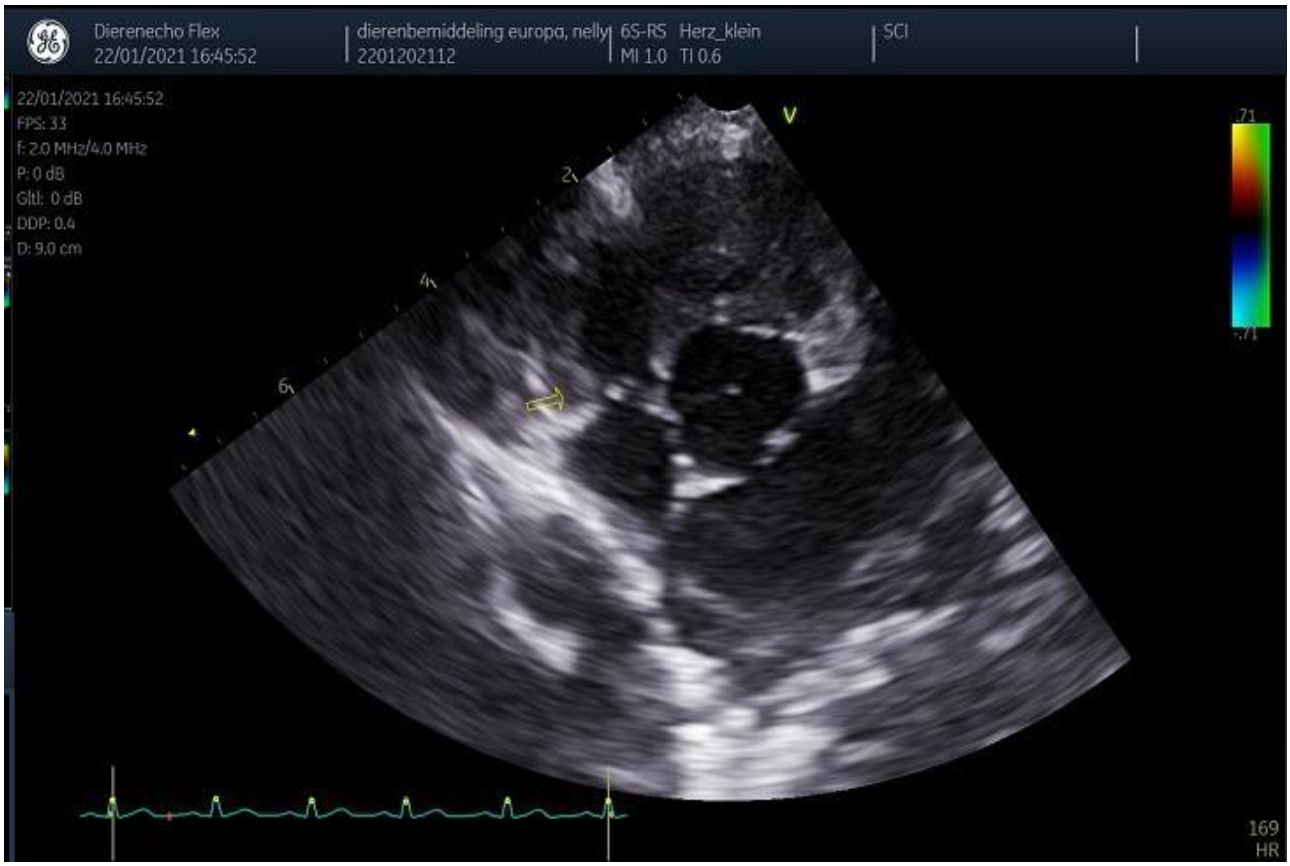
33. Left apical 4-chamber view with open atrioventricular valves. Valves appear normal.



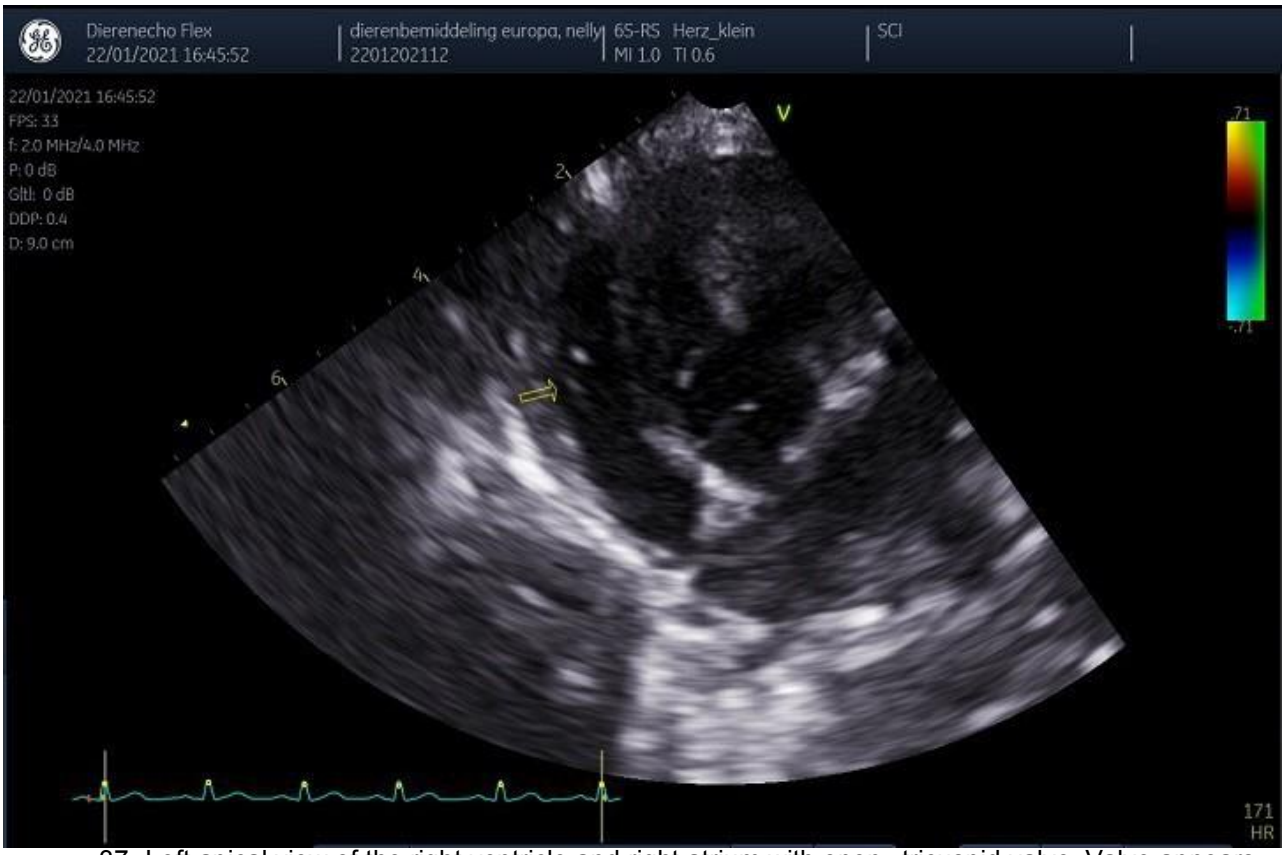
34. Left apical 5-chamber view with closed aortic valve. Valve appears normal.



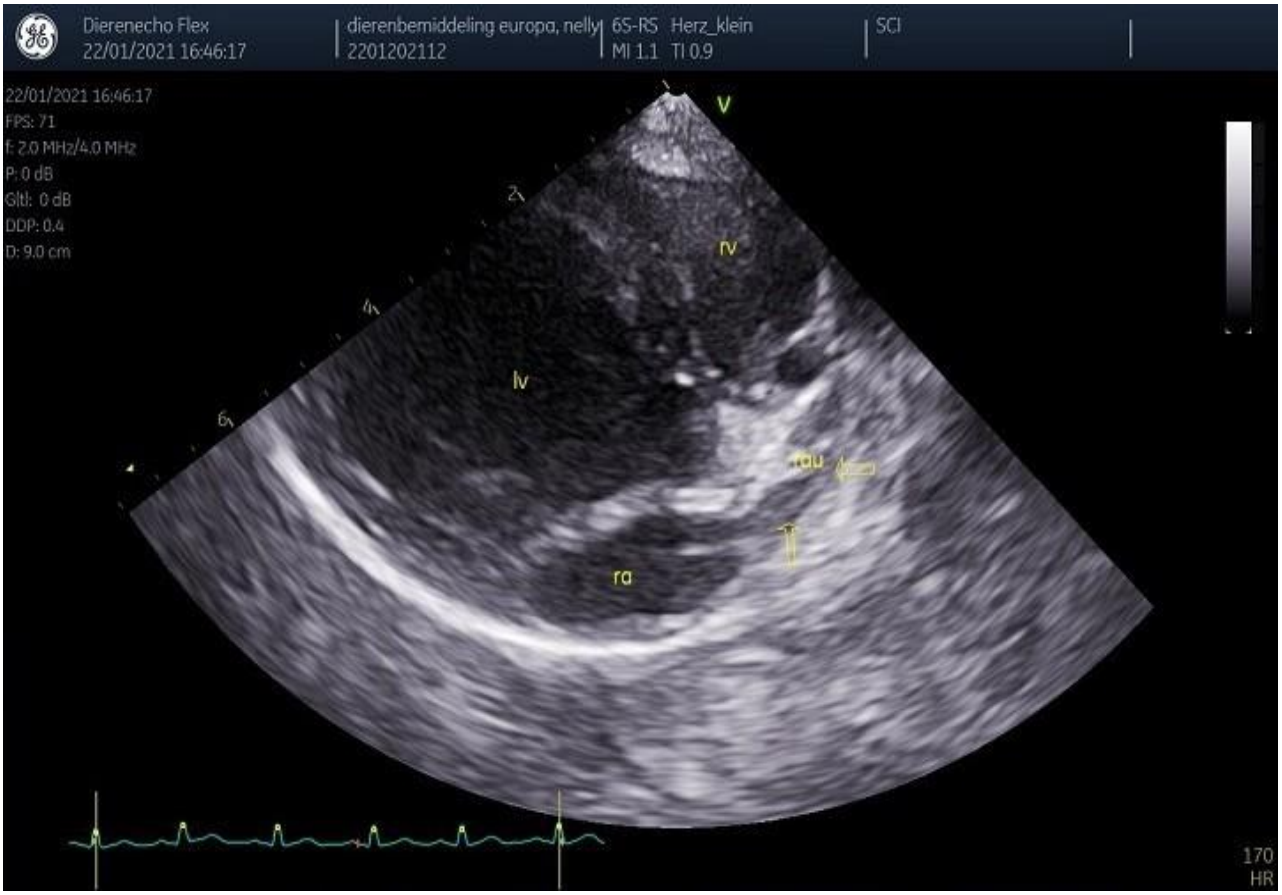
35. Left apical 5-chamber view with open aortic valve.



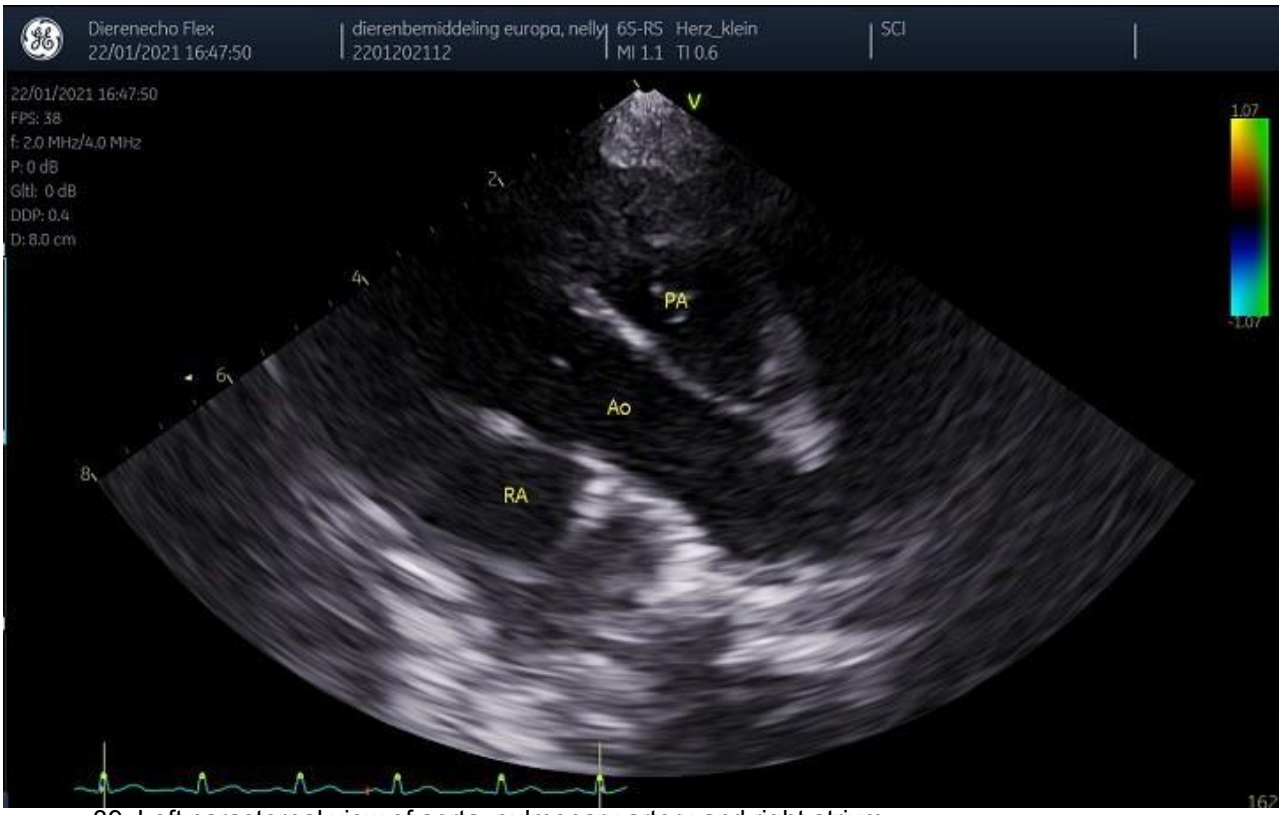
36. Left apical view of the right ventricle and right atrium with closed tricuspid valve. Valve appears normal.



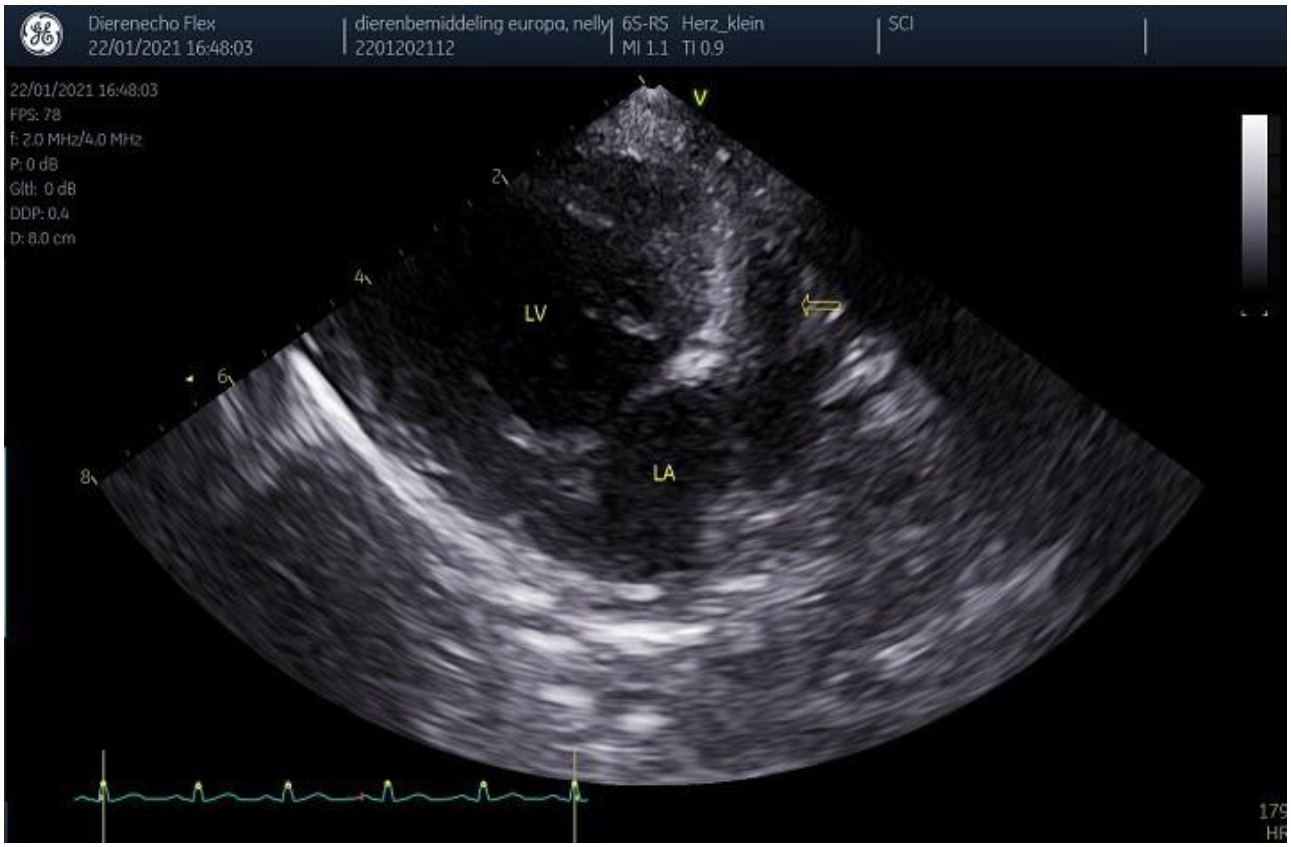
37. Left apical view of the right ventricle and right atrium with open tricuspid valve. Valve appears normal.



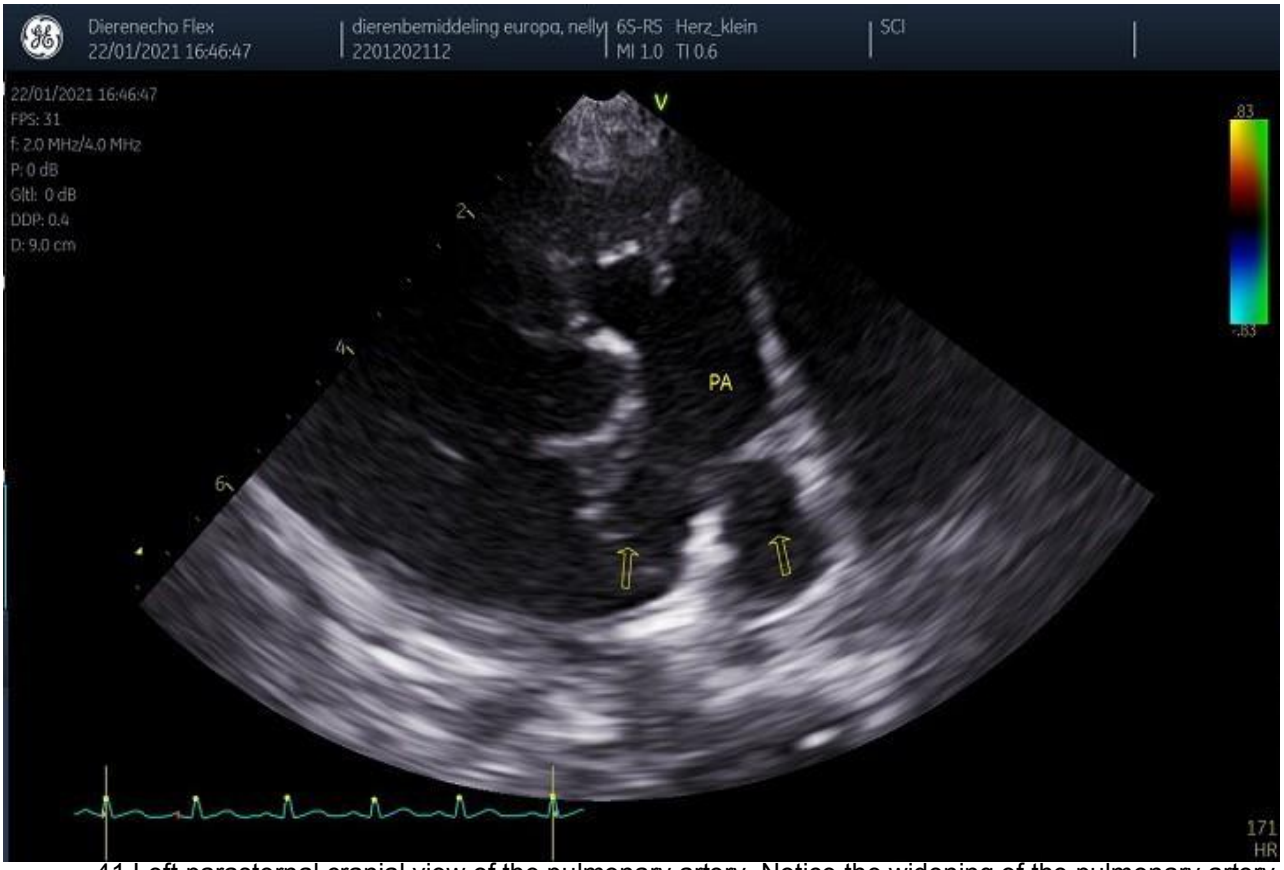
38. Left apical view of right ventricle and right atrium and auricle (arrow) – no masses to be detected.



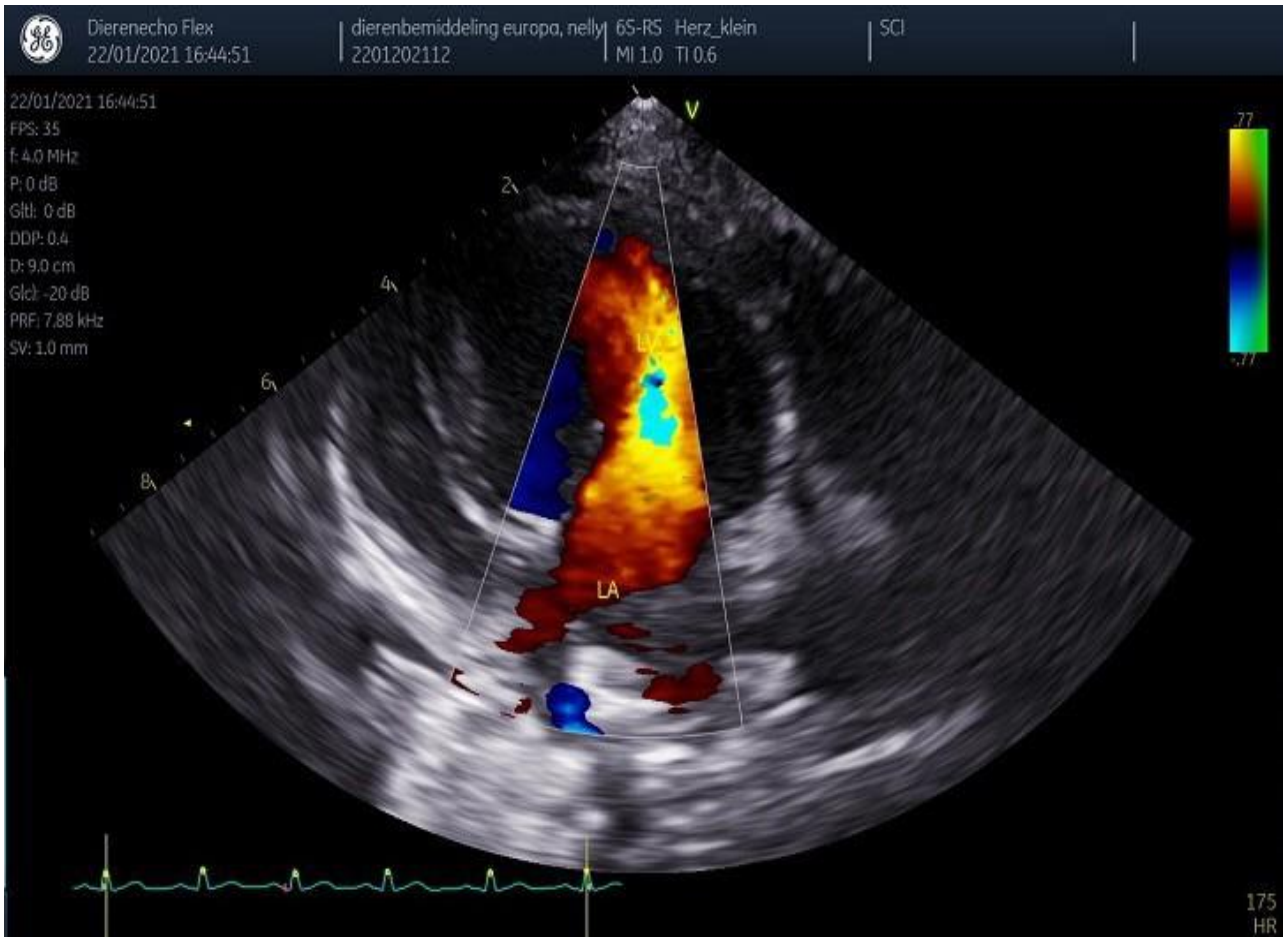
39. Left parasternal view of aorta, pulmonary artery and right atrium.



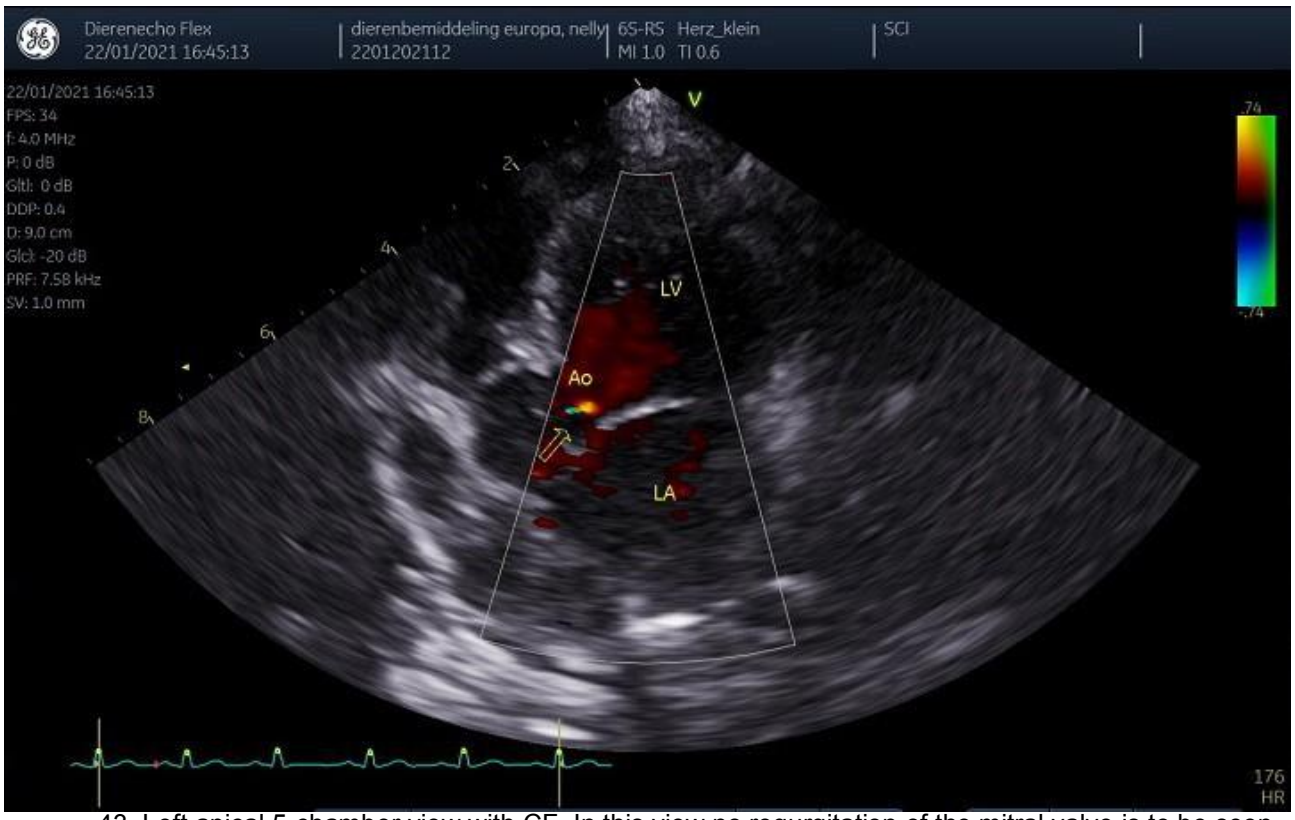
40. Left parasternal cranial view of the left atrium with auricle (arrow). No masses to be seen.



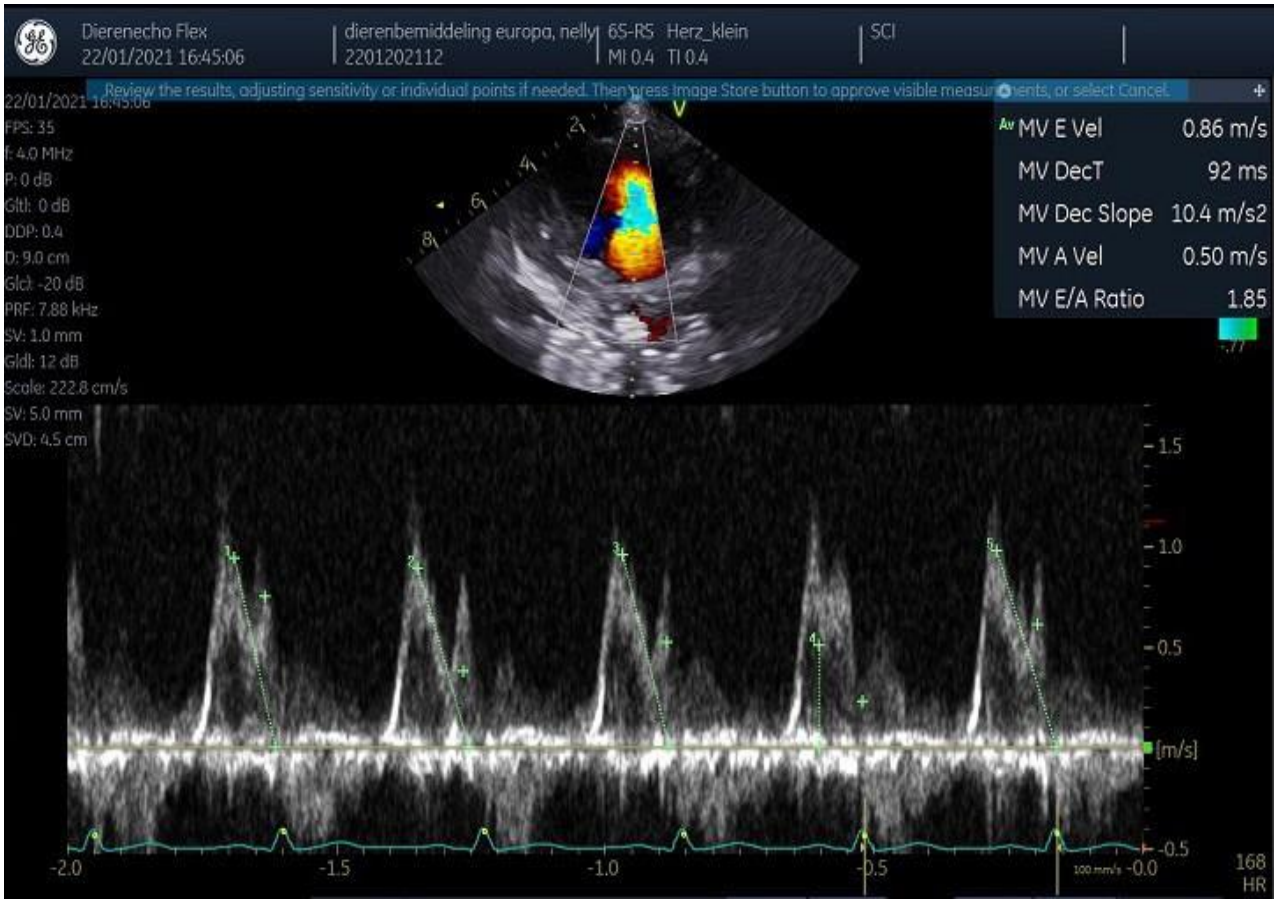
41.Left parasternal cranial view of the pulmonary artery. Notice the widening of the pulmonary artery and the branches (arrows).



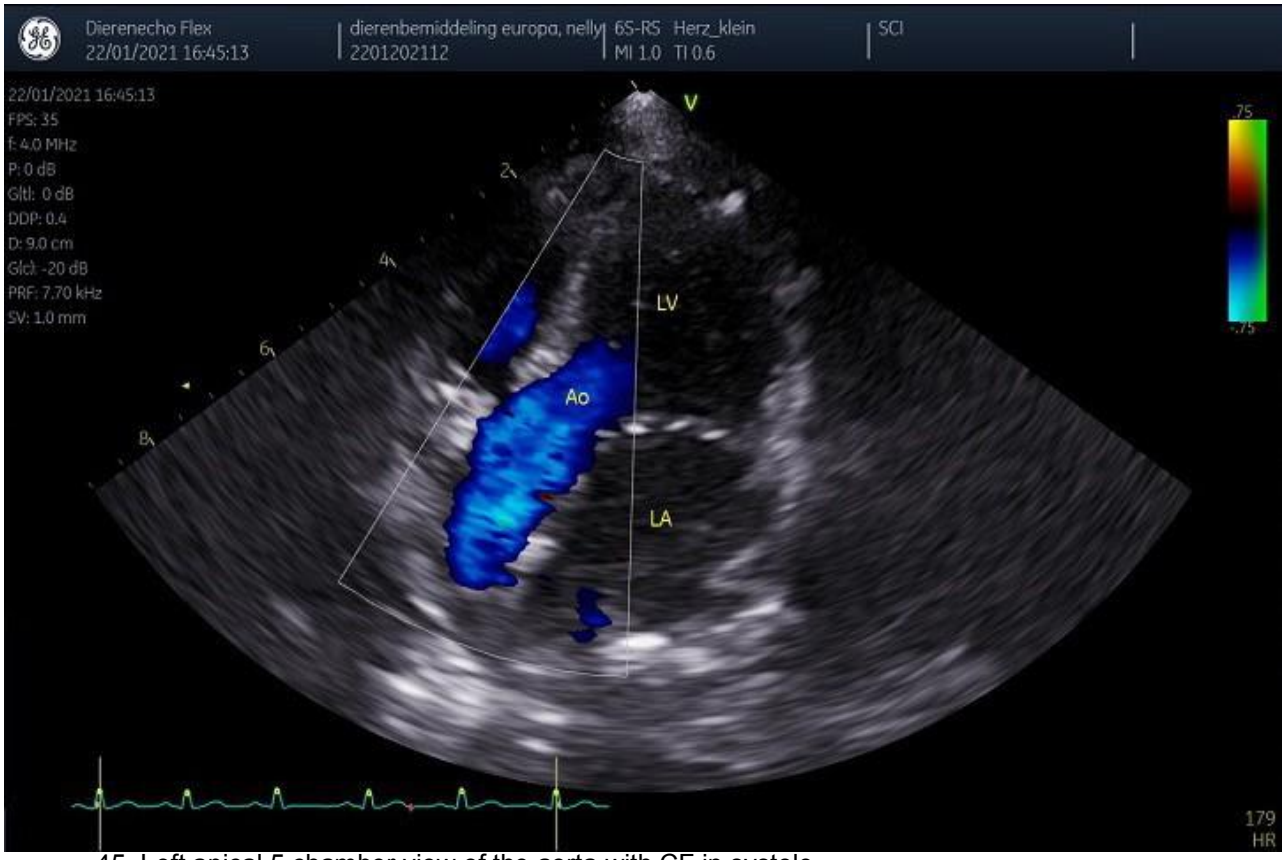
42. Left apical 4-chamber view with CF at the level of the mitral valve with open valves.



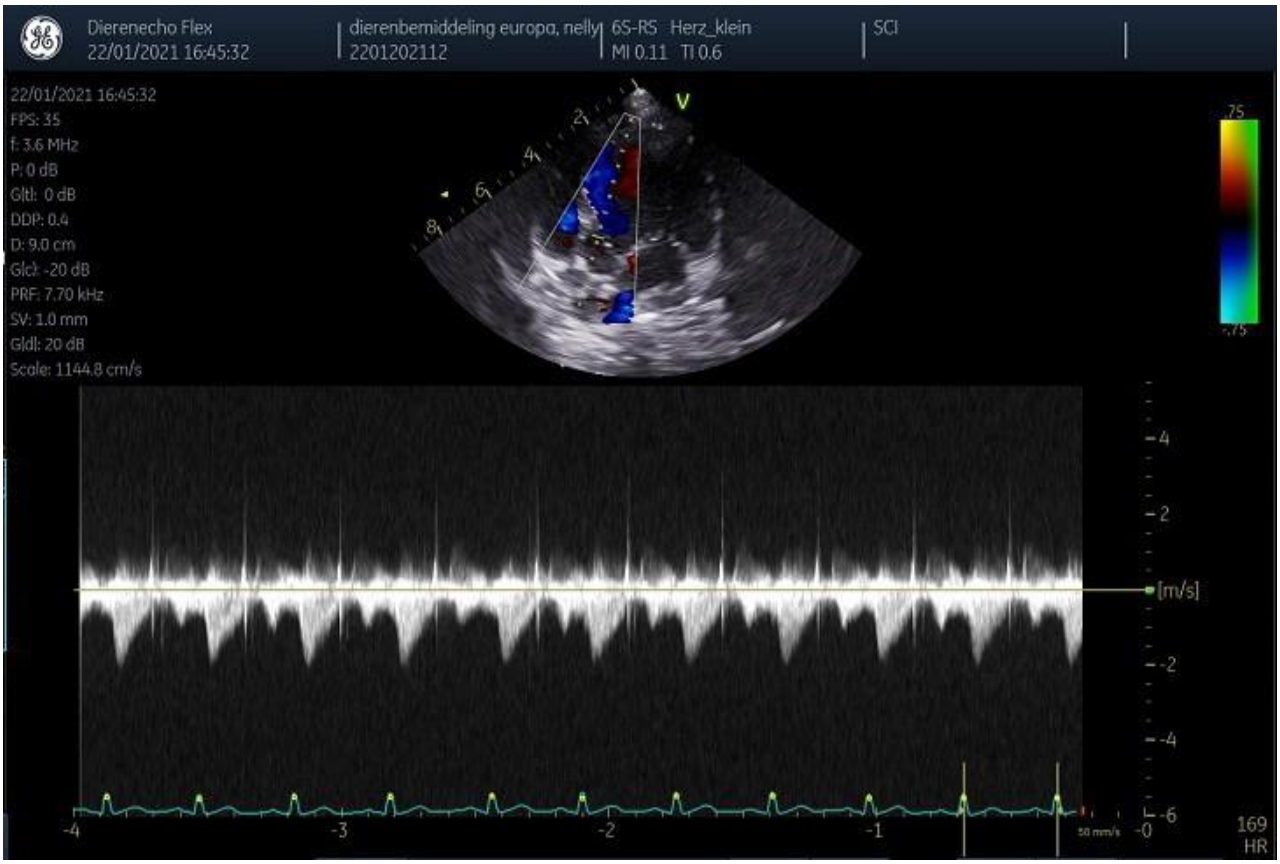
43. Left apical 5-chamber view with CF. In this view no regurgitation of the mitral valve is to be seen, but trivial regurgitation is to be seen at the level of the aortic valve (arrow).



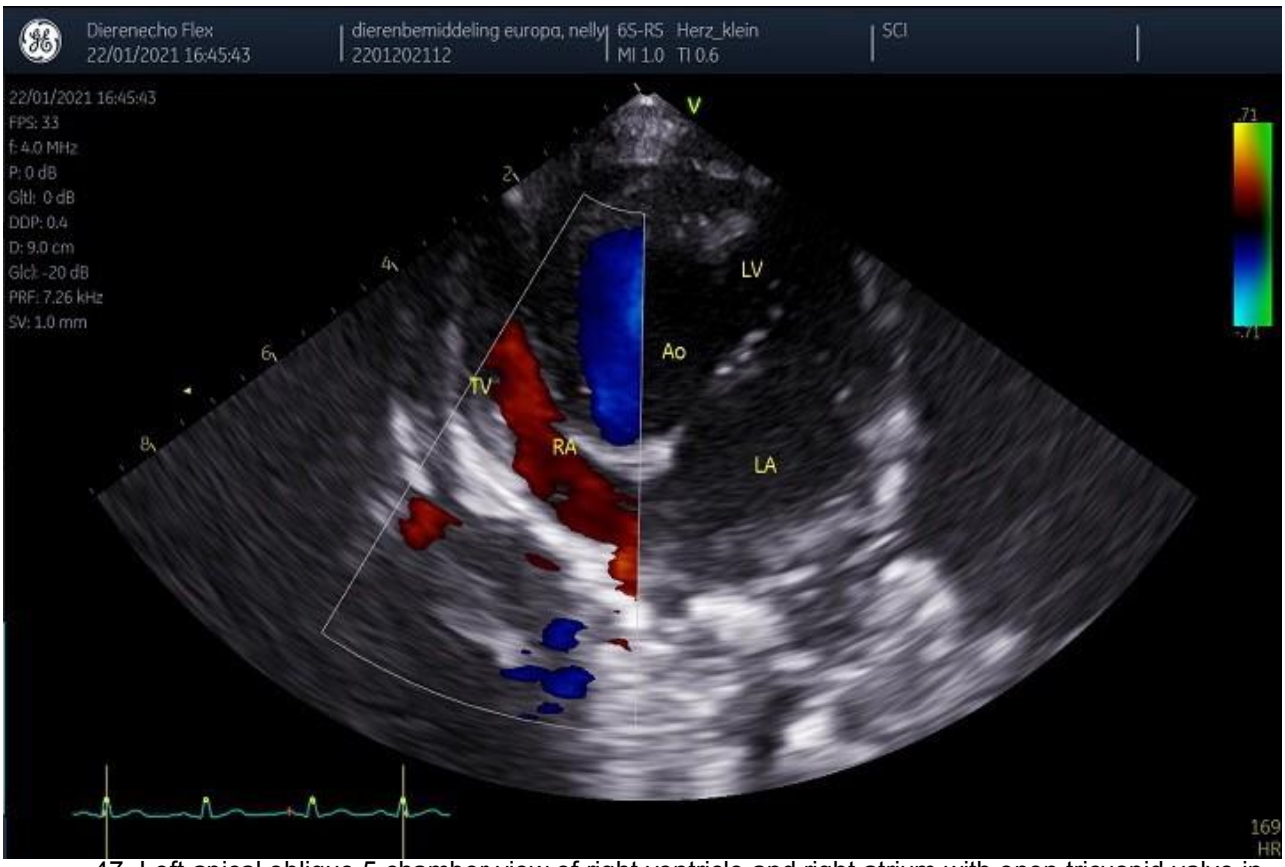
44. Left apical 4-chamber view with PW doppler of the mitral valve flow. Mitral valve flow profile is normal.



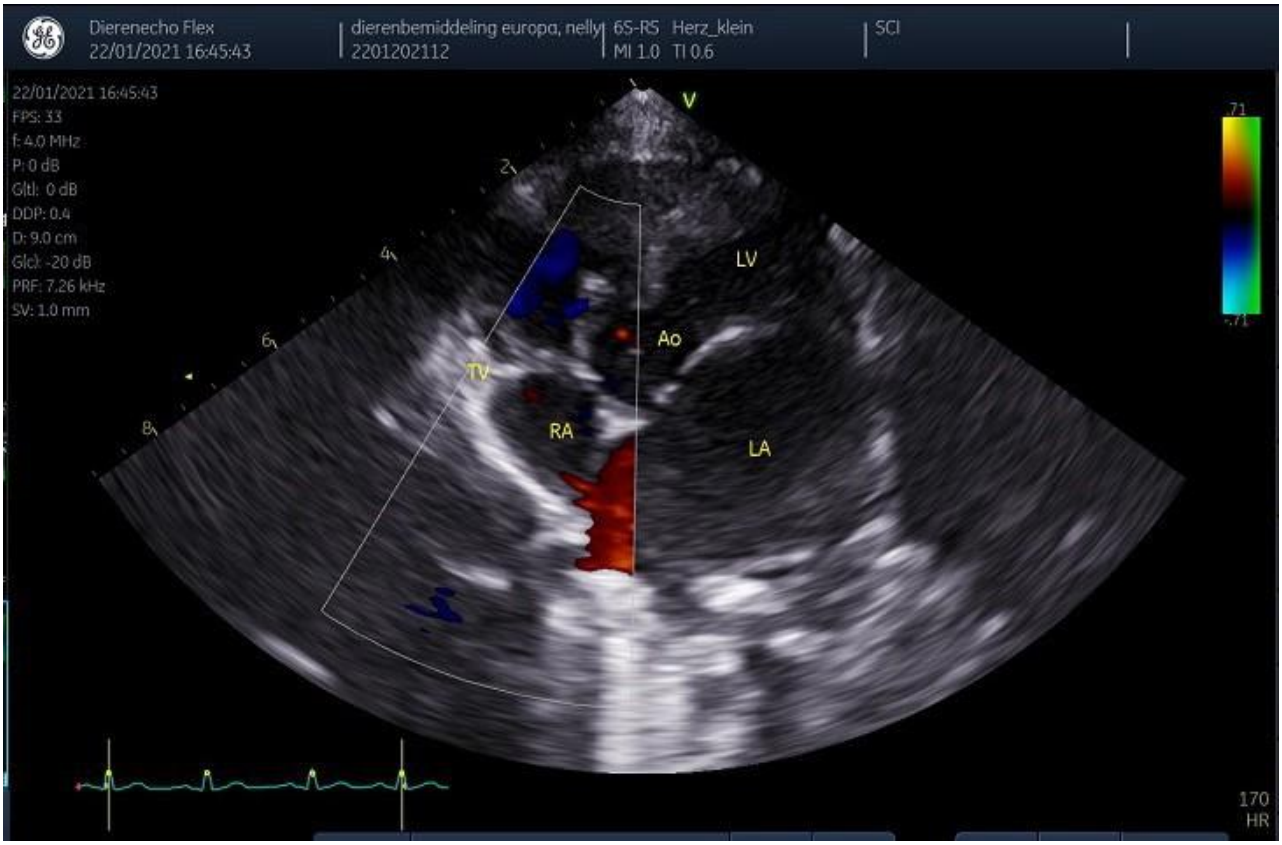
45. Left apical 5 chamber view of the aorta with CF in systole .



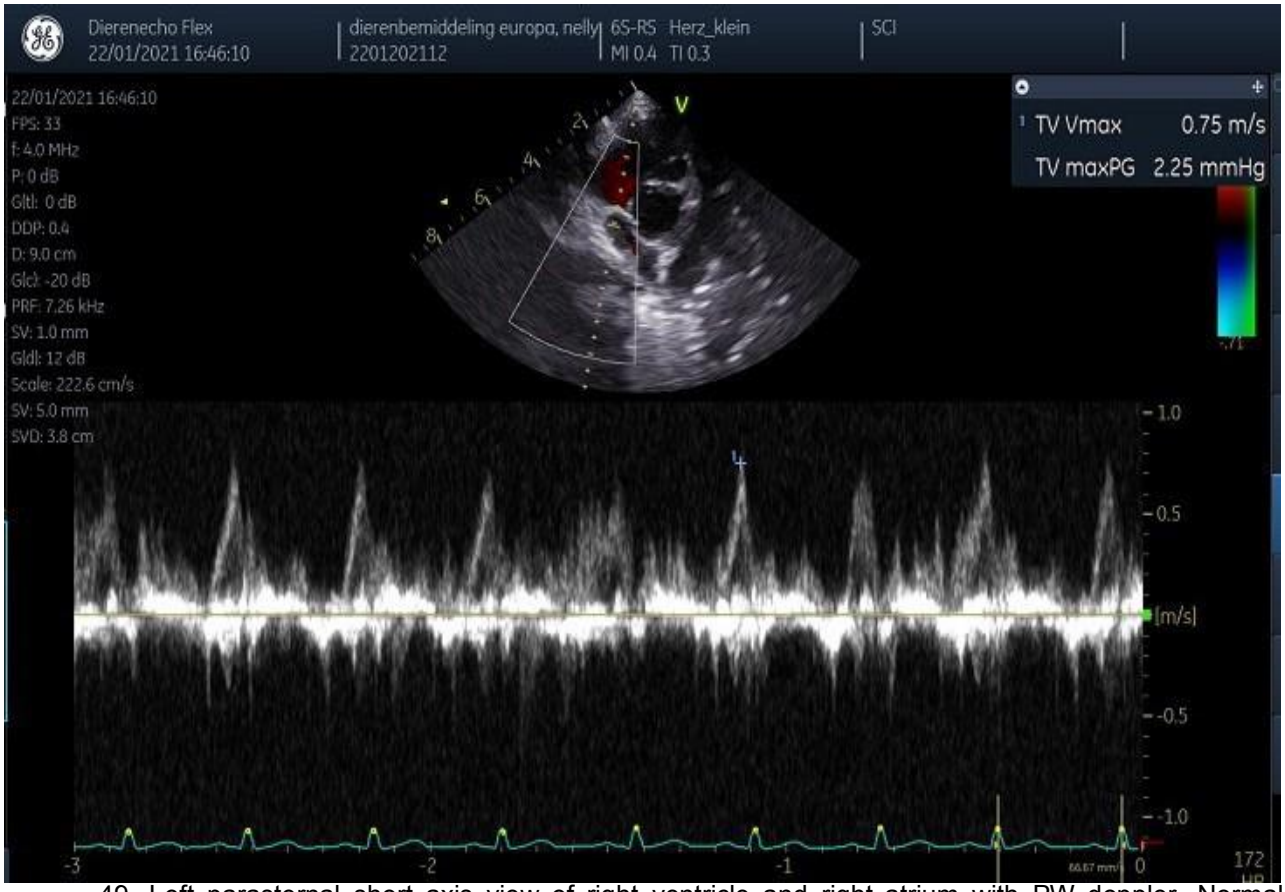
46. Left apical 5 chamber view of the aorta with CW doppler. Normal flow velocity of 1,9 m/sec can be appreciated.



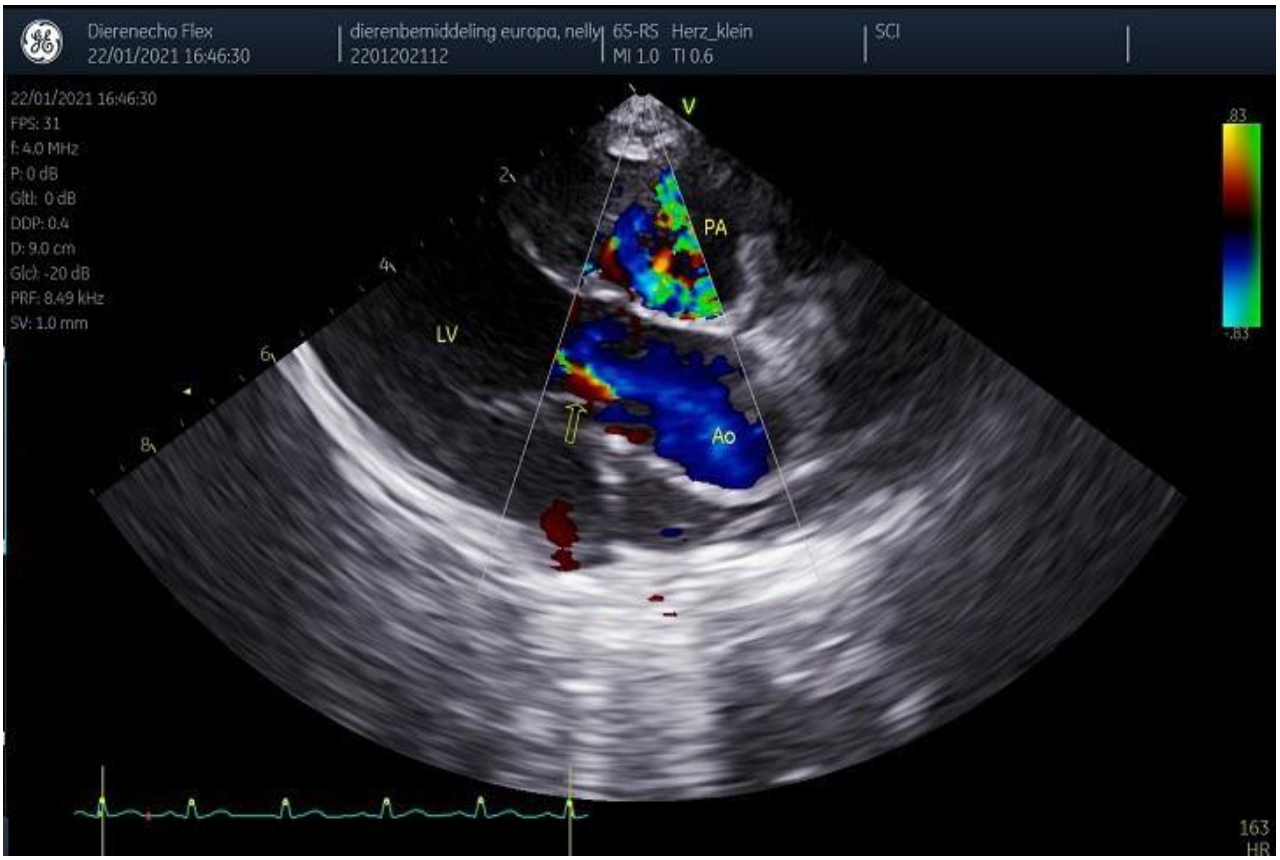
47. Left apical oblique 5 chamber view of right ventricle and right atrium with open tricuspid valve in CF.



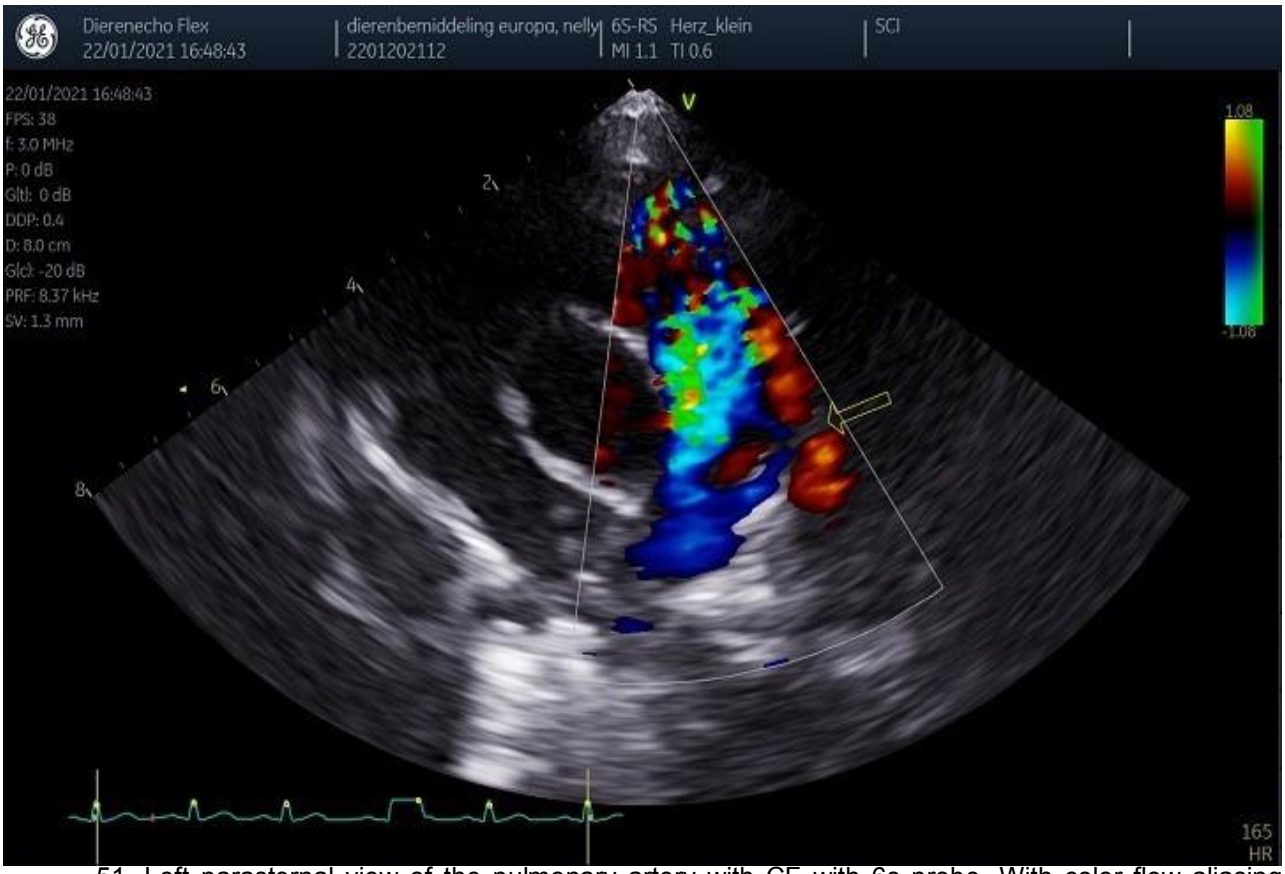
48. Left apical oblique 5 chamber view of right ventricle and right atrium tricuspid valve in CF – no regurgitation jet to be seen.



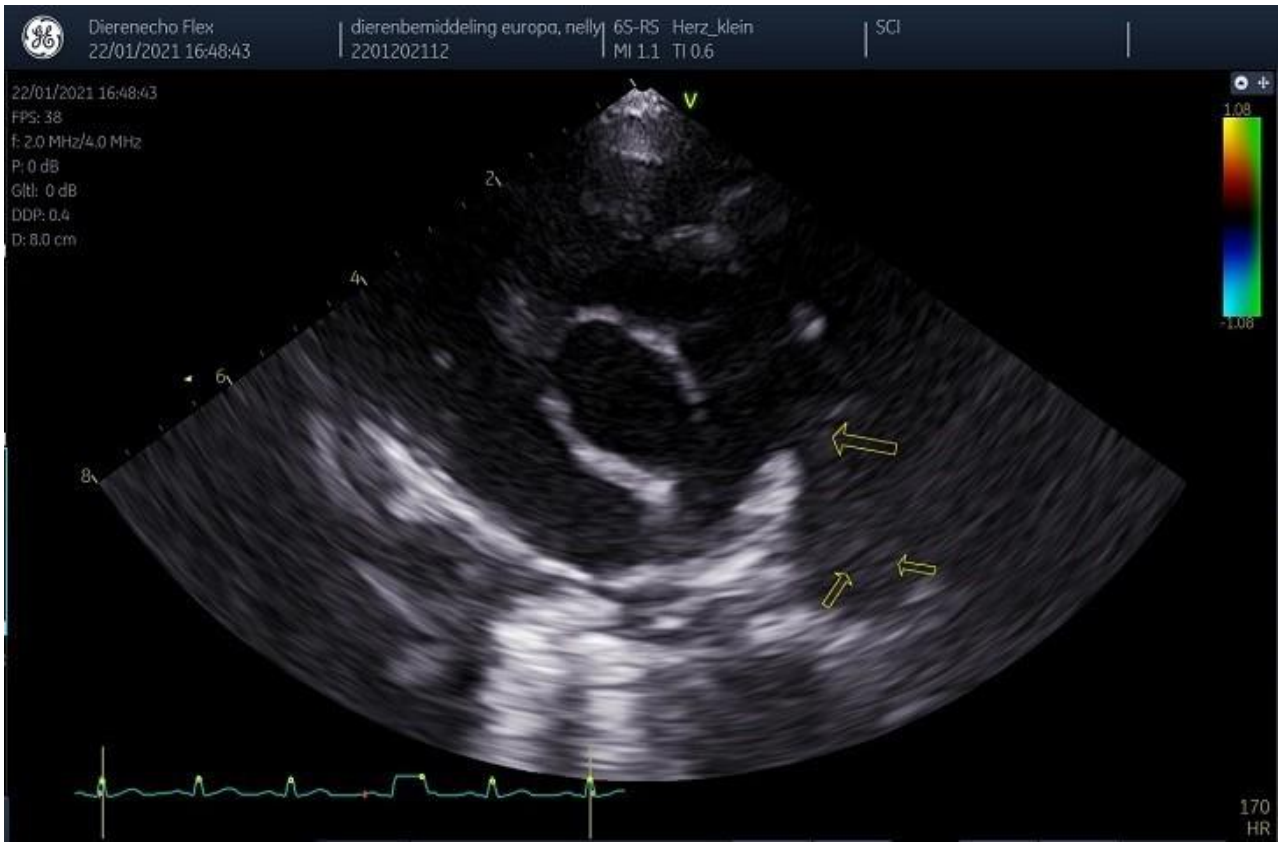
49. Left parasternal short axis view of right ventricle and right atrium with PW doppler. Normal tricuspid valve flow profile can be appreciated.



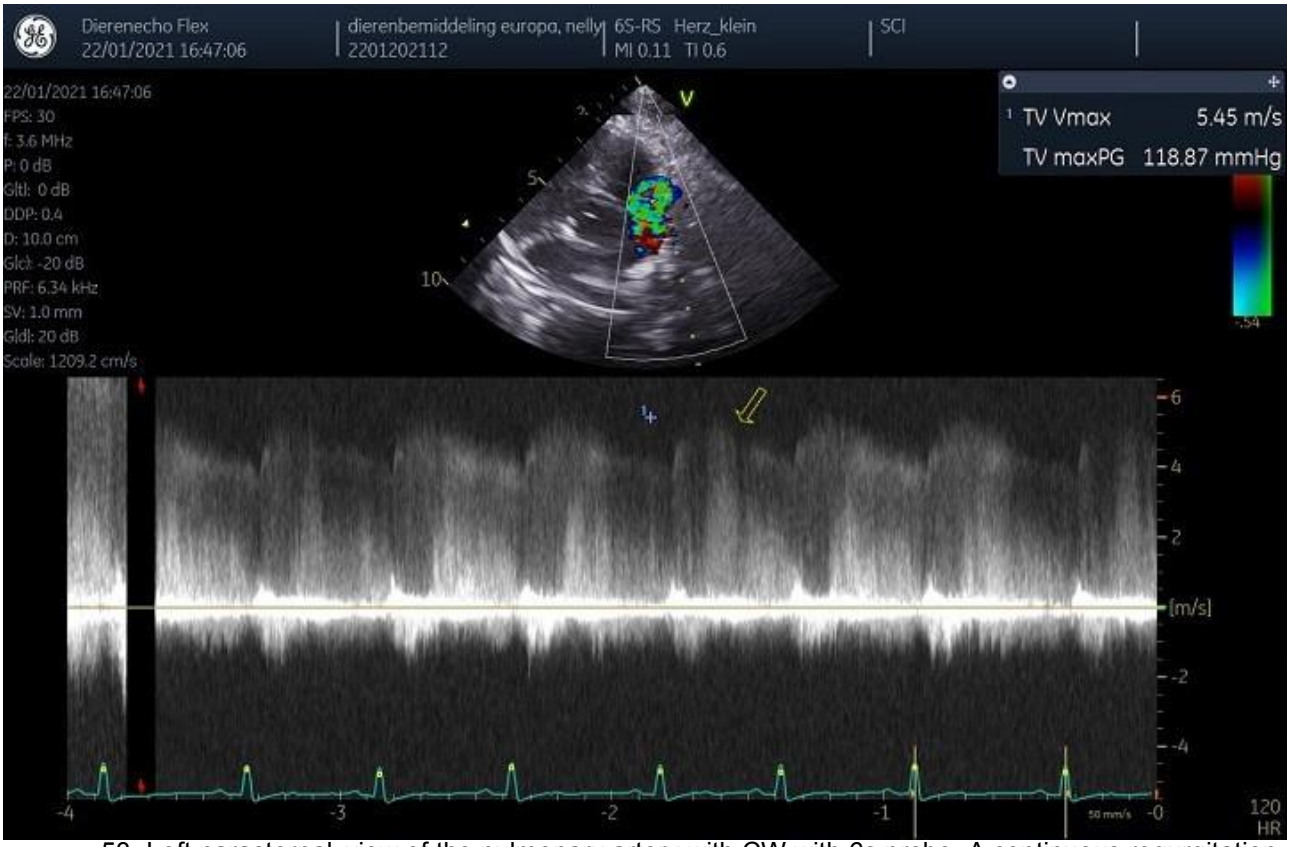
50. Left parasternal view of the aorta . With color flow a trivial regurgitant jet at the level of the aorta (arrow) and also aliasing within the pulmonary artery can be appreciated.



51. Left parasternal view of the pulmonary artery with CF with 6s probe. With color flow aliasing within the whole pulmonary artery can be appreciated. The yellow arrow points to the PDA lesion where red colored flow indicates blood flow from the PDA towards the pulmonic valve (towards the transducer).



52. Left parasternal view of the pulmonary artery with CF with 6s probe. After color has been removed one can appreciate the PDA opening (one larger arrow) and the wider ampulla of the PDA (two smaller arrows).



53. Left parasternal view of the pulmonary artery with CW with 6s probe. A continuous regurgitation jet with a maximum velocity of 5,45m/sec can be appreciated.

Measurements

		measurements Nelly	
Mmode	**J. Boon in mm** (4,5kg)	**long axis MM**	**short axis MM**
IVSd (mm)	5,97-7,7	6	6
LVIdD (mm)	18,83-21	36	38
LVFWd(mm)	5,75-6,17	6	6
IVSs (mm)	8,99-10,92	8	9
LVIDs (mm)	10,52-12,42	23	24
LVFWs (mm)	7,99-9,77	10	9
FS%	33,7-45,9 %	35	36
La (mm)	12,78-15,63		19
Ao (mm)	12,76-14,78		16
La:Ao	0,95-1,65	1,29	1,24
EPSS	1,7-5,7	3	4
wLVIDD	<1,7	2,31	2,44

2-D Mode			
La diameter	16-26,5 (ref 2,11 in 4-chamber view)	34	
La:Ao	<1,6 (scandinavian method)		1,29
Mpa:Ao	0,8-1,15 (Chetboul)		1,26

Doppler	**J.Boon** (Chetboul Gen.)	**right**	**left**
Ao Vmax (m/sec)	<2	2,43 (subcostal)	1,9
Pa Vmax (m/sec)	<1,3	2,58	
MV E (m/sec)	0,58-1,17		0,86
MV A (m/sec)	0,39-0,86		0,5
E:A mitral	0,92-2,72		1,85
TV E (m/sec)	0,5-0,98		0,65
TV A (m/sec)	0,29-0,7		0,26
E:A tricuspid	1,09-2,8		2,53
tricuspid regurge			0,75
pulmonic regurge		4,09	5,45

* values deviating from the norm

Interpretation

Measurements of the left ventricular study reveal an enlarged left ventricular endsystolic and enddiastolic volume, thus volume overload of the left ventricle. A (continuous) regurgitant jet across the pulmonic valve with a high velocity leads to the diagnosis persistent ductus arteriosus (PDA). The high flow velocity and more or less continuous flow across the pulmonic valve in right parasternal view is most likely due to ductal flow swirling back toward the bifurcation and can thus not be measured correctly. The high flow velocity across the aortic valve in transabdominal view can be explained with volume overload created by the shunting lesion (increased venous return). The Mpa/Ao relation is increased due to widening of the pulmonary artery typically seen in patients with PDA.

Echocardiogram report

During the echocardiographic examination Nelly has a normal sinus rhythm within 120-170 BPM.

On the right and left parasternal views Nelly's echocardiogram shows that the size of the left ventricle is severely increased (volume overload) with normal wall measurements. The systolic and diastolic function of the left ventricle is normal shown by a normal fractional shortening value and normal mitral wave flow profile. Both atria are normal in size, although the left atrium appears a little wider in right parasternal view – probably due to the large volume of the left ventricle which seems to push the left atrium dorsally. The aorta is normal proportioned. No shunting between the ventricles or atria can be visualized.

Widening of the entire pulmonary artery and its branches can be appreciated in right parasternal short axis and left parasternal cranial view. In left parasternal cranial view a patent ductus ateriosus (PDA) with a narrow pulmonary opening and a wider aorta-faced ampulla can be seen in 2D image. With color flow doppler aliasing within the whole pulmonary artery and a continuous regurgitation jet at the level of the pulmonary valve can be detected. Also in left parasternal cranial view color flow brings a jet flowing through the PDA opening towards the pulmonary valve into picture. With CW doppler the PDA-typical continuous regurgitation jet within the pulmonary artery can be measured at a maximum of 5,45m/sec (118mmHg).

The leaflets of the mitral valve, tricuspid valve, pulmonic and aortic valve do not show any dysplasia or thickening. Only trivial regurgitation at the aortic valve and mitral valve can be appreciated in some views. Tricuspid valve flow profile is normal. Mitral valve flow profile shows no signs of diastolic dysfunction. Flow velocity across the aortic valve is high probably due to high left ventricular volume due to the PDA shunting lesion.

Diagnosis

- **Patent ductus ateriosus**
  - **secondary left ventricular eccentric hypertrophy**
  - **secondary high aortic flow velocity**
  - **trivial aortic and mitral valve insufficiency**

Discussion

Epidemiology

A patent ductus arteriosus (PDA) is a postnatally preserved opening between the descending aorta and pulmonary artery. Usually higher pressure within the aorta leads to a left-right shunt, thus blood flow going from the aorta through the PDA into the pulmonary artery. Due to consequent high volume overload within the left ventricle, congestive left heart failure is to follow and a high mortality rate within the first year after birth in untreated animals is common (1,14). The PDA is one of the most common congenital heart diseases in the dog and is diagnosed in up to 11-37% of all congenital defects and in 9% in combination with other congenital defects (1,3,10). The PDA is 3-4x more often seen in female dogs (1,9), predominantly in small breeds as the Chihuahua, Keeshound, Malthesian, Poodle, Bichon Frisee, Cavalier King Charles, Pon, Shelty, Yorkshire ,

Springer Spaniel and recently also found more often in the Dutch Stabyhound. A genetic base could be found in the (Miniature) Poodle and Border Collie (1,3,10,13,14). With 3-7% a PDA is of all congenital defects less often seen in cats (1,19).

Pathophysiology

The ductus arteriosus (DA) is a connection in between the aorta and pulmonary artery during fetal development. Through the DA oxygenated maternal blood flows from the pulmonary artery directly into the aorta, sparing the non functional fetal lungs. Normally the DA consists of mainly smooth muscle tissue (98%) and only of a very small amount of elastic fibers (2%). Postnatally the onset of breathing and oxygenation lead to a quick drop of the pulmonary vascular resistance, which (in combination with low prostaglandine levels) leads to contraction of the ductal muscle tissue and thus to closure of the duct. The process of closure starts at the level of the pulmonary artery and emerges to the aortic part. It usually takes minutes to hours, in individuals it may take up to a week (1).

In patients with a PDA the amount of smooth muscle tissue within the duct is significantly lower and replaced by elastic, non-contractile fibers leaving the DA unable to constrict (1,10,13,14). Depending on the amount of muscular tissue one defines different grades of PDA (1):

- in **grade 1-2** the pulmonic part of the ductus is closed and only a small aortic diverticulum is left, leaving the patient without any symptoms
- in **grade 3-5** up to 50% of the muscular tissue is replaced by elastic fibers, leading to a common PDA with left-right shunting
    - severity of left ventricular volume overload is depending on the size of the shunt with mild in grade 3, moderate in grade 4 and severe in grade 5
- in **grade 6** more than 50% of the muscular tissue is replaced by elastic fibers leaving behind a large PDA lesion which causes right-left shunting
    - in these patients no narrowing of the pulmonic part of the PDA can be visualized, postnatal pulmonary hypertension occurs, leading to reverse shunting

Previous histologic studies in Miniature Poodles with hereditary PDA identified varying degrees of hypoplasia and asymmetry of ductus-specific smooth muscle and the presence of aorta-like elastic tissue in the ductus wall enable to contraction and thus closure. To determine if similar structural abnormalities cause PDA in other dogs, histology of ductal architecture was studied in 8 non-Poodle purebred dogs with PDA with no immediate family history of PDA. Essentially the same morphologic abnormalities were observed in 7 of 8 dogs with PDA as those in dogs known to have a hereditary form of PDA. These findings suggest that apparently sporadic PDA in these breeds is caused by a genetic defect in the structure of the ductus arteriosus that is similar or identical to that in the Poodle (14).

Shunt flow through the PDA is depending on systemic and pulmonic resistance and thus the pressure gradient of those two compartments. Expecting a physiologically higher pressure in the aorta than in the pulmonary artery, this leads to a left-right shunt. The shunt volume and velocity are also dependent on the anatomical feature and the size of the PDA. Usually only one narrowing at the level of the PA can

be detected, leading to high shunting volume and velocity. If several regions of narrowing within the PDA are present, this may lead to a lower pressure gradient and thus to a smaller shunt volume (1).

In a patient with only one narrowing and a normal size shunt, blood will flow from the aorta through the PDA to the pulmonary artery, through the lungs and via the left atrium and left ventricle back into the ascending aorta. This leads to volume overload of the left ventricle (eccentric hypertrophy). After a while also left atrial volume overload may occur in untreated patients. Depending on the size of the PDA opening (grade 3,4,5), shunt volume rises eventually leading to congestive left heart failure and lung edema. Also systolic dysfunction may occur. Mitral valve insufficiency may be seen secondary to the large end-diastolic volume of the left ventricle (mitral valves will be pulled away from each other due to enlargement of the left ventricle). The larger end-diastolic volume may cause elevated systolic blood pressure and also higher flow velocity measured at the aortic valve. Depending on the shunting volume a maximum velocity of up to 3.5m/sec may be appreciated within the aorta. On the contrary, the diastolic aortic pressure will drop due to volume loss via the PDA (1).

A reversed PDA (rPDA) is very rarely seen (in only 0,03% of all PDA patients) and usually detected in combination or due to primary pulmonary hypertension. In grade 6 PDA patients the very large duct opening transfers the aortic pressure directly onto the pulmonary system. Thus postnatally pulmonary resistance won't drop as expected and pulmonary pressure will align with aortic pressure probably causing pulmonary hypertension. In very rare cases a primary left-right-shunting PDA might also cause pulmonary hypertension due to constant volume overload and eventually also a reversed right-left-shunt. Patients with a rPDA will show the typical signs of pulmonary hypertension (as right ventricular concentric hypertrophy, small left ventricle, paradox septal movement) on echocardiography. The rPDA itself will then not be visible with color doppler, but will be diagnosed with contrast echocardiography (bubble study): with air bubbles enriched saline is injected into a vein - through the right-left shunt the air bubbles will then spare the lungs being passed from the pulmonary artery directly into the aorta and will then be detected ultrasonographically in the abdominal aorta (1,18).

Clinical presentation and echocardiographic diagnosis

Most of the patients with PDA will be presented with clinical signs of congestive heart failure. Coughing, dyspnea, exercise intolerance, lethargy, premature appearance and poor growth are common features. Only about 25% of the PDA population will stay free of clinical signs up until the age of 12 months. If a patient suffers from rPDA typical signs are collapse, tachypnea, weakness in the hind legs, differential cyanosis and polycythemia (1,6). Cats with PDA however often do not display clinical signs at all and may not have the characteristic physical examination findings typical of PDA in dogs (19).

A PDA can already be detected by auscultation - mostly at a very young age – when a typical continuous loud murmur can be heard in the 3rd left intercostal space at the level of the pulmonary valve or at the base of the sternum. Precordial buzzing can be appreciated in about 80% of the PDA patients. The palpable pulse is usually very strong/hyperkinetic. In patients with rPDA no/or just a very mild murmur will be detected

Electrocardiography usually shows signs of left-ventricular and left-atrial enlargement with a high R-amplitude and a wide P-wave (1,9). Atrial fibrillation and/or (supra)ventricular extrasystoles and tachycardia may also be appreciated (1).

Depending on the shunt volume radiographs reveal generalized cardiomegaly and left atrial

enlargement. In dorsoventral recumbency a widened descending aorta, a dilation of the pulmonary artery base and an enlarged left auricle may be seen – known as “the ductal triad”(1,9).

Echocardiography – especially doppler echocardiography – is the diagnostic modality of choice for diagnosing a PDA with its morphology and dimension and staging its severity.

In 2-D the first morphological change to be visualized will be the left ventricular volume overload (eccentric hypertrophy) with high end-diastolic diameter (in 84% of the patients), whereas left atrial enlargement will be seen in only about 30% of the patients (2,9). Volume overload will correlate with the shunt volume whereas the left ventricle wall thickness will be within reference range (1,2). In some patients bowing of the inter-ventricular and inter-atrial septum towards the right side may be appreciated (2). End-systolic volume may be normal or enlarged. Fractional shortening usually stays normal (and if decreased will be a sign for systolic dysfunction due to high afterload) but will typically not be elevated (2). Also depending on shunt volume, the examiner will be able to appreciate widening of all of the structures that have to deal with the high shunt volume (starting at the pulmonary artery and its branches, going on to the pulmonary veins, the left atrium and ventricle, and eventually also the aorta up to the level of the PDA). The PDA with the pulmonary narrowing and the aortal ampulla might in most of the patients also be visualized – preferably form the left side with a visualization rate of 96%(1,2). A few case studies have been presented showing that a PDA with typical narrowing and ampulla can not always be identified and although the clinical presentation and preoperative echocardiogram in these patients were consistent with a standard PDA, a ductal vessel was not be identified during surgical dissection. In one patient for example necropsy identified a recess within the wall of the aorta communicating with the pulmonary artery via an ostium at the heart base which determined this structure as an intramural PDA (15).

The use of color doppler will eventually verify the presence of a PDA. A red colored jet might be appreciated going from the PDA lesion, which is originated just proximal to the bifurcation of the pulmonary artery, towards the ultrasound probe (towards the pulmonary valve) in some patients (1,2). Within the pulmonary artery aliasing will be present and usually extend from the bifurcation to the pulmonary valve. Pulmonary insufficiency will also be seen. Spectral doppler shows a very classic flow pattern within the pulmonary artery, that is not seen in any other congenital defect. With CW doppler the typical positive continuous flow profile (with a zig-zag or saw-tooth appearance) with a maximum end-systolic velocity of 4,5-6m/sec and a maximum end-diastolic velocity of 4m/sec should be detected(1,2). Best images and highest ductal flows are also obtained from cranial left transverse imaging plane (2). Negative flow may also be continuous from ductal flow that is swirling back towards the bifurcation, making estimation of pulmonary artery velocity less accurate (2). Prolapse of the pulmonary artery valve cusps as ductal flow strikes them might be appreciated (2).

Secondary to the volume overload and high stroke volume, trivial to mild mitral valve insufficiency and aortic insufficiency may be appreciated in up to 60% of the patients using color and/or PW/CW doppler (1,2,9).

Given the typical changes in PDA patients found with echocardiography, vascular catheterization is not necessary to make a diagnosis. The only advantage using catheterization is that the whole anatomic circumstances of the PDA (form of the ampulla, one or multiple narrowings) and pressure gradients as well as shunt volume can be estimated more exactly (1).

Studies show however, that transesophageal echocardiography TEE provides most accurate anatomic information regarding PDA morphology and closely approximated angiographic ductal dimensions. Three-dimensional transesophageal echocardiography (TEE-3D) even provides a full view of the exact

form and shape of the PDA that cannot be replicated with any other echocardiographic technique. A study using TEE-3D also revealed that most PDA have an oval shape in the majority of dogs, which might have to be considered choosing the right closure device (1,2,7,8).

Treatment

There are basically 4 therapeutical approaches to a PDA (1).

1. **No therapy**: If a patient has a very small PDA defect (or even a silent PDA as described in the Pon) with only a very small amount of shunt volume which is hemodynamically not relevant, no therapy is needed.
2. **Symptomatic medical treatment**: In patients where surgical intervention or catheterization is not an option (for example in patients with multiple defects) or in patients that have already gone into congestive heart failure, medical treatment is the treatment of choice to prolong survival time or to stabilize patients before they undergo causal therapy. Depending on the extend of the symptoms, diuretics, pimobendan, ACE-inhobitors, amlodipin and antiarrhythmics might be indicated.
3. **Surgical closure:** In the dog surgical closure of the ductus has become a standard procedure with good results. There is a wide variety of surgical techniques involving different methods of dissection and suture passage. Intraoperative hemorrhage during dissection is the most serious potential complication and can be life-threatening. Different options, as double ligature or hemostatic clips, are possible. Greatest disadvantage is open thorax surgery with higher risk of peri- or post-surgical complications (as severe hemorrhage) and longer recovery time, which nowadays can be minimized using thoracoscopic occlusion instead of open surgery (1,4).
4. **Catheter-guided intervention**: Interventional closure is nowadays the therapy of choice, showing very good results, and at the same it has to deal with lower risks than surgical closure. Depending on the size of the shunt opening different systems for closure (as the Coil or the Amplatzer) are used and placed into the shunt opening trans-venously or trans-arterially by implantation catheter. The implanted system will then cause a mechanical and thrombotic closure of the shunt defect.

The European Cardiology Association has publishes guidelines for the management of congenital heart disease which recommend device closure in all PDA patients with volume overload but without pulmonary hypertension (PH). In patients with PH closure might also be considered if there is still left-right-shunt present. In patient with PH and reverse shunt closure is irrelevant (4,16).

Prognosis

Prognosis depends on the type and/or size of the PDA as well as the treatment. As mentioned above, patients with a very small shunting lesion or a grade 1-2 PDA that does not cause any volume overload, usually do not require treatment and they are not expected to ever show any clinical signs.

The prognosis of patients with grade 3-5 PDA with left-right shunting depends on cardiac function, severity of volume overload, presence of heart failure and also choice of closure (surgical or interventional). The earlier a patient gets treated the better. Best results are expected when the patient is treated within the first 6 months of age, or at least before the patient has gone into cardiac failure. If the patient has already gone into cardiac failure, it needs to be stabilized first. Also these patients

require a longer rehabilitation time post intervention and might need to stay on additive long term medical treatment afterwards as well (1).

A more recent study showed however, that also older patients and patients that have concurrent heart disease - as mitral valve disease - which may complicate a PDA, still benefit from treatment. Even in those patients clinical signs can improve or resolve with PDA closure (16). Clinical signs at presentation, concurrent congenital heart disease, and severe mitral regurgitation due do mitral valve disease however are proven to negatively affect survival (16,17).

Success rate with surgical treatment is 75-85%, given that peri- and post-surgical risks have to be considered. With catheter-guided intervention success rate is 80% when a Coil is used and even 87-100% when the Amplatzer method is used. General complications as embolisation, arrhythmias, bleeding due to perforation, infections or recanalization are seen in less than 20% of the patients treated with a Coil, and only in 3-7% of the patients treated with an Amplatzer. The post-mortality rate of 0-2% in all patients undergone catheter-guided intervention is extremely low (1). A more recent retrospective study showed reviews, that although major complications were more common with surgical ligation, the incidence of minor complications was not significantly different choosing surgical closure or the coil, but less seen with the Amplatzer method. There was no difference in survival between dogs treated with surgical ligation and dogs treated with a catheter guided procedure. Thus according to this study both, surgical ligation and catheterization, are still both equably suitable techniques for PDA closure, although major complications were more common with surgical ligation. The Amplatzer method appears to be the method with the least complications and thus can be considered the safest method (12).

In patients with a reversed PDA (due to grade 6 PDA or any other reason for pulmonary hypertension leading to a reverse shunt) surgical or catheter guided intervention is contra-indicated. With closure of the shunt pulmonary hypertension would rise in these patients causing acute right heart failure and death of the patient. The patient would need to be treated symptomatically. The pulmonary hypertension may be treated with sildanefil. Right congestive heart failure should be treated if present (with diuretics, ACE-inhibitors and eventually pimobendan). To decrease polycythemia the patient can be treated with hydroxyurea, but phlebotomy in intervals of 4-6 weeks should be considered as the symptomatic treatment of choice as it decreases clinical signs in frequency and severity very successfully (1,6,18). A retrospective study of rPDA patients showed that the mean survival time of untreated rPDA patients was 626 days. Dogs with right sided congestive heart failure had a shorter median survival time (58 days). Dogs treated with sildenafil only survived longer (1839 days) but best results were seen in patients treated with phlebotomy (up to 8 years) (1,6,18).

Given the findings that suggest that sporadic PDA in several breeds is caused by a genetic defect in the structure of the ductus arteriosus, the relatives of dogs with PDA, particularly parents, offspring, and siblings, should be screened for evidence of PDA. Dogs with PDA should not be used for breeding, regardless of breed (14).

Conclusion

Nelly's echocardiogram showed typical changes of a PDA with left-right-shunting. Volume overload of the left ventricle, an enlarged pulmonary artery with aliasing in color flow and a typical saw-tooth continuous flow profile with a maximum of 5,45m/sec within the pulmonary artery created by the shunting lesion could be appreciated. In 2D-mode the PDA could be imaged from the left cranial side as well.

Given the clear diagnose, it was recommended to perform a surgical or catheter-guided closure of the PDA with a good longterm prognosis for a normal life after complete closure. Due to high costs of the catheter-guided closure the animal shelter chose for surgical closure. As to my knowledge, the procedure went well and Nelly recovered as expected.

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