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Electrophysiologic features of fetal ventricular aneurysms and diverticula

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Abstract

Objective—Congenital ventricular wall defects are very rare and include congenital ventricular aneurysms (CVAs) and diverticula (CVDs).

Method—We report a series of five fetuses: three with CVAs and two with CVDs referred due to fetal arrhythmia. In addition to routine fetal echocardiography, fetal magnetocardiography (fMCG) was used. The literature in CVA and CVD is reviewed.

Results—Incessant premature ventricular contractions (PVC), mainly bigeminy and trigeminy were found in three fetuses with CVAs and in one with CVD, who also had ventricular couplets. The other fetus with CVD, referred because of PVCs, had only sinus tachycardia. ST elevation was noted in two. Fetal movement had a variable impact on PVC's. Postnatal evaluation demonstrated two persistent left ventricular aneurysms and one persistent right CVD; one CVD resolved at 35 weeks gestation. Two neonates had incessant PVCs. Both arrhythmias resolved spontaneously while being treated with propranolol.

Conclusion—FMCG is complementary to echocardiographic imaging. In fetuses with left ventricular wall defects, additional electrophysiological diagnosis can be made by fMCG,

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including the complexity of ventricular ectopy, arrhythmic response to fetal movement, presence of ST-T wave abnormalities, and atrial amplitude increases. Prenatal risk factor assessment using fMCG can additionally support post-natal treatment and follow-up.

Keywords

electrophysiology; fetal magnetocardiography (fMCG); ventricular aneurysm; fetus; premature ventricular contractions; ventricular diverticulum

Introduction

Congenital ventricular aneurysms and diverticula are uncommon cardiac malformations[1]. Mainly case reports exist in the literature. The first case was reported antenatally by Gembruch and colleagues in a 32 week gestational age (GA) fetus that developed ventricular extrasystoles[2]. Previous published data specified a prevalence of 0.5 in 100.000 born with an equal distribution in gender[3]. Because of the low prevalence of the disease, the two terms congenital ventricular diverticulum (CVD) and aneurysm (CVA) have often been used interchangeably.

CVAs have a loss of integrity of the myocardium and lack of one or more elements of the cardiac muscle. Mostly they are fibrotic and appear sac-like with paradoxical ballooning during ventricular systole. In contrast to these congenital defects, pseudoaneurysms have a portion which is walled off pericardium. Diverticula appear as dilation of the myocardium but with all three muscle layers retained. CVDs are dys- or akinetic and can spontaneously resolve. The etiology of CVAs and CVDs is unknown. An intrinsic abnormality in embryogenesis may lead to a focal defect of the muscular ventricular wall[4]. Aneurysms and diverticula can be acquired in the prenatal period from a viral infection[5], inflammatory diseases, or coronary anomalies with stenosis[6].

In the human adult, CVA and CVD are known to be associated with complex re-entrant ectopy and ventricular tachyarrhythmias, and impart a higher risk of sudden cardiac death. Of 41 fetal cases of CVA and CVD, eight of them (~20%) had cited arrhythmia (Table 1). We report a series of five fetuses presenting clinically with arrhythmias due to left ventricular wall defects, and we review the published literature.

Materials and Methods

Patients

The fMCG records of pregnant women with fetal ventricular wall defects referred to the Biomagnetism Laboratories at the Department of Medical Physics, University of Wisconsin-Madison from 2002 to 2012 were retrieved from our database.

Informed consent was obtained from each participant and the University of Wisconsin Institutional Review Board reviewed and approved the fMCG protocol.

The study included three subjects diagnosed with left ventricular wall aneurysm and two with diverticulum. We called them diverticula when there was as a continuous muscle on all

edges. If they appeared to have interruption of the muscle element leaving only fibrous tissue, we called them an aneurism. The median gestational ages were 33 weeks (Range 22–34 weeks). The fMCG data were re-evaluated by two pediatric cardiologists for rhythm, cardiac time intervals, ST segment abnormalities, and signal amplitudes. Neonatal outcomes were reviewed.

Methods

fMCG is the magnetic analogue of the fetal ECG, but provides better signal quality and favorable signal transmission properties. A 37-channel monoaxial (Magnes, 4D Neuroimaging, Inc., San Diego, Calif., USA) and/or a 21-channel (Tristan Technologies, USA) vector superconducting quantum interference device (SQUID) was used to record the fMCG from the maternal abdomen. A SonoSite M-Turbo (Bothwell, Wash., USA) portable ultrasound scanner equipped with a 60-mm broadband (2–5 MHz) curved array transducer was first used to determine preliminary rhythm, and location of the fetal heart. The SQUID was placed directly above and in direct contact with the mother's abdomen. Four to seven recordings of 10 minutes duration were obtained. Post processing required construction of an actocardiogram (simultaneous tracing of fetal activity and fetal heart rate), using fMCG data, to monitor and characterize fetal movement[7]. Spatial filtering was used to remove maternal interference. All fMCG recordings were reviewed by at least two pediatric cardiologists. Custom Matlab programs were used to measure cardiac time intervals and signal amplitudes. The precision of the electronic measurement calipers was approximately 0.001 sec. The P and QRS amplitude ratios (including both positive and negative components of each wave) were plotted against 68 health normal control fetuses. The ratios in the subjects at < 1 week of age were available in 3 infants during sinus rhythm and 1 during ventricular bigeminy. These were displayed electronically using Muse ECG storage and cardio-analytic system (GE Medical, Milwaukee, WI), and using the superimposition median view, the P:QRS ratio was again obtained using the Limb leads. These ratios, and the neonatal cardiac time intervals, are displayed in Table II. Linear regression was used to assess the dependence of the amplitude parameters on gestational age[8].

Results

Fetal echo and fMCG results

All five fetuses presented to the Biomagnetism Laboratory due to irregular rhythm patterns (Table 2). They showed left ventricular wall defects confirmed by fetal and neonatal echocardiography. Three of them were located in the left ventricular apex, one in the right lateral ventricle, and one in the posterior LV submitral region. Left and right ventricular function was normal in all fetuses except in the region of the wall defect.

Electrophysiologic patterns were complex, and varied considerably. In addition to rare sinus rhythm, four fetuses presented with incessant premature ventricular contractions. One had couplets and bigeminy, two had ventricular bigeminy as the predominant pattern, and one had ventricular trigeminy. Coupling intervals (PVC onset to prior QRS onset) were not stable. One fetus with CVD and history of ventricular ectopy by echocardiography had only sinus tachycardia (Figure 1). Three of four fetuses exhibited a change in PVC response

during fetal movement. In two fetuses the ectopy was suppressed temporarily by fetal activity and in one it was exacerbated (Figure 2).

Four fetuses had normal cardiac time intervals, and the fetus with an extensive right ventricle (RV) aneurysm had QRS prolongation. QTc intervals were impacted by QRS duration, but were not prolonged. Two fetuses had mild ST- elevation. The main statistical data are presented in Table 2. The most striking observation was that P: QRS amplitude ratio in the left-sided aneurysms ranged between 0.22–0.31 (normal value P: QRS ratio = 0.1), indicating early signs of atrial hypertrophy or dilatation, which was not evident by echocardiography. The fetus with RV aneurism had a less dramatic P:QRS amplitude ratio increase of 0.18.

Neonatal echo and electrophysiologic outcome

Postnatal evaluation by echocardiography demonstrated three persistent left ventricular aneurysms and one CVD. One CVD resolved at 35 weeks gestation. Two neonates had incessant PVCs after birth and were treated with propranolol (2–3 mg/day) for 3 and 9 months (Figure 3) and the remainder were not treated. Of the two with ST elevation pre-natally, one had persistent ST elevation postnatally. Neonatal P:QRS ratio's were available in 3 of 5 subjects, and were not as large as fetal ratios. In one neonate (case 1) the P:QRS during ventricular bigeminy was larger than 1 week later during sinus rhythm. In no case, however was the postnatal P:QRS ratio larger than the fetal ratio.

Intermediate-term follow - up

Follow- up examinations were available in all infants at age ranges from 3–35 months. One fetus showed LV CVD resolution in utero. In two infants, left ventricular wall defects were stable, whereas in the other two infants (RV-1, LVapex-1) the defects became smaller. Therefore surgical resection was not necessary in any patient. Arrhythmias resolved in all subjects, confirmed by Holter monitoring. One infant with an LV apical aneurism and persistent ventricular arrhythmia had moderate global LV hypokinesis at birth which improved (except in the region of the aneurism) over 3 weeks. No neonatal deaths have occurred.

Discussion

The main findings in this study are that left or right ventricular wall defects in utero are associated with a high incidence of arrhythmia (5 in 5 cases), specifically premature ventricular contractions. All subjects had PVCs—four of them in utero and one postnatally. The one with PVCs postnatally, had a history of echo-documented PVCs and fMCG-documented sinus tachycardia in utero. One could speculate that the tachycardia had suppressed the PVCs in utero. In addition to the PVCs, we found couplets, bi/trigeminy, atrial amplitude increases and ST elevation. QRS prolongation was noted in one fetus. Ventricular wall defects in our series were not associated with QT prolongation.

The combined use of fetal MCG and echocardiography detected not only a higher frequency of ectopy and other conduction abnormalities (tachycardia, ST-T changes, bundle branch block, and atrial hypertrophy), but also the size, morphology, and contraction characteristics

of the CVAs and CVDs. Both methods complement one another in prenatal cardiac monitoring.

The clinical outcome in our series was far better than has been reported in the literature (Table 1), which is largely comprised of case studies. No patients experienced intrauterine fetal demise, cardiac arrest, or sudden infant death, and in several patients, fetal CVAs and CVDs regressed over time. While only non-malignant arrhythmias were detected in this series, we speculate that arrhythmic deaths can be one of several mechanisms of fetal demise. Complications of ventricular wall defects such as hydrops, large pericardial effusions, and poor ventricular function, may all increase the likelihood of unstable arrhythmias when an arrhythmic substrate exists. Lethal arrhythmias may go undetected in echo/Doppler imaging due to its intermittency, whereas monitoring by fMCG allows continuous capture of all QRS complexes, similar to cardiac telemetry. In consequence, if the arrhythmia can be characterized, premature delivery can be avoided and if neonatal cardiac output can be maintained, surgical resection may not be necessary. Different treatment strategies for fetal arrhythmias, according to the precise electrophysiological findings, are recommended by Donofrio and colleagues in the new Scientific Statement of the American Heart Association “Diagnosis and Treatment of Fetal Cardiac Disease”[9].

Li and colleagues have established P and QRS amplitude values in normal subjects by fetal magnetocardiography[8]. Increased amplitudes were found in subjects with fetal arrhythmias. This implied that arrhythmias in utero can be accompanied by hypertrophy, similar to the MCG and ECG findings, and that these may include atrial hypertrophy. Atrial hypertrophy is a critical adaptation of the fetus during low cardiac output states such as those associated with severe acute bradycardia[8]. It commonly accompanies incessant arrhythmias with AV dissociation where the atrium contracts against a closed AV valve. In this study we found four of five patients with elevated P and QRS amplitudes. A lack of increase was found in the fetus with sinus tachycardia. P:QRS amplitude might initially be increased, and the incessant tachycardia might be the resolution as another sign of recovery of the heart, since this fetus’s CVD resolved in utero. Similar to the findings of Li and colleagues[8], fMCG evidence of atrial hypertrophy was not associated with visible atrial hypertrophy or distension during sinus rhythm. The neonatal ECGs in three (cases 1, 2, and 3), for whom measurements could be measured using superimposition views, were not associated with significant P:QRS amplitude increase, except in one fetus (Fetus 2) where the P:QRS amplitude ratio during ventricular bigeminy significantly increased compared with sinus rhythm (though not to ratios seen prenatally in that fetus). These findings suggest, that the flow dynamics of transitional circulation, or of acute hemodynamic changes during bigeminy and other conduction disturbances, likely impact the atrial signal size relative to the ventricle during normally conducted beats in the neonate.

Ventricular aneurysms appear to be heterogenous, and this may account for the variable post-natal findings and outcomes. In support of this, the electrophysiologic characteristics also appeared to be heterogenous. ST elevation was observed in only two of five, sinus tachycardia in one. In two fetuses, the ectopy appeared to suppress with fetal movement Changes in autonomic activity based on the gestation of the fetus might also influence fetal

presentation. The timing of resolution of the arrhythmias also varied with birth being an important transition point.

Conclusion

In summary, we have shown for the first time that precise electrophysiological diagnosis of CVA's and CVDs can be made in utero by using fMCG. This procedure should be considered, in addition to fetal echocardiography, in fetuses with ventricular aneurysms or diverticula to characterize the extent of ventricular arrhythmias, predict prognosis, and assess potential need for antiarrhythmic treatment post-natally. CVAs and CVDs appear to have a favorable prognosis and conservative prenatal management is advised. The P:QRS amplitude abnormalities, QRS duration, and ST-T wave abnormalities may be useful in further risk-stratification.

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List of abbreviations

fMCG	fetal magnetocardiography
SQUID	superconducting quantum interference device
CVD	congenital ventricular diverticulum
CVA	congenital ventricular aneurysm
PVCs	premature ventricular contractions
fCTIs	fetal cardiac time intervals
GA	gestational age

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Bulleted Statement

In the human adult, CVA and CVD are known to be associated with complex re-entrant ectopy and ventricular tachyarrhythmias, and impart a higher risk of sudden cardiac death. In this study we could show, that in fetuses with left ventricular wall defects, additional electrophysiological diagnosis can be made by fetal magnetocardiography, including the complexity of ventricular ectopy, arrhythmic response to fetal movement, presence of ST-T wave abnormalities, and atrial amplitude increases.

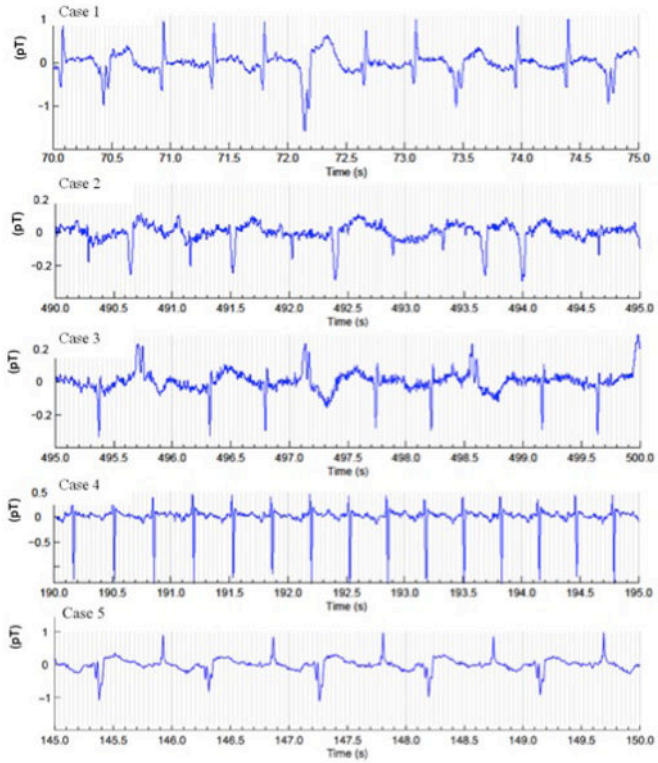

Figure 1. fMCG findings (Case 1–5)

Four fetuses presented incessant premature ventricular contractions. Trigeminy is demonstrated in Cases 1 and 3. Ventricular couplets are noted in Case 2, and bigeminy is demonstrated in Cases 2 and 5. Case 4 had sinus tachycardia. Case 1 and 4 demonstrate ST elevation.

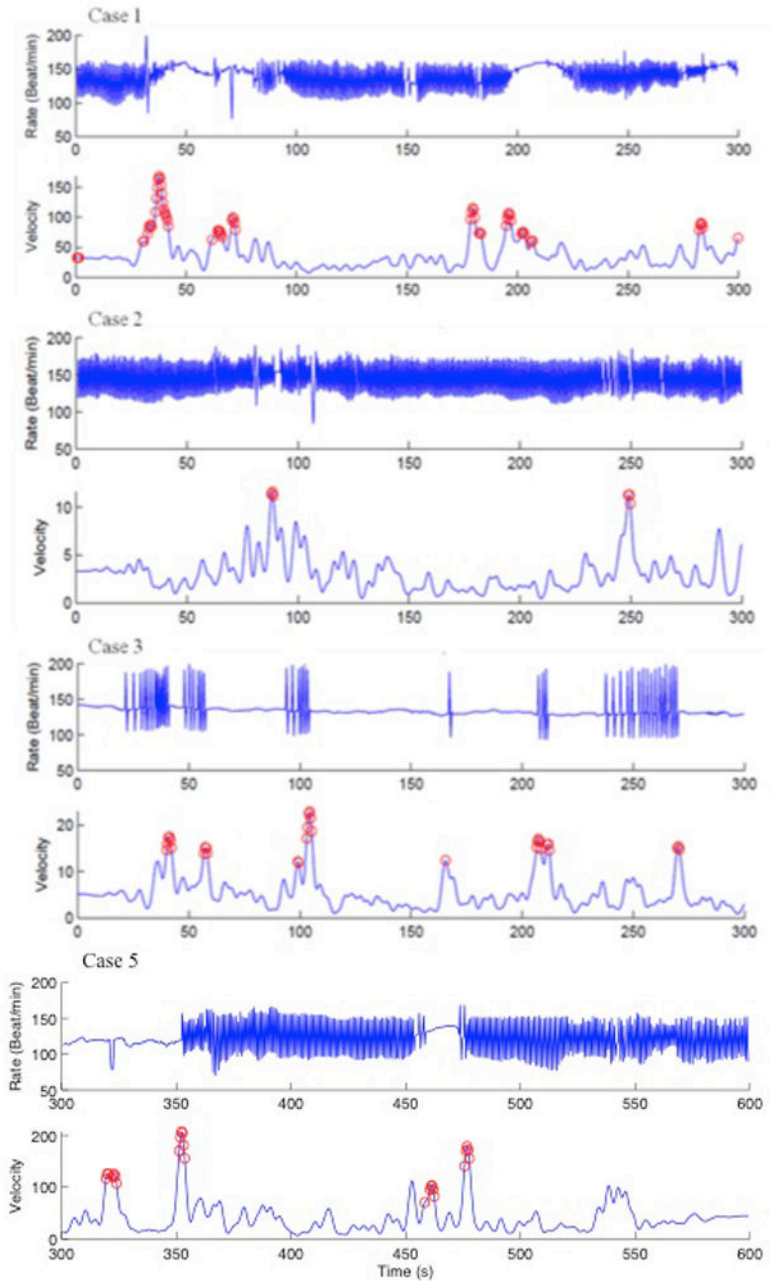

Figure 2. PVC response during fetal movement (Cases 1–3, 5)

For each case, the top panel shows the heart rate trend (Rate, beats/min) over five minutes, and the bottom panel (velocity) shows the fetal actogram, with maximum slope activity marked by open circles. In Cases 1 and 5, fetal movement abolished PVCs, Case 2 showed no effect, while in case 3 fetal movement induced PVCs.

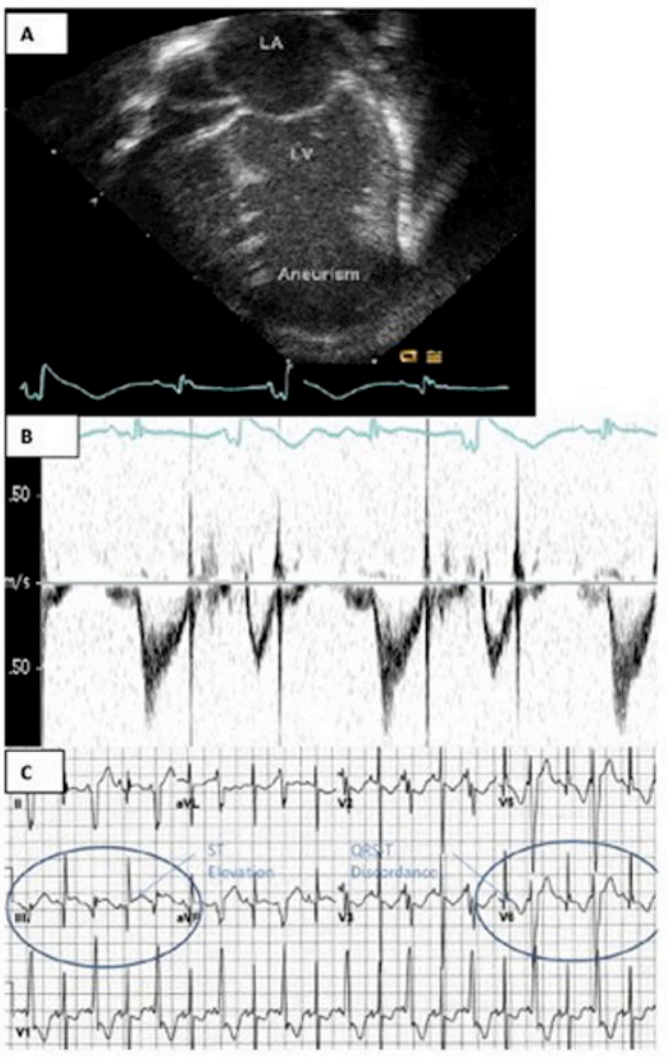

Figure 3. P:QRS ratio

All 4 cases with PVCs (shown as X's) compared to healthy controls (black boxes).

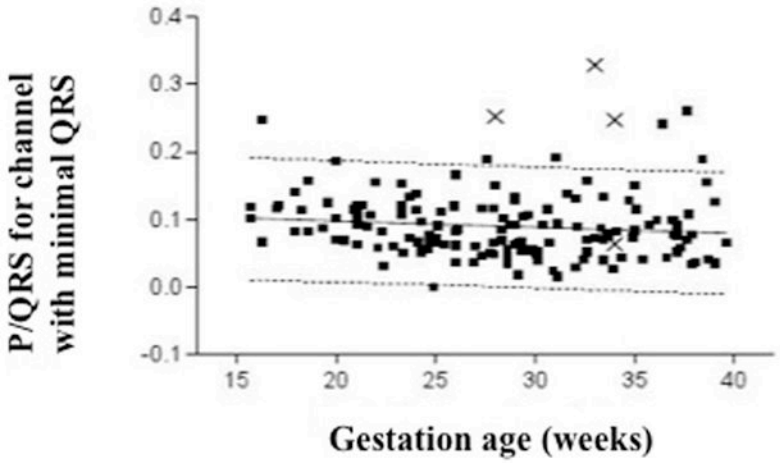

Figure 4.

Postnatal findings of a left ventricular aneurysm (Case 1): a) Neonatal echocardiography b) Neonatal Doppler c) ECG. Areas of ST elevation of the sinus QRS (Lead II) and reciprocal T wave inversion (V5) are shown within the ovals.

Table 1

Literature review

of fetal presentation of ventricular aneurysms and diverticuli

Reference	GA (wks)	Location	Symptoms & Signs	Medication	Surgery cited	Outcome
Balakumar et al.[10]	24	Aneurysm	Abnormal 4-chamber view, mitral & tricupsid regurgitation	None cited	No	N/A
Barberato et al.[11]	16	Apical aneurysm	Pericardial effusion	None cited	No	IUFD (37 wks)
Bernasconi et al.[12]	24	Submitral diverticulum	Pericardial effusion	None cited	No	IUFD (26 wks)
Brachlow et al.[13]	32	Apical diverticulum	Normal cardiac function	None cited	No	Clinically well (3 yrs)
Carles et al.[14]	13	Apical diverticulum	Pericardial effusion	None cited	No	TOP (14 wks)
Cavalle-Garrido et al.[15]	18	Apical aneurysm	Abnormal 4- chamber view	Digoxin	No	Clinically well (2 yrs)
	19	Sub-mitral aneurysm	Abnormal 4-chamber view, hydrops	None cited	No	IUFD (31 wks)
	21	Apical aneurysm	Abnormal 4-chamber view	None cited	No	Clinically well (2 yrs)
	20	Submitral diverticulum	Pericardial effusion, hydrops	None cited	No	IUFD (26 wks)
Cesko et al.[16]	17	Apical diverticulum	Pericardial effusion	None cited	No	TOP (22 wks)
Chaubal et al.[17]	21	Apical aneurysm	Abnormal 4-chamber view	None cited	No	IUFD (27 wks)
Chiang et al.[18]	34	Apical aneurysm	Cardiomegaly	None cited	No	Clinically well (1 yr)
El Kady et al.[19]	17	Apical aneurysm	Pericardial effusion	Nifedipine and methyldopa-mother's hypertension	No	IUFD (27 wks)
Fujita et al.[20]	26	Apical aneurysm	Arrhythmia (ventricular extrasystoles)	None cited	No	Clinically well (2 yrs)
Gembruch et al.[2]	32	Apical aneurysm	Arrhythmia (ventricular extrasystoles), dilated left ventricular	Propafenone for 4 months Antithrombotic prophylaxis with low-dose aspirin	No	Clinically well (2 yrs)
Espinoza et al.[3]	29	Apical aneurysm	Low-lying placenta, cardiac failure	Digoxin	No	IUFD (31 wks)
Hornberger et al.[22]	25	Apical aneurysm	Premature atrial contractions	Only abstract available	No	Clinically well (10 mo)
	28	Apical aneurysm	Premature atrial contractions	Only abstract available	No	Clinically well (4 mo)
Jacobson et al.[23]	33	Free wall aneurysm	Pericardial effusion	None cited	Yes (8.5 mo)	Clinically well
Kitchiner et al.[24]	33	Apical diverticulum	Cardiomegaly, mitral regurgitation	None cited	No	Clinically well
Lupoglazoff et al.[25]	28	Apical aneurysm	Abnormal 4-chamber view	Only abstract available	No	Neonatal death
Marijon et al.[26]	>26	Apical aneurysm	Left ventricular dysfunction	None cited	No	Perinatal death (1 d)
	>26	Apical aneurysm	Normal cardiac function	None cited	No	Perinatal death (1 d)

Reference	GA (wks)	Location	Symptoms & Signs	Medication	Surgery cited	Outcome
Matias et al.[27]	21	Apical aneurysm	Pericardial effusion	Only abstract available	No	Perinatal death
	22	Free wall aneurysm	Pericardial effusion	Only abstract available	No	TOP (23 wks)
	26	Apical aneurysm	Pericardial effusion, cardiomegaly	Only abstract available	No	IUFD (33 wks)
McElhinnery et al.[28]	22	Apical aneurysm	Hydrops	Only abstract available	No	TOP (23 wks)
	39	Apical aneurysm	Asymptomatic	Only abstract available	No	Clinically well
Nam et al.[29]	21	Apical diverticulum	Cardiac malformation	None cited	No	TOP (22 wks)
Papagiannis et al.[30]	23	Free wall aneurysm	Multiple congenital anomalies	Only abstract available	No	TOP (23 wks)
	32	Apical aneurysm	Arrhythmia	Only abstract available	No	Clinically well (7 yr)
Patel et al.[31]	25	Apical aneurysm	Abnormal 4-chamber view	Digoxin and Captopril	No	Clinically well (3.5 mo)
Prefumo et al.[34]	12	Free wall diverticulum	Pericardial effusion	None cited	No	Clinically well (17 mo)
Pipitone et al.[32]	25	Apical aneurysm	Abnormal 4-chamber view, mitral regurgitation	None cited	No	Clinically well (1 yr)
Pradhan et al.[33]	28	Apical diverticulum	Abnormal 4-chamber view, arrhythmia (ventricular extrasystoles)	Digoxin	No	Clinically well (1 yr)
Seo et al.[35]	21	Apical aneurysm	Abnormal 4-chamber view, mitral regurgitation	None cited	Yes°	Clinically well (21 mo)
Sepulveda et al.[36]	19	Apical aneurysm	Hydrops, Abnormal 4-chamber view	Only abstract available	No	TOP (19 wks)
Sharma et al.37	29	Apical aneurysm	Pericardial effusion, cardiomegaly	None cited	Yes (3 d)Δ	Clinically well (4 mo)
Sherman et al.[38]	24	Apical aneurysm	Pericardial effusion	Digoxin	No	IUFD (31 wks)
Tsujimoto et al.[39]	37	Apical diverticulum	Arrhthmia (ventricular bigeminy)	None cited	No	Clinically well (2 yrs, 8 mo)
Weichert et al.[1]	32	Free wall aneurysm	Arrhythmia (ventricular extrasystoles), cardiomegaly	None cited	No	Clinically well (2 yrs)

°modified Damus-Kaye-Stansel

Δresection of the aneurysm

Table 2

Pre and postnatal echo and fMCG findings in CVA and CVD

Case	GA (weeks)	Location and Echo findings	fMCG findings	fCTIs (ms)	Neonatal Outcome nCTI's (ms)	Follow-up
1	33	CVA, LV apex, moderate wall defect with adjacent hypokinesis	PVCs bigeminy, mild ST elevation Fetal movement suppressed ectopy large amplitude P wave	RR= 421 PR= 58 QRS= 61 QTc =447 P:QRS Ampl. 0.329:1	irregular PVCs, QRST discordance, ST elevation Transient global LV dysfunction P:QRS Ampl ratio ECG −0 days, bigeminy = 0.211:1 ECG −7 days, Sinus rhythm P:QRS 0.078:1 RR=491 PR=98 QRS=66 QTc=481	2 ½ year: aneurysm smaller arrhythmia resolved, propanolol 2mg/kg/day until 6 months of age
2	28	CVD, LV Submitral	PVCs, couplets minimum change in ectopy with movement large amplitude P wave	RR= 488 PR= 99 QRS= 44 QTc =391 P:QRS Ampl. 0.252:1	PVCs spontaneously resolved ECG at 0 days Sinus rhythm, RR=451 PR=156 QRS=54 QTc=429 P:QRS Ampl ratio 0.17:1	3 months: abnormal segment of the left ventricular wall no arrhythmia
3	34	CVA, LV apex, normal cavity size and function	PVCs trigeminy ectopy aggravated by fetal movement large amplitude P wave	RR= 423 PR= 124 QRS= 59 QTc =390 P:QRS Ampl. 0.247:1	Sinus rhythm, no arrhythmia ECG at 0 days RR=540 PR=110 QRS=62 QTc=427 P:QRS Amp ratio 0.066:1	2 ½ months: unchanged aneurysm no arrhythmia
4	34	CVD, LV apex, large wall defect	sinus tachycardia, mild ST - elevation	RR= 309 PR= 102 QRS= 42 QTc =349 P:QRS Ampl. 0.063:1	sinus rhythm, normal conduction intervals, frequent monomorphic PVCs ST elevation	15 months: aneurysm smaller arrhythmia resolved, propanolol 2 mg/kg/day until 9 months of age
5	22	CVA, RV lateral, large wall defect	PVCs bigeminy Fetal movement suppressed ectopy	RR= 478 PR= 96 QRS= 77 QTc= 446 P:QRS Ampl. 0.182:1	Sinus rhythm, Few PVC's	3 months: aneurism smaller Arrhythmia resolved at birth. No medication.

Abbreviations: Amplitude (Ampl), fetal cardiac time intervals (fCTIs), congenital ventricular diverticulum (CVD), congenital ventricular aneurysm (CVA), premature ventricular contractions (PVCs), left ventricle (LV)