



Published in final edited form as:

*J Perinatol.* 2016 October ; 36(10): 823–827. doi:10.1038/jp.2016.90.

## Maternal arrhythmia and perinatal outcomes

Dana Henry, MD<sup>1</sup>, Juan M Gonzalez, MD<sup>2</sup>, Ian, S. Harris, MD<sup>3</sup>, Teresa Sparks, MD<sup>2</sup>, Molly Killion, RN, CNS<sup>2</sup>, Mari-Paule Thiet, MD<sup>2</sup>, and Katherine Bianco, MD<sup>4</sup>

<sup>1</sup>Department of Obstetrics and Gynecology, California Pacific Medical Center, San Francisco, California

<sup>2</sup>Department of Obstetrics, Gynecology & Reproductive Sciences, University of California, San Francisco. San Francisco, California

<sup>3</sup>Department of Internal Medicine, University of California, San Francisco. San Francisco, California

<sup>4</sup>Department of Obstetrics and Gynecology, Stanford University, Stanford, California

### Abstract

**Objective**—To determine if arrhythmia in the setting of maternal cardiac disease (MCD) affects perinatal outcomes.

**Study Design**—This is a retrospective cohort study of pregnant women with MCD who delivered from 2008 to 2013. Perinatal outcomes among women with an arrhythmia were compared to those without.

**Result**—Among 143 women; 36 (25%) had an arrhythmia. Those with an arrhythmia were more likely to have a spontaneous vaginal delivery (64% vs. 43%,  $p < 0.05$ ) and required fewer operative vaginal births (8% vs. 27%,  $p=0.02$ ). Pregnancies were more likely to be complicated by IUGR (17% vs. 5%,  $p < 0.05$ ) although there were no differences in the rate of small for gestational age. The risk of IUGR remained increased after controlling for confounding (aOR 6.98, 95% CI 1.59–30.79,  $p=0.01$ ). Two cases of placental abruption were identified among mothers with arrhythmia while none were identified in the controls ( $p < 0.05$ )

**Conclusion**—Patients with arrhythmias were more likely to have a spontaneous vaginal delivery. Our data suggests that these pregnancies were an increased risk for IUGR.

### Introduction

Maternal cardiac disease is a major contributor to maternal morbidity and mortality (1), leading to calls for a specialized multidisciplinary care approach for these women (2). Among those with cardiac disease, women with an arrhythmia have increased risk for cardiac complications in pregnancy (3). It has also been suggested that the alterations in

---

Users may view, print, copy, and download text and data-mine the content in such documents, for the purposes of academic research, subject always to the full Conditions of use:[http://www.nature.com/authors/editorial\\_policies/license.html#terms](http://www.nature.com/authors/editorial_policies/license.html#terms)

**Corresponding Author:** Dana Henry, MD, California Pacific Medical Center, Perinatal Services, 3700 California Street, San Francisco, CA 94118, Phone: (415) 515-0374, Fax: (415) 600-2376, [danaemhenry@gmail.com](mailto:danaemhenry@gmail.com).

Conflicts of Interest: The authors have no conflicts of interest to disclose.

maternal physiology during pregnancy including increased heart rate and cardiac output, reduced systemic vascular resistance, increase plasma catecholamine and adrenergic receptor sensitivity, atrial stretch, and increased end-diastolic volume due to intravascular volume expansion, may exacerbate or trigger maternal arrhythmias through the electrophysiological effects of hormones, hemodynamic changes, and effects on underlying heart disease (4). Furthermore, these alternations in hemodynamics may have effects on the developing fetus, and it is well documented that several anti-arrhythmic medications may have teratogenic potential, and thus must be used with extreme caution in pregnancy (4–5).

Despite the established literature that maternal cardiac disease poses increased maternal risk during pregnancy, the degree of fetal and neonatal risk is less well understood. In the large prospective study of women with cardiac disease (CARPREG), Siu and colleagues found that NYHA class >II or cyanosis, smoking, multiple gestation, maternal left heart obstruction, and use of anticoagulants during pregnancy were predictive of adverse neonatal events (3). Though these factors were associated with poor neonatal outcomes, the relationship to specific diagnoses is not clear. There is little available research evaluating the risks associated with specific types of cardiac disease, and few evidence-based recommendations regarding the differential management of these patients (6–9).

Thus, we sought to determine if women with a cardiac arrhythmia are at increased risk for adverse perinatal outcomes compared to women with other types of cardiac disease who do not also have an arrhythmia. Evaluating outcomes among these patients may aid in the identification of specific risk factors or disease features associated with adverse outcomes, suggesting potential intervention that could ultimately lead to improved maternal, obstetric and neonatal outcomes.

## Materials and methods

We designed a retrospective cohort study of pregnant women with maternal cardiac disease delivered at the University of California, San Francisco between August 2008 and May, 2013. We included all women who were cared for by the Pregnancy and Cardiac Treatment (PACT) program, a multidisciplinary group consisting of Perinatology, Cardiology, Anesthesiology, and Nursing specialists. Women cared for in this program include those with primary arrhythmia, congenital heart defects, or noncongenital structural heart defects (e.g. valvular heart disease or cardiomyopathy). Antenatal, intrapartum, and postpartum management of these patients was discussed on a monthly basis, and care recommendations were provided to the managing clinicians. For women who had multiple pregnancies during the study period, only the first pregnancy was included. Resident and attending physicians managed all patients in our cohort according to institutional standards of care. Resident physicians under the direct supervision of attending obstetricians performed all deliveries. The Committee on Human Research approved this study at the study institution.

Maternal demographic information, labor characteristics and outcomes were collected prospectively at the time of delivery by the managing clinicians and maintained within a perinatal database. Similarly neonatal outcomes were collected and recorded by the managing pediatricians. This information was abstracted into maternal and neonatal

datasets, respectively. The maternal and neonatal databases were then linked using two unique identifiers and crosschecked for complete linkage. Additionally, trained abstractors performed daily chart review and data abstraction to ensure information accuracy and to minimize missing data. The database also underwent monthly review by trained physicians for quality assurance. In addition to information obtained through this perinatal database, information regarding specifics of cardiac diagnoses was obtained through individual chart review. Women were classified as having primary arrhythmias or structural heart defects with or without arrhythmias. Structural heart defects were classified as either congenital or non-congenital, and the congenital defects were further categorized as simple or complex according to definitions outlined by Hoffman, *et al*(7). Simple defects included atrial septal defect, ventricular septal defect, patent ductus arteriosus, aortic stenosis, pulmonic stenosis, and coarctation of the aorta, while complex defects included Ebstein anomaly, conotruncal anomalies (specifically transposition of the great vessels, tetralogy of Fallot, and double outlet right ventricle) and Eisenmenger syndrome. Cardiac diagnoses and classifications were confirmed by an attending Cardiologist.

We compared maternal and neonatal outcomes between women with a diagnosis of a cardiac arrhythmia, and those with cardiac disease and no arrhythmia. Obstetric outcomes of interest included mode of delivery, intrauterine fetal demise (IUFD), preeclampsia, intrauterine growth restriction (IUGR, defined as estimated fetal weight for gestational age less than or equal to the 10<sup>th</sup> percentile), gestational diabetes (GDM), chorioamnionitis, post-partum hemorrhage (defined as estimated blood loss >500 mL after vaginal delivery or >1000 mL after cesarean delivery), need for blood transfusion, clinical diagnosis of placental abruption, and intensive care unit (ICU) admission. Neonatal outcomes included gestational age at delivery, preterm birth less than 37 weeks, birth weight, NICU admission, 5 minute Apgar < 7, and small for gestational age (SGA). Comparisons were made using chi-squared and t-tests for univariable outcomes as appropriate. In groups with sample size less than 30, the Fisher's test was used. We performed multivariable logistic regression to control for potential confounding, using those patients who did not have any arrhythmia as the reference group. Covariates included in the regression model were: maternal age, parity, gestational age, chronic hypertension, and mode of delivery. We analyzed the data as composite maternal outcomes which included diagnosis of pre-eclampsia, IUGR, IUFD and abruption placenta, and for neonatal composite outcomes included: preterm birth, SGA, 5 minute Apgar < 7, small for gestational age (SGA), umbilical artery pH < 7.0, NICU admissions. We defined statistical significance if  $p < 0.05$  or if 95% confidence interval (CI) did not include unity. Statistical analysis was performed using STATA v11.0 (StataCorp, College Station, TX).

## Results

There were 143 women included in the study. Of these, 36 (25%) carried a diagnosis of a cardiac arrhythmia. The distribution of these diagnoses is illustrated in Figure 1. The most common arrhythmia diagnosis was supraventricular tachycardia (18 cases (50%)). Other arrhythmias included atrial fibrillation, ventricular tachycardia, Wolf-Parkinson White syndrome, and arrhythmogenic right ventricular dysplasia. In addition, sixteen (44%) patients with arrhythmia also carried a diagnosis of an underlying structural lesion. The

remainder of the cohort was comprised of women without an arrhythmia diagnosis but with congenital or acquired cardiac lesions. The most common diagnosis among this group was mitral valve disease, followed by atrial septal defects, aortic valve disease, and transposition of the great arteries. A full list diagnoses is provided in Supplementary Table 1.

The demographics of the cohort are described in Table 1. Women who carried a diagnosis of an arrhythmia did not significantly differ in mean age, ethnicity, or pre-pregnancy BMI as compared with women without arrhythmia. Women with arrhythmia were less likely to be nulliparous, 39% vs. 61% ( $p=0.02$ ). There were no differences in the rates of prior cesarean delivery between the two groups. Similarly, the rates of other medical comorbidities such as chronic hypertension and diabetes did not differ between the two groups of women. Not surprisingly, women with an arrhythmia were less likely to have a congenital cardiac defect (31% vs. 70%,  $p < 0.001$ ), or a structural lesion (44% vs. 89%,  $p < 0.001$ ). The frequency of complex structural lesions (19% vs. 36%), cardiomyopathy (19% vs. 9%), and conotruncal abnormalities (17% vs. 18%), was not significantly different between the groups.

Next, we compared mode of delivery between women with arrhythmia to those without. These results are displayed in Figure 2. Women with an arrhythmia were more likely to have a spontaneous vaginal delivery, 64% compared to 43% ( $p=0.03$ , OR 2.35 (1.08–5.07)). This appeared to be accounted for by an increase in spontaneous vaginal delivery, as women with arrhythmia were less likely to require any type of operative vaginal delivery (8% vs. 27%,  $p=0.02$ , OR 0.24 (0.07–0.90)) and forceps assisted vaginal delivery (5% vs. 21%,  $p=0.04$ , OR 0.23 (0–.92)). The rates of vacuum assisted vaginal delivery and cesarean delivery were similar between the two groups.

Rates of adverse obstetrical outcomes are displayed in Table 2. Remarkably, women with arrhythmia were significantly more likely to have a diagnosis of intrauterine growth restriction (IUGR) than those with other types cardiac disease (17% vs. 5%,  $p=0.02$ , OR 4.08 (1.23–13.54), and placental abruption (6% vs. 0%,  $p=0.01$ ). The increased risk of fetal growth restriction remained statistically significantly elevated in a multivariable logistic regression model controlling for gestational age at delivery, maternal age, and hypertension, aOR 6.98 (1.59–30.79). Other outcomes evaluated, including fetal demise, preeclampsia, gestational diabetes, chorioamnionitis, postpartum hemorrhage, need for blood transfusion and ICU admission were not significantly different between the two groups. Despite these differences, there were no statistically significant differences in the rates of adverse neonatal outcomes (Table 3) among women with and without arrhythmia. Interestingly, despite differences in the diagnosis of IUGR, there did not appear to be a statistically significant difference in the birth weights of neonates between the two groups, or a difference in the rates of small for gestational age (SGA) infants.

With regards to maternal composite outcomes in a multivariable analysis controlling for maternal age and parity, we found no difference in the group of patients affected with arrhythmia (OR: 2.03, CI: 0.8–5.2;  $p=0.14$ ). In addition, we investigated neonatal composite outcomes in our cohort. We found no difference in the multivariable analysis controlling for maternal age, parity and mode of delivery. However, we observed an increased risk for

adverse neonatal outcomes if the infant was delivered by cesarean section (OR: 4.3, CI: 2.0–9.4  $p < 0.001$ ).

Finally, we evaluated the rates of adverse maternal and neonatal outcomes among only those women with a diagnosis of a cardiac arrhythmia. We compared outcomes based upon method of treatment and well as presence of concurrent structural disease. Among women with an arrhythmia, 25 (69%) required treatment during the pregnancy. Of those, 17 (47%) received medication, 8 (22%) required ablation procedures during or prior to the pregnancy, 3 (8%) had an ICD in place, and 2 (6%) had pacemakers. Rates of adverse obstetric and neonatal outcomes were not statistically different among these women depending upon arrhythmia treatment modality, nor were outcomes different depending upon the presence of concurrent structural disease (Table 4). Lastly, we compared all patients treated with beta-blockers ( $n=15$ ) in our cohort, with patients not requiring beta-blockers ( $n=21$ ). We investigated maternal outcomes such as placental abruption, ICU admissions, preterm delivery, and IUGR. The data didn't show statistical significance. We addressed neonatal outcomes such as SGA, 5 min Apgar's score and NICU admission. We found similar results, the data didn't show statistical significance. However, we considered that we were underpowered to find any differences within this small sub-group.

## Discussion

We undertook a retrospective cohort study of women with cardiac disease in pregnancy to evaluate the effect of arrhythmia on obstetric and adverse maternal and neonatal outcomes. Our study illustrates that women with an arrhythmia were more likely to achieve a spontaneous vaginal delivery than those women without an arrhythmia. Of note, however, women with an arrhythmia were also more likely to be parous, which likely contributed to the increased odds of achieving a vaginal delivery. Importantly, we found that women with cardiac disease that also had arrhythmia had a significantly increased risk of intrauterine growth restriction and placental abruption. However, other adverse maternal or neonatal outcomes were not significantly different between these two groups of women.

There is biologic plausibility to suspect that cardiac arrhythmia may affect placentation and subsequent fetal-placental development leading to our observation of intrauterine growth restriction and placental abruption(4). Placental abruption was very rare in our cohort of women, and thus our estimate of the association with cardiac arrhythmia and placental abruption may be unstable. In our cohort, only two women experienced placental abruption, both of which also had an arrhythmia. Both patients developed significant vaginal bleeding in labor, and a clinical diagnosis of abruption was made. It will be important therefore, to look closely at this association among a larger cohort of women where more outcomes may be observed to further characterize the relationship between arrhythmia and placental abruption.

Regarding fetal growth, previous work has described an association with antiarrhythmic medication and fetal growth restriction (4,8–9). In a recent study of beta-blocker therapy among women with cardiac disease in pregnancy, Ersboll et al. (10) found that beta-blockers were independently associated with increased risk of delivering an SGA infant; our findings

are consistent with these results. Though we did not identify medication treatment as a risk factor for IUGR among our cohort, our sample was limited by small size, and was not powered to detect a potential difference among these women. Yet, the finding of a potential medication effect on fetal growth again underscores the importance of a multidisciplinary approach to determine the optimal management strategy to optimize fetal conditions while assuring that therapy is not withheld in situations where maternal benefit will exceed fetal risk. Although our data suggests a difference in IUGR, we found no difference in SGA. SGA is defined by according to reference population standards if the newborn is constitutionally small but otherwise normal. Alternatively, a fetus can be delayed in fetal growth late in gestation if asymmetric and may not have a reduction in birth weight significant enough to be classified as SGA. The latter suggest a plausible explanation of our observation. Another possibility is antenatal ultrasound measurements can be spurious and associated with measurement error.

Spontaneous vaginal delivery was more common in patients with arrhythmia, with and without congenital or acquired cardiac lesions. Operative vaginal birth was more likely in mothers with congenital or acquired cardiac lesions without arrhythmia. Our study also illustrates that vaginal delivery was successful and appears safe among our cohort of women with cardiac diseases and arrhythmia. These findings are consistent with larger studies and reviews that have suggested that cesarean delivery be reserved for usual obstetric indications as well as those patients with severe heart failure, aortic root dilatation, or aortic dissection (11–12). Among the women in our cohort, those without an arrhythmia had a greater representation of patients with congenital or acquired cardiac lesions and were more likely to undergo operative vaginal birth. This is likely representative of the clinical team's assessment of a patients' ability to tolerate the second stage of labor in the setting of congenital or acquired cardiac lesions. Shortening the second stage of labor by use of forceps or vacuum extraction has been advocated in congenital or acquired cardiac lesions that are preload dependent. However, these recommendations are based on expert opinion. Their benefit has not been systematically studied and research is urgently needed to better understand intrapartum cardiac physiology. We observed that spontaneous vaginal delivery was more common in patients with arrhythmia with and without congenital or acquired cardiac lesions. However, these findings can be associated with the increase use of operative vaginal birth in mothers with congenital or acquired cardiac lesions without arrhythmia.

This study has limitations based on a small and heterogeneous sample of patients. Our small sample may limit our ability to see small differences in rare adverse outcomes. Furthermore, our patients carried a wide variety of cardiac diagnoses limiting our ability to draw conclusions regarding optimal management of specific diagnoses, or the effect of treatment choices on perinatal outcomes among the group of diagnosis or arrhythmia. Though our study attempts to categorize patients by the presence of an arrhythmia, even with this classification, these patients remain a heterogeneous group. It is possible that those with a primary arrhythmia syndrome differ from those with structural disease and concurrent arrhythmia. Among these patients, arrhythmia may be a marker for more severe disease, yet our small sample size limits our ability to detect differences between these small groups. Finally, the retrospective nature of our study limits our ability to understand the etiology of the relationship between cardiac arrhythmia and adverse perinatal outcomes. Prospective

trials among these women are needed to help understand the timing of any potential fetal-placental compromise and guide antepartum treatment decisions.

From a clinical perspective, our study supports the importance of a multidisciplinary approach to the care of pregnant women with cardiac disease. Our approach includes experts from Cardiology, Maternal-Fetal Medicine, Obstetrics, Anesthesiology, and Nursing. For patients with a history of arrhythmias we assure they have been evaluated with transthoracic echo and a baseline electrocardiogram. Pharmacological interventions are used judiciously and administered in the lowest effective dose. The patient and fetus are observed carefully during the pregnancy and delivery. In certain cases, intrapartum and postpartum telemetry may be recommended, and maternal electrolytes are also monitored.

Our study illustrates a potentially important relationship between maternal arrhythmia and intrauterine fetal growth restriction and suggests an association of placental abruption. These findings suggest that women who carry a diagnosis of an arrhythmia with cardiac disease might benefit from increased antenatal fetal growth assessment, including serial ultrasound examinations and antenatal testing. Understanding of this risk may be important for pre-conception counseling as well as antepartum and intrapartum management.

## Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

## Acknowledgments

### Funding:

Dr. Katherine Bianco was supported by the Eunice Kennedy Shiever NIH/NICHD Clinical Investigator Award (K08HD069518-01).

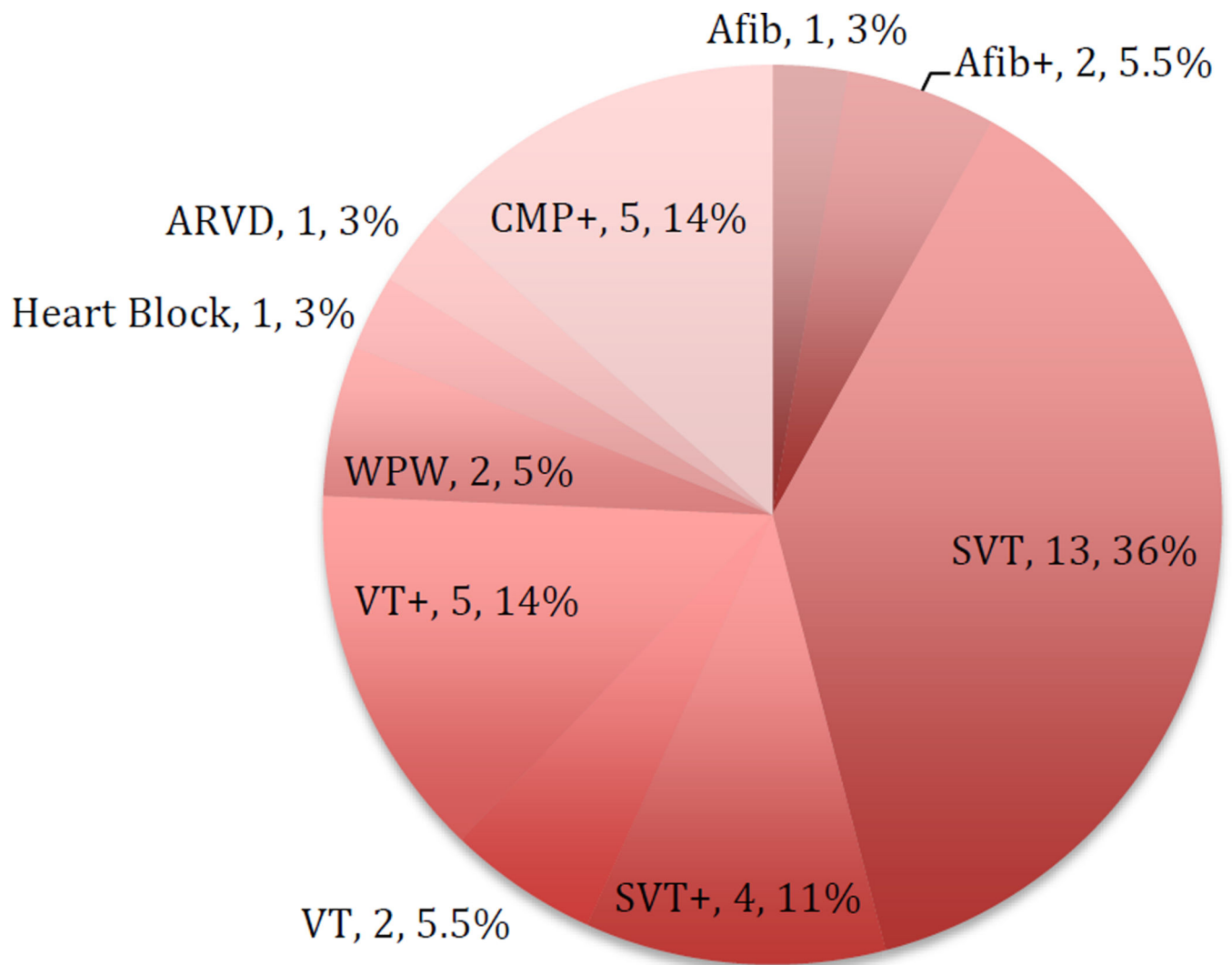
The authors would like to acknowledge Ms. Valerie Bosco, NP; for her invaluable contribution to the study and her tireless dedication to UCSF PACT program patients.

## References

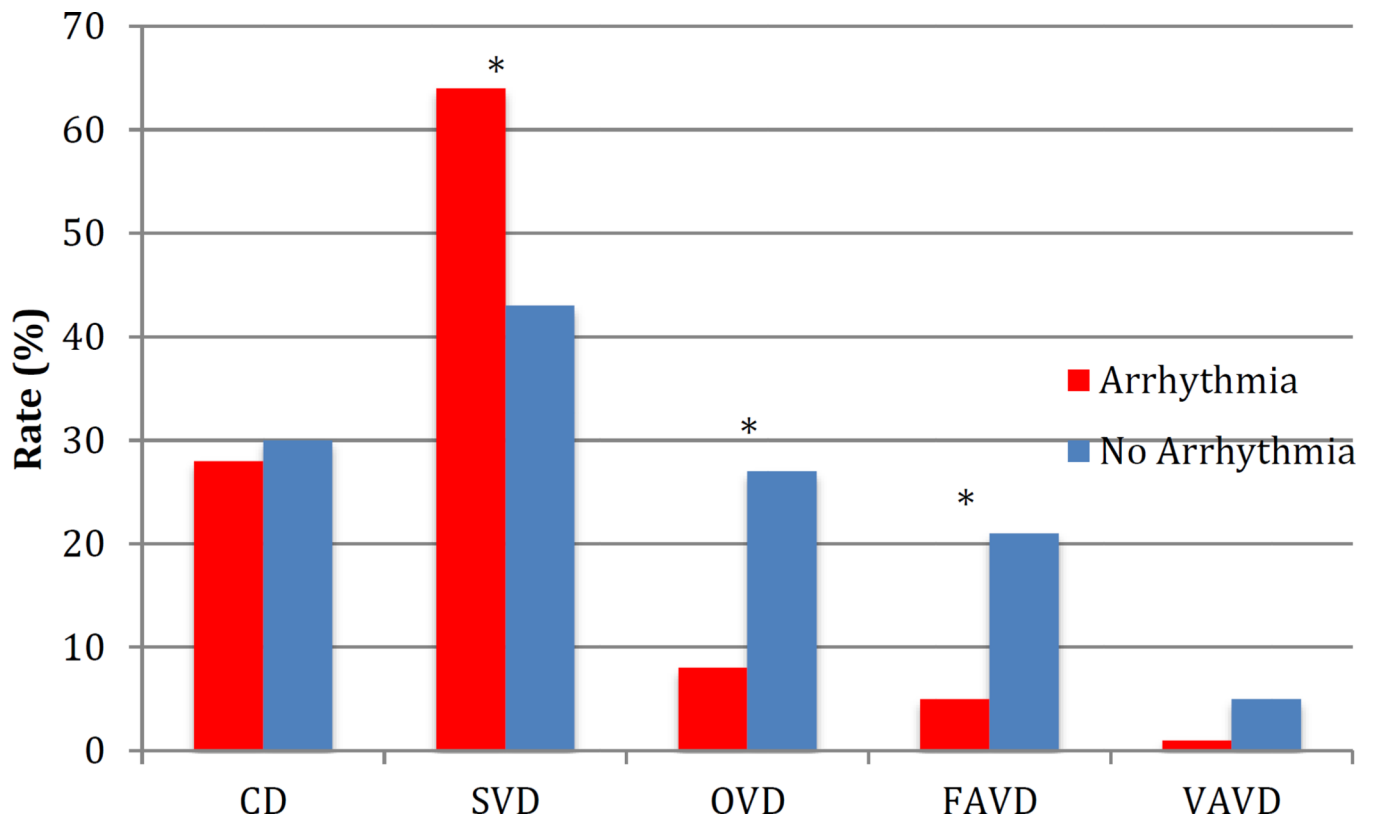
1. Huisman, CM.; Zwart, JJ.; Roos-Hesselink, JW.; Duvekot, JJ.; van Roosmalen, J. Incidence and Predictors of Maternal Cardiovascular Mortality and Severe Morbidity in the Netherlands: A Prospective Cohort Study. In: Frasch, MG., editor. PLoS ONE. Vol. 8. 2013 Feb 14. p. e56494
2. Alonso-Gonzalez R, Swan L. Treating cardiac disease in pregnancy. Women's Health. 2014 Jan; 10(1):79–90.
3. Siu, SC.; Sermer, M.; Colman, JM.; Alvarez, AN.; Mercier, LA.; Morton, BC., et al. Circulation. Vol. 104. Lippincott Williams & Wilkins; 2001 Jul 31. Prospective multicenter study of pregnancy outcomes in women with heart disease; p. 515-521.
4. Gowda, RM.; Khan, IA.; Mehta, NJ.; Vasavada, BC.; Sacchi, TJ. Int J Cardiol. Vol. 88. Elsevier; 2003 Apr. Cardiac arrhythmias in pregnancy: clinical and therapeutic considerations; p. 129-133.
5. Laksman Z, Harris L, Silverside CK. Cardiac arrhythmias during pregnancy: a clinical approach. Fetal and Maternal Medicine Review. 22:123–143.
6. Enriquez AD, Economy KE, Tedrow UB. Contemporary management of arrhythmias during pregnancy. Circ Arrhythm Electrophysiol. 2014 Oct; 7(5):961–967. [PubMed: 25336366]
7. Hoffman JIE, Kaplan S, Liberthson RR. Prevalence of congenital heart disease. American Heart Journal. 2004 Mar; 147(3):425–439. [PubMed: 14999190]

8. Ray JG, Vermeulen MJ, Schull MJ, Redelmeier DA. Cardiovascular health after maternal placental syndromes (CHAMPS): population-based retrospective cohort study. *The Lancet*. 2005 Nov; 366(9499):1797–1803.
9. Ray JG, Schull MJ, Kingdom JC, Vermeulen MJ. Heart failure and dysrhythmias after maternal placental syndromes: HAD MPS Study. *Heart*. 2012 Jul 5; 98(15):1136–1141. [PubMed: 22591737]
10. Ersbøll AS, Hedegaard M, Søndergaard L, Ersbøll M, Johansen M. Treatment with oral beta-blockers during pregnancy complicated by maternal heart disease increases the risk of fetal growth restriction. *BJOG: Int J O & G*. 2014 Jan 9; 121(5):618–626.
11. Regitz-Zagrosek V, Blomstrom Lundqvist C, Borghi C, Cifkova R, et al. Endorsed by the European Society of Gynecology (ESG), the Association for European Paediatric Cardiology (AEPC), and the German Society for Gender Medicine (DGesGM), Authors/Task Force Members. ESC Guidelines on the management of cardiovascular diseases during pregnancy: The Task Force on the Management of Cardiovascular Diseases during Pregnancy of the European Society of Cardiology (ESC). *European Heart Journal*. 2011 Dec 14; 32(24):3147–3197. [PubMed: 21873418]
12. McNulty JH. Arrhythmias in pregnancy. *Cardiol Clin*. 2012 Aug; 30(3):425–434. [PubMed: 22813367]





**Figure 1.** Distribution of diagnoses among women with arrhythmia. Afib: Atrial fibrillation, SVT: supraventricular tachycardia, VT: ventricular tachycardia, WPW: Wolf-Parkinson-White, ARVD: Arrhythmogenic Right Ventricular Dysplasia, CMP: Cardiomyopathy with arrhythmia (the + is the arrhythmia diagnosis in addition to concurrent structural cardiac disease).



**Figure 2.** Mode of delivery by arrhythmia status. CD: Cesarean delivery, SVD: spontaneous vaginal delivery; OVD: operative vaginal delivery, FAVD: forceps assisted vaginal delivery, VAVD: vacuum assisted vaginal delivery. \* P-value < 0.05, represents Chi-2 test for comparisons of proportions.

**Table 1**

Maternal Demographics among patients with cardiac disease

	<b>Arrhythmia N=36 N (% or <math>\pm</math>SD)</b>	<b>No Arrhythmia N= 107 N (% or <math>\pm</math>SD)</b>	<b>P-value*</b>
Age (yrs)	31.5 ( $\pm$ 5.6)	30.2 ( $\pm$ 6.7)	0.27
Ethnicity			0.13
Latina	5 (13)	17 (16)	
Caucasian	24 (63)	45 (43)	
Black	2 (5)	12 (11)	
Asian	5 (13)	11 (10)	
Native American	0 (0)	3 (2)	
Other	0(0)	12 (13)	
Unknown	0 (0)	7 (7)	
Pre-pregnancy BMI (kg/m <sup>2</sup> )	25.2 ( $\pm$ 7.3)	25.6 ( $\pm$ 6.0)	0.77
Nulliparous	14 (39)	65 (61)	0.02
Previous CD	7 (19)	13 (12)	0.59
Chronic HTN	3 (8)	13 (12)	0.53
Pre-gestational DM	0 (0)	3 (3)	0.31
Gestational DM	2 (6)	8 (7)	0.69

\* P-value represents t-test for comparisons of means or Chi-2 test for comparisons of proportions.

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

**Table 2**

Adverse obstetrical outcomes by arrhythmia status

	<b>Arrhythmia N=36 N (% or <math>\pm</math>SD)</b>	<b>No Arrhythmia N= 107 N (% or <math>\pm</math>SD)</b>	<b>P-value*</b>	<b>OR (95% CI)</b>
Gestational age at delivery (weeks)	37.9 ( $\pm$ 2.2)	37.3 ( $\pm$ 3.3)	0.48	
IUFD	0 (0)	2 (2)	0.41	0 (0–5.78)
Preeclampsia	2 (6)	10 (9)	0.48	0.57 (0–2.46)
IUGR	6 (17)	5 (5)	0.02	4.08 (1.23–13.54)
GDM	2 (6)	8 (7)	0.70	0.73 (0–3.22)
Blood transfusion	1 (3)	13 (12)	0.10	0.21 (0–1.29)
Chorioamnionitis	0 (0)	8 (7)	0.09	0 (0–.36)
Abruption	2 (6)	0 (0)	0.01	** $\infty$ (1.59– $\infty$ )
Postpartum hemorrhage	6 (17)	29 (27)	0.21	0.54 (0.21–1.39)
ICU admission	2 (6)	18 (17)	0.09	0.29 (0–1.20)

\*P-value represents t-test for comparisons of means or Chi-2 test for comparisons of proportions.

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

**Table 3**

## Neonatal Outcomes by arrhythmia status

	<b>Arrhythmia</b> <b>N=36</b> <b>N (% or <math>\pm</math>SD)</b>	<b>No Arrhythmia</b> <b>N= 107</b> <b>N (% or <math>\pm</math>SD)</b>	<b>P-value*</b>	<b>OR (95% CI)</b>
Gestational age at delivery (weeks)	37.9 ( $\pm$ 2.2)	37.3 ( $\pm$ 3.3)	0.48	
Preterm delivery	7 (19)	25 (23)	0.63	0.79 (0.32–1.99)
Birth weight (gms)	3079 ( $\pm$ 661)	2873 ( $\pm$ 773)	0.15	
SGA	2 (6)	12 (11)	0.33	0.47 (0.10–2.19)
5 min Apgar <7	6 (17)	12 (11)	0.39	1.58 (0.57–4.46)
NICU admission	10 (28)	33 (31)	0.73	0.86 (0.38–1.97)

SGA: Small for gestational age, less than 10% by Alexander et al 1996, Obstetrics & Gynecology.

\* P-value represents t-test for comparisons of means or Chi-2 test for comparisons of proportions.

**Table 4**

Outcomes among patients with arrhythmia with and without concurrent structural disease

	<b>Arrhythmia with structural lesion N=16 N (%)</b>	<b>Arrhythmia without structural lesion N= 20 N (%)</b>	<b>P-value*</b>
IUGR	3 (19)	3 (15)	0.76
Abruption	0 (0)	2 (10)	0.19
ICU Admission	1 (6)	1 (5)	0.87
Preterm delivery	3 (19)	4 (20)	0.93
SGA	1 (6)	1 (5)	0.87
Apgar 5 <7	3 (19)	3 (15)	0.76
NICU Admission	4 (25)	6 (30)	0.74

\* P-values represent Chi-2 test of significance.

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript