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**Conventional HeartDefects Cut Output in Pregnancy**

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**Toronto** — Pregnant women with congenital heart defects are not able to ramp up their cardiac output to handle the stresses of pregnancy, according to a small study out of the Netherlands. Dr. Jolien Roos-Hesselink reported the study results in a poster presentation at the 18th International Symposium on Adult Congenital Heart Disease.

“We know that cardiac output generally rises during pregnancy and we felt it important to see what happens in women with congenital heart disease as a means of explaining the increased risk of complications seen in these women and their offspring,” Dr. Roos-Hesselink, a cardiologist, said in an interview. “We suspected that maybe cardiac output wasn’t increasing equivalently in these patients,” she said.

Pregnancy is a major concern in the management of women with congenital heart disease, complicated by a greater risk of fetal growth retardation, premature birth, and perinatal mortality. Normally, cardiac output increases by 40%–50% during pregnancy.

Dr. Roos-Hesselink’s team at the Thoraxcenter of Erasmus Medical Center in Rotterdam, the Netherlands, studied three patient groups before, during, and after pregnancy. These patients had a variety of congenital heart defects, including aortic valve replacement, tetralogy of Fallot, Ebstein anomaly, ventricular septal defect, atrioventricular septal defect, and pulmonic stenosis. Subjects underwent cardiac MRI at 20 weeks’ gestation and again at 32 weeks. Measurements were compared with measurements taken 6–12 months postconception and post partum.

“In the first eight patients we studied, we saw that in seven of the patients, cardiac output did not rise as the pregnancy progressed, but rather fell,” she reported. Although the data presented are limited, Dr. Roos-Hesselink’s team have now tested 25 patients and will be presenting further data soon.

Previous echocardiography studies have hinted at the issue of cardiac output in pregnancy complicated by coronary heart disease, but until cardiac MRI was determined to be safe for women in pregnancy, there was no means of accurately measuring cardiac output during gestation, said Dr. Roos-Hesselink. “It seems to be that the ventricular function in these women is not capable of handling the stress of pregnancy, although it does increase from baseline during the first trimester.”

After increasing appropriately from pre- and postpregnancy baseline values up to 20 weeks’ gestation, cardiac output decreased significantly, from 6.9 L/min at 20 weeks to 5.4 L/min at 32 weeks of gestation. In addition, a significant reduction in end-dias tonic volume and stroke volume between 20 and 32 weeks of gestation was observed, along with a decline in left ventricular ejection fraction from 53% to 49%. Left ventricular mass increased from 87.5 grams at 20 weeks to 94.4 grams at 32 weeks.

“Pulmonary valve replacement (PVR) should only be undertaken in patients in whom all three reasons are present,” Dr. Gary Webb said at the 18th International Symposium on Adult Congenital Heart Disease. The clinical situations that might drive a decision to replace a leaky pulmonary valve include exercise intolerance attributable to the pulmonary regurgitation or congenital heart defect, sustained atrial flutter or fibrillation, sustained ventricular tachycardia or re-suspected sudden death, or an asymptomatic patient with “excessive” right ventricular dilation.

“Ten years ago, we replaced the pulmonary valve for exercise intolerance, sustained arrhythmias, and ‘progressive’ right ventricular dysfunction,” said Dr. Webb, director of the Philadelphia Adult Congenital Heart Center. “However, using these criteria for surgery, we learned that we had waited too long for many of these patients, and in the end they had suboptimal results.”

The decision of when to intervene in pulmonary regurgitation has evolved and continues to evolve, said Dr. Webb. “Even mild left ventricular systolic dysfunction is an indication for surgery for aortic and mitral regurgitation,” he said, and if we could see that right ventricular systolic function was declining from normal, then we could apply the same criteria. But, of course, it doesn’t work in tetralogy patients because the right ventricle is not normal.”

Efforts to risk-stratify these patients, therefore, have focused instead on right ventricular diastolic volumes. Although there are risks to waiting too long before PVR, Dr. Webb does not think a low threshold for replacing a leaking pulmonary valve is wise. Indeed, 10-year and 30-year survival after repair of tetralogy of Fallot in one large study was 97% and 89% in patients surviving at least 1 year (J. Am. Coll. Cardiol 1997; 30:1374-83).

“If we have 11% mortality over 30 years in this cohort of survivors we don’t want to be pulling the trigger too impulsively on these patients,” he said.