# [Fetal cardiac findings and hemodynamic changes associated with severe lower urinary tract obstruction in utero.](https://www.ncbi.nlm.nih.gov/pubmed/30908816)

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**Take Home Points:**

* Fetuses with lower urinary tract obstruction (LUTO) have a shorter indexed mitral valve inflow time and higher left ventricular myocardial performance index suggesting left ventricular dysfunction.
* LUTO fetuses had higher right ventricular to left ventricular cardiac index ratio, which might explain the finding of smaller left-heart structures in a subset of fetuses with LUTO.



***Commentary from Dr. Inga Voges (Kiel, Germany), section editor of Pediatric Cardiology Journal Watch:*** Fetuses with lower urinary tract obstruction (LUTO) can develop cardiomegaly, cardiac hypertrophy and pericardial effusion. In this interesting retrospective study, the authors analyzed echocardiograms of a cohort of 25 fetuses with LUTO with a focus on ventricular function and hemodynamic assessment. 25 gestational age (GA)-matched control fetuses were included. Five patients had serial postnatal echocardiograms, that were analysed.

Fetuses with LUTO commonly presented with cardiomegaly, pericardial effusion as well as right and left ventricular (RV, LV) hypertrophy demonstrated by increased LV, RV and septal wall thickness. Six Fetuses had right ventricular dysfunction and four fetuses had small left-heart structures.

Myocardial performance index was significantly increased, and mitral valve inflow time indexed to cardiac cycle length was significantly reduced, both suggesting LV diastolic dysfunction. RV cardiac index was higher, and LV cardiac index was lower compared to controls, resulting in an increased RV to LV cardiac index ratio in LUTO fetuses. Ascending aortic and aortic isthmus z-scores were lower in LUTO fetuses compared to controls. See Table 2 and Figure 1.

During follow-up there were 17 liveborn patients; two of the 25 pregnancies were lost to follow-up, three had elective termination of pregnancy and there was intrauterine fetal demise in three cases.

One out of the four fetuses with small left-sided structures demised in utero. Two of the remaining three fetuses presented with slightly small left-sided structures postnatally that did not require any intervention, the third one was diagnosed with coarctation and died a few hours after birth due to respiratory distress. In those patients with serial postnatal echocardiograms, systolic function normalized by 3–10 days and ventricular hypertrophy normalized by 20 days to four months.

The authors nicely explain possible reasons for their finding of small left-heart structures (Figure 2) and hereby motivate others to perform future studies.





