Editorial

Fetal Postsystolic Shortening Assessment by Myocardial Deformation Imaging
Sign of Cardiac Dysfunction?

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Fetal growth restriction (FGR) is defined as seriously lower estimated fetal weight and (later) confirmed birth weight (lower than the 10th percentile according to local reference curves). This whole group is associated with poorer perinatal outcome (eg, preeclampsia and stillbirth) and has serious potential implications on their health status from early childhood to adult life, for example, hypertension and increased intima-media thickness.1–3

 restricted infants was carefully conducted, incorporating many structural and functional parameters (including Doppler tissue imaging).3 Fetuses with FGR showed signs of more globular hearts and increased thickness of the myocardial walls. Furthermore, decreased longitudinal motion at the base of the myocardial walls, increased ratio between peak early pulsed Doppler velocity (E) and early diastolic peak myocardial velocity (E'), and isovolumic relaxation time were found as signs of diastolic dysfunction.

General antepartum obstetric ultrasound has become a standard part of gestational care and is commonly used for the determination of fetal age, size, sex, and well-being and for the detection of congenital anomalies. Update on the diagnosis and classification of FGR and proposal of a stage-based management protocol has been published recently,4 integrating current evidence to classify stages of fetal deterioration and establishes follow-up intervals and optimal delivery timings, which might help preventing the medical squeals of this complex clinical condition. The protocol is based on umbilical artery Doppler parameters (eg, umbilical artery pulsatility index) to detect early onset of severe clinical condition and the presence of any of the factors associated with a poorer prenatal outcome, including Doppler cerebroplacental ratio, uterine artery Doppler, a growth percentile below the third percentile, and possibly maternal and environmental factors. Indices of myocardial tissue velocities and deformation were up to now not included in this protocol.

Myocardial velocities do not truly represent regional myocardial function, because of cardiac translational motion and segmental interactions. Nevertheless, they are often used to assess diastolic function. Strain and strain rate imaging are available as tools that provide the noninvasive assessment of (local) myocardial deformation and as a marker of subclinical cardiac disease. There is a wealth of studies indicating a relation between myocardial disease and strain. Myocardial strain is decreased not only in patients with overt cardiomyopathy, but also in asymptomatic patients with diabetes mellitus, hypertension, smoking, obesity, and subclinical atherosclerosis and even in asymptomatic survivors of childhood cancer, during and short after anthracyclines therapy.4 Just recently, Cordero-Reyes et al5 provided significant evidence that myocardial contraction, as defined by noninvasive strain imaging, is correlated with the expression of elements involved in force generation and relaxation at the cellular level in patients with dilated cardiomyopathy. Initially, cardiac strain imaging was based on Doppler tissue imaging data and involved cumbersome analysis to obtain a strain estimate in 1 direction and inherently angle dependent. With the commercial availability...
of 2-dimensional (2D) speckle-tracking echocardiography, strain estimation in multiple directions became feasible by using conventional B-mode echograms. Interestingly, speckle-tracking-derived deformation measurements assessed using different ultrasound machines and software packages are not always similar. The applications and limitations of deformation imaging in healthy children and in congenital heart disease were recently published.10

Normal strain and strain rate values in a healthy fetal cohort are scarce. Earlier published correlations of global and regional strain and strain rate data with advancing pregnancy are based on cross-sectional data (eg, gestational age range, 13–40 weeks). Gestational age largely determines the risk of perinatal mortality in early-onset FGR.11 Therefore, the establishment of second-trimester 2D speckle-tracking echocardiographic reference values for global and regional strain, strain rate, and time to peak global left and right ventricular strain in a healthy fetal cohort is a mandatory prerequisite for its use in evaluating (pathological) changes in both left and right ventricular function during pregnancy.12 Furthermore, there is a need to use age-specific reference values, assessed with the same ultrasound and software package, for the adequate interpretation of 2D speckle-tracking echocardiographic measurements and their normal changes during the same pregnancy (between the second- and third-trimester screenings).13

Regional strain alterations in only 1 segment of the myocardium are of limited value. It would be of interest to examine changes in regional diastolic function in fetuses with FGR. In the study by Crispi et al, FGR was defined as birth weight below 10th percentile, without differentiating between early- and late-onset FGR. Strain and strain rate were determined using both Doppler tissue imaging and 2D speckle-tracking echocardiography techniques. Longitudinal myocardial velocities were found to be decreased as reported earlier, yet adjustment for cardiac size, which was reported earlier by the same group to change the results, was not performed. Whereas peak strain and strain rate values of the left ventricle were not significantly different between FGR cases and controls, postsystolic shortening (PSS) as assessed by deformation imaging in the basal segment of the septal ventricular wall was observed in 57% of the FGR cases and in none of the controls. The authors conclude that PSS support increase pressure overload as a mechanism for cardiovascular programming in FGR. This study is the first report that addresses the possible linkage between PSS and diastolic dysfunction in FGR. The authors were not able to detect PSS in tissue Doppler velocity curves. The study is limited by the relatively small case number, yet the absence of no PSS at all in the control group is certainly an interesting finding.

PSS, defined as myocardial shortening after the point of aortic valve closure, can also be observed physiologically in healthy individuals.15,16 Therefore, it has not always been a marker of disease. However, the incidence and magnitude of PSS in myocardial disorders, for example, adults with hypertrophic cardiomyopathy,17 is significantly increased. The number of segments having PSS correlated significantly with the isovolumic relaxation time. In patients with hypertrophic cardiomyopathy, pathological PSS is found more regularly. In patients with hypertrophic obstructive cardiomyopathy,18 PSS can delay myocardial relaxation resulting in an increased left ventricular filling pressure, defined as increased E/E′ ratio, with a reduction in the average value of PSS and E/E′ ratio already 6 months after alcohol septal ablation. Presence of pathological PSS, which may exceed far over aortic valve closure with a reduced overall strain, was observed in both ischemic and scarred segments in coronary artery disease or ischemia.19 In ischemic patients, it is suggested that the presence of (radial) postsystolic thickening does not characterize contractility in the ischemic segment, but it is a measure of preserved elasticity without fibrosis and thus potential functional recovery after appropriate restoration of perfusion if the tissue is not irreversibly damaged. Whether the presence of PSS, detected (only) by 2D strain in longitudinal myocardial layers, is a direct measure of viability remains to be unraveled. However, one has to keep in mind that subclinical alterations in the longitudinal myocardial velocities and strain are often reported as early sign of cardiac dysfunction because of ischemia of the endothelial layers. No causal relationships (eg, myocardial biopsies or molecular tests) were established in the present study. Yet, fetuses with FGR and PSS showed absence of hypertrophic response, a poorer perinatal outcome (described as lower gestational age and birth weight, containing all case of perinatal mortality), and higher absolute values of blood pressure 6 months after birth. Absence of hypertrophic response might occur because of relative ischemia of the endothelial layer or as a sign of myocardial fibrosis correlating PSS with ischemia.18

Finally, indices of myocardial deformation were also not significantly different between FGR cases with and without PSS. These 2 subgroups are small, the myocardial indices were not corrected for gestational age, and little is known about the feasibility and reliability of the strain and strain rate measurements, optimal region of interest size (in fetuses of different gestational age), strain length, and beat to beat variation. Furthermore, because the accuracy of the estimated isovolumic relaxation time is affected by the ratio between frame rate and heart rate, it might be difficult to observe significant differences in isovolumic relaxation time. In a study of adults with untreated hypertension, the degree of PSS relative to the end-systolic strain (calculated as post-systolic strain index) was associated with increased procollagen fibers and delayed diastolic lengthening which contribute to diastolic dysfunction. However, these patients still had normal left ventricular longitudinal strain. Whether PSS, in the presence of normal left ventricular longitudinal strain, is an earlier sign of myocardial dysfunction in fetuses with FGR is not yet clear.

We think that the present conventional echocardiographic and myocardial deformation results may have pathogenic contribution to the functional heterogeneity of this disease entity, especially diastolic dysfunction. The first step should be attempted to discriminate pathological from physiological changes in strain and PSS. Presence (or absence) of PSS in fetal hearts during normal pregnancies should be studied, using the same myocardial deformation techniques. Not only its presence, but also the magnitude and number of segments having PSS during both the second and third trimester, as well as during the first year of life, should be further assessed and followed in time. Further large-scale studies
should be conducted to assess the clinical significance of PSS in FGR cases.

Disclosures

None.

References


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