● Editorial

EFFECT OF FETAL HYPOXIA ON CARDIAC FUNCTION AND STRUCTURE

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It is currently recognized that the quality of the fetal environment during early development is critical for programming of cardiovascular health and disease later in life. Fetal hypoxia is one of the most common consequences of complicated pregnancies worldwide, as it is a central pathophysiologic feature of placental insufficiency in fetal growth restriction, fetal anemia, twin-to-twin transfusion syndrome, maternal smoking or even in maternal inflammatory conditions.

Chronic hypoxia *in utero* alters myocardial development by a direct inhibitory effect on cardiomyocyte proliferation and coronary vessel growth, and results also in a fetal blood redistribution known as "brain-sparing effect". This central redistribution of the cardiac output occurs in order to maintain perfusion to the key organs – brain, heart and adrenals. It involves peripheral vasoconstriction due to activation of a selective carotid body chemoreflex and up-regulation of renin-angiotensin system. It is currently being questioned, however, whether these circulatory changes, classically understood as adaptive to an adverse intrauterine environment, might become maladaptive and permanently alter cardiac structure and function with persistent consequences for the long-term health of the offspring[1].

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Clinical and experimental studies on fetal growth restriction, and consequent low birth weight, showed that this population presents, as early as in childhood and adolescence, signs of endothelial dysfunction measured by aorta and carotid intima-media thickness and stiffness, central pulse wave velocity and blood pressure. In adulthood, hypertension, impaired glucose tolerance, insulin resistance, obesity, metabolic syndrome and coronary artery disease are well known consequences since the initial epidemiological studies by Barker and colleagues in the 80s[2].

Assessment of fetal cardiovascular status and cardiac function has the potential to improve clinical surveillance in these conditions and to provide clinical insight into diseases course. Hopefully it may help in early identification of special risk groups for cardiovascular diseases later in life. Although routine assessment of fetal cardiac function has often been qualitative, as the field has evolved, quantitative

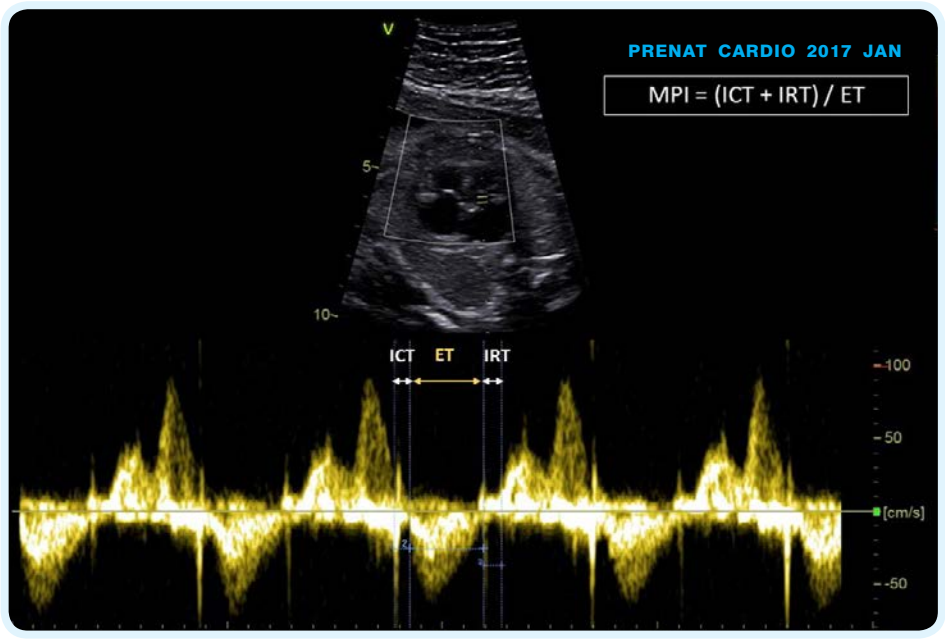

Fig 1. Myocardial performance index (MPI) using pulsed-wave Doppler.

Doppler waveform displays the opening and closing clicks of the mitral and aortic valves. ICT - isovolumic contraction time; IRT - isovolumic relaxation time; ET - ejection time.

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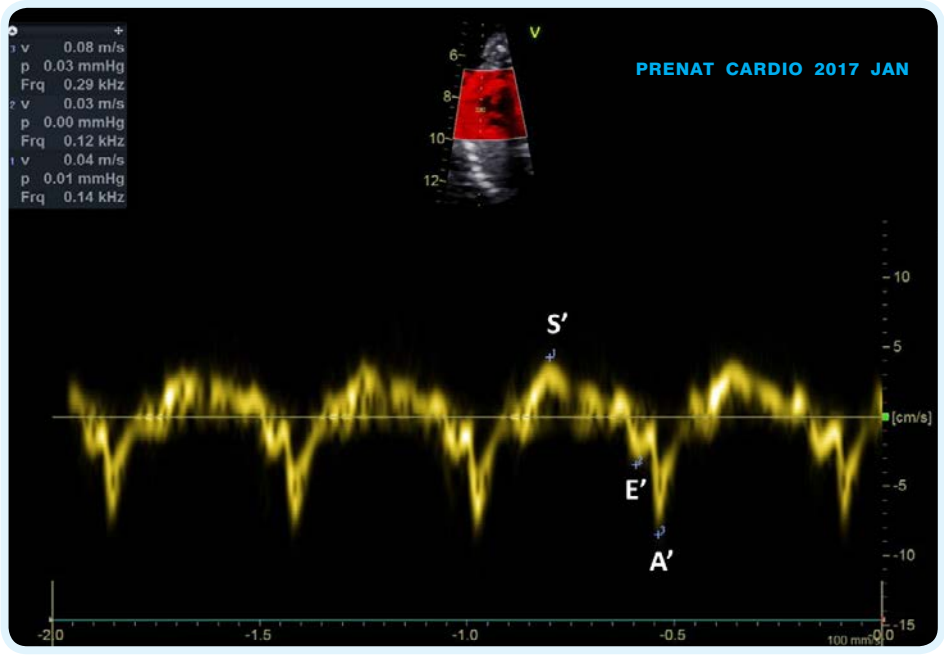

Fig. 2. Tissue Doppler Imaging.

Pulsed-wave tissue Doppler interrogation of the right ventricle. S´- peak annular velocity in systole; E´- peak velocity in early diastole; A' - peak velocity in late diastole during atrial contraction.

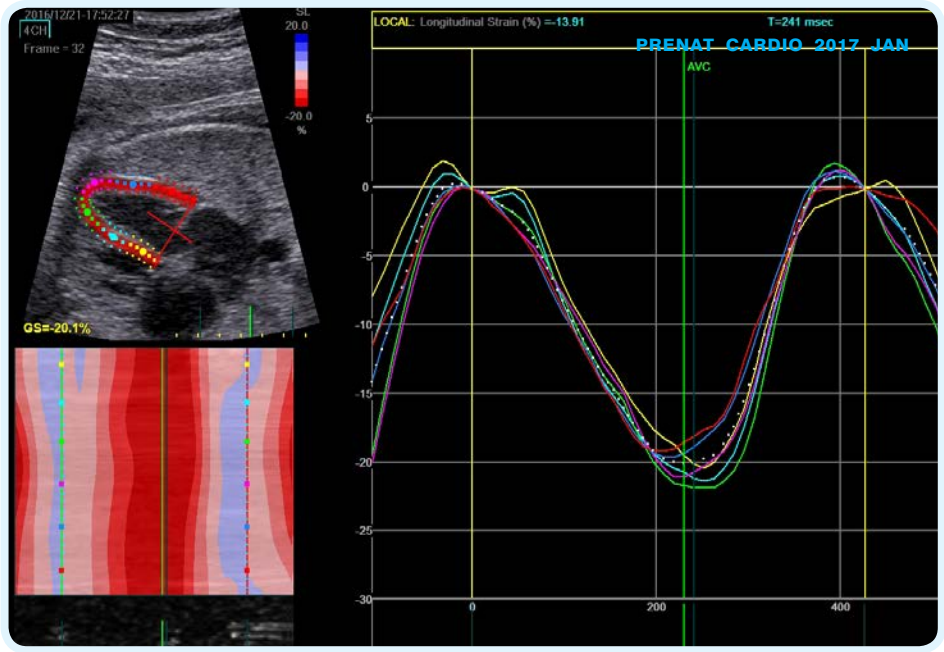

Fig. 3. Deformation study using 2D speckle tracking.

On the top left, tracking of the left ventricle borders in a four-chamber view of a fetal heart. On the right, speckle tracking longitudinal strain curves from the six left ventricle segments, displayed as a function of time. GS – Global strain.

assessment of functional parameters has assumed greater importance. Beside the traditional ultrasound imaging modalities for assessing cardiac morphometry and blood flow – M-mode, B-mode and conventional Doppler –, tissue Doppler imaging and 2D-speckle tracking have more recently been proposed for the evaluation of global and regional myocardial motion and deformation in fetus[3] (Figure 1, 2 and 3). Considering the uniqueness of fetal cardiovascular physiology and the particularities of proper acquisition and interpretation of functional echocardiography parameters, an advanced training and a critical approach are fundamental for fetal cardiology practitioners[4].

Despite the great research efforts on the understanding of fetal hemodynamic adaptation and long term consequences of fetal growth restriction, little is currently known on how the cardiovascular remodeling develops throughout early life and progresses into chronic disease, and on the susceptibility factors for cardiovascular diseases. Likewise, a wide variety of other fetal conditions, like maternal diabetes, maternal cardiovascular risk factors or twin-to-twin transfusion syndrome, for example, require further investigation by both in utero and longitudinal studies, probably taking advantage of the recently proposed techniques for cardiac function evaluation.

In conclusion, the interaction between the timing and quality of stress during pregnancy and the developmental outcomes is not yet well understood. In future studies, more careful use of functional echocardiographic parameters in both normal and stressed pregnancies will help our understanding of the biological mechanisms involved in the adverse changes of fetal hemodynamics.

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