ARTYKUŁ ORYGINALNY / ORIGINAL ARTICLE

Myocardial performance index (Tei index) in term and preterm neonates during the neonatal period

Renata Bokiniec¹, Paweł Własienko², Maria K. Borszewska-Kornacka¹, Dariusz Madajczak¹, Joanna Szymkiewicz-Dangel¹

¹Neonatal and Intensive Care Department, Medical University of Warsaw, Warsaw, Poland

Abstract

Background: The myocardial performance index (MPI) is a noninvasive method to measure global systolic and diastolic myocardial function. In both term and premature neonates, changes in the systolic and diastolic function of the left ventricle (LV) and right ventricle (RV) reflect the degree of neonatal myocardial immaturity and the co-existence of foetal circulation.

Aim: To assess MPI (or Tei indices) of both ventricles in term and preterm newborns, and to observe MPI trends throughout the neonatal period.

Methods: Heart ultrasound imaging was performed on the first day of life (DOL), after patent ductus arteriosus (PDA) closure, and on the 28th DOL, in 29 term and 29 preterm newborns. RVMPI and LVMPI were measured within the preterm group at 40 weeks of post-conception age (PCA).

Results: A statistically significant reduction in RVMPI was observed in both term and preterm newborns. In term newborns, the RVMPI value on the first DOL was 0.42 ± 14 , dropping to 0.29 ± 0.09 after PDA closure, and finally reaching 0.22 ± 0.09 on the 28^{th} DOL. The respective RVMPI values for the preterm newborns were 0.44 ± 0.15 , 0.30 ± 0.12 , and 0.21 ± 0.08 . Little variability in the mean values of LVMPI was observed in both groups throughout the neonatal period. The LVMPI for term neonates in successive measurements was 0.37 ± 0.10 , 0.39 ± 0.07 , and 0.37 ± 0.11 , respectively, and for the preterm neonates it was 0.37 ± 0.10 , 0.35 ± 0.09 , and 0.36 ± 0.10 , respectively. The MPI values from preterm newborns taken at 40 weeks PCA (RVMPI = 0.28 ± 0.09 ; LVMPI = 0.37 ± 0.05) were comparable to those measured in term newborns after PDA closure.

Conclusions: Observed postnatal changes in RVMPI correspond to changes in ventricular function, reflecting the haemodynamic changes of the transitional circulation. The relatively small postnatal changes in LVMPI in term and preterm newborns may reflect an immature myocardium. The RVMPI and LVMPI values at 40 weeks PCA in preterm newborns correlate best with MPI values in term newborns just after PDA closure.

Key words: myocardial performance index, neonates, Doppler

Kardiol Pol 2016; 74, 9: 1002-1009

INTRODUCTION

In both term and preterm neonates, changes in the systolic and diastolic function of the left ventricle (LV) and right ventricle (RV) reflect the degree of neonatal myocardial immaturity, the co-existence of foetal circulation, and the presence of

concurrent diseases. For example, impairment of myocardial systolic and diastolic function accompanies intrauterine and secondary infections, and affects preterm neonates with bronchopulmonary dysplasia (BPD), hypoxia, and intrauterine growth retardation [1–5]. Conventional methods of evaluation

Address for correspondence:

Renata Bokiniec, MD, PhD, Neonatal and Intensive Care Department, Medical University of Warsaw, ul. Karowa 2, 00–315 Warszawa, Poland, e-mail: r.bokiniec@wp.pl Received: 19.11.2015 Accepted: 22.03.2016 Available as AOP: 25.04.2016

Kardiologia Polska Copyright © Polskie Towarzystwo Kardiologiczne 2016

²Perinatal Cardiology Unit, Medical University of Warsaw, Warsaw, Poland

of myocardial function have limitations in adults, which become even more problematic in the immature myocardium of the preterm neonate. Firstly, there is a limitation in the evaluation of the haemodynamics of the circulatory system if the assessment of the cardiac output of the right ventricle (RVCO) is performed in the presence of an open patent foramen ovale, and, similarly, if the left ventricle (LVCO) is measured when there is leakage through the ductus arteriosus (DA). The RVCO solely reflects the pulmonary flow when the foramen ovale is closed, and the LVCO reflects the systemic flow only with a closed DA. Secondly, evaluation of the fractional shortening (FS) and the ejection fraction (EF) in M-mode echocardiography (ECHO) also has its limitations. M-mode recording solely reflects movement of the anterior and posterior walls of the LV. Furthermore, weak movement of the anterior wall of the LV can be observed during systole, as opposed to the posterior and lateral walls in the preterm neonate. Therefore, LV function is often underestimated using these conventional methods, which are largely dependent on the myocardial geometry, cardiac function, preload, and afterload. For these reasons, non-invasive, bedside methods are being developed and applied more frequently in both neonatal and adult medicine for the early evaluation of global systolic and diastolic myocardial function.

The myocardial performance index (MPI), introduced during the mid-nineties by Tei et al. [6], is a non-invasive way to measure global systolic and diastolic myocardial function. The MPI, also known as the Tei index, was introduced for the assessment of myocardial function in adults with dilated cardiomyopathy [6]. Initially, Tei et al. [6] used two measurements for the calculation of the index: (a) isovolumetric time, measured from the end of the A wave to the initiation of the E wave, and (b) ejection time (ET), measured as the outflow from the LV or RV [6]. The formula (a-b)/b defines the Tei index [6]. The MPI value is inversely proportional to myocardial function; an increase in MPI points towards a deterioration in global myocardial function. The methodology for the measurement of MPI has been developed over time by researchers studying various foetal pathologies. For example, Freidman et al. [7] proposed that LVMPI should be calculated from a single recording of the Doppler wave, representing both the outflow and inflow to the LV. This would allow simultaneous calculation of the isovolumetric contraction time (ICT) and the isovolumetric relaxation time (IRT) [7]. To improve accuracy of the MPI measurement, Raboisson et al. [8], and later Hernandez-Andrade et al. [9], developed the MPI modification (Mod-MPI), in which the opening and closing movements of the aortic valve (AV) and mitral valve (MV), or clicks, were used as the reference points for the measurement of ICT and IRT. The record of the Doppler wave is obtained by placing a Doppler gate on the medial wall of the ascending aorta, and from this projection it is possible to record the movement of both the AV and MV. Harada et

al. [10] proposed another method for the evaluation of MPI in the foetus and neonate, in which myocardial movement in tissue Doppler was recorded. The aim of the study is to assess MPI (or Tei indices) of both ventricles in term and preterm newborns, and to observe MPI trends throughout the neonatal period.

METHODS

The study involved term (n = 29) and preterm (n = 29) newborns. The 29 healthy, term infants were born at 37–41 weeks of gestation with a mean gestational age of 39 weeks. This group (18 boys and 11 girls) had a mean birth weight of 3443 g (min 2700 g, max 4200 g). Twenty-nine preterm neonates (15 boys and 14 girls) were selected with a mean gestational age of 26 ± 2 weeks and mean birth weight of 916 ± 310.5 g. In this group of preterm neonates, seven had mild BDP on the 28^{th} day of life (BPD28DOL), and three had severe BPD in the 36^{th} week post-conception age (BPD36PCA). Thus, only 19 newborns were included in the group of preterm neonates born without BPD for statistical comparison with the term neonates.

Neonates were included in the study if they had normal cardiac morphology and if the mother had a normal perinatal history. Selected clinical parameters-of all neonates are presented in Table 1. Prior parental consent was obtained for all neonates involved in the study. The study was approved by the Ethical Committee of the Medical University of Warsaw.

Imaging was obtained using the Philips HD 11XE Ultrasound with a 12-MHz sector probe. The ICT, IRT, and ET were measured, and the MPI calculated using the formula (ICT + IRT)/ET, and easily calculated as (a–b)b as per the Hernandez-Andrade et al. modification [8, 9]. We used the Doppler ECHO (clicks) of the opening and closing of the AV and MV as reference points to estimate the timing of the ejection period (Fig. 1) [8, 9]. During the ultrasound examination, it was possible to obtain a continuous record of electrocardiogram (ECG). In order to reduce examination times, the MPIs for the RV and LV were calculated using Cardiac Arena cardiology software.

The tricuspid inflow waves were recorded from the apical four-chamber view with the pulsed-wave Doppler sample volume positioned at the tips of the tricuspid leaflets in diastole (Fig. 2). Right ventricular ET was measured separately from the parasternal short-axis scan plane with a pulse-wave Doppler signal placed at the pulmonary valve annulus in the RV outflow tract (Fig. 3). The ECG was recorded continuously during the examination. The calculation of the Tei index (RVMPI) was considered meaningful for the RV if the difference in heart rate for the inflow and outflow path was 5 bpm or more.

A gated pulsed Doppler sample volume was placed in the LV outflow tract in the apical five-chamber view (Fig. 2). For the Tei index (LVMPI), the Doppler sample volume was placed below the MV towards the ventricular septum with the

Table 1. Clinical characteristics of infants enrolled in the study

	Term (n = 29)	Preterm (n = 29)
Gestational age [weeks]	39 (min 37; max 41)	26 (min 24; max 32)
Male	18	15
Female	11	14
Birth weight [g]	3443 (min 2700; max 4200)	916 (min 468; max 1920)
Caesarean delivery	11 (38%)	18 (62%)
One-minute Apgar score	Median 10 (min 5; max 10)	Median 5 (min 1; max 10)
Five-minute Apgar score	Median 10 (min 7; max 10)	Median 7 (min 2; max 10)
PDA surgical ligation	No	5 (17.2%)
PDA pharmacological ligation	No	8 (27.5%)
BPD (28 th day of life)	No	7 (24.1%)
BPD (36 th PCA)	No	3 (10.3%)

BDP — bronchopulmonary dysplasia; PCA — post-conception age; PDA — patent ductus arteriosus

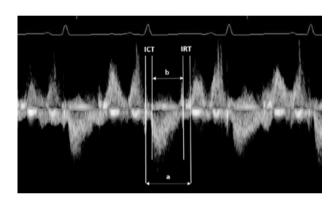


Figure 1. Pulsed Doppler tracing obtained from the apical five-chamber view with echocardiography tracing for timing above. Measurement of myocardial performance index (MPI; Tei index) of left ventricle. MPI (Tei index) = ICT + IRT/ET = a-b/b; ICT — isovolaemic contraction time; IRT — isovolaemic relaxation time; ET — ejection time

pulsed Doppler trace including the E/A waveform (positive) and the aortic (negative) blood flow waveforms.

The ICT, IRT, and ET were measured in term neonates three times. The first measurement was after birth within the first DOL, the second measurement was within the third DOL prior to discharge home (after patent ductus arteriosus [PDA] closure), and the third measurement was at the end of the neonatal period within the 28th DOL. Four children failed to attend for examination on the 28th DOL.

In the preterm neonates, four measurements of the ICT, IRT, and ET were recorded. The first measurement was taken as soon as possible after birth, the second after PDA closure, the third on the 28th DOL, and the final measurement in the 40th week post-conception. The latter serves to compare the MPI values between preterm and term neonates.

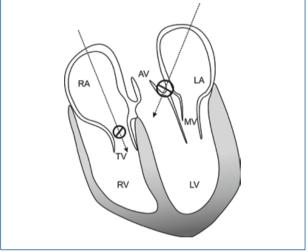


Figure 2. Apical five-chamber view. The arrows indicate the position of the Doppler gate for recording the Doppler wave when measuring myocardial performance index for the left ventricle (LV) and for recording the diastolic waveform for the right ventricle (RV); AV — aortic valve; LA — left atrium; MV — mitral valve; RA — right atrium; TV — tricuspid valve. Figure based on Skinner et al. [18]

Statistical analysis

Statistical tests were used to analyse the significance of any observed differences. For analysis of continuous variables, two types of Wilcoxon tests were used. For independent samples, the changes in variables during the neonatal period were analysed using the Wilcoxon signed-rank tests. Chi-square or Fisher's exact tests (depending on the number of cases) were used for analysis of the relationship between quantitative variables. A p value < 0.05 was taken as statistically significant.

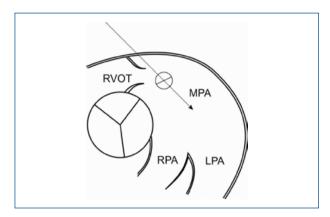


Figure 3. Five-chamber view in short parasternal projection. The arrow indicates the position of the Doppler gate at the pulmonary valve or recording the systolic waveform when measuring myocardial performance index; RVOT — right ventricular outflow tract; LPA — left pulmonary artery, MPA — main pulmonary artery; RPA — right pulmonary artery. Figure based on Skinner et al. [18]

Calculations were made using the statistical analysis system (SAS) software system.

In this study we used heart ultrasound and the Mod-MPI method developed by Hernandez-Andrade et al. [9] to determine RVMPI and LVMPI in term and preterm newborns, in order to identify any trends in the MPI that occur throughout the neonatal period.

RESULTS

In term neonates on the first DOL, the value of the MPI for the LV was 0.37 ± 0.10 . A follow-up measurement performed after closure of the PDA revealed that the LVMPI had risen only slightly to 0.39 ± 0.07 , but this increase was not statistically significant, with a p value of 0.549. On the 28^{th} DOL, at the end of the neonatal period, the mean value of the LVMPI remained unchanged $(0.37 \pm 0.11, p = 0.524)$ compared to the measurement performed on the first DOL and after PDA closure. In the first DOL, the RVMPI was 0.42 ± 0.14 , which was higher than that for the LV. After closure of the PDA, there was a dramatic decrease in RVMPI to 0.29 ± 0.09 (p < 0.05),

and on the 28^{th} DOL the mean value of the RVMPI had fallen further to 0.22 ± 0.09 (p < 0.05) (Table 2, Fig. 4).

In preterm neonates the MPI was higher for the RV (0.44 \pm 0.15) than for the LV (0.37 \pm 0.10) on the first DOL (Tables 2, 3). The MPI for the LV remained unchanged after PDA closure (0.35 \pm 0.09, p = 0.4392), but there was a statistically significant decrease in the index for the RV (from 0.44 \pm 0.15 to 0.30 \pm 0.12, p = 0.0002). In the preterm neonates on the 28th DOL, the mean LVMPI value remained relatively stable at 0.36 \pm 0.10. For the RV, however, it decreased even further to 0.21 \pm 0.08 (p < 0.05) (Fig. 4).

Term neonates with measurements made during the first DOL were also compared to a group of preterm neonates in week 40 PCA (Table 3). No statistically significant differences were observed in the term neonates LVMPI (0.37 \pm 0.10) compared to the preterm neonates (0.37 \pm 0.05, p = 0.6740). In contrast to the LV measurements, a statistically significant difference was observed for the RVMPI (term RVMPI = 0.42 \pm 0.14; preterm RVMPI = 0.28 \pm 0.09; p = 0.0305).

When comparing the RVMPI in term neonates after PDA closure (0.29 \pm 0.09) with the preterm group at 40 weeks PCA (0.28 \pm 0.08), no difference was observed (p = 0.9964) (Table 3). A decrease in the heart rate was observed between the first measurement and those made at the end of the first week of life.

Some of the preterm newborns were born with mild (BPD28DOL) or severe (BPD36PCA) BPD. The mean RVMPI values in the first DOL were comparable to those in preterm newborns not suffering from BPD (0.44 \pm 0.15), in both the mild (0.48 \pm 0.16) and severe (0.47 \pm 0.23) BPD groups. On the 28th DOL, the RVMPI was markedly higher in the BPD36PCA group (0.31 \pm 0.10) than in the BPD28DOL (0.22 \pm 0.06) and healthy groups (Fig. 4). Due to the small sample size of the BPD group, statistical analysis was not conducted.

DISCUSSION

This study analysed the changes in MPI for the RV and LV for term and preterm neonates throughout the neonatal period up until the 28th DOL. The differences in MPI, which can act as a proxy of global myocardial function, reflect the differences in myocardial maturity in both study groups. In preterm

Table 2. Comparison of RVMPI and LVMPI in term and preterm neonates during the neonatal period

Time point		1 st DOL	After closure PDA	P	28 th DOL	P
Term neonates	RVMPI	0.42 ± 0.14	0.29 ± 0.09	< 0.05	0.22 ± 0.09	< 0.05
	LVMPI	0.37 ± 0.10	0.39 ± 0.07)	NS	0.37 ± 0.11	NS
Preterm neonates	RVMPI	0.44 ± 0.15	0.30 ± 0.12	0.0002	0.21 ± 0.08	< 0.05
	LVMPI	0.37 ± 0.10	0.35 ± 0.09	NS	0.36 ± 0.10	NS

Data shown as mean \pm standard deviation. DOL — day of life; LVMPI — left ventricular myocardial performance index; RVMPI — right ventricular myocardial performance index; PDA — patent ductus arteriosus

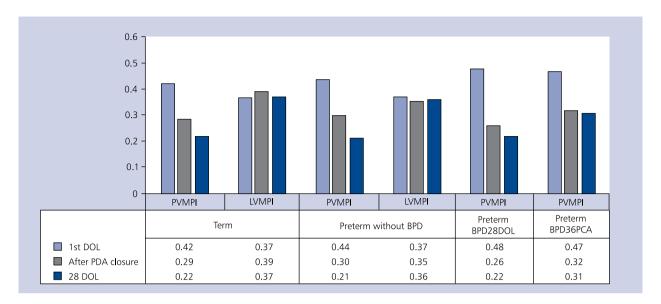


Figure 4. Mean values of myocardial performance index for right and left ventricles in both study groups (term and preterm neonates). In the preterm bronchopulmonary dysplasia (BPD) group statistical analysis was omitted due to low group sample size; DOL — day of life; LVMPI — left ventricular myocardial performance index; RVMPI — right ventricular myocardial performance index; PCA — post-conception age; PDA — patent ductus arteriosus

Table 3. Comparison of RVMPI and LVMPI between preterm neonates at 40 weeks post-conception age (40th PCA) and term neonates

	Term 1 st DOL	Preterm 40 th PCA	P
RVMPI	0.42 ± 0.14	0.28 ± 0.09	0.0305
LVMPI	0.37 ± 0.10	0.37 ± 0.05	NS
	Term after closure PDA	Preterm 40 th PCA	Р
RVMPI	0.29 ± 0.09	0.28 ± 0.09	NS
LVMPI	0.39 ± 0.07	0.37 ± 0.05	NS

Data shown as mean ± standard deviation. DOL — day of life; LVMPI — left ventricular myocardial performance index; RVMPI — right ventricular myocardial performance index; PCA — post- conception age; PDA — patent ductus arteriosus

neonates, the myocardium has more water and less contractile mass, leading to diminished compliance and force generated per sarcomere compared to term neonates. Furthermore, the immature myocardium is more sensitive to increases in afterload immediately following PDA ligation, due to an increase in systemic vascular resistance [11].

These differences may contribute to the MPI discrepancies between both groups to a greater extent than the haemodynamic changes of the transitional circulation observed immediately after birth. The haemodynamic changes due to the presence of a PDA can influence myocardial function to a greater extent than the differing MPI values of the ventricles observed in neonates of differing maturity. For example, in cases of postnatal significant PDA left-to-right shunting, the systemic pressure is already high, causing increased flow through the pulmonary circulation. Increment of blood flow into the left atrium leads to its enlargement, with subsequent LV expansion.

In our study, the highest RVMPI values in both term and preterm neonates were obtained on the first DOL. This was followed by large reductions in the mean value of the RVMPI after PDA closure, and a further significant decline by the 28th DOL. In preterm neonates the RVMPI was raised once more in week 40 PCA.

Our results are similar to those of other researchers only in part, most likely due to the different DOL on which the measurements were recorded. For example, Malakan-Rad and Momtazmanesh [12] analysed the MPI index for the RV in 51 term neonates at up to 72 h of life and obtained an value of 0.23 \pm 0.14. In our study, such low values were only obtained towards the end of the neonatal period after PDA closure. On the third DOL the RVMPI was 0.28 \pm 0.8, while on the first DOL the values were much higher (0.42 \pm 0.14). One can suspect that the measurements performed by Malakan-Rad and Momtazmanesh [12] were taken closer to the third DOL, rather than the first.

Elevated pressure within the pulmonary circulation (high afterload) observed in newborns, particularly in the first three DOL, reflects the so-called transitional circulation. This elevated pressure adequately explains the high RVMPI values during the early period of life. Throughout the gestational period, the development of the RV and LV in human foetuses is similar. Although the stroke volume is higher in the RV compared to the LV, which is reflected in the higher mean RVMPI values observed in human foetuses (0.35 ± 0.07) [13].

Even though MPI declines in both ventricles with increasing gestational age (Tsutsumi et al. [13]), immediately after birth the RVMPI will peak, and then this is followed by a fall in MPI during the subsequent hours and days of life. The most striking changes occur in the first two DOLs, as shown by Murase et al. [14] in very low birth weight neonates. The authors demonstrated a significant drop in RVMPI between the 12th and 24th hours of life (from 0.42 ± 0.33 to 0.30 ± 0.16), and a further drop to 0.24 ± 0.14 by the 36th hour. Respective values observed for LVMPI were 0.45 ± 0.21 , 0.38 ± 0.15 , and 0.32 ± 0.15 . This work is in partial accordance with our own results. While we observed RVMPIs averaging 0.44 ± 0.15 in the first DOL in preterm neonates, the mean LVMPI was 0.37 ± 0.10 on the first DOL and only declined slightly in subsequent days (although this was not statistically significant). We did not observe an increase in LVMPI immediately after birth in preterm neonates.

The differences observed in our study compared to Murase et al. [14] could be explained by our choice of measurement times. As opposed to Murase et al. [14], we only assessed the MPI once in the first DOL, and secondly we chose PDA closure as our second measurement time, which can vary in preterm neonates weighing under 1000 g. In term neonates, we did not observe a statistically significant increase in LVMPI after PDA closure (only a slight increase from 0.37 ± 0.10 to 0.39 ± 0.07). These values remained reasonably constant in subsequent days. The available literature suggests the increase in LVMPI in the early neonatal period is caused predominantly by changes in diastolic LV function due to an increase in the IRT [6, 14, 15].

Among our 29 preterm neonates, only four required operative PDA closure, eight were treated medically with ibuprofen, and the remaining 17 underwent spontaneous PDA closure. Noori et al. [11] previously assessed the LVMPI after PDA ligation in 23 preterm neonates and observed a significant increase in LVMPI 2 h after the procedure. If we compare this to our study, it can be infered that spontaneous or pharmacological DA closure does not dramatically influence the changes in LVMPI.

A few studies support the notion that increased pressure in the pulmonary circulation, including neonates with pulmonary hypertension of any cause, results in an elevated RVMPI [5, 16, 17]. However, literature regarding pulmonary

hypertension in neonates suffering from BPD is still scarce. Czernik et al. [2] compared the RVMPI value in preterm neonates without BPD to those who developed BPD by 36 weeks PCA. In neonates who had developed BPD at 36 weeks PCA the authors did not observe a decrease in RVMPI on the 7th, 14th, and 28th DOLs compared to the second DOL. The RVMPI on the second DOL was equal in both groups and averaged 0.39 (min 0.33, max 0.55). These values are lower compared to those obtained in our study (RVMPI = 0.47 ± 0.23). Nevertheless, we confirmed the results obtained by Czernik et al. [2] after PDA closure (RVMPI = 0.32 ± 0.08 on the 7th DOL), and those obtained on the 28th DOL (RVMPI = 0.31 ± 0.10). Similarly to Czernik et al. [2], we observed increased RVMPI (0.31 ± 0.1) values on the 28th DOL in BPD sufferers diagnosed at 36 weeks PCA, compared to the mild BPD (0.22 ± 0.06) and healthy term neonates (0.23 ± 0.1) . Such elevated RVMPI values in children with severe BPD reflect a persistently elevated pressure in the pulmonary circulation, resulting from a lower number of blood vessels surrounding the pulmonary alveoli, hypertrophy of the vessel media, muscularisation of small peripheral vessels, and inappropriate vasoreactivity [2]. Although the authors are aware of the limitations of the study, the aim was to present the dynamics of the changes in the mean values of LVMPI and RVMPI in different groups of neonates depending on their maturity. In clinical practice, knowledge of MPI trends in the neonatal period may provide a basis for comparison of mean values for LVMPI and RVMPI in other important pathologies of the neonatal period such as in the case of the challenges concerning echocardiographic diagnosis of BPD hypertension. In view of the increasing use of the Tei index in the youngest group of patients, and following the inclusion of a larger group of subjects, it seems appropriate to establish a net of centiles for this index, both at the start and at the conclusion of the neonatal period for term neonates and for preterm neonates in relation to gestational age.

Limitations of the study

A limitation of the study was the small sample sizes of both study groups (term, n=29, preterm, n=29).

CONCLUSIONS

Myocardial performance index measurement is feasible in neonates. Postnatal changes in RVMPI in both term and preterm newborns reflect both systolic and diastolic RV function well, which is influenced by the haemodynamic changes of the transitional circulation. The low magnitude of postnatal LVMPI changes in term and preterm newborns may reflect myocardial immaturity. Both PDA closure and co-existent BPD may affect MPI values in neonates. Comparing systolic and diastolic myocardial function between term and preterm newborns is not straightforward. RVMPI and LVMPI measured at 40 weeks PCA in preterm newborns correlate best with MPI values in term neonates after PDA closure. Further studies with

larger numbers of neonates throughout the whole neonatal period are indicated.

Acknowledgements

Language assistance was provided by Proper Medical Writing Ltd.

Conflict of interest: none declared

References

- Mourani PM, Ivy DD, Rosenberg AA et al. Left ventricular diastolic dysfunction in bronchopulmonary dysplasia. J Pediatr, 2008; 152: 291–293. doi: 10.1016/j.jpeds.2007.11.006.
- 2. Czernik C, Rhode S, Metze B et al. Persistently elevated right ventricular index of myocardial performance in preterm infants with incipient bronchopulmonary dysplasia. PloS One, 2012; 7: e38352. doi: 10.1371/journal.pone.0038352.
- Ichihashi K, Yada Y, Takahashi N et al. Utility of a Doppler-derived index combining systolic and diastolic performance (Tei index) for detecting hypoxic cardiac damage in newborns. J Perinat Med, 2005; 33: 549–552. doi: 10.1515/JPM.2005.098.
- Karaarslan S, Alp H, Baysal T et al. Is myocardial performance index useful in differential diagnosis of moderate and severe hypoxic-ischaemic encephalopathy? A serial Doppler echocardiographic evaluation. Cardiol Young, 2012; 22: 335–340. doi: 10.1017/S104795111200011X.
- Altın H, Karaarslan S, Karataş Z et al. Evaluation of cardiac functions in term small for gestational age newborns with mild growth retardation: a serial conventional and tissue Doppler imaging echocardiographic study. Early Hum Dev, 2012; 88: 757-764. doi: 10.1016/j.earlhumdev.2012.04.003.
- Tei C, Ling LH, Hodge DO et al. New index of combined systolic and diastolic myocardial performance: a simple and reproducible measure of cardiac function - a study in normals and dilated cardiomyopathy. J Cardiol, 1995; 26: 357–366.
- Friedman D, Buyon J, Kim M, Glickstein JS. Fetal cardiac function assessed by Doppler myocardial performance index (Tei Index). Ultrasound Obstet Gynecol, 2003; 21: 33–36. doi: 10.1002/uog.11.

- Raboisson MJ, Bourdages M, Fouron JC. Measuring left ventricular myocardial performance index in fetuses. Am J Cardiol, 2003; 91: 919–921.
- Hernandez-Andrade E, López-Tenorio J, Figueroa-Diesel H et al. A modified myocardial performance (Tei) index based on the use of valve clicks improves reproducibility of fetal left cardiac function assessment. Ultrasound Obstet Gynecol, 2005; 26: 227–232. doi: 10.1002/uog.1959.
- Harada K, Tamura M, Toyono M, Yasuoka K. Comparison of the right ventricular Tei index by tissue Doppler imaging to that obtained by pulsed Doppler in children without heart disease. Am J Cardiol, 2002; 90: 566–569.
- Noori S, Friedlich P, Seri I, Wong P. Changes in myocardial function and hemodynamics after ligation of the ductus arteriosus in preterm infants. J Pediatr, 2007; 150: 597–602. doi: 10.1016/j.jpeds.2007.01.035.
- Malakan-Rad E, Momtazmanesh N. Doppler-derived right ventricular myocardial performance index in neonates normal values. Acta Medica Iranica, 2002; 40: 226–229.
- Tsutsumi T, Ishii M, Eto G et al. Serial evaluation for myocardial performance in fetuses and neonates using a new Doppler index. Pediatr Int, 1999; 41: 722–727.
- Murase M, Ishida A, Morisawa T. Left and right ventricular myocardial performance index (Tei index) in very-low-birth-weight infants. Pediatr Cardiol, 2009; 30: 928–935. doi: 10.1007/s00246-009-9464-8.
- Tei C, Nishimura RA, Seward JB, Tajik AJ. Noninvasive Doppler-derived myocardial performance index: correlation with simultaneous measurements of cardiac catheterization measurements. J Am Soc Echocardiogr, 1997; 10: 169–178.
- Patel N, Mills JF, Cheung MM. Use of the myocardial performance index to assess right ventricular function in infants with pulmonary hypertension. Pediatr Cardiol, 2009; 30: 133–137. doi: 10.1007/s00246-008-9285-1.
- Murase M, Morisawa T, Ishida A. Postnatal myocardial performance index (MPI) in recipient infants of twin-twin transfusion syndrome: A pulsed-Doppler study. J Clinic Experiment Cardiol, 2011; 2: 145. doi: 10.4172/2155-9880.1000145
- Skinner J, Alverson D, Hunter S eds. Echocardiography for the neonatologist. Churchill Livingstone; London 2000; 135–138.

Cite this article as: Bokiniec R, Własienko P, Borszewska-Kornacka MK et al. Myocardial performance index (Tei index) in term and preterm neonates during the neonatal period. Kardiol Pol, 2016; 74: 1002–1009. doi: 10.5603/KP.a2016.0056.

Wskaźnik sprawności mięśnia sercowego (wskaźnik Tei) w okresie noworodkowym u dzieci urodzonych o czasie i u wcześniaków

Renata Bokiniec¹, Paweł Własienko², Maria Katarzyna Borszewska-Kornacka¹, Dariusz Madajczak¹, Joanna Szymkiewicz-Dangel¹

¹Klinika Neonatologii i Intensywnej Terapii Noworodka, Warszawski Uniwersytet Medyczny, Warszawa

Streszczenie

Wstęp: Wskaźnik sprawności tkankowej (MPI, wskaźnik Tei) to nieinwazyjna metoda służąca do oceny globalnej, zarówno skurczowej, jak i rozkurczowej funkcji miokardium. U noworodków donoszonych oraz u wcześniaków zmiany funkcji skurczowej i rozkurczowej komory lewej (LV) i prawej (RV) odzwierciedlają stopień niedojrzałości noworodkowego miokardium oraz współistnienie krążenia płodowego.

Cel: Celem pracy była ocena MPI dla komory prawej (RVMPI) i lewej (LVMPI) u noworodków donoszonych i wcześniaków w okresie noworodkowym.

Metody: W grupie 29 noworodków donoszonych i 29 wcześniaków wykonano badania echokardiograficzne w pierwszej dobie życia, po zamknięciu przewodu tętniczego oraz w 28. dobie życia. Dodatkowo w grupie wcześniaków wykonano pomiar RVMPI i LVMPI w 40. tygodniu życia postkoncepcyjnego.

Wyniki: W obydwu grupach noworodków zaobserwowano istotne statystycznie obniżenie wartości RVMPI. U noworodków donoszonych w pierwszej dobie życia wartość RVMPI obniżyła się z 0.42 ± 0.14 do 0.29 ± 0.09 po zamknięciu przewodu tętniczego, a w 28. dobie życia do 0.22 ± 0.09 . Odpowiednio dla noworodków przedwcześnie urodzonych wartości RVMPI wynosiły: 0.44 ± 0.15 ; 0.30 ± 0.12 i 0.21 ± 0.08 . W obydwu grupach zaobserwowano małą zmienność wartości LVMPI. U noworodków donoszonych wartości LVMPI wynosiły odpowiednio: 0.37 ± 0.10 ; 0.39 ± 0.07 ; 0.37 ± 0.11 , a w przypadku wcześniaków: 0.37 ± 0.10 ; 0.35 ± 0.09 i 0.36 ± 0.10 . Wartości MPI uzyskane u wcześniaków w 40. tygodniu życia postkoncepcyjnego dla RVMPI wynosiły 0.28 ± 0.09 , a dla LVMPI — 0.37 ± 0.05 i były porównywalne z wartościami MPI uzyskanymi u noworodków donoszonych po zamknięciu przewodu tętniczego.

Wnioski: Istotne obniżenie wartości RVMPI odzwierciedla zmiany hemodynamiczne zachodzące w układzie sercowo-naczyniowym noworodka ("krążenie przejściowe") po urodzeniu. Stosunkowo małe zmiany w zakresie wartości LVMPI mogą świadczyć o niedojrzałości noworodkowego miokardium. Wartości RVMPI i LVMPI uzyskane u noworodków przedwcześnie urodzonych w 40. tygodniu życia postkoncepcyjnego najlepiej korelują z wartościami MPI noworodków donoszonych uzyskanymi po zamknięciu przewodu tętniczego.

Słowa kluczowe: wskaźnik sprawności mięśnia sercowego, noworodki, badanie doplerowskie

Kardiol Pol 2016; 74, 9: 1002-1009

Adres do korespondencji:

dr n. med. Renata Bokiniec, Klinika Neonatologii i Intensywnej Terapii Noworodka, Warszawski Uniwersytet Medyczny, ul. Karowa 2, 00–315 Warszawa, e-mail: r.bokiniec@wp.pl

Praca wpłynęła: 19.11.2015 r. Zaakceptowana do druku: 22.03.2016 r. Data publikacji AoP: 25.04.2016 r.

²Poradnia Perinatologii i Kardiologii Perinatalnej, II Katedra i Klinika Położnictwa i Ginekologii, Warszawski Uniwersytet Medyczny, Warszawa