Tei index in neonatal respiratory distress and perinatal asphyxia

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Received 30 September 2013; accepted 9 December 2013
Available online 13 January 2014

Abstract  Cardiovascular compromise is a common complication of neonatal respiratory distress and perinatal asphyxia. Tei index is a Doppler-derived index for the assessment of overall left ventricular function that combines systolic and diastolic time intervals.

Aim: Assess the role of MPI versus cardiac troponin I as early indicator of hypoxic cardiac damage in neonates with respiratory distress or perinatal asphyxia. The present work was conducted on forty neonates, 15 with neonatal respiratory distress (group I), 15 with perinatal asphyxia (group II), and 10 apparently healthy neonates as a control (group III). All have: Detailed history-thorough clinical examination-Plain X-ray-ECG-Two dimensional, M-mode and Doppler echocardiographic examination with the measurement of both myocardial performance index (MPI) of the right and left ventricle-Serum cardiac troponin I. 

Results: There was statistically significant increase in serum cardiac troponin I in groups I and II than group III. Left and right ventricular myocardial performance index (MPI) were increased in group I and II than the control group. No correlation between Tei index and each of postnatal age, apgar score at 5-min, heart rate, serum cardiac troponin I, ejection fraction and fractional shortening, but there was direct relationship between MPI and LVEDD and inverse relationship between MPI and each of EF% and FS%. But there was significant correlation between L.V. MPI and gestational age.

Conclusion: Tei index was higher in neonates with respiratory distress and neonates with perinatal asphyxia than in normal neonates despite normal or even increased ejection fraction which indicates that these patients may have subclinical ventricular dysfunction which should be followed up carefully.

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1. Introduction

Respiratory distress is a common emergency responsible for 30-40% of admission in the neonatal period. The clinical presentation of neonatal respiratory distress includes apnea, cyanosis, grunting, inspiratory stridor, nasal flaring, poor feeding and tachypnea (respiratory rate more than 60 beats per
minute). There may be also retractions in the intercostals, subcostal, or suprasternal spaces. Most cases of neonatal respiratory distress are caused by transient tachypnea of newborn, respiratory distress syndrome, or meconium aspiration syndrome, but other various cases are possible.1 (see Tables 1–5)

Perinatal asphyxia is a common cause of neonatal morbidity and mortality. Perinatal asphyxia can be defined as the impaired respiratory gas exchange accompanied by development of acidosis. The incidence of perinatal asphyxia varies from 1 to 8 per 1,000 live birth.2 Among the indicators of perinatal asphyxia commonly used to diagnose this condition are neonatal respiratory distress, delayed onset of spontaneous respiration, low Apgar score (< 6 at 5 min), need for resuscitation and/or ventilation and metabolic acidosis (cord blood PH < 7.0 or 7.0 and base deficit >12 mmoL/L). Post natal indicators include neonatal encephalopathy, multi organ failure and abnormal findings on brain imaging.3

Cardiovascular compromise is a common complication of neonatal respiratory distress and perinatal asphyxia. This reduced cardiovascular reserve may present clinically with hypotension, which is associated with increased mortality and adverse neurological outcome.4 To evaluate cardiac involvement in neonates with respiratory distress, ECG and Echocardiography recording were performed and cardiac enzymes were determined. These data were related to clinical presentation and patients’ outcome.5

Recently the myocardial performance index (MPI) or Tei index has been proposed to be useful indicator of cardiac involvement in neonates with respiratory distress and perinatal asphyxia, as it is useful in the evaluation of both systolic and diastolic function of left ventricle in combination, and it provides an objective assessment of persistent pulmonary hypertension of newborn with perinatal asphyxia.6 Tei index is a simple Doppler-derived index, can be easily obtained as non-invasive technique with good diagnostic value of cardiac injury in neonates suffering from perinatal asphyxia.7

1.1. Aim of the work

To evaluate the role of Doppler-derived index combining both systolic and diastolic performance (Tei index) versus cardiac troponin I in the assessment of ischemic cardiac injury in neonates suffering from respiratory distress and perinatal asphyxia.

2. Patients and methods

This study was performed on forty neonates, 22 males and 18 females, their gestational age ranges from 28 to 36 weeks and their post natal age ranges from 1 to 7 days in the neonatal intensive care unit of Menoufia University Hospital in the period from October 2010–2011. The study included neonates with neonatal respiratory distress and perinatal asphyxia with post natal age ranging from 1–7 days with exclusion of neonates with congenital heart diseases, neonates with inborn errors of metabolism and neonates with sepsis. They were classified into three groups: Group I: Fifteen neonates with respiratory distress (8 males and 7 females), with mean gestational age 33 weeks and mean post natal age 5 days (Apgar score at 5 min ranging between 7 and 9 and PH between 7.2 and 7.3). Group II: Fifteen neonates with severe asphyxia, with mean gestational age 32 weeks and mean post natal age 3 days (Apgar score at 5 min < 7, and PH < 7.2), Group III (control group): Ten apparently healthy neonates without asphyxia (5 males and 5 females), with mean gestational age 36 weeks and mean post natal age 4 days (Apgar score at 5 min > 9 and PH > 7.3).

All neonates in the study were subjected to the following: Detailed perinatal history: Maternal diseases and drug intake, high risk pregnancy, fetal presentation, mode of delivery, risk factors of prematurity, labor (prolonged, obstructed, abnormal presentation), presence of meconium, antenatal ultrasound and risk factors of infection. – Full clinical examination including: Gestational age in weeks, Post natal age in days. Apgar score at 1 min and at 5 min, anthropometric measures performed on percentile charts, general examination, cardiac examination, chest examination, abdominal examination and neurological examination, and mean arterial blood pressure. – Laboratory investigations include: Complete blood count (CBC) by colter apparatus, c-reactive protein, arterial blood gases, serum lactate dehydrogenase, serum creatine kinase MB and serum cardiac troponin I. – Radiological investigation include: Echocardiogram using TOSHIBA (SSH140) echo Doppler apparatus and Acuson XP-128 phased array system equipped with a 2.0, 2.5, 3.5 and 5 MHz

Routinely the examination consisted of M-mode and two-dimensional echocardiography, pulsed and continuous wave Doppler and color flow mapping.

1. Left ventricular measurements were obtained at end-systole and end diastole according to the recommendation of the American society of echocardiography.
2. Left ventricular end-systolic and end-diastolic diameters and volumes (LVESD, LVEDV, LVEDD, and LVEDV) were computed using the Simpson rule.
3. Left ventricular ejection fraction was calculated as: % EF = (EDV–ESV)/EDV.
4. Left ventricular fractional shortening was calculated as: % FS = (EDD–BSD)/EDD.
5. Peak velocities of early (E) and late (A) filling were derived from atrioventricular valve inflow velocity profiles. The ratio of early to late peak velocities (E/A) was calculated.
6. Deceleration time (DT) was measured as the time from peak b velocity to the intercept of the deceleration of flow with the baseline.

2.1. Measurement of myocardial performance index

Doppler time intervals were measured from the atrioventricular valve inflow and ventricular outflow tracings.1 The interval “a” from cessation to the onset of atrioventricular valve inflow is equal to the sum of isovolumic contraction time (ICT), ejection time (ET), and isovolumic relaxation time (IRT). The interval “b” which equals the ejection time is derived from the duration of ventricular outflow Doppler velocity profile. MPI was calculated as: (a–b)/b which equals (ICT–IRT)/ET.
2.2. Myocardial performance index (MPI)

Interval (a) represents the duration in milliseconds from atroventricular valve closure to opening for either the tricuspid or the mitral valve interval, (b) represents the ventricular ejection time in milliseconds for either the right or the left ventricle valve; ET = ejection time; ICT = isovolamic contraction time; IRT = isovolamic relaxation time.9

2.3. Measurement of the myocardial performance index of the right ventricle

For the evaluation of the R.V Tei index the interval, from the cessation to the onset of tricuspid valve inflow (the interval from the end of the A wave to the start of the E wave), is obtained from the apical 4-chamber view with the pulse-wave Doppler signal located at the tips of the tricuspid valve leaflets. The b interval (right ventricular ejection time) is measured from the parasternal long-axis view, with the sample volume located just below the pulmonary valve.10

2.4. Measurement of the myocardial performance index of the left ventricle

For the evaluation of the L.V Tei index the interval, from the cessation to the onset of mitral valve inflow (the interval from the end of the A wave to the start of the E wave), is obtained from the apical 4-chamber view with the pulse-wave Doppler signal located at the tips of the mitral valve leaflets. The b interval (left ventricular ejection time) is measured from apical 5-chamber view with the pulse wave Doppler signal located just below the aortic valve.8

3. Results

Table 1  Cardiac troponin I in the studied groups.

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Group I Mean ± SD</th>
<th>P-value</th>
<th>Group II Mean ± SD</th>
<th>P-value</th>
<th>Group III Mean ± SD</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac troponin I (ng/ml)</td>
<td>0.58 ± 0.16</td>
<td>P1 &lt; 0.01</td>
<td>0.33 ± 0.01</td>
<td>P3 &lt; 0.05</td>
<td>0.25 ± 0.16</td>
<td>P &lt; 0.01</td>
</tr>
</tbody>
</table>

P1 comparison between groups 1 & 2.  
P2 comparison between groups 1 & 3.  
P3 comparison between groups 2 & 3.  
P comparison between groups 1–3.  
Significant at level of 0.05.  
Highly significant at level of 0.01.

Table 2  Echocardiographic measurements of the left ventricle (2-D & Echo-Doppler).

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Group I Mean ± SD</th>
<th>P-value</th>
<th>Group II Mean ± SD</th>
<th>P-value</th>
<th>Group III Mean ± SD</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>L.V.E.T</td>
<td>229 ± 4</td>
<td>P1 &gt; 0.05</td>
<td>228 ± 27</td>
<td>P3 &gt; 0.05</td>
<td>240 ± 6</td>
<td>P &gt; 0.05</td>
</tr>
<tr>
<td>ICT + IRT of L.V</td>
<td>85 ± 3</td>
<td>P1 &lt; 0.05</td>
<td>89 ± 4</td>
<td>P3 &lt; 0.05</td>
<td>84 ± 5</td>
<td>P &lt; 0.01</td>
</tr>
<tr>
<td>L.V. MPI</td>
<td>0.49 ± 0.02</td>
<td>P1 &gt; 0.05</td>
<td>0.47 ± 0.14</td>
<td>P3 &lt; 0.05</td>
<td>0.35 ± 0.02</td>
<td>P &lt; 0.01</td>
</tr>
<tr>
<td>L.V. E/A ratio</td>
<td>1.22 ± 0.13</td>
<td>P1 &lt; 0.05</td>
<td>1.33 ± 0.14</td>
<td>P3 &lt; 0.01</td>
<td>1.50 ± 0.08</td>
<td>P &lt; 0.01</td>
</tr>
<tr>
<td>L.V.D.T</td>
<td>109.67 ± 9.61</td>
<td>P1 &lt; 0.01</td>
<td>121.67 ± 8.80</td>
<td>P3 &lt; 0.01</td>
<td>132.50 ± 2.27</td>
<td>P &lt; 0.01</td>
</tr>
</tbody>
</table>

P1 comparison between groups 1 & 2.  
P2 comparison between groups 1 & 3.  
P3 comparison between groups 2 & 3.  
P comparison between groups 1–3.  
Significant at level of 0.05.  
Highly significant at level of 0.01.  
IRT: isovolamic relaxation time, L.V. MPI: left ventricular myocardial performance index, L.V.E.T: left ventricular ejection time, ICT: isovolamic contraction time, L.V.D.T: left ventricular deceleration time.
Table 3  Echocardiographic measurements of the right ventricle (2-D & Doppler).

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Group I Mean ± SD</th>
<th>P-value</th>
<th>Group II Mean ± SD</th>
<th>P-value</th>
<th>Group III Mean ± SD</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>R.V.E.T</td>
<td>276.73 ± 6.12</td>
<td>P1 &gt; 0.05</td>
<td>276.07 ± 5.42</td>
<td>P3 &gt; 0.05</td>
<td>280.20 ± 14.87</td>
<td>P &gt; 0.05</td>
</tr>
<tr>
<td>ICT + IRT of R.V</td>
<td>77.67 ± 2.58</td>
<td>P1 &lt; 0.01</td>
<td>70.27 ± 7.58</td>
<td>P3 &lt; 0.01</td>
<td>65.30 ± 3.80</td>
<td>P &lt; 0.01</td>
</tr>
<tr>
<td>R.V. MPI</td>
<td>0.27 ± .02</td>
<td>P1 &gt; 0.05</td>
<td>0.26 ± .02</td>
<td>P3 &lt; 0.01</td>
<td>0.23 ± .01</td>
<td>P &lt; 0.01</td>
</tr>
<tr>
<td>R.V. E/A ratio</td>
<td>21.60 ± 41.96</td>
<td>P1 &gt; 0.05</td>
<td>1.29 ± .08</td>
<td>P3 &gt; 0.05</td>
<td>1.32 ± .01</td>
<td>P &gt; 0.05</td>
</tr>
<tr>
<td>R.V.D.T</td>
<td>101.13 ± 5.97</td>
<td>P1 &gt; 0.05</td>
<td>101.80 ± 4.89</td>
<td>P3 &gt; 0.05</td>
<td>105.10 ± 2.85</td>
<td>P &gt; 0.05</td>
</tr>
</tbody>
</table>

P2 comparison between groups 1 & 3.
P3 comparison between groups 2 & 3.
P1 comparison between groups 1 & 2.
P comparison between groups 1–3.
Significant at level of 0.05.
Highly significant at level of 0.01.


Correlation between L.V. MPI and gestational age

Table 4  Correlation between L.V. MPI and R.V. MPI and each of postnatal age, gestational age, Apgar score, EF% and FS% of the studied groups.

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Group I</th>
<th>R.V. MPI</th>
<th>Group II</th>
<th>R.V. MPI</th>
<th>Group III</th>
<th>R.V. MPI</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Pearson correlation</td>
<td>P-value</td>
<td></td>
<td>Pearson correlation</td>
<td>P-value</td>
</tr>
<tr>
<td>R.V. MPI</td>
<td>0.259</td>
<td>&gt;0.05</td>
<td>&gt;0.05</td>
<td>0.358</td>
<td>&gt;0.05</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Post natal age (days)</td>
<td>0.245</td>
<td>&gt;0.05</td>
<td>0.286</td>
<td>&gt;0.05</td>
<td>0.405</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Gestational age (weeks)</td>
<td>0.511</td>
<td>&lt;0.05</td>
<td>0.475</td>
<td>&lt;0.05</td>
<td>0.458</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Apgar at 5 min</td>
<td>0.186</td>
<td>&gt;0.05</td>
<td>0.179</td>
<td>&gt;0.05</td>
<td>0.403</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Cardiac troponin 1</td>
<td>0.254</td>
<td>&gt;0.05</td>
<td>0.132</td>
<td>&gt;0.05</td>
<td>0.357</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>EF%</td>
<td>0.129</td>
<td>&gt;0.05</td>
<td>0.203</td>
<td>&gt;0.05</td>
<td>0.023</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>FS%</td>
<td>0.283</td>
<td>&gt;0.05</td>
<td>0.395</td>
<td>&gt;0.05</td>
<td>0.234</td>
<td>&gt;0.05</td>
</tr>
</tbody>
</table>

Group II

Table 5  Correlation between Tei index and heart rate.

Parameters of correlation x ± SD Pearson’s correlation coefficient (r) P-value
Tei index 0.35 ± 0.02 0.18 >0.05
Heart rate 104.1 ± 14.6

Correlation between Tei index and heart rate.

R.V. MPI: right ventricular myocardial performance index, EF%: ejection fraction, FS%: fraction shortening.
4. Discussion

Cardiovascular compromise is a common complication of neonatal respiratory distress and perinatal asphyxia. This reduced cardiovascular reserve may present clinically with hypotension, which is associated with increased mortality and adverse neurological outcome.1

To evaluate cardiac involvement in neonates with respiratory distress, ECG and Echocardiography recording were performed and cardiac enzymes were determined. These data were related to clinical presentation and patients’ outcome.2

As regards cardiac troponin I, there is statistical significant difference between the three studied groups, where P-value was < 0.05 with higher level in neonates with respiratory distress and neonates with perinatal asphyxia than neonates of the control group indicating more cardiac damage in neonates with respiratory distress and neonates with perinatal asphyxia. Similar results were obtained by5 study.

The mean L.V. MPI in the three studied groups was 49, 47 and 35 in groups I, II and III, respectively, which is in comparison to value of L.V. MPI of normal neonate obtained by other studies which shows that the value of MPI of both left and right ventricles is higher in neonates with respiratory distress or perinatal asphyxia than the control group. These results agree with results obtained by.12

Little was known about Tei index being influenced by aging14 and so its diagnostic utility in newborns was unclear, however our study reported that Tei index of both left and right ventricles is not influenced by post natal age, this is in agreement to15 who concluded that Tei index of the left and right ventricles increased immediately and transitory after birth then decreased and stabilized after 24 h of birth in apparently healthy 50 neonates. It is suggested that the early increase of MPI immediately after birth is due to intrapartum hypoxia then the decrease in the Tei index most likely reflects the alterations in cardiac loading that result from the increase in pulmonary blood flow attendant to the onset of pulmonary ventilation. The transition to postnatal circulation includes a sudden decrease in pulmonary vascular resistance and shift of cardiac output from the right to left ventricle.16

In our study, we proved that MPI of both left and right ventricles is influenced by gestational age, where there is statistical significant correlation between Tei index and maturity of neonates, this is in contrast to12 study which reported no significant correlation between MPI and gestational age.

As regards MPI and gender, no statistical significant difference was found on comparing males with females of the same gestational and postnatal ages. These results agree with those obtained by17 who reported no significant correlation between MPI and gender.

Our study demonstrated that there is no significant correlation between MPI of both left and right ventricles and heart rate in the three studied groups. Similar results were found by11 who reported that the effects of body surface area, ventricular geometry and heart rate on Tei index were not significant. Major advantage of the Tei index is that it is independent of both ventricular geometry and heart rate.12

5. Conclusion

1- Tei index was higher in neonates with respiratory distress and neonates with perinatal asphyxia than in normal neonates despite normal or even increased ejection fraction which indicates that these patients may have subclinical ventricular dysfunction which should be followed up carefully.

2- Tei index is a simple Doppler derived index that combines both systolic and diastolic function and could be easily obtained as non-invasive technique with good diagnostic value.

3- The myocardial performance index (MPI) is used to assess ventricular function. The index is independent of heart rate and blood pressure and does not rely on geometric assumptions. Preterm neonates with the evidence of perinatal asphyxia or neonatal respiratory distress have an increased MPI compared with a group of control neonates without evidence of physis or distress.

Conflict of interest statement

None to be declared.

References