Transmural Conduction Velocity Index in Healthy Schoolchildren

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Transmural conduction velocity index (TCVI) was obtained in 174 healthy children. TCVI was defined as the echocardiographically determined inter-ventricular septal thickness (IVST) divided by the ventricular activation time (VAT) measured by body surface potential mapping. TCVI ranged from 14 to 49 cm/sec and was highly correlated with IVST (r = 0.75, TCVI = 3.25x IVST + 0.56). It was concluded that the left ventricular hypertrophy with muscular thickness does cause the greater conduction velocity in healthy children.

TOYOSHIMA et al.1 reported the conduction velocity obtained by echocardiography and body surface potential mapping in patients with right bundle branch block. They concluded that the ventricular hypertrophy itself cause the greater conduction velocity. The purpose of the present study was to clarify the relationship between ventricular activation time (VAT) and left ventricular muscle thickness in healthy schoolchildren using the index described by Toyoshima.

MATERIALS AND METHODS

1) Study Population
Healthy schoolchildren aged 6—13 years were examined by echocardiography and body surface potential mapping. The children who have organic heart disease or post-operative state of heart were excluded by history taking, physical examination, auscultation, electrocardiogram and chest X-ray. The precise data for analyses were obtained from 174 among 181 subjects examined. The details of study population are shown in Table I.

2) Examination
M-mode echocardiography was obtained by Fukuda Denshi SSD-110S using a transducer with frequency of 3.5 or 2.25 MHz. The transducer was held strictly in the position where the echo beam transversed the interventricular septal wall vertically. The echocardiogram was

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Boys</th>
<th>Girls</th>
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</thead>
<tbody>
<tr>
<td>6—7</td>
<td>30</td>
<td>28</td>
</tr>
<tr>
<td>9—10</td>
<td>20</td>
<td>18</td>
</tr>
<tr>
<td>12—13</td>
<td>40</td>
<td>38</td>
</tr>
<tr>
<td>Total</td>
<td>90</td>
<td>84</td>
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TABLE I

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recorded on the stripchart with a paper speed of 100 mm/s using an oscillographic recorder (ECO-12SS type, Fukuda Denshi). Interventricular septal thickness (IVST) and left ventricular dimension at end diastole (LVDd) were measured at the onset of QRS complex.

Body surface potential mapping was recorded by a Heart Potential Mapper (model 5100A, Chunichi Denshi Kogyosh Co., Ltd.). All the electrocardiographic data for mapping were sampled simultaneously at intervals of 1 and 4 msec. Electrode position and number were followed the method described by Yamada.

Transmural conduction velocity index (TCVI) was calculated by the following formula in children with the same thickness of ventricular septum and left ventricular free wall; TCVI = IVST/VAT. The onset of QRS complex was defined as the time just before maximum potential successively increasing exceed 0.1 mV in lead V4. Ventricular activation time (VAT), which was considered to be the begining of
intrisicoid deflection, was arbitrarily measured at the peak of R wave of in lead V5 electrocardiogram digitized at 1 msec interval.

RESULTS

IVST and LVDd were increased with age. The mean values (mm) of IVST and LVDd were 6.8 and 33.8 for 6–7 years of age, 7.9 and 35.6 for 9–10 years, 8.0 and 41.1 for 12–13 years, respectively.

VAT had a tendency to increase with age, but correlation was not so good as for IVST and LVDd. The mean values (msec) were 28.7 for 6–7 years of age, 29.6 for 9–10 years and 32.5 for 12–13 years (Fig. 1).

To clarify which of LVDd and IVST was important as a determining factor for VAT, the VAT was plotted against both LVDd and IVST in all children. There was no significant correlation between each variables. When the VAT was plotted against IVST in 23 cases with almost the same LVDd (38–40 mm), there was a negative correlation between the two variables as shown in Fig. 2 ($y = -2.10x + 47.6, r = 0.61$).

These results suggested that TCVI might be increased with IVST. In order to prove the assumption, TCVI was plotted against IVST in all children (Fig. 3). TCVI was positively correlated with IVST; $y = 3.25x + 0.56$ (y: TCVI, x: IVST), $r = 0.75$ and $N = 125$.

When the VAT was plotted against LVDd in the 37 cases with almost the same IVST (6–7 mm), there was a positive correlation between the two variables as shown in Fig. 4 ($y = 0.55x + 10.9$, y: VAT, x: LVDd, $r = 0.61$, $N = 37$).

TCVI was not related to age in both sexes as shown in Fig. 5. This value in all age groups was $25.2 \pm 5.71$ (mean ± S.D.) and increased with the septal thickness. Maximum value of TCVI was 49 cm/sec.

DISCUSSION

The mean conduction velocity index (25.2 cm/sec) in the present study was lower than the value reported by Durrer et al. This may be caused by the difference in age or other biologic conditions. Ventricular activation time tended to increase together with left ventricular diastolic dimension. These results suggested that the ventricular activation time might be more related to the left ventricular enlargement. Velocity index increased with septal thickness. It was concluded that the cardiac hypertrophy itself caused the greater conduction velocity in the healthy children.

REFERENCES