Left Ventricular Activation Time in Normal Men

By TAKASHI WADA, M.D.

Simultaneous recordings from right and left precordial leads in 50 normal individuals were obtained. The duration of rS in lead V1 was always equal to or greater than the duration of qR in lead V2. Evidence is presented to support the thesis that the duration of rS in lead V1 is a more accurate index of left ventricular activation time than the duration of qR in the left precordial leads.

In direct leads obtained through a small electrode attached to the epicardium, there is a nearly perpendicular return of the R wave from its peak to the base line. This intrinsic deflection marks the extinction of the electric potential of the small core of ventricle to which the electrode is applied. In precordial leads, where a relatively large electrode is at a considerable distance from the heart, there is a more gradually sloping downstroke, or intrinsicoid deflection, since electric activity does not disappear simultaneously in all parts of the wide area subtended by the electrode.

In clinical electrocardiography, the time from the onset of the initial deflection to the peak of the R wave in precordial leads over the left ventricle is widely used for timing and measuring left ventricular activation. An electrode placed at V5 or V6 registers an R wave because of the predominating positive potentials derived from the anterior and lateral wall of the left ventricle. Negative potentials derived from the activation of the posterior wall of the left ventricle influence the duration of the positive phase in these leads and cause slurring or notching on the descending limb of the R wave. Thus these leads reflect counteracting positive and negative potentials. A reversed situation occurs at V8 where the positive deflection created by the activation of the posterior wall is altered by the negativity of the anterior wall. Therefore clinical left precordial electrocardiograms do not reflect precisely the entire left ventricular activation.

It may be assumed, then, that unless the descending limb of the R wave in the left precordial leads is nearly perpendicular, activation of some portion of the left ventricle is still continuing after the R wave has reached its peak. Measurements made on these leads then would not represent the true activation time of the entire left ventricle.

On the other hand, negative potentials over the right precordium (S wave) are believed to be reciprocals of the positive potentials created by the activation of the entire left ventricle. Therefore it is believed that this reflected negativity might be a more accurate measurement of complete left ventricular activation.

The purpose of this study was to examine the precordial leads in a large group of normal individuals, and to determine which lead most accurately measured the total activation time of the left ventricular wall.

Materials and Methods

Fifty men with normal cardiovascular systems, as judged by a careful history and examination, were selected for study. The group consisted of 14 resident physicians, 19 medical students, 7 hospital employees, and 10 convalescent noncardiac patients. Their ages ranged from 22 to 39 years with a mean of 29 years.

Precordial electrocardiograms, V3R through V8 were taken on each subject in the recumbent position with a Sanborn Twin-Beam Cardieter. An initial recording consisted of lead V5 on 1 channel.
taken simultaneously with each lead from $V_{3R}$ through $V_7$ on the other channel. Lead $V_8$ was later changed to $V_7$ as the constant, since there was no difference in the duration of the R wave in $V_8$ and $V_9$. However, the magnitude of the complex was usually greater in $V_7$ than in $V_8$, thus facilitating measurements. The second run consisted of $V_1$ as the constant reference lead taken simultaneously with $V_{3R}$ through $V_7$. Standardization was the customary 1 mv. per 10 mm. Paper speed was 75 mm. per second, so that the space between each vertical line of photographic paper was 3 mm. in width and 0.04 second in duration. With use of 3X magnifying lens the duration of the various components of the QRS complex was estimated to 0.005 second. The final figure represented an average of 3 independent measurements on 3 separate occasions. A total of 450 individual readings was made; 330 of these showed identical results in 3 separate determinations. The maximum difference in repeated measurements of the same tracing was 0.01 second. Measurements were made primarily in leads $V_1$ and $V_2$ because of their opposite locations in relation to the heart.

**RESULTS**

**Configuration and Timing of Initial Deflection in $V_1$ and $V_7$**

In all tracings the initial deflection in $V_1$ was upright. In 46, the initial deflection in $V_7$ was a q wave and began simultaneously with the r in $V_1$. In the remaining 4, the q wave was not present in $V_7$ and the R wave began later than the initial deflection in $V_1$. The delay was long enough to allow for a q wave if electromotive forces had been strong enough to produce one (fig. 1).

Of the 46 tracings with a measurable q in $V_7$, the r wave in $V_1$ was notched or slurred in 27 instances. This notch or slur, which represents the peak of the initial r in $V_1$, coincided in time with the nadir of the q wave in $V_7$ (fig. 2). Nineteen of the 46 tracings presented a smooth r in $V_1$. In 3 of these the nadir of q$V_7$ occurred simultaneously with the peak of r$V_1$ (figs. 3 and 4). In the remaining 16 tracings the nadir of q$V_7$ occurred earlier than the peak of r$V_1$, presumably coinciding in time with the forces that were insufficient to cause a notch or slur in $V_1$ (fig. 5).

Thus it is seen that in all instances manifesting a q wave in $V_7$, the duration of this deflection was equal to or less than the duration of the r wave in lead $V_1$.

**Time Relationship of the Nadir of SV$_1$ and Peak of RV$_7$**

In 12 of the 50 tracings studied, the nadir of SV$_1$ coincided with the peak of RV$_7$ (fig. 3). In 15, the nadir of SV$_1$ fell at various points on the descending limb of the R wave in $V_7$ or immediately after its return to the isoelectric line (figs. 1, 2, 4, and 5). In 28, a small s wave was present in $V_7$, the nadir of which coincided in time with the nadir of SV$_1$ in 16 instances. In the remaining 12 tracings the nadir of SV$_1$ fell at various points on the descending limb of RV$_7$. It should be emphasized that the nadir of the S wave in $V_1$ never occurred earlier than the peak of the R wave in $V_7$ or in any other lead over the left ventricle.

**Duration of QRS Complex in Leads $V_1$ and $V_7$**

Details of this analysis are presented in table 1. There was a significant difference in duration between rS, measured from the onset of the r to the nadir of the S wave, in lead $V_1$ and qR, measured from the onset of the
q to the peak of the R wave in lead V7. The duration of the former ranged from 0.035 to 0.055 second with a mean of 0.048 second. The duration of the latter ranged from 0.030 to 0.050 second, with a mean of 0.042, thus indicating that a greater potential was reflected by the negativity in lead V1 than the positivity in lead V7. In 6 instances, rS in V1 was as much as 0.015 second greater than qR in V7 (table 1, cases 5, 15, 21, 33, 39, and 44).

In no instance was the duration of rS in lead V1 shorter than the duration of qR in lead V7.

**Discussion**

It has been accepted generally that in normal individuals the activation of the septum occurs from both directions. However, the left side of the septum is activated from 0.01 to 0.015 second earlier than the right. This vectorial force produces a small r in the right ventricular leads that occurs simultaneously with a small q in the left ventricular leads. This was demonstrated in 46 tracings of the 50 subjects studied in this series.

In 27 instances in which the r in lead V1 was noted, the notch coincided with the nadir of the q wave in lead V7 and presumably was derived from the same force, that of the septal activation. As the nadir of the q wave in V7 is passed, the activation of the free wall of the left ventricle has progressed sufficiently to replace the downstroke of the q wave.

The negativity recorded as S in V1 is due to the reciprocal transmission of positive potentials derived from the forces created by activation of the left ventricular wall. In 12 of the 50 tracings studied here, the nadir of the S wave in V1 coincided with the peak of the R wave in V7. In the remaining 36, there was no coincidence; the nadir of SV1 always occurred after the peak of RV7. This lack of coincidence and longer duration of rS in lead V1 were noted by Rapaport and his associates although no actual measurements were made. It has been noted also that in general the magnitude of QRS complex in lead V1 is greater than that in V7.

It appears that the larger amplitude of the QRS complex in lead V1 and its longer duration provide a more accurate reflection of the entire left ventricular activation time than the potential registered by lead V7. Because of

---

**Fig. 3** Top. Simultaneous onset of the initial deflection in V1 and V7. The peak of a smooth r in V1 coincides with the nadir of q in V7 indicating a common source of forces, clearly the septal activation. The duration is identical from the onset of the r to the nadir of the S and from the onset of q to the peak of R in V7. (Patient 2)

**Fig. 4** Middle. Simultaneous onset of the initial deflection in V1 and V7. However, the initial deflection in V7, a q wave, is very small and its nadir occurred earlier than the peak of the r wave in V1. The duration from the onset of the q to the peak of R in V7 is shorter than the duration from the onset of the r to the nadir of S in V1. This suggests that some activation of the left ventricle is still going on after the inscription of the peak of RV7. (Patient 12)

**Fig. 5** Bottom. Simultaneous onset of the initial deflection in V3 and V7. The tall, smooth r wave in V1 and its peak coincides with the nadir of the q wave in V7. The duration from the onset of the r to the nadir of the S in V1 is slightly longer than the duration from the onset of q to the peak of R in V7. Note a delay in the inscription of the initial deflection in V3, the transitional area. If this were not present, the longer duration of RS in V3 might be a better index for the total activation time of the ventricles. (Patient 31)
TABLE 1.—Duration of QRS Complex in Leads $V_1$ and $V_7$

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Duration of rs in $V_1$</th>
<th>Duration of qR in $V_1$</th>
<th>Case no.</th>
<th>Duration of rs in $V_7$</th>
<th>Duration of qR in $V_7$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.045</td>
<td>0.045</td>
<td>26</td>
<td>0.045</td>
<td>0.035</td>
</tr>
<tr>
<td>2</td>
<td>0.050</td>
<td>0.050</td>
<td>27</td>
<td>0.055</td>
<td>0.045</td>
</tr>
<tr>
<td>3</td>
<td>0.045</td>
<td>0.040</td>
<td>28</td>
<td>0.045</td>
<td>0.045</td>
</tr>
<tr>
<td>4</td>
<td>0.050</td>
<td>0.045</td>
<td>29</td>
<td>0.045</td>
<td>0.040</td>
</tr>
<tr>
<td>5</td>
<td>0.055</td>
<td>0.040</td>
<td>30</td>
<td>0.050</td>
<td>0.045</td>
</tr>
<tr>
<td>6</td>
<td>0.055</td>
<td>0.050</td>
<td>31</td>
<td>0.050</td>
<td>0.045</td>
</tr>
<tr>
<td>7</td>
<td>0.055</td>
<td>0.045</td>
<td>32</td>
<td>0.055</td>
<td>0.045</td>
</tr>
<tr>
<td>8</td>
<td>0.045</td>
<td>0.045</td>
<td>33</td>
<td>0.055</td>
<td>0.040</td>
</tr>
<tr>
<td>9</td>
<td>0.040</td>
<td>0.040</td>
<td>34</td>
<td>0.040</td>
<td>0.035</td>
</tr>
<tr>
<td>10</td>
<td>0.040</td>
<td>0.040</td>
<td>35</td>
<td>0.045</td>
<td>0.035</td>
</tr>
<tr>
<td>11</td>
<td>0.045</td>
<td>0.040</td>
<td>36</td>
<td>0.045</td>
<td>0.040</td>
</tr>
<tr>
<td>12</td>
<td>0.050</td>
<td>0.040</td>
<td>37</td>
<td>0.050</td>
<td>0.040</td>
</tr>
<tr>
<td>13</td>
<td>0.040</td>
<td>0.040</td>
<td>38</td>
<td>0.045</td>
<td>0.040</td>
</tr>
<tr>
<td>14</td>
<td>0.045</td>
<td></td>
<td>39</td>
<td>0.050</td>
<td>0.035</td>
</tr>
<tr>
<td>15</td>
<td>0.050</td>
<td>0.035</td>
<td>40</td>
<td>0.045</td>
<td>0.040</td>
</tr>
<tr>
<td>16</td>
<td>0.050</td>
<td>0.045</td>
<td>41</td>
<td>0.050</td>
<td>0.040</td>
</tr>
<tr>
<td>17</td>
<td>0.040</td>
<td>0.040</td>
<td>42</td>
<td>0.040</td>
<td>0.040</td>
</tr>
<tr>
<td>18</td>
<td>0.050</td>
<td>0.045</td>
<td>43</td>
<td>0.045</td>
<td>0.045</td>
</tr>
<tr>
<td>19</td>
<td>0.050</td>
<td>0.050</td>
<td>44</td>
<td>0.050</td>
<td>0.035</td>
</tr>
<tr>
<td>20</td>
<td>0.055</td>
<td></td>
<td>45</td>
<td>0.045</td>
<td>0.045</td>
</tr>
<tr>
<td>21</td>
<td>0.055</td>
<td>0.040</td>
<td>46</td>
<td>0.050</td>
<td>0.045</td>
</tr>
<tr>
<td>22</td>
<td>0.050</td>
<td>0.040</td>
<td>47</td>
<td>0.045</td>
<td></td>
</tr>
<tr>
<td>23</td>
<td>0.050</td>
<td>0.040</td>
<td>48</td>
<td>0.045</td>
<td>0.040</td>
</tr>
<tr>
<td>24</td>
<td>0.050</td>
<td>0.040</td>
<td>49</td>
<td>0.050</td>
<td>0.045</td>
</tr>
<tr>
<td>25</td>
<td>0.035</td>
<td>0.035</td>
<td>50</td>
<td>0.045</td>
<td>0.045</td>
</tr>
</tbody>
</table>

An average measurement of 3 separate readings is shown above. There were 4 instances in which the q wave was absent in $V_7$, which accounts for the blank spaces in the table.

its position in relation to the left ventricular muscle mass, lead $V_1$ reflects the over-all negativity during the activation of the free wall of this chamber. On the other hand, the location of the electrode at $V_7$ is such that it cannot record the potentials of all portions of the left ventricle. The fact that the electrode at $V_1$ is closer to the heart and that there is less interposition of lung between this electrode and the heart than there is between the heart and the electrode at $V_7$ seems to account for the greater amplitude of the QRS complex in lead $V_1$.

Twenty-eight of the tracings in this study presented a qRs type of complex in lead $V_7$, in which the s wave reflected the activation of some portion of the heart other than the anterolateral wall beneath the electrode. In 16 of these, the nadir of the s wave in $V_1$ coincided with the nadir of the s wave in $V_7$. This suggests a common source, presumably from forces arising from the activation of the posterior wall of the left ventricle. In the other 12 instances with a small s wave in lead $V_7$, the nadir of the S$V_1$ fell at any point from the peak of the R wave in $V_7$ to its isoelectric line. Activation of the crista supraventricularis causes this late negativity in the left precordial leads and at the same time relative positivity in the right precordial leads, but is manifested as the ascending limb of the S wave in lead $V_1$.

The quantitative measurements of the QRS complexes described above are shown in table 2. The most pertinent measurements are those of the duration of rS, from the onset of the r to the nadir of the S wave in lead $V_1$, and the duration of qR, from the onset of the q to the peak of the R wave in lead $V_7$. The $p$ value of the differences was less than 0.001. It is concluded, therefore, that lead $V_1$ may be used routinely for more accurate determination of the left ventricular activation time in normal men than the usual leads over the left precordium. The reliability of the peak of the R wave in left ventricular
leads as an intrinsic or intrinsicoid deflection has also been questioned by others.5, 6

Wilson suggested that the true intrinsic deflection would be the lowest or most negative point and not the peak of the R wave.5 Sodi-Pallares believed that the true intrinsic deflection occurs somewhere on the lower one half of the descending limb of the R wave. The results of the present study seem to indicate that the nadir of the S in V1 appears to approximate most closely the instant of arrival of the activation potential at the left ventricular epicardial surface. Thus an accurate measurement of the left ventricular activation time is provided.

**Summary and Conclusions**

Simultaneous electrocardiographic recordings using right and left precordial leads were taken in 50 normal individuals.

Detailed analyses of QRS complexes were made with special reference to leads V1 and V7.

The duration of rS in lead V1, from the onset of the r to the nadir of the S wave was always the same or greater than the duration of qR in lead V7, from the onset of the q to the peak of the R wave.

The advantage of choosing lead V1 for the measurement of left ventricular activation time was discussed.

It is concluded that the duration of rS in lead V1 appears to be a more accurate index for the measurement of left ventricular activation time than the generally used measurement of left precordial complexes.

**Acknowledgment**

The author wishes to express his gratitude to Drs. Gordon B. Myers and Harper K. Hellem, Detroit, Mich., and to Dr. James Baer, Dearborn, Mich., for their valued advice and cooperation in the performance of this study. The author is also grateful to Dr. John Ord for the statistical analyses and to Dr. Kouichi Tanaka for his assistance in writing the manuscript.

**Summario in Interlingua**

Electrocardiogrammas simultaneae a derivatio dextero- e sinistro-precordial esseva obtenite ab 50 individuos normal.

Detaliate analyses de complexos QRS esseva effectuate con refe:ntia special al derivationes V1 e V7.

Le duration de rS in derivation V1 (i.e. le intervallo ab le declaration del unda r usque al nadir del unda S) esseva semper al minus equal al duration de qR in derivation V7 (i.e. al intervallo ab le declaration del unda q usque al zenit del unda R).

Es discutite le avantage de seliger le derivation V1 pro le mesuration del tempore de activation sinistro-ventricular.

Es conclusite que le duration de rS in le derivation V1 es apparentemente un plus accurate indice pro le mesura del tempore de activation sinistro-ventricular que le usualmente empletate parametros in le complexos precordial.

**References**


Left Ventricular Activation Time in Normal Men
TAKASHI WADA

Circulation. 1959;19:868-872
doi: 10.1161/01.CIR.19.6.868

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1959 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/19/6/868

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org/subscriptions/