Some Pitfalls of Vectorcardiographic Diagnosis of Myocardial Infarction with Particular Respect to Emphysema

By Kazuhiko Murata, M.D., Satoru Matsushita, M.D., Hiroshi Kurihara, M.D., and Masaji Seki, M.D.

SUMMARY

Vectorcardiographic changes indicating old myocardial infarction were observed in eight of 288 patients who at autopsy did not show extensive myocardial disease. Of these eight patients four had been given a misdiagnosis of anteroseptal infarction, while four had been suspected of having anterolateral or anterior infarction on the basis of an abnormal configuration of the initial portion of the QRS loop. The possibility of overdiagnosis of myocardial infarction should be borne in mind in reading vectorcardiograms, especially in the absence of clear-cut history of the condition.

A prominent Q wave simulating anteroseptal or anterior infarction was observed in lead V2 or V3, or in both in 12-lead electrocardiograms in all these eight cases. A high incidence of coronary sclerosis, scattered myocardial fibrosis and pulmonary emphysema was noted in these false positive cases at autopsy, but right ventricular hypertrophy, significant septal hypertrophy, or giant right atrium was not demonstrated.

ADDITIONAL INDEXING WORDS:
Myocardial fibrosis

Diagnostic error of myocardial infarction

ALTHOUGH vectorcardiography has been claimed to be a sensitive tool in diagnosis of myocardial infarction,1-4 thorough studies on interpretative errors are necessary for precise appraisal of the accuracy of this method. The possibility of vectorcardiographic overdiagnosis of myocardial infarction has been pointed out by few investigators,3, 5-8 and to these is added the following report of eight cases diagnosed by vectorcardiogram as myocardial infarction when none was present at postmortem examination.

Methods

All available Frank lead vectorcardiograms of 332 patients, including 43 patients with autopsy-proved myocardial infarction, who came to autopsy either at the Yokufukai Geriatric Hospital or at the University of Tokyo were reviewed. In 37 cases the vectorcardiograms were believed indicative of myocardial infarction according to the criteria described by previous investigators.2, 3, 9-11

The autopsy records of these 37 cases initially selected were reviewed and the 28 cases in which myocardial infarction was found at autopsy were excluded. A myocardial infarct was defined as extensive fibrosis of the myocardium extending at least 2 cm in one dimension. Another case of extensive amyloid infiltration of the myocardium was also excluded. Thus, there remained eight cases, and these constitute the group studied.

The method of recording the vectorcardiograms was the same as previously described.* 12, 13 The chest electrodes were placed at the level of the fifth intercostal space. The three planar projections of the vectorcardiogram were photographed with a Canon camera unit from the oscilloscope screen. The QRS loop was interrupted 500 or 800 times per second. The sagittal plane was viewed from the left.

Examinations of the hearts were performed according to our routine method, cross-sectioning the organ at intervals of 0.5 cm. The coronary

From the Third Department of Internal Medicine, Faculty of Medicine, University of Tokyo and Yokufukai Geriatric Hospital, Tokyo, Japan.

*Frank lead vectorcardiograms were recorded by using FVC-3 of Fukuda Medical Electronic Co. in Japan.

Circulation, Volume XXXV, January 1967

172
arteries were sectioned transversely at intervals of 0.3 to 0.5 cm. A reduction in diameter of the coronary artery of more than 50% in any section was designated as moderate sclerosis, while severe sclerosis was diagnosed when the diameter was reduced more than 75%. Left ventricular hypertrophy was diagnosed when (1) the heart weight was 300 g or more and the thickness of the left ventricular wall excluding papillary and trabecular muscles was 1.0 cm or more, or (2) the heart weight was 250 g or more and the thickness of the left ventricular wall was 1.1 cm or more.12

**Results**

Among the 288 autopsy cases not associated with myocardial infarction or amyloidosis reviewed, there were eight in which vectorcardiograms were interpreted as indicative of old myocardial infarction. All the patients were more than 59 years of age, and five were more than 80.

**Vectorcardiograms**

Two different patterns were observed in the vectorcardiograms. The abnormal findings were most obvious in the horizontal plane in these cases, although the sagittal plane was helpful for the determination of the orientation of the initial QRS vector.

In four cases (cases 1 to 4), the QRS loop started directly posteriorly and no QRS vector was directed anteriorly as shown in figure 1. In this group, the entire QRS loop was inscribed counterclockwise in the horizontal plane. In these cases diagnosis had been anteroseptal infarction. The T loop was oriented to the right in two cases and posteriorly in one case.

The initial QRS vector was directed to the right and slightly anteriorly in the remaining four cases (cases 5 to 8). The initial portion of the QRS loop was inscribed clockwise in these cases. The remainder of the loop was inscribed counterclockwise in three, while a figure-of-eight configuration was observed in one. No QRS vector was directed to the left and anteriorly. An illustrative vectorcardiogram of this type is shown in figure 2. Old anterior or anterolateral infarction was seriously suspected in these four cases on the basis of the abnormal configuration of the initial portion of the QRS loop, although the entire configuration of the loop was not very typical. The direction of the T loop was within normal range in three, while a rightward displacement of the loop was seen in one.

**Electrocardiograms**

Routine 12-lead electrocardiograms in the eight cases were also consistent with old anteroseptal or anterior infarction. As shown in table 1, QS deflection in leads V1 through V3 was observed in four cases, whereas a poor progression of R waves in the right precordial leads with QS in lead V3 was demonstrated in two. QS pattern limited to leads V1 and V2 was seen in two cases.

**Autopsy Findings**

As shown in table 1, mild left ventricular hypertrophy was present in three cases, but

### Table 1

<table>
<thead>
<tr>
<th>Case</th>
<th>Age</th>
<th>Sex</th>
<th>Electrocardiogram</th>
<th>Heart weight (g)</th>
<th>LVH</th>
<th>RVH</th>
<th>Coronary sclerosis</th>
<th>Myocardial fibrosis</th>
<th>Pulmonary emphysema</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>59</td>
<td>M</td>
<td>QS in V1-3</td>
<td>400</td>
<td>+</td>
<td>−</td>
<td>++++</td>
<td>+</td>
<td>+++</td>
</tr>
<tr>
<td>2</td>
<td>81</td>
<td>M</td>
<td>QS in V1-3</td>
<td>230</td>
<td>−</td>
<td>−</td>
<td>+</td>
<td>−</td>
<td>+</td>
</tr>
<tr>
<td>3</td>
<td>83</td>
<td>F</td>
<td>QS in V1-3</td>
<td>240</td>
<td>−</td>
<td>−</td>
<td>++++</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>4</td>
<td>90</td>
<td>F</td>
<td>QS in V1-3</td>
<td>300</td>
<td>+</td>
<td>−</td>
<td>++++</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>5</td>
<td>61</td>
<td>M</td>
<td>QS in V1-2</td>
<td>330</td>
<td>+</td>
<td>−</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>6</td>
<td>76</td>
<td>F</td>
<td>QS in V3</td>
<td>235</td>
<td>−</td>
<td>−</td>
<td>++++</td>
<td>−</td>
<td>+</td>
</tr>
<tr>
<td>7</td>
<td>87</td>
<td>F</td>
<td>QS in V1-2</td>
<td>245</td>
<td>−</td>
<td>−</td>
<td>++++</td>
<td>−</td>
<td>+</td>
</tr>
<tr>
<td>8</td>
<td>89</td>
<td>F</td>
<td>QS in V3</td>
<td>180</td>
<td>−</td>
<td>−</td>
<td>++</td>
<td>+</td>
<td>+++</td>
</tr>
</tbody>
</table>

Abbreviations: LVH = left ventricular hypertrophy; RVH = right ventricular hypertrophy. The pathological findings were graded into mild (+), moderate (++), and severe (+++) degree.
marked hypertrophy of the interventricular septum was not observed. Right ventricular hypertrophy or giant right atrium was not present in any case. In all the cases coronary sclerosis of moderate or severe degree was associated. Scattered fibrotic lesions of the myocardium were observed in five cases. Apart from the cardiac findings, a high incidence of pulmonary emphysema was demonstrated, lung disease of moderate to severe degree being demonstrated in six cases.

**Discussion**

It is well recognized that significant-appearing Q waves simulating myocardial infarction may be seen not infrequently in scalar electrocardiograms, but less attention seems to have been directed to the possibility of overdiagnosis of myocardial infarction by vectorcardiography. Hugenholtz and associates reported that a large initial rightward force suggestive of lateral infarction may be seen in septal hypertrophy, whereas a vectorcardiographic pattern simulating anteroseptal infarction is occasionally observed in the presence of giant right atrium. Banta and Estes stated that a vectorcardiogram may mimic

---

**Figure 1**

Case 2: As seen in the magnified view of the horizontal plane, the QRS loop started posteriorly and no QRS vector was directed anteriorly. The T loop was directed anteriorly to the right. The QRS loop was interrupted 800 times per second. QS pattern was present in leads V through V in the routine electrocardiogram. The vectorcardiogram and electrocardiogram were interpreted as diagnostic of old anteroseptal infarction. At autopsy, the heart weight was 230 g and no myocardial fibrosis was observed, although moderate coronary sclerosis was present.
MYOCARDIAL INFARCTION

Figure 2

Case 6: The QRS loop was interrupted 500 times per second. Although the initial QRS vector was directed to the right and slightly anteriorly, a 0.02-second vector was obviously directed posteriorly. The initial portion of the QRS loop was inscribed clockwise. The direction of the T loop was within the normal range. There were tiny r waves in leads V₁ and V₂ in 12-lead electrocardiogram, but no r wave was present in lead V₃. The vectorcardiogram was interpreted as strongly suggestive of anterolateral infarction, while the electrocardiogram was thought to be consistent with anterior infarction. The heart was not enlarged at autopsy and no significant myocardial fibrosis was demonstrated.

myocardial infarction in cases of idiopathic myocardial hypertrophy. On the other handAbramson,⁵ Surawicz and associates,⁷ and Toyama⁸ observed abnormal posterior or superior orientation of the initial QRS vector in apparently normal people.

In the present study, abnormal configuration of the initial portion of the QRS loop simulating myocardial infarction was observed in eight of 288 cases in which extensive myocardial disease was not demonstrated later at autopsy. Anteroseptal infarction was diagnosed in four of these eight cases, while anterior or anterolateral infarction was suspected in the remaining four cases. Particularly noteworthy in this group was the high incidence of coronary sclerosis and pulmonary emphysema. Scattered myocardial fibrosis was also frequently demonstrated.

Differentiation of these false positive cases from cases of “true” myocardial infarction is of great clinical importance, but a detailed
comparative analysis of the vectorcardiograms was not attempted in the present study because of the rather small number of the cases. Our present impression, however, is in agreement with the description by Surawicz and associates who considered this differentiation extremely difficult.

The mechanism of development of the vectorcardiographic findings simulating myocardial infarction in the present series is not entirely clear, but two alternative explanations may be possible. The high incidence of pulmonary emphysema suggests the first possibility that the abnormal configuration of the QRS loop is a result of altered position of the heart. It is well known that significant Q waves simulating anteroseptal infarction are not uncommon in patients with severe pulmonary emphysema, and this finding has been explained as a result of downward displacement of the heart. The electrode position at the level of fifth intercostal space may be too high for patients with severe pulmonary emphysema. On the other hand, the high incidence of coronary sclerosis and myocardial fibrosis, though scattered, raises a second possibility described by Abramson and Toyama that abnormal intraventricular conduction is responsible. No other evidence of conduction delay, however, was demonstrated in the present series. A systematic study of the conduction system was not performed.

References

infarction: I. Significant Q wave in leads V\textsubscript{1} to V\textsubscript{4}. Jap Circ J 20: 201, 1956. (In Japanese.)


Imperfect Logic in a Nobelate

The name laevogram indicates that the curve corresponds to a systole in which the contraction begins in the left side of the heart and passes from this to the right heart. A dextrogram gives the reverse picture.

It has been doubted whether in man a diphasic action current of the heart with downwardly directed phase I in Leads II and III corresponds to a laevogram and it has been believed on theoretical grounds that the position may be the reverse. But we have by chance had the opportunity to apply a direct control in man.

There was a hitherto quite healthy man whose sternum had been surgically removed. His heart was covered only by a thin layer of skin. Light tapping on the area of the heart caused an extrasystole. Because the right half of the heart lies in man on the anterior side and thus must be stimulated mechanically by the tapping this must cause a dextrogram. Fig. 24 [included] confirmed this prediction.—WILLIAM EINTHOVEN: The String Galvanometer and the Measurement of the Action Currents of the Heart. In Nobel Lectures: Physiology or Medicine. New York, Elsevier Publishing Co., 1965, p. 108.

Man without a sternum. The heart was as good as naked and covered only by a thin layer of skin. A light tapping on the heart area caused an extrasystole. This is a pure dextrogram which is in agreement with the theory that the segments of the heart muscle are electrically to be regarded as being individual entities.
Some Pitfalls of Vectorcardiographic Diagnosis of Myocardial Infarction with Particular Respect to Emphysema

KAZUHIKO MURATA, SATORU MATSUSHITA, HIROSHI KURIHARA and MASUJI SEKI

Circulation. 1967;35:172-177
doi: 10.1161/01.CIR.35.1.172

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1967 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/35/1/172

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/