Electrocardiograms displaying QS or rS complexes in precordial leads V1 to V3 are subject to varying interpretation. Some authorities believe that, in the presence of other electrocardiographic evidence for left ventricular hypertrophy, these changes do not necessarily justify the additional diagnosis of anteroseptal infarction. Others consider such QS or rS complexes with diminutive r waves to be always indicative of concomitant anterior myocardial infarction, whereas they regard a deep S in V2 and tall R in V6 as additional evidence for the diagnosis of left ventricular hypertrophy. In most instances this differential point remains unsolved. Recent interest in aortic valve surgery has accentuated the need for an accurate diagnosis of superimposed myocardial infarction and for a precise electrocardiographic differentiation between left ventricular hypertrophy and infarction of the anterior wall of the left ventricle.

The clinical application of corrected lead systems in vectorcardiography has made possible the exact delineation of early QRS vectors and thereby the identification of specific areas of infarction. It has recently been demonstrated, moreover, that the diagnosis of left ventricular hypertrophy may be made with greater accuracy by the use of the vectorcardiogram.

In anteroseptal and anterolateral wall infarction the 0.01- and 0.02-second QRS vectors as projected on the horizontal and left sagittal planes are displaced significantly posteriorly in relation to their orientation in normal individuals. The direction of the 0.03-second and maximum QRS vectors, on the other hand, showed less significant variation from the normal. Diagnoses based on these criteria were highly reliable in a series of autopsied patients.

Left ventricular hypertrophy has been shown to affect in particular the orientation of the 0.03-second and maximum QRS vectors toward a more posterior, superior, and leftward direction. In addition, a marked increase in magnitude of these vectors has been found. Significant correlation of both measurements with left ventricular systolic pressures has been demonstrated in patients with aortic stenosis in whom the severity of the lesion was known from data obtained by cardiac catheterization. Thus, increasing left ventricular pressure leads to left ventricular hypertrophy and affects particularly QRS vectors in the mid-period of the total QRS interval.

The reliability of these criteria was evaluated in 60 autopsied patients, all of whom were shown to have left ventricular hypertrophy but 24 of whom demonstrated anteroseptal or anterolateral infarction as well. The results have also been compared with data obtained in 60 normal individuals in this laboratory and with other control series reported elsewhere. Eleven of these patients have been the subject of an earlier report.
ANANTERIOR WALL INFARCTION

Methods and Materials

Vectorcardiograms were recorded by the Frank technic as previously described in detail. In nearly every instance the recording was made in the 2 weeks preceding autopsy. Twenty patients were studied at the Mallory Institute of Pathology, Boston City Hospital, and form a part of a larger series presently under study. Ten of these had coronary injection studies. Forty patients were studied at the Pathology Department, Peter Bent Brigham Hospital, Boston. About half of these patients had undergone cardiac surgery for rheumatic heart disease. In all instances the heart was carefully studied by parallel slicing and detailed microscopic examination of affected areas. Infarcted regions were carefully mapped in accordance with a system previously described. Left ventricular hypertrophy was considered to be present when, in addition to an increased heart weight, the left ventricular wall at the level of the anterior papillary muscle measured more than 11 mm.; right ventricular hypertrophy was diagnosed when the free wall of the right ventricle at the right anterior papillary muscle measured more than 7 mm. in thickness.

The patients ranged from 18 to 80 years in age and consisted of 40 men and 20 women. Twelve-lead electrocardiograms recorded at or about the same time as the vectorcardiograms were reviewed and interpreted according to standard criteria. The horizontal plane vectorcardiograms were analyzed with regard to the direction of the 0.01-, 0.02-, 0.03-, 0.04-second, and maximum QRS vectors. The magnitude of the latter was also determined. The material was analyzed in the manner of earlier studies. Student's t test was used to evaluate the significance of the results.

Results

At postmortem examination 13 of the 36 patients with left ventricular hypertrophy without anterior wall infarction, were found to have right ventricular hypertrophy as well. They were considered as a separate category. A second subgroup consisted of six patients in whom, in addition to left ventricular hypertrophy, an isolated posterobasal infarction was found. These two subgroups and the 17 patients with pure left ventricular hypertrophy were contrasted individually and as a whole with the group of normal subjects (fig. 1).

Of the 24 patients with left ventricular hypertrophy and anterior wall infarction, the infarct was restricted to the anteroseptal region in 15 patients; the remaining nine demonstrated at times quite extensive involvement of the anterior wall including part of the anterolateral wall. Again, each of the two subgroups as well as the combined infarction group was compared with the normal group.

Table 1 gives the direction of the 0.01-, 0.02-, 0.03-second, and maximum horizontal plane QRS vector as well as the control values in 60 adults previously reported.

In table 2 the category with pure left ven-
Table 1

<table>
<thead>
<tr>
<th>Category</th>
<th>Pure LVH</th>
<th>LVH with postero-basal infarction</th>
<th>LVH with RVH</th>
<th>Total LVH</th>
<th>Normals</th>
<th>Anteroseptal infarction</th>
<th>Anterolateral infarction</th>
<th>Anteroseptal and anterolateral infarction combined</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. patients</td>
<td>17</td>
<td>6</td>
<td>13</td>
<td>36</td>
<td>60</td>
<td>15</td>
<td>9</td>
<td>24</td>
</tr>
<tr>
<td>0.01-second vector</td>
<td>97</td>
<td>149</td>
<td>82</td>
<td>100</td>
<td>98</td>
<td>337</td>
<td>258</td>
<td>NC†</td>
</tr>
<tr>
<td>S.D.</td>
<td>41.7</td>
<td>22.6</td>
<td>33.8</td>
<td>43.1</td>
<td>25.6</td>
<td>W8§</td>
<td>W8</td>
<td>W8</td>
</tr>
<tr>
<td>T test*</td>
<td>—; NS‡</td>
<td>5.2; p &lt; 0.001</td>
<td>—; NS</td>
<td>—</td>
<td>—; NS</td>
<td>—</td>
<td>—</td>
<td>—; NS</td>
</tr>
<tr>
<td>0.02-second vector</td>
<td>44</td>
<td>83</td>
<td>49</td>
<td>52</td>
<td>50</td>
<td>316</td>
<td>267</td>
<td>298</td>
</tr>
<tr>
<td>S.D.</td>
<td>29.8</td>
<td>39.0</td>
<td>20.9</td>
<td>32.0</td>
<td>24.2</td>
<td>31.9</td>
<td>44.6</td>
<td>43.8</td>
</tr>
<tr>
<td>T test</td>
<td>—; NS‡</td>
<td>—; NS‡</td>
<td>—; NS‡</td>
<td>—; NS‡</td>
<td>—</td>
<td>—</td>
<td>&gt;10; p &lt; 0.001</td>
<td>&gt;10; p &lt; 0.001</td>
</tr>
<tr>
<td>0.03-second vector</td>
<td>340</td>
<td>4</td>
<td>5</td>
<td>353</td>
<td>8</td>
<td>313</td>
<td>290</td>
<td>304</td>
</tr>
<tr>
<td>S.D.</td>
<td>22.2</td>
<td>72.8</td>
<td>22.6</td>
<td>33.3</td>
<td>15.6</td>
<td>31.7</td>
<td>23.5</td>
<td>30.9</td>
</tr>
<tr>
<td>T test</td>
<td>4.9; p &lt; 0.001</td>
<td>—; NS‡</td>
<td>—; NS‡</td>
<td>2.4; p &lt; 0.05</td>
<td>8.9; p &gt; 0.001</td>
<td>&gt;10; p &lt; 0.001</td>
<td>9.62; p &lt; 0.001</td>
<td></td>
</tr>
<tr>
<td>Max. QRS vector</td>
<td>316</td>
<td>349</td>
<td>310</td>
<td>319</td>
<td>346</td>
<td>303</td>
<td>305</td>
<td>304</td>
</tr>
<tr>
<td>S.D.</td>
<td>30.4</td>
<td>41.8</td>
<td>52.9</td>
<td>43.9</td>
<td>30.5</td>
<td>32.3</td>
<td>20.9</td>
<td>28.6</td>
</tr>
<tr>
<td>T test</td>
<td>3.6; p &lt; 0.001</td>
<td>—; NS‡</td>
<td>2.35; p &lt; 0.05</td>
<td>3.2; p &lt; 0.001</td>
<td>6.1; p &lt; 0.001</td>
<td>5.8; p &lt; 0.001</td>
<td>5.9; p &lt; 0.001</td>
<td></td>
</tr>
</tbody>
</table>

*All measurements compared with their control counterparts.
†NC, not calculated.
‡NS, not significant.
§WS, wide scatter.
tricular hypertrophy and the group whose left ventricular hypertrophy might be associated with right ventricular hypertrophy, but not with myocardial infarction, are compared with each of the infarction groups and with the infarction group as a whole. The differences found are presented in figures 1, 2, and 3. Analysis of the electrocardiograms in these 60 patients showed three with QS complexes in $V_1$, $V_2$, and $V_3$ (fig. 4) and nine in whom diminutive or small and decreasing r waves were seen in these same leads (figs. 5 and 6).

The two major groups were also clearly separated by their respective heart weights. In the group of pure left ventricular hypertrophy the average heart weight was 700 Gm.; in the subgroup with additional right ventricular hypertrophy the average was 610 Gm. By contrast, no infarcted heart weighed more than 600 Gm. The average weight in the group with anteroseptal infarction was 473 Gm.; with anterolateral infarction, 491 Gm. In the small subgroup of left ventricular hypertrophy with posterobasal infarction the heart averaged 530 Gm.

**Discussion**

**0.01-Second Vector**

The direction of the 0.01-second vector of the left ventricular hypertrophy group pointed anteriorly and was nearly identical with the control value. Only the subgroup with posterobasal infarction showed a significant difference from the normal ($p < 0.001$). In these six patients the vector pointed much more rightward than normal. Although this could be expected theoretically, previous studies on autopsied patients with posterobasal infarction had not indicated significant differences until the time of inscription of the 0.03- and 0.04-second vector.

Comparison of the left ventricular hypertrophy group with the anterior wall infarct group proved impossible, since extreme varia-
tion in the latter category rendered calculations meaningless. However, the mean values of the anteroseptal and anterolateral subgroups (337° and 258°, respectively) were close to those reported earlier. Thus, although no statistical significance can be attached to the direction of the 0.01-second vector, great differences from the normal are found in most instances. The relative importance of this point in the vectorcardiographic diagnosis of anteroseptal infarction has been stressed previously.

0.02-Second Vector

In contrast, attention to the direction of the 0.02-second vector permits a sharp differentiation between the left ventricular hypertrophy group and the infarct group. All patients in the left ventricular hypertrophy group showed anteriorly directed vectors in close proximity to each other (fig. 2), although again a slightly more rightward tendency prevailed in the subgroup with posterobasal infarction. This vector could be almost perfectly superimposed upon the corresponding vector for the normal horizontal plane loop. Thus, as previously suggested, left ventricular hypertrophy did not significantly alter the early 0.01- and 0.02-second vectors.

Table 2

<table>
<thead>
<tr>
<th>Category</th>
<th>Pure LVH</th>
<th>Total LVH</th>
<th>Anteroseptal infarction</th>
<th>Anterolateral infarction</th>
<th>Total infarction group</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. patients</td>
<td>17</td>
<td>36</td>
<td>15</td>
<td>9</td>
<td>24</td>
</tr>
<tr>
<td>0.01-Second vector</td>
<td>97</td>
<td>100</td>
<td>337</td>
<td>258</td>
<td>NC</td>
</tr>
<tr>
<td>S.D.</td>
<td>41.9</td>
<td>43.1</td>
<td>11</td>
<td>0</td>
<td>WS</td>
</tr>
<tr>
<td>T test</td>
<td></td>
<td></td>
<td>9.7; p &lt; 0.001</td>
<td>&gt;10; p &lt; 0.001</td>
<td>&gt;10; p &lt; 0.001</td>
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<tr>
<td>0.02-Second vector</td>
<td>44</td>
<td>52</td>
<td>44.6</td>
<td>44.6</td>
<td>39.9</td>
</tr>
<tr>
<td>S.D.</td>
<td>29.8</td>
<td>32</td>
<td>44.6</td>
<td>44.6</td>
<td>39.9</td>
</tr>
<tr>
<td>T test</td>
<td></td>
<td></td>
<td>9.7; p &lt; 0.001</td>
<td>5.7; p &lt; 0.001</td>
<td>5.7; p &lt; 0.001</td>
</tr>
<tr>
<td>0.03-Second vector</td>
<td>340</td>
<td>353</td>
<td>313</td>
<td>290</td>
<td>304</td>
</tr>
<tr>
<td>S.D.</td>
<td>222</td>
<td>33.3</td>
<td>31.7</td>
<td>23.5</td>
<td>30.9</td>
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<tr>
<td>T test</td>
<td></td>
<td></td>
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<td>5.7; p &lt; 0.001</td>
<td>5.7; p &lt; 0.001</td>
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<tr>
<td>Max. QRS</td>
<td>316</td>
<td>319</td>
<td>305</td>
<td>305</td>
<td>305</td>
</tr>
<tr>
<td>S.D.</td>
<td>30.4</td>
<td>43.9</td>
<td>32.3</td>
<td>29.9</td>
<td>28.6</td>
</tr>
<tr>
<td>T test</td>
<td></td>
<td></td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
</tbody>
</table>

*Comparing total LVH group with each of the groups in whom LVH was associated with anterior wall infarction.

1WS, wide spread prevented calculation of S.D.

2Non-Gaussian distribution prevented calculation.
0.03-Second Vector

By contrast, analysis of the 0.03-second vector permits the diagnosis of left ventricular hypertrophy. The mean value of the entire left ventricular hypertrophy group (353°) differed significantly from the normal (8°, \( p < 0.05 \)). The difference is even more striking (figs. 1 and 2) when one examines only the subgroup with pure left ventricular hypertrophy (340° v. 8°, table 1). The latter value agrees closely with measurements in patients with aortic stenosis reported elsewhere.\(^7\)\(^{10}\)

Comparison with other control series\(^15\),\(^16\) in which the age groups are more closely comparable with the present series, indicates an even greater separation. These normal data have not been incorporated in table 1 because they were recorded with the SVEC III system.\(^16\) If they were used as the normal standard with which the present data are compared, the latter would take on even greater significance. Mori and co-workers\(^16\) found the normal 0.02-second vector directed at 71° and the 0.03-second vector at 26°. Pipberger, in a study of 100 normal subjects with a slightly younger average age, likewise showed a more anterior direction of these vectors than those.

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Figure 4

Vectorcardiogram of a 56-year-old man with significant aortic stenosis. In the horizontal plane the 0.02-second vector is directed at 30°, the 0.03-second vector at 360°, and the maximum QRS vector at 336° (magnitude 8 millivolts)—establishing a diagnosis of left ventricular hypertrophy without anterior infarction. On the other hand, deep QS complexes from \( V_4R \) to \( V_6 \) make it impossible to rule out associated anterior wall infarction on the electrocardiogram. At postmortem examination the heart weighed 800 Gm., no infarct was present (in this illustration only, is the sagittal plane viewed from the right side).
found in the 60 patients studied by Forkner and co-workers,\textsuperscript{11} which were used as controls in the present study.

As with the 0.01- and 0.02-second vector, associated posterobasal infarct directs the initial forces anteriorly for a longer time. Associated right ventricular hypertrophy acts in much the same manner (figs. 1, 2, and 3). Thus, conditions such as increased right ventricular mass, located anteriorly, or decreased left ventricular mass, situated posteriorly, either of which would increase the anterior and rightward salience of the initial part of the QRS loop, will, in isolated instances (fig. 2), result in overlap with the normal. If these cases are excluded, the 0.03-second vector becomes a highly reliable indicator of left ventricular hypertrophy ($p < 0.001$).

At the same time the infarct group demonstrated a 0.03-second vector directed even further posteriorly (fig. 2, table 2). Thus, the difference between the left ventricular hypertrophy and infarct groups remains a significant one, both for anteroseptal ($313^\circ$) and anterolateral ($290^\circ$) infarction ($p < 0.01$). Anterior wall infarction complicating hypertrophy results in the disappearance of electromotive forces in the anterior wall, induces a more posterior orientation of the 0.03-second vector than that which results from left ventricular hypertrophy consequent to the increased left ventricular mass. In this respect

*Figure 5*

Vectorcardiogram shows definite anteroseptal infarct, since the horizontal plane 0.02-second vector is directed at $317^\circ$, and maximum QRS vector at $323^\circ$. Left ventricular hypertrophy is suggested by direction of the 0.03-second vector at $323^\circ$ and by the increased magnitude of the maximum QRS vector. Neither diagnosis can be made with certainty on the electrocardiogram although anterior infarction may be suspected. Autopsy showed both.
it behaves much like the 0.02-second vector. Only the patients with the greatest increase in heart weight showed such a degree of posterior direction of their 0.03-second vector as to result in overlap with the infarct group (figs. 5 and 7).

**Maximum QRS Vector**

Study of the maximum QRS vector revealed a distribution of values similar to the 0.03-second vector (fig. 2). The total left ventricular hypertrophy group had a mean direction of 319°, which was close to that of the pure left ventricular hypertrophy group (316°), despite the persistent finding of a less posterior direction in the subgroup with postero-basal infarction. These findings are almost identical with the value of 318° found by Bristow and co-workers in a group of 41 patients with left ventricular hypertrophy without infarction, studied by vectocardiogram with the Frank lead system. They are also quite close to the 328° value found in 40 young patients with aortic stenosis studied by the cube vectocardiogram. Thus, the influence of increased ventricular mass is most pronounced at the time of the apogee of the QRS loop (figs. 1, 6, and 7). Its effect is similar to that of either anteroseptal (mean 303°) or anterolateral (mean 305°) myocardial infarction, since these conditions also deflect the vector loop in a posterior direction by virtue of absent anterior forces (fig. 5). The latter measurements, in turn, correspond very closely to those reported earlier in larger

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Electrocardiogram of a 46-year-old man with severe aortic stenosis. Small r waves in $V_{1R}$ to $V_2$ are associated with normal R waves in $V_3$ to $V_6$ and nonspecific T-wave inversion. Left ventricular hypertrophy can be suspected, particularly in view of left axis deviation, but not diagnosed with certainty on the electrocardiogram. The 0.02-second vector is directed at 78°, the 0.03 at 335°, and the maximum at 340°, thus establishing the diagnosis of left ventricular hypertrophy. This was confirmed at autopsy; the heart weighed 500 Gm.

Magnitude of Maximum QRS Vector

The magnitude of the maximum QRS vector, although variable, is usually decreased in infarction, and was always increased in left ventricular hypertrophy. The mean value in the left ventricular hypertrophy group was 3.4 millivolts, a value in close agreement with Bristow’s finding of 3.29 millivolts. In the total left ventricular hypertrophy group, which included patients with associated right ventricular hypertrophy, this value was slightly lower, 2.1 millivolts, but still significantly different from the normal measurement of 1.58 millivolts ($p < 0.001$).

It appears, therefore, that although an increase in over-all left ventricular mass consequent to hypertrophy, and a decrease in the anterior ventricular mass resulting from infarction, exert different influences upon the direction of the initial horizontal plane QRS forces, this difference is much less apparent at the time of inscription of the maximum QRS vector. At this moment, however, a difference in its effect on the magnitude becomes most evident. The configuration of the remainder of the loop and the direction of the 0.04-second vector as well as later vectors remain quite similar in both groups, indicating that no significant differences develop subsequently.

Comments

It follows from these observations, that the direction of the 0.02-second vector is the most useful measurement in separating left ven-
tricular hypertrophy from anterior wall infarction, with or without left ventricular hypertrophy (figs. 5 and 7). In no instance were forces directed anteriorly to the 180°-360° axis in the infarction group, whereas all patients with left ventricular hypertrophy did have such anterior forces. In turn, the direction of the 0.03-second vector separated left ventricular hypertrophy from the normal and from the anterior wall infarction group. Finally, the direction of the maximum QRS vector allows the separation of either left ventricular hypertrophy or anterior wall infarction from the normal but not from one another. But this differentiation can be made with confidence by measurement of the magnitude of this vector.

The longer duration of anteriorly directed forces in the vectorcardiograms of the nine patients who showed diminutive r waves in leads V1 to V3 of the electrocardiogram demands explanation. It is possible that the exact measurement of the duration of the r wave in the electrocardiograms may be subject to errors introduced by the lower frequency response of the direct-writing electrocardiograph. By contrast, it is the opinion of Sodi-Pallares¹⁷ that the use of the semi-direct precordial lead offers a much more accurate assessment of localized infarction than the remote leads used in most orthogonal vectorcardiographic systems. Bryant and Kossmann¹⁸ have discussed the role of incomplete left bundle-branch block in the reduction of the initial r waves recorded over the right precordium. Wallace and co-workers¹⁹ have recently published their findings in left ventricular hypertrophy studied by the Frank vectorcardiogram and believe that changes in the spatial orientation of initial forces must be held responsible. Although such changes certainly have been found in our cases, it must be emphasized that in three patients who had pure QS complexes in their electrocardiograms anteriorly directed forces were found by the vectorcardiogram (fig. 4). Furthermore, there were no patients in whom the electrocardiogram indicated infarction but the vectorcardiogram did not, in whom infarct was demonstrated at autopsy. These findings suggest that in certain instances the reference system itself must be responsible for the discrepancies observed. It has been suggested on theoretical grounds that marked differences between complexes recorded by the standard electrocardiographic reference system and those obtained by the corrected lead systems are to be expected.²⁰ Vectorcardiographic studies of large series of patients with myocardial infarction, some of whom were studied at autopsy, have borne out this contention.⁶,¹⁴

Observations on other planar projections of the spatial vectorcardiographic loop in these patients did not show differences of similar clarity, although the direction of rotation of the QRS loop in the left sagittal plane projection has always been in a counterclockwise direction in left ventricular hypertrophy uncomplicated by infarction, whereas anterior wall infarction, and inferior infarction, often change this rotation into a clockwise direction. In this respect the direction of rotation in the sagittal plane also remains a useful screening criterion.

Summary

Horizontal plane projections of vectorcardiograms registered by the Frank lead system were analyzed in 36 patients with left ventricular hypertrophy and in 24 with left ventricular hypertrophy and associated anterior wall infarction. All were studied at autopsy. The 0.01-, 0.02-, 0.03-second, and maximum QRS vectors were compared with similar measurements in 60 control subjects previously reported. New criteria for the vectorcardiographic diagnosis of left ventricular hypertrophy have been outlined. Close agreement with earlier reported groups of patients with anteroseptal and anterolateral infarction was found, and findings by others in patients with left ventricular hypertrophy were confirmed.

The direction of the 0.02-second QRS vector allowed the separation of the infarct group from the left ventricular hypertrophy group and from the normal, whereas the direction of the 0.03-second and maximum QRS vectors
separated left ventricular hypertrophy from the normal. The magnitude of the maximum QRS vector is another highly reliable indicator of left ventricular hypertrophy and also serves to separate this group from those with associated anterior wall infarct. The influence of associated right ventricular hypertrophy or posterobasal infarction on left ventricular hypertrophy has been discussed.

The accuracy of this technic of vectorcardiographic differentiation between left ventricular hypertrophy on the one hand and left ventricular hypertrophy with anterior infarction on the other in every instance was confirmed at autopsy. In all 12 patients whose electrocardiograms presented the dilemma of Q5 or rS complexes in leads V1 to V3 of the standard precordial electrocardiogram, the vectorcardiographic diagnosis was confirmed at autopsy. The Frank vectorcardiogram is a sensitive and reliable method to recognize left ventricular hypertrophy and may prove to be a useful adjunct to the ordinary electrocardiogram in cases where the electrocardiographic diagnosis is questionable.

Acknowledgment

We are indebted to Dr. Felix L. Rodriguez of the Mallory Institute of Pathology and Harvard Medical School for allowing us the use of his detailed pathologic studies and coronary injection data in many of these patients who constitute part of another study.

References

Recognition of Anterior Wall Infarction in Patients with Left Ventricular Hypertrophy: A Study by the Frank Vectorcardiogram
PAUL G. HUGENHOLTZ, THOMAS J. RYAN, THOMAS WOERNER and HAROLD D. LEVINE

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