Secular Trends in Q Wave and Non–Q Wave Acute Myocardial Infarction

The Minnesota Heart Survey

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The Minnesota Heart Survey examined trends of Q wave and non–Q wave acute myocardial infarction (AMI) using a 50% random sample of all hospital discharges of patients with AMI or another acute coronary disease from 35 of 36 hospitals in 1970 and 30 of 31 hospitals in 1980 in the Minneapolis–St. Paul metropolitan area. A total of 1,901 and 1,864 potential AMI cases were abstracted in 1970 and 1980, respectively. Electrocardiograms were coded according to the Minnesota code. AMIs were validated by computerized algorithm based on chest pain, enzymes, electrocardiograms, and autopsy. This study shows that with the use of a consistent, standard diagnostic algorithm, attack rates for Q wave AMI did not change significantly between 1970 and 1980 and that attack rates for non–Q wave AMI decreased significantly during the same decade. However, when the more sensitive cardiac enzymes creatine phosphokinase and creatine phosphokinase–MB were considered, attack rates of both Q wave and non–Q wave AMIs increased. This research documents four important trends for community AMI rates that are at variance with those reported by others. There was a decline in non–Q wave AMI attack rates from 1970 to 1980; women had outcomes equal to or worse than those for men for both case–fatality and 7-year survival rates; patients with non–Q wave AMIs had worse in-hospital prognoses than those with Q wave AMIs; and 7-year survival rates were worse for Q wave AMI in 1980. These findings demonstrate the need for standard diagnostic criteria for Q wave and non–Q wave AMI if trends are to be monitored. In the future, as new trials of operative and nonoperative therapies of AMI are undertaken, these considerations will increase in importance. (Circulation 1991;83:492–503)

Coronary heart disease (CHD) mortality rates have decreased 41% in the past 20 years, but acute CHD admission rates have not decreased similarly. The Minnesota Heart Survey (MHS) found that age-adjusted attack rates for definite myocardial infarction (MI) were similar in 1970 and 1980 (174.2 versus 179.9 per 100,000, respectively).1 The Worcester Heart Attack Study reported increased rates of initial and recurrent acute myocardial infarctions (AMIs) between 1975 and 1981.2,3 The Mayo Clinic study of CHD in residents of Rochester, Minn., reported decreased incidence rates of initial MI for men but increased rates for women between 1950 and 1982.4,5 All three of these community surveillance programs reported decreases in case–fatality rates over time and suggested that the observed decline in CHD mortality rates may reflect decreases in out-of-hospital coronary deaths or improved in-hospital case–fatality rates.2,4,6

The reported trends in AMI attack rates are not uniform for Q wave and non–Q wave AMIs. Some investigators2,4 observed an increase in AMI rates primarily in non–Q wave AMIs and particularly among persons 65 years of age and older. They suggest that the increase in non–Q wave AMI incidence rates and the decrease in case–fatality rates may be due to the expanded use of serum creatine phosphokinase (CPK) and CPK-MB between 1975 and 1981 and the subsequent detection of “milder” infarctions. They also hypothesized that these temporal trends might reflect changes in the natural history of AMI, with a higher proportion of milder (non–Q wave) cases presenting to the hospital. Additionally, improvements in medical care and patient education may have resulted in earlier or more effective interventions.

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The MHS was initiated in 1979 in the seven-county Twin Cities metropolitan area to investigate the causes of declining cardiovascular disease mortality rates by monitoring trends in community CHD morbidity, mortality, and risk factors. Trends of in-hospital attack and case-fatality rates for patients with AMI and stroke have been reported for 1970 and 1980.1,6 MHS findings are used to investigate trends in Q wave and non-Q wave AMI attack, case-fatality, and long-term survival rates in 1970 and 1980.

Methods

The MHS has been described in detail by Gillum et al.7,8 The population of the Twin Cities metropolitan area was 1,874,380 in 1970 and 1,985,873 in 1980. The Twin Cities population 30–74 years old was 744,022 in 1970 and 866,814 in 1980. Records of patients with discharge diagnoses of AMI were obtained from 35 of 36 Twin Cities hospitals in 1970 and 30 of 31 hospitals in 1980. One 194-bed private hospital with four coronary care unit beds and four additional telemetry beds declined to participate in the survey. Participating hospitals provided the study with discharge reports coded according to the International Classification of Diseases (the eighth revision was used in 1970 and the ninth was used in 1980). A 50% random sample of discharge diagnoses coded 410 (AMI) or 411 (other acute coronary diseases) was selected and abstracted from seven-county Twin Cities residents 30–74 years old. The total number of potential AMI cases abstracted was 1,901 in 1970 and 1,864 in 1980.

Hospital records of potential AMIs were abstracted by trained nurse-abstractors who collected demographic information as well as data on therapeutic and diagnostic procedures.7,8 Information was collected on history of chest pain, electrocardiograms (ECGs), enzyme levels [CPK and its isoenzyme CPK-MB, serum lactate dehydrogenase (LDH), and serum aspartate aminotransferase (AST)], and autopsy records.

Chest Pain

History of chest pain was defined as the presence or absence of typical cardiac chest pain in the hospital record. When the notes of different health-care providers were inconsistent determinators of presence or absence of chest pain, a hierarchical rule was applied; the hierarchy was attending physician, resident, intern, and then nurse.

Enzymes

Although all four enzyme levels were abstracted from the hospital record, only AST and LDH were used for the standard algorithm. Levels of AST and LDH were considered in the diagnostic algorithm as follows. For AST or LDH, a peak value exceeding twice the upper limit of the laboratory normal was considered abnormal unless the patient's record showed active liver disease, trauma within 1 week of admission, or surgery. If either of the two enzymes were abnormal, the individual was considered to have abnormal enzymes.

To further examine trends in AMI rates, we also calculated attack rates using CPK and CPK-MB as well as AST and LDH. CPK-MB was coded as present or absent by the laboratories or considered present if the value was above the upper limit of normal. Presence of CPK-MB, regardless of liver disease, trauma, or surgery, was considered abnormal. In these analyses, if any of the four enzymes was abnormal, the individual was considered to have abnormal enzymes.

Electrocardiogram Coding

All ECGs up to a maximum of 12 were copied and sent to the Minnesota coding center for visual Minnesota reading.11 When more than 12 recordings were available, the first six and the last six by date and time were used. The Minnesota code Q wave and non-Q wave classifications for ECG patterns are presented in the “Appendix.”

Q code criteria. Criteria for a Q wave pattern requires either the appearance of new diagnostic Minnesota Q codes or change from a minor Q code to a diagnostic Minnesota Q code accompanied by new T wave inversion, new ST elevation, or new ST depression. Q code and ST-T changes must occur in the same lead group (see “Appendix”). Among the ECG patterns shown in the “Appendix,” only evolving diagnostic was considered sufficient to document AMI by itself. A diagnostic Q code on the baseline record in any lead group precluded this latter diagnosis.

Non-Q code criteria. The “Appendix” also displays the criteria used for non-Q wave AMI. This determination was made when there were no measurable Q waves. All non-Q wave AMI patterns required confirmation by abnormal peak enzyme levels, chest pain, or autopsy.

Diagnostic Algorithm

Figure 1 presents the diagnostic algorithm for definite Q wave and non-Q wave AMI. The four major elements used to validate an AMI were 1) the presence or absence of acute chest pain, 2) peak levels of cardiac enzymes, 3) ECG patterns compatible with an AMI, and 4) the presence of an AMI by necropsy (AMI documented within 8 weeks of death). As previously stated, an evolving diagnostic Q code was always considered evidence of a Q wave AMI. The diagnostic outcomes of this algorithm were definite Q wave AMI, definite non-Q wave AMI, definite AMI not classifiable, and no AMI. Typical cardiac pain was not required to validate a Q wave AMI but was necessary to validate non-Q wave AMI when autopsy findings of AMI were not present.

Results

The availability of diagnostic information in 1970 and 1980 is shown in Table 1. No significant difference was noted in the use of AST between 1970 and 1980. However, usable ECGs and levels of LDH, CPK, and CPK-MB, all measures of cardiac ischemia, were significantly more often present in the
hospital charts in 1980. Autopsy rates for in-hospital deaths decreased by 28% (from 61% to 44%) during this decade.

We abstracted 1,901 records in 1970 and 1,864 records in 1980. Of these, 36.3% (891) were validated by our criteria as definite AMIs in 1970 and 45.5% (848) were validated in 1980. Table 2 shows the frequency of records validated in each diagnostic category by year and sex.

Acute Myocardial Infarction Rates

Age-adjusted attack rates for patients 30–74 years old for definite Q wave and non–Q wave AMIs are shown in Table 3. Age-adjusted rates for Q wave AMI (ages 30–74 years) were similar in 1970 and 1980. This finding held for men and women, except for a significant decrease in attack rates for women in the 30–64-year-old group (p<0.05). Attack rates for non–Q wave AMIs decreased significantly between 1970 and 1980 (p<0.001). This finding was true for men (p<0.001) and women (p<0.01) and at ages 30–64 (p<0.001) and 65–74 years (p<0.001).

In-Hospital Case-Fatality Rates

Table 4 displays overall age- and gender-specific case-fatality rates for 1970 and 1980. Overall, Q wave AMI case-fatality rates were significantly lower for 1980 than for 1970 (p<0.05) and were lower for men than for women (p<0.05). There was a smaller, statistically nonsignificant decrease in female Q wave case-fatality rates. The pattern of decreasing case fatality in 1980 was also observed for non–Q wave AMI case-fatality rates. The decrease in male rates was larger than the decrease in female rates. However, because of the smaller number of non–Q wave AMIs, the trends in non–Q wave case-fatality rates were not statistically significant.

Age-adjusted case-fatality rates for patients 30–74 years old were higher for women than for men for both Q wave and non–Q wave AMI in 1970 and for non–Q wave AMI in 1980. The female/male difference was statistically significant only for Q wave AMI in 1980 (14.5% versus 7.8%, p<0.05). Female/male differences persisted at ages 30–64 and 65–74 years.

Age-adjusted case-fatality rates for patients 30–74 years old were higher for non–Q wave AMI than for Q wave AMI in 1970 and 1980. The overall difference was statistically significant in 1980 (p<0.05) but not in 1970 (p<0.08). For men, non–Q wave case-fatality rates were significantly higher than Q wave rates for both years (p<0.05). For women, non–Q wave case-fatality rates were higher in both years, but the differences were not statistically significant.

**FIGURE 1. Minnesota Heart Survey Q wave and non–Q wave acute myocardial infarction diagnostic algorithm.**
Table 1. Availability of Variables for Validating Myocardial Infarction in 1970 and 1980: Minnesota Heart Survey

<table>
<thead>
<tr>
<th>Variable</th>
<th>1970 (n=901)</th>
<th></th>
<th>1980 (n=1,864)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>%</td>
<td>Values per case* (n)</td>
<td>%</td>
<td>Values per case* (n)</td>
</tr>
<tr>
<td>Cardiac enzymes</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AST</td>
<td>85.8</td>
<td>3.5</td>
<td>91.4</td>
<td>3.0</td>
</tr>
<tr>
<td></td>
<td>(84.2, 87.4)</td>
<td></td>
<td>(90.1, 92.7)</td>
<td></td>
</tr>
<tr>
<td>LDH</td>
<td>67.2</td>
<td>2.6</td>
<td>92.3</td>
<td>2.8</td>
</tr>
<tr>
<td></td>
<td>(65.1, 69.3)</td>
<td></td>
<td>(91.1, 92.7)</td>
<td></td>
</tr>
<tr>
<td>CPK</td>
<td>45.1</td>
<td>3.0</td>
<td>88.4</td>
<td>3.3</td>
</tr>
<tr>
<td></td>
<td>(42.9, 47.3)</td>
<td></td>
<td>(86.9, 89.9)</td>
<td></td>
</tr>
<tr>
<td>CPK-MB</td>
<td>0.0</td>
<td>0.0</td>
<td>75.2</td>
<td>2.7</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(73.2, 77.2)</td>
<td></td>
</tr>
<tr>
<td>12-lead ECG</td>
<td>94.1</td>
<td>4.8</td>
<td>97.5</td>
<td>4.8</td>
</tr>
<tr>
<td></td>
<td>(93.0, 95.2)</td>
<td></td>
<td>(96.8, 98.2)</td>
<td></td>
</tr>
<tr>
<td>Autopsy†</td>
<td>61.0</td>
<td>...</td>
<td>44.0</td>
<td>...</td>
</tr>
<tr>
<td></td>
<td>(59.2, 63.2)</td>
<td></td>
<td>(41.7, 46.3)</td>
<td></td>
</tr>
</tbody>
</table>

AST, serum aspartate transaminase; LDH, lactate dehydrogenase; CPK, creatine phosphokinase; ECG, electrocardiogram. Numbers in parentheses indicate 95% confidence intervals. Values are age-adjusted. Percentages exclude those with missing levels, which amount to 1% for AST, LDH, and CPK, and 0% for CPK-MB, 12-lead ECG, and autopsy. Percentages of those with missing values of CPK-MB, 12-lead ECG, and autopsy are 0%. Percentages of those with missing values for CPK-MB, 12-lead ECG, and autopsy are 0%.

Case–Fatality Rates by Serum Aspartate Aminotransferase Levels

Table 5 gives age-adjusted case–fatality rates by level of serum AST and by type of AMI for patients hospitalized 2 or more days and with AST levels equal to or more than twice the upper limit of normal. The population was stratified by level of AST (levels twofold to fourfold that of the upper limit of normal or levels fivefold or more than that of the upper limit of normal) to compare case–fatality rates for patients with similar degrees of cardiac damage. Patients discharged within 2 days were excluded from this analysis because of uncertainty in peak enzyme levels and missing values among short-stay individuals.

Overall, case–fatality rates for Q wave and non-Q wave AMI were positively associated with level of serum AST in both 1970 and 1980. The 1970 non-Q wave AMI case–fatality rates for both sexes with AST levels at least fivefold that of the upper limit of normal were significantly higher compared with those with AST levels from twofold to fourfold of the upper limit of normal (p<0.05). When case–fatality rates by type of AMI were compared within levels of AST, there were no significant differences. The highest case–fatality rates for both men and women occurred in those with non-Q wave AMIs in 1970 with AST levels at least fivefold that of the upper limit of normal.

Predictors of In-Hospital Case Fatality

Multiple logistic regression analysis was used to identify predictors of in-hospital case fatality among patients whose AMIs were diagnosed by other data than autopsy alone. This analysis identified pulmonary edema as the only consistent significant predictor of in-hospital case fatality for Q wave and non-Q wave AMI in 1970 and 1980. Age was a significant

Table 2. Distribution of Definite Acute Myocardial Infarction by Type of Infarct: Minnesota Heart Survey, 1970 and 1980

<table>
<thead>
<tr>
<th>AMI classification</th>
<th>1970</th>
<th>1980</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>%</td>
<td>n</td>
</tr>
<tr>
<td>Men</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-Q wave AMI</td>
<td>19.9</td>
<td>112</td>
</tr>
<tr>
<td>Q wave AMI</td>
<td>67.0</td>
<td>377</td>
</tr>
<tr>
<td>Nonclassifiable AMI</td>
<td>13.1</td>
<td>74</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>563</td>
</tr>
<tr>
<td>Women</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-Q wave AMI</td>
<td>25.9</td>
<td>59</td>
</tr>
<tr>
<td>Q wave AMI</td>
<td>59.7</td>
<td>136</td>
</tr>
<tr>
<td>Nonclassifiable AMI</td>
<td>14.5</td>
<td>33</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>228</td>
</tr>
</tbody>
</table>

AMI, acute myocardial infarction.
predictor for Q wave AMI in 1970 and 1980 and for non-Q wave AMI in 1980. Other variables (i.e., sex, recorded pulse rate, AST level, systolic blood pressure, previous MI history, presence of an S₃, and admission to the cardiac care unit) were inconsistent predictors. Among the latter, only pulse rate and AST level were predictors more than once.

**Long-term Survival**

Figures 2 and 3 display 7-year survival curves by year, sex, and type of AMI. The survival rate of men and women combined was significantly lower among Q wave AMI patients for 1980 (p<0.05, using the log rank test). Seven-year survival rates for women were significantly worse for Q wave than for non-Q wave AMI in 1980 (p<0.05).

**Effect on Case–Fatality Rates of Excluding Autopsy-Identified Acute Myocardial Infarctions**

We examined the possibility that the use of autopsy information may have differentially affected one type of AMI more than the other. In 1970 and 1980, significantly more non-Q wave AMIs than Q wave AMIs would have been excluded if autopsy findings had not been considered (in 1970, 10.5% of non-Q wave versus 4.3% of Q wave AMIs, p<0.01; in 1980, 8.0% versus 2.6%, p<0.01). Exclusion of autopsy-identified AMIs significantly decreased non-Q wave AMI case–fatality rates from 19.6% to 10.1% (48.5% reduction, p<0.05) in 1970 and from 15.2% to 8.5% (44.1% reduction, p<0.05) in 1980, whereas autopsy exclusion nonsignificantly reduced Q wave case–fatality rates from 13.8% to 9.9% (28.3% reduction, p=NS) in 1970 and from 7.3% to 7.3% (24.7% reduction, p=NS) in 1980. Furthermore, when autopsy findings were excluded from the diagnostic criterion and Q wave and non-Q wave AMI case–fatality rates among individuals with elevated serum enzymes were compared, the rates were equivalent: 10.1% for non-Q wave and 11.4% for Q wave AMIs in 1970 and 7.4% for non-Q wave and 7.9% for Q wave AMIs in 1980. Seven-year mortality rates re-

**Table 3. Age-Adjusted Acute Myocardial Infarction Attack Rates per 100,000 for 1970 and 1980 in Twin Cities Residents 30–74 Years Old: Minnesota Heart Survey**

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Q wave acute myocardial infarction</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30–64</td>
<td>180.9</td>
<td>178.7</td>
<td>35.8</td>
<td>29.8*</td>
<td>105.3</td>
<td>102.3</td>
</tr>
<tr>
<td>65–74</td>
<td>555.9</td>
<td>567.4</td>
<td>277.1</td>
<td>281.8</td>
<td>390.5</td>
<td>403.2</td>
</tr>
<tr>
<td>Total</td>
<td>229.7</td>
<td>229.3</td>
<td>67.1</td>
<td>62.6</td>
<td>142.4</td>
<td>141.4</td>
</tr>
<tr>
<td>Non-Q wave acute myocardial infarction</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30–64</td>
<td>54.2</td>
<td>47.6*</td>
<td>18.1</td>
<td>14.9*</td>
<td>35.4</td>
<td>30.8‡</td>
</tr>
<tr>
<td>65–74</td>
<td>162.6</td>
<td>127.0‡</td>
<td>104.4</td>
<td>84.5*</td>
<td>128.0</td>
<td>102.6‡</td>
</tr>
<tr>
<td>Total</td>
<td>68.3</td>
<td>57.9‡</td>
<td>29.3</td>
<td>23.9‡</td>
<td>47.5</td>
<td>40.1‡</td>
</tr>
</tbody>
</table>

Rates are age adjusted to 1970 Twin Cities population 30–74 years of age. Definite acute myocardial infarctions determined using pain, electrocardiogram, autopsy, and enzymes.

* p<0.05, ‡p<0.01, §p<0.001.

**Table 4. Age-Adjusted In-Hospital Acute Myocardial Infarction Case–Fatality Rates by Type of Infarction for 1970 and 1980 in Twin Cities Residents 30–74 Years Old: Minnesota Heart Survey**

<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>Q wave acute myocardial infarction</th>
<th>Non-Q wave acute myocardial infarction</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>CFR</td>
<td>n</td>
</tr>
<tr>
<td>Men</td>
<td></td>
<td></td>
</tr>
<tr>
<td>30–64</td>
<td>271</td>
<td>7.2</td>
</tr>
<tr>
<td>65–74</td>
<td>106</td>
<td>21.7</td>
</tr>
<tr>
<td>Total</td>
<td>377</td>
<td>12.7</td>
</tr>
<tr>
<td>Women</td>
<td></td>
<td></td>
</tr>
<tr>
<td>30–64</td>
<td>59</td>
<td>8.2</td>
</tr>
<tr>
<td>65–74</td>
<td>77</td>
<td>28.6</td>
</tr>
<tr>
<td>Total</td>
<td>136</td>
<td>15.9</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td></td>
</tr>
<tr>
<td>30–64</td>
<td>321</td>
<td>7.2</td>
</tr>
<tr>
<td>65–74</td>
<td>183</td>
<td>24.6</td>
</tr>
<tr>
<td>Total</td>
<td>513</td>
<td>13.8</td>
</tr>
</tbody>
</table>

Rates are age adjusted to 1970 distribution of hospital discharges of patients 30–74 years old with myocardial infarctions.

* p<0.05.

CFR, case–fatality rate.
mained higher for Q wave than for non-Q wave AMI. Female case-fatality rates remained higher than male rates for Q wave AMI in 1970 and 1980 and for non-Q wave AMI in 1980.

**Discussion**

This study shows that using a consistent standardized diagnostic algorithm, attack rates for Q wave AMI did not change significantly between 1970 and 1980 and that attack rates for non-Q wave AMI decreased significantly during the same decade. Initially, MHS findings appear to be contrary to trend data from the Worcester and Olmsted County cardiovascular surveillance studies. These studies, using all available enzyme data for AMI diagnosis, found sharp increases in the incidence of non-Q wave AMI between the mid-1970s and 1980. These and other investigators suggested that the sharp increase in the incidence of non-Q wave AMI between 1975 and 1981 may be due to increased use of serum CPK-MB isoenzyme subfractions to diagnose AMI.

We investigated the impact of considering CPK and CPK-MB in our diagnostic algorithm. Inclusion of CPK and CPK-MB in the diagnostic algorithm had only minimal impact on 1970 AMI attack rates. In 1980, both Q wave and non-Q wave AMI attack rates were significantly higher when CPK and CPK-MB were considered; Q wave AMI rates increased 16.8% ($p<0.01$), and non-Q wave AMI rates increased 94.0% ($p<0.001$). Figure 4 clearly illustrates that inclusion of the more sensitive enzymes that were available in 1980 had a larger impact on secular trends of non-Q wave AMI attack rates than on Q wave rates.

These data provide an example of stage migration, or the “Will Rogers phenomenon” described by Feinstein et al. who cautioned against the possibility of reaching fallacious conclusions about trends when diagnostic tests improve over time. This admonition clearly applies to interpretation of trends of Q wave and non-Q wave AMI rates based on changing diagnostic enzyme information. We believe that sec-

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**Table 5. Age-Adjusted In-Hospital Case-Fatality Rates by AST Level for Acute Myocardial Infarctions With Length of Stay of More Than 2 Days for 1970 and 1980: Minnesota Heart Survey**

<table>
<thead>
<tr>
<th>AST (upper limit of normal)</th>
<th>Q wave acute myocardial infarction</th>
<th>Non-Q wave acute myocardial infarction</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Male</td>
<td>Female</td>
</tr>
<tr>
<td>1970</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2–4</td>
<td>9.5</td>
<td>150</td>
</tr>
<tr>
<td>≥5</td>
<td>17.1</td>
<td>137</td>
</tr>
<tr>
<td>1980</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2–4</td>
<td>4.0</td>
<td>157</td>
</tr>
<tr>
<td>≥5</td>
<td>9.1</td>
<td>185</td>
</tr>
</tbody>
</table>

AST, serum aspartate aminotransferase.
FIGURE 3. Plot of survival after acute myocardial infarction. From the Minnesota Heart Survey, 1980. Diagnostic criteria for definite acute myocardial infarction are based on electrocardiogram, pain, autopsy, serum aspartate aminotransferase, and serum lactate dehydrogenase.


...ular trends cannot be properly assessed unless consistent diagnostic criteria for AMI are applied.

Case Fatality

In the present study, the non-Q wave AMI inhospital case-fatality rates were uniformly higher than those for Q wave AMI, although the differences were statistically significant only in 1970. Several studies have identified subgroups with non-Q wave AMI case-fatality rates higher than those for Q wave AMI. Scheinman and Abbott\textsuperscript{15} reported higher in-hospital non-Q wave AMI than Q wave AMI case-fatality rates for patients with elevated enzymes; Rigo et al\textsuperscript{16} found significantly higher rates for non-Q wave infarcts with abnormal QRS complexes (i.e., conduction defects not diagnostic of AMI); and Maisel et al\textsuperscript{17} reported higher non-Q wave AMI case-fatality rates for patients with infarct extension during hospitalization. However, most studies reported lower case-fatality rates for non-Q wave...
In-hospital case-fatality rates were statistically lower for non-Q wave AMI in half of the above studies ($n = 16$).

The purpose of the MHS AMI diagnostic algorithm was to document in a standardized manner all acute AMI cases that occurred in the Twin Cities surveillance area. Because of this focus on community surveillance, we investigated four possible explanations for the higher non-Q wave AMI case-fatality rates observed in the present study. 1) Milder Q wave AMIs, based on evolving Q waves without enzyme elevations, may have been captured. 2) The mildest non-Q wave AMIs, those with ST-T wave changes and normal enzymes, may have been removed. 3) The Minnesota code criteria may have excluded small Q waves (less than 1 mm in depth), and thus some AMIs that clinically would have been classified as Q wave may have been classified as non-Q wave. 4) The use of autopsy as prima facie evidence of definite AMI may have differentially affected one type of AMI (Q wave or non-Q wave AMI).

First, we considered the possibility that a group of milder Q wave AMIs were included. Eleven of the 17 studies that calculated in-hospital fatality rates and published diagnostic criteria required elevated enzymes for a diagnosis of definite Q wave AMI. We examined the possibility that the MHS algorithm captured milder Q wave AMIs that were based on evolving Q wave codes not supported by enzyme elevation. Because enzyme elevations are considered more sensitive indicators of the extent of MI damage, those cases might be milder. We investigated this potential by removing from the analyses Q wave AMI documented by ECG pattern change only (no autopsy evidence of recent AMI or elevated enzymes). When these cases were removed, Q wave AMI case-fatality rates decreased nonsignificantly from 13.8% to 12.3% in 1970 and from 9.7% to 8.5% in 1980. Exclusion of these cases did not significantly affect the comparisons of Q wave and non-Q wave AMI case-fatality rates reported in the present study. Thus, bias in the algorithm toward selecting less severe Q wave AMIs by not requiring enzyme elevation in all cases does not appear to explain the observed differences in case-fatality rates.

The possibility that the standardized algorithm excluded the mildest non-Q wave AMI cases, that is, cases with ST-T wave changes and normal enzymes, was considered next. This prospect was examined because the majority of studies also required enzyme elevations for diagnosis of non-Q wave infarction (14 of 18 reviewed). For 1970 and 1980 combined, only 40 individuals were in this category. However, adding these individuals to the non-Q wave AMI group did not significantly change the case-fatality rates.

Third, we considered whether the Minnesota code classification of ECGs excluded small Q wave changes thought to be clinically important and subsequently misclassified some Q wave AMIs as non-Q wave AMIs. A cardiologist (R.C.) read each ECG identified by Minnesota code classification as non-Q wave AMI to determine whether clinical evaluation produced a different classification. Considering both years together, inclusion of the clinical reading resulted in reclassifying 13.3% of non-Q wave AMIs to Q wave AMIs. Case-fatality and 7-year survival rates were minimally affected by this reclassification, and none of the relations reported here was changed. In 1981, Marmor et al.32,33 applied a diagnostic algorithm based on the Minnesota code to a sample group of 200 AMIs. Their definition of non-Q wave AMI was nearly identical to ours. They found case-fatality rates for Q wave AMIs higher than for non-Q wave AMIs. Bayley et al.25 also used Minnesota code classification to diagnose survival and observed higher in-hospital case-fatality rates in Q wave AMI patients. Those results suggest that the differences in case-fatality rates observed in the present study are not primarily because of the sensitivity and specificity of AMI definition using Minnesota coded ECGs.

We also examined the likelihood that the MHS diagnostic algorithm's use of autopsy information may have differentially affected one type of AMI more than the other. In 1970 and 1980, significantly more non-Q wave AMIs than Q wave AMIs would have been excluded if autopsy findings were not considered. Exclusion of autopsy-identified AMIs significantly reduced non-Q wave AMI case-fatality rates and nonsignificantly reduced Q wave case-fatality rates.

One possible explanation for the large effect of autopsy on non-Q wave AMI case-fatality is that the MHS algorithm differentially mislabeled early Q wave cases that did not have time to evolve. When a definite AMI was found by autopsy, we documented non-Q wave AMI if there were at least two ECGs and no measurable Q wave on any ECG. Thus, an autopsy-confirmed recent AMI could be classified as a non-Q wave AMI when only one ECG in the hospital chart showed a nonspecific ST-T wave pattern. Conversely, if a single ECG showed a Q wave, the AMI could be classified as Q wave. In patients dying early, it is obvious that either Q wave or non-Q wave patterns could have evolved if death had not intervened. In the Beta-Blocker Heart Attack Trial,34 although the presence of abnormal Q waves was used to define the type of AMI, an ECG taken 5–21 days after the acute event showed that 17.4% of patients with Q wave AMIs lost their Q waves, whereas 32% of patients with non-Q wave AMIs developed significant Q waves during the interval. It would seem that individuals who died rapidly could have eventually evolved in either direction but that classification of type of AMI based on available information is reasonable.

We conclude that MHS's attempt to capture all definite AMIs using standardized diagnostic criteria has included a subgroup of high-risk non-Q wave AMIs that are generally not considered. With the exception of the Worcester studies of Goldberg et al.2,3 and the 1966 Israeli studies of Schor et al.35 most studies of Q wave and non-Q wave AMI survival
exclude patients who do not live long enough in the hospital to exhibit enzyme elevations and/or evolving ECG patterns. Goldberg et al found higher in-hospital and Schor et al found higher 21-day (after admission) mortality rates for Q wave AMI. Neither study specified how autopsy-identified AMIs were classified when only minimal clinical data were available. We believe that because the purpose of the present study was to monitor secular trends for the entire community, it is appropriate to include events identified primarily through autopsy.

Inclusion of Recurrent Events

Our case series includes both first and recurrent AMIs. Non–Q wave AMIs are more often associated with a history of previous AMI than are Q wave AMIs, and case–fatality rates for recurrent non–Q wave AMIs have been shown to exceed rates for incident Q wave AMIs. Because of the nature of hospital records, accurate data on the mixture of first and recurrent events were not available for the present study. Logistic regression analysis did not identify history of prior MI as a predictor of in-hospital case fatality. However, we cannot exclude the possibility that our findings of higher non–Q wave case–fatality rates are because of the case mix of patients with first and recurrent AMIs hospitalized in the Twin Cities in 1970 and 1980 and may limit the ability to generalize these findings to studies with different case mixes.

It is generally thought that non–Q wave AMI has a prognostic advantage in-hospital but that over time this advantage is lost. A number of investigators have suggested that this is because patients with non–Q wave AMIs have “incomplete infarctions” (i.e., they have more residual viable but unstable myocardium within the perfusion zone of the infarct-related vessel than do patients with Q wave AMIs). This is supported by the observation that reinfarction, postinfarction angina, and the need for coronary bypass surgery appear to be higher in patients with non–Q wave AMIs.

Seven-Year Survival

There appears to be general consensus that long-term survival is comparable for patients with Q wave and non–Q wave AMIs. Of 18 studies reviewed, three studies reported significantly worse long-term survival rates for patients with non–Q wave AMIs, and three reported significantly worse long-term survival rates for patients with Q wave AMIs. Bayley et al reported worse survival rates at 1 year in non–Q wave patients with a history of previous infarction; Cannom et al reported worse non–Q wave survival rates at 36 months in persons discharged alive from the hospital; and Mahony et al reported worse 30-month survival rates for non–Q wave AMI patients with abnormal QRS complexes. Strauss et al found 1-year survival, Pohjola et al found 5-year survival, and DeWood et al found 8-year survival rates significantly worse for patients with Q wave AMIs. The remaining studies reported no significant differences in long-term survival.

In the present study, 7-year survival rates were significantly worse in 1980 for men and women combined and for women with Q wave AMIs. A Cox regression analysis was performed to investigate predictors of long-term survival for persons alive 30 days after hospital discharge. Ten variables were assessed (age, sex, length of stay, level of AST, systolic blood pressure, heart rate, history of previous MI, pulmonary edema, presence of an S3, and cardiac care unit stay). Age, heart rate, pulmonary edema by radiography, and admission to a cardiac care unit during hospitalization were consistent predictors of long-term survival for 1970 and 1980. Type of AMI was a significant predictor in 1980.

Study Limitations

The present study is based on hospital record abstraction and did not include direct surveys of health-care providers, patients, or patients’ families. Thus, it was not possible to verify symptoms or history of prior AMI. In the Twin Cities, patients do not necessarily return to the same hospital for care after an initial cardiac event. Because of these limitations, we were unable to differentiate between initial and recurrent MI events. Because of the reported higher prevalence of prior MI among non–Q wave patients, it is possible that our non–Q wave MI patients had a greater proportion of prior MIs than did those with Q wave MIs. We recognize that there also may be unexamined variables that contributed to the observed differences between Q wave and non–Q wave AMI mortality (i.e., cardiogenic shock, ventricular ectopy, location of Q wave MI, hypotension, heart block, and different therapeutic strategies for Q wave and non–Q wave AMI). Finally, some caution is warranted whenever enzyme levels are analyzed. The use of peak enzyme as a measure of equivalency of myocardial damage has limitations in that peak enzyme may not actually represent the true peak for that individual. The peak level depends on the time relation between MI onset to the time of enzyme collection. This information was not consistently available in the present study. Other measures of equivalency may include coronary anatomy, ejection fraction, and cardiac output. Categorical enzyme levels have been used for this purpose. Goldberg et al examined type of MI by tertile of peak CPK and found that case–fatality rates were lower among patients with non–Q wave MIs at each CPK level examined. We found that case–fatality rates for Q wave and non–Q wave AMI patients dichotomized by two enzyme categories were not statistically different in 1970 and 1980. We were surprised by the high case–fatality rates in 1970 among non–Q wave AMI patients with AST levels at least fivefold that of the upper limit of normal. These high rates may be due to the relatively small number of non–Q wave AMI patients in this category (n = 50).
In summary, any study that uses hospital records faces certain limitations with regard to lack of availability of data that would ideally be collected in a prospective clinical trial and MI registry. We acknowledge the limitations of hospital record data and suggest the optimal strategy to minimize these limitations is to apply standardized approaches to collecting, categorizing, and interpreting the data.

Conclusions

Four important trends for community AMI rates are at variance with those reported by others. There was a decline in non-Q wave AMI attack rates from 1970 to 1980; women had an outcome equal to or worse than that of men for both case-fatality and 7-year survival rates; non-Q wave AMIs had worse in-hospital prognoses than Q wave AMIs; and 7-year survival was worse for patients with Q wave AMIs in 1980.

We believe the reason for our differing results is that the MHS used a standardized and consistent diagnostic algorithm instead of clinical judgment. The MHS diagnostic algorithm also classifies ECGs according to Minnesota code as part of its diagnostic structure. This ECG classification system provides comparability to other studies and minimizes ascertainment bias by eliminating the clinical judgment of referee cardiologists.

Montague et al. pointed out that part of the present controversy about the significance of non-Q wave infarction derives from the clinical “use of ill-defined pathological nomenclature.” Even though there now appears to be a consensus that a non-Q wave infarction is determined by the absence of “infarct-associated Q waves” on the ECG, unless the same criteria are used, different subgroups of non-Q wave AMI patients may be identified. The MHS adopted a case definition for the type of AMI that classified all AMIs, even those with minimal ECG information. Although the MHS used a “wide net” approach to identify events, the application of consistent diagnostic criteria over time resulted in an observed significant decrease in non-Q wave attack rates between 1970 and 1980. Other community surveillance based on all available clinical data has shown increasing non-Q wave attack rates during this decade. Again, these findings clearly demonstrate the need for standard diagnostic criteria for Q wave and non-Q wave AMIs if changes over time are to be monitored. In the future, as new trials of operative and nonoperative therapies in patients with AMIs are undertaken, these conditions will increase in importance.

Appendix

Electrocardiogram Definitions for Q Wave and Non-Q Wave Myocardial Infarctions

Criteria for Q Wave Myocardial Infarction Patterns

Evolving Diagnostic Electrocardiogram

Requires more than one ECG

Definitions Using the Minnesota Code

| Q codes | Diagnostic:1-1-1 through 1-1-7, 1-2-1 through 1-2-5 plus 1-2-7 |
| ST segment elevation code | :9-2 |
| T wave inversion codes | No T wave inversion:5-0 or 5-4 |
| Minor or flat T wave inversion:5-3 |
| Moderate T wave inversion:5-2 |
| Major T wave inversion:5-1 |
| ST depression codes | No ST depression:4-0 or 4-4 |
| Minor ST depression:4-3 |
| Moderate ST depression:4-2 |
| Major ST depression:4-1 |

ECG present with no Q code

Diagnostic Q code in first ECG in a lead group precludes an evolving pattern in that lead group

Later ECG with diagnostic Q code or

ECG present with no Q code and no major or moderate ST depression

Later ECG with equivocal Q code and a major or moderate ST depression or

ECG present with no Q code and no ST segment elevation

Later ECG with an equivocal Q code and an ST segment elevation or

ECG present with no Q code and no major or moderate T wave inversion

Later ECG with an equivocal Q code and a major or moderate T wave inversion or

ECG present with an equivocal Q code and no major or moderate ST depression

Later ECG with diagnostic Q code and a major or moderate ST depression or

ECG present with an equivocal Q code and no ST segment elevation

Later ECG with diagnostic Q code and an ST segment elevation or

ECG present with an equivocal Q code and no major or moderate T wave inversion

Later ECG with diagnostic Q code and a major or moderate T wave inversion

Diagnostic Electrocardiogram

Any ECG with a diagnostic Q code
Equivocal Electrocardiogram
Any ECG with an equivocal Q code

Normal Electrocardiogram
Initial value assigned by program
ECG does not have codes that cause it to be classified into any other category
All leads are codable

Normal Electrocardiogram With Technical Problems
One or more ECGs in a series with uncodable leads; may be questionable
Problem may be across some or all leads (see problem list below)
No Electrocardiograms Remaining After Removing Uncodable Ones
ECGs with problems across all leads are removed from analysis
Reasons for removal include
Complete atrioventricular block
Artificial pacemaker
Asystole or ventricular fibrillation
Technical problems

Criteria for Non–Q Wave Myocardial Infarction Patterns
Absence of codable Q waves using Minnesota code; a codable Q wave is 1 or more mm in depth and 0.02 or more seconds in duration.

Major Evolving ST-T Code
No ST segment elevation in one ECG to ST segment elevation in another ECG, plus changing T codes
No T wave inversion code to major or moderate T wave inversion
Minor T wave inversion or flat to major T wave inversion
(These codes may be present on consecutive records of different dates.)

Minor Evolving ST-T Code
No ST segment elevation to ST segment elevation
No T wave inversion code to major or moderate T wave inversion
Minor T wave inversion or flat to major T wave inversion

No ST depression code to major or moderate ST depression
Minor ST depression to major ST depression

Stable ST-T Codes (ST-T codes that do not change in any record)
Persistent ST segment elevation
Persistent minor T wave inversion to flat to major T wave inversion
Persistent minor ST depression to major ST depression

Normal Electrocardiogram
No major or minor ST-T wave changes
*ST-T code change can occur in either direction.
†ST segment elevation does not have to be present in the same lead group as the T progression, nor does it have to be simultaneous.

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