Stroke is a serious potential complication of acute myocardial infarction (AMI). Several publications have outlined the incidence and clinical significance of stroke occurring during AMI in the prethrombolytic and thrombolytic eras. The frequency of this complication has apparently not increased with the advent of thrombolytic treatment but nevertheless remains an important cause of morbidity and mortality in patients with AMI. Data on the incidence and prognostic significance of stroke in survivors from AMI, however, are very scarce and limited to select subgroups of patients.

The purposes of the present study were (1) to assess the incidence of stroke or transient ischemic attack (TIA) after hospital discharge in a large unselected population of survivors from AMI, (2) to identify the characteristics of MI patients at risk, and (3) to assess the impact on mortality of stroke/TIA occurring after AMI.

Materials and Methods

From August 1981 to July 1983, 4808 hospital survivors from a cohort of 5839 consecutive AMI patients hospitalized in 13 of the 21 existing coronary care units in Israel were screened for inclusion in SPRINT (Secondary Prevention Reinfarction Israeli Nifedipine Trial). No effect of nifedipine on mortality or reinfarction after AMI was observed during the trial.

Demographic, historical, and clinical data were collected on study forms for all registered AMI patients by the study physicians in the coronary care units. Historical characteristics were based on self report by patients at the time of hospitalization. The diagnosis of AMI was based on the presence of typical symptoms, including severe pain in the region of the sternum, the precordium, or the upper abdomen lasting for at least 1 hour.

Keywords: cerebrovascular disorders, myocardial infarction, prognosis, risk factors
unless relieved by major analgesics; unequivocal electrocardiographic (ECG) findings (Minnesota Code interpretations); and elevated serum levels (at least 1.5 times the upper normal limit in the local laboratory) of creatine phosphokinase (CK)-MB or of two of the enzymes: following CK, glutamic oxaloacetic transaminase (GOT), and lactate dehydrogenase (LDH). Diagnosis was established on the basis of the presence of one of three sets of conditions: (1) typical symptoms, elevated serum levels of cardiac enzymes, and ECG findings of either Q-QS with major ST- and/or T-wave abnormalities, or dynamic ST depression or elevation, or dynamic T-wave changes, on comparison of two recent ECGs; (2) atypical symptoms, elevated serum levels of cardiac enzymes, and one of the following Q-QS changes on comparison of two recent ECGs: the appearance of Q-QS findings on the second ECG that were not present on the first, or the appearance of major Q-QS findings that were previously only minor; or (3) typical symptoms and ECG findings required under (2), without enzyme elevations. Assessment of heart failure on admission was based on the Killip classification: class I, no heart failure; class II, mild to moderate heart failure; class III, pulmonary edema; and class IV, cardiogenic shock.

Chronic atrial fibrillation was diagnosed by history, comparison with previous ECGs (when available), and persistent presence of the arrhythmia during hospitalization and on discharge from hospital.

One-year morbidity and mortality follow-up was completed for 99% of the patients by periodic medical visits, hospital records, and in a small group of patients, by telephone interviews of the patients or their family physician. The diagnosis of stroke/TIA was based on the sudden development of a focal neurological deficit persisting for at least 24 hours (stroke) or recovering earlier (TIA). Diagnoses were made by attending physicians in emergency rooms or during hospitalization and confirmed by study physicians during follow-up visits of patients who survived the stroke. In addition, all individual records or hospital reports for which stroke/TIA was quoted in the year after hospital discharge were reviewed for this study by one of the authors (D.T.). No attempt was made to differentiate between cerebral hemorrhage, embolism, or thrombosis because data on neuroradiological imaging was available for only a limited number of patients. Patients without stroke/TIA during the first year of follow-up formed the reference group.

Mortality follow-up was conducted for up to 7 years (mean, 5.5 years) for 99% of hospital survivors. Anticoagulation or antiaggregant drugs were not routinely given to survivors from AMI at discharge. Thrombolysis was not provided to any patient in this study, covering hospitalization experience from 1981 to 1983.

Statistical Analysis

The prevalence of attributes correlating with stroke/TIA was compared between patients with and without stroke/TIA, and the differences were examined by the \( \chi^2 \) test. Mortality rates were adjusted for age according to the total study distribution in age groups of \( \leq 50 \) years, 50 to 69, and \( \geq 70 \) years. By exponentiating risk coefficients derived from multiple logistic regression, we obtained estimates of the covariate-adjusted relative odds for the occurrence of stroke/TIA during the year after AMI, associated with presence versus absence of chronic atrial fibrillation; past MI; anterior MI site; past stroke; GOT level more than four times above upper normal limit; and for a 10-year age increment (namely, the factors that significantly predicted the incidence of stroke/TIA). The SAS software was used, specifically the FREQ and CATMOD procedures for this analysis. The actuarial survival curves were computed by the LIFETEST procedure.

Results

Forty-eight (1%) of the 4808 hospital survivors from AMI experienced stroke/TIA in the year after the MI. Thirty-seven of these cases were diagnosed as stroke and 11 as TIA. Rates did not differ significantly among men (0.9%) and women (1.3%). However, rates were significantly elevated among older patients (>70 years, 1.9%) and in those with a history of previous MI (1.8%), hypertension (1.4%), stroke in the past (4.1%), or chronic atrial fibrillation (9%) in comparison with younger patients (\( \leq 50 \) years; 0.5%; \( P<.001 \)), patients with a first MI (0.8%; \( P<.01 \)), and in those free of hypertension (0.7%; \( P=.06 \)), previous stroke (0.9%; \( P<.001 \)), or chronic atrial fibrillation (1%, \( P<.001 \)). Thirty-one percent (15 of 48) of events occurred in the first month and 63% (30 of 48) during the first half year after hospital discharge. The characteristics and hospital course of patients with and without stroke/TIA in the year after the MI are given in Tables 1 and 2. Patients developing stroke/TIA were 6 years older and more often had a past history of MI, hypertension, chronic atrial fibrillation, or stroke. However, a past history of angina pectoris, diabetes mellitus, or smoking were nearly equally prevalent in both groups. Anterior site of MI and high levels of the cardiac enzyme GOT were more frequent in patients subsequently developing stroke/TIA. Patients who developed stroke/TIA presented more often in a higher Killip class and had pulmonary congestion or edema on admission more often than counterparts without stroke/TIA. All other in-hospital complications were nearly equally prevalent in both groups (Table 2). The age-adjusted 1-year and long-term mortality rates (4.5 to 7 years; mean, 5.5 years) were significantly higher in patients with stroke/TIA (Table 3) than in those without (31% and 62% vs 9% and 31%, respectively; \( P<.01 \)). Fourteen of the 19 deaths (74%) that occurred during the first year among patients with stroke/TIA occurred within 2 weeks of the cerebral event. Figure 1 shows the respective survival curves of patients with and without stroke/TIA.

At discharge from the hospital, 13 patients (27%) who subsequently developed stroke/TIA and 759 patients (16%) in the reference group received anticoagulant or antiaggregant drugs.

To assess the independent parameters predicting the appearance of stroke/TIA in the year after AMI, multivariate analysis was conducted among 4808 survivors adjusting for the following: age, gender, past history of MI, angina pectoris, hypertension, diabetes mellitus, chronic atrial fibrillation, previous stroke, smoking, anterior MI site, congestive heart failure on admission to hospital, pulmonary congestion, or cardiomegaly on chest x-ray, as well as in-hospital complications including congestive heart failure, paroxysmal atrial fibrillation, ventricular arrhythmia, advanced atrioventricular block, and high serum enzyme levels. The independent
TABLE 1. Characteristics of Patients With and Without Stroke/TIA in the First Year Postdischarge After Acute MI

<table>
<thead>
<tr>
<th></th>
<th>Stroke/TIA (n=48)</th>
<th>Reference Group (n=4760)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>No.</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>%</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>33</td>
<td></td>
<td>777555</td>
</tr>
<tr>
<td><strong>%</strong></td>
<td>69</td>
<td></td>
<td>.001</td>
</tr>
<tr>
<td>Mean age, y</td>
<td>67.7 (+9.5)</td>
<td>61.6 (+10.6)</td>
<td></td>
</tr>
<tr>
<td><strong>History</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Recurrent MI</td>
<td>19</td>
<td>1058</td>
<td>.01</td>
</tr>
<tr>
<td>AP in the past</td>
<td>24</td>
<td>2273</td>
<td>.NS</td>
</tr>
<tr>
<td>Hypertension</td>
<td>27</td>
<td>1902</td>
<td>.06</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>11</td>
<td>902</td>
<td>.NS</td>
</tr>
<tr>
<td>CAF</td>
<td>3</td>
<td>30</td>
<td>.001</td>
</tr>
<tr>
<td>Previous stroke</td>
<td>10</td>
<td>210</td>
<td>.001</td>
</tr>
<tr>
<td>Smoker</td>
<td>13</td>
<td>1700</td>
<td>.NS</td>
</tr>
<tr>
<td><strong>MI Location</strong></td>
<td></td>
<td></td>
<td>.05</td>
</tr>
<tr>
<td>Anterior</td>
<td>27</td>
<td>1980</td>
<td>.43</td>
</tr>
<tr>
<td>Inferior</td>
<td>15</td>
<td>1939</td>
<td>.43</td>
</tr>
</tbody>
</table>

TIA, transient ischemic attack; NS, not significant; MI, myocardial infarction; AP, angina pectoris; CAF, chronic atrial fibrillation.

The six risk factors, the estimated likelihood of developing stroke/TIA in hospital survivors from AMI rose from 0.29% in patients without any risk factors to 3.18% among patients with three and 6.76% among patients with four of the above risk factors (Fig 2). None of the patients had more than four risk factors.

TABLE 2. Hospital Course of Patients With and Without Stroke/TIA in the First Year Postdischarge After Acute MI

<table>
<thead>
<tr>
<th></th>
<th>Stroke/TIA (n=48)</th>
<th>Reference Group (n=4760)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>No.</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>%</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Admission</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Killip class II-IV</td>
<td>19</td>
<td>881</td>
<td>.003</td>
</tr>
<tr>
<td><strong>Chest X-Ray</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pulmonary congestion or edema</td>
<td>19</td>
<td>1000</td>
<td>.002</td>
</tr>
<tr>
<td>Cardiomegaly</td>
<td>18</td>
<td>1252</td>
<td>.09</td>
</tr>
<tr>
<td><strong>Complications</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VT</td>
<td>5</td>
<td>766</td>
<td>.NS</td>
</tr>
<tr>
<td>VF</td>
<td>1</td>
<td>188</td>
<td>.NS</td>
</tr>
<tr>
<td>PAF</td>
<td>9</td>
<td>543</td>
<td>.NS</td>
</tr>
<tr>
<td>Advanced AVB</td>
<td>6</td>
<td>373</td>
<td>.NS</td>
</tr>
<tr>
<td>CHF</td>
<td>9</td>
<td>809</td>
<td>.NS</td>
</tr>
<tr>
<td><strong>Enzymes (≥4× upper normal limit)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CK</td>
<td>24</td>
<td>2636</td>
<td>.NS</td>
</tr>
<tr>
<td>GOT</td>
<td>25</td>
<td>1906</td>
<td>.06</td>
</tr>
<tr>
<td>LDH</td>
<td>8</td>
<td>529</td>
<td>.NS</td>
</tr>
</tbody>
</table>

TIA, transient ischemic attack; VT, ventricular tachycardia; NS, not significant; VF, ventricular fibrillation; PAF, paroxysmal atrial fibrillation; AVB, atrioventricular block; CHF, congestive heart failure; CK, creatine phosphokinase; GOT, glutamic oxaloacetic transaminase; LDH, lactate dehydrogenase.
Discussion

Stroke, a relatively rare complication after AMI, is associated with high mortality and represents one of the major causes of morbidity among hospital survivors. In the prethrombolytic era, the incidence of stroke during AMI varied from 0.9% to 2.4%. Although lower rates of stroke were sometimes reported after the advent of thrombolytic therapy, this may be partially explained by the exclusion of such therapy of patients at high risk.

Incidence of Stroke in Survivors of AMI

Few studies have examined the incidence of stroke among MI survivors. The Sixty-Plus Reinfarction Study reported 19 stroke cases among 439 elderly MI patients in the placebo arm of the trial during a mean follow-up period of 6 years. In the early 1970s McAllen and Marshall reported a 6% incidence of stroke/TIA among 260 MI survivors followed for 5 years. In a 20-year population-based study, only during the first 2 months after AMI was a significant difference noted between observed and expected probabilities of stroke; after that time no difference emerged over 10 years of follow-up. These data are in accordance with the present study in which 1% (48 of 4808) of MI survivors experienced stroke/TIA in the year after hospital discharge, nearly a third of these during the first month of follow-up. In a more recent trial on the effect of warfarin after MI, 44 of 607 (7%) patients experienced stroke in the placebo arm of the study during an average follow-up time of 37 months.

The incidence of stroke in the general population was evaluated extensively in the population of Rochester, Minn. The average annual age and sex adjusted incidence of stroke was 0.14% for the years 1980 to 1984 (0.21% for the 55- to 64-year-old group). In the Jewish population of northern Israel, the incidence of stroke in 1984 was higher: the age standardized incidence rates were 0.61% and 0.50% for men and women, respectively (for the 60- to 64-year-old group, 0.56% and 0.49%, respectively). Thus, although the incidence of stroke is relatively low in the year after the acute phase of MI, it nevertheless remains higher in comparison with the general population.

Clinical Predictors of Stroke

The most reliable predictors of stroke during AMI as previously reported by our group and others were older age, anterior site of MI, high enzyme levels, impaired

![Graph](image-url)
left ventricular function, atrial arrhythmias, and prior cerebrovascular disease.\textsuperscript{2,8,10,12,13} In the thrombolytic era few studies have examined the clinical factors associated with stroke appearance and then only during AMI. O'Connor et al\textsuperscript{a} found that cerebral infarction was related to anterior site MI and poor left ventricular function, but intracranial hemorrhage remained an unpredictable risk in these patients. Older age, a higher Killip class, and the occurrence of anterior infarction significantly increased the risk of stroke as shown by Maggioni et al.\textsuperscript{10} Both studies demonstrate that non-hemorrhagic stroke remains the most common form seen in patients with AMI on thrombolytic therapy.

To the best of our knowledge the clinical factors associated with increased risk of stroke in patients after discharge from AMI were not previously assessed. In the present study the factors identified as contributing independently to increased risk of stroke/TIA after hospital discharge were chronic atrial fibrillation, older age, past MI, anterior site of MI, serum GOT levels more than four times above upper normal limits, and previous stroke. The likelihood of developing stroke/TIA after hospital discharge increased proportionally to the number of risk factors present.

Chronic atrial fibrillation was present in 6\% (3 of 48) of stroke/TIA patients versus less than 1\% (32 of 4760) of the reference group. In a previous study we reported that chronic atrial fibrillation increased the risk of subsequent stroke/TIA ninefold.\textsuperscript{24} In several studies, nonrheumatic chronic atrial fibrillation was associated with approximately fivefold increased risk for stroke in comparison with matched population with sinus rhythm.\textsuperscript{25,26} Moreover, low intensity anticoagulation with warfarin can prevent most of the additional stroke risks caused by atrial fibrillation.\textsuperscript{27}

Echocardiographic studies have shown that the majority of patients with visible mural thrombi have had an anterior infarction, whereas patients with good left ventricular function and inferior infarction rarely had detectable thrombi. Mural thrombi are known to be associated with an increased embolic risk in the first few weeks after acute MI; however, there are conflicting results regarding their long-term embolic risk.\textsuperscript{26-30} Weinreich and coworkers\textsuperscript{28} followed 43 patients with left ventricular thrombi after AMI and identified embolic events only during the first 4 months after infarction in a mean follow-up of 15 months. However, in a more recent prospective study, following 85 patients with left ventricular thrombi, all embolic events occurred later than 1 month after MI (range, 1 to 96 months), suggesting that chronic thrombi continue to embolize.\textsuperscript{29} In the SPRINT Registry echocardiogram was not routinely performed on all hospital survivors. However, anterior site of MI was found to independently increase the risk of stroke after AMI.

**Clinical Implications**

The mortality of patients developing stroke during hospitalization for AMI is very high. We\textsuperscript{1} and others\textsuperscript{8,9} have previously reported an estimated threefold inhospital mortality among patients with stroke/TIA during AMI in comparison to those without. Mortality was also significantly elevated among the 48 patients of this study who developed stroke/TIA after discharge. The majority of deaths up to an average of 5.5 years after discharge in those patients occurred during the first year of follow-up. Moreover, nearly three quarters of the deaths during the first year (14 of 19) occurred within 2 weeks of the cerebral event.

Long-term anticoagulant treatment possibly has a favorable effect on the incidence of stroke after acute MI,\textsuperscript{11} although associated with a variable degree of hemorrhagic complications. Identification of high versus low risk patients for stroke might help in the selection of patients who could benefit most from this treatment.

**Appendix**

**Sprint Study Group**

**Executive Board:** Henry N. Neufeld, MD, Chairman (deceased); Jacob Agmon, MD, Vice-Chairman; Solomon Behar, MD; Uri Goldbourt, PhD; Henrietta Reicher-Reiss, MD; Edward Abinader, MD; Jacob Barzilay, MD; Natalio Cristal, MD; Yaacov Friedman, MD; Nissim Kauli, MD; Yechekiel Kishon, MD; Abraham Palant, MD; Benyamin Peled, MD; Leonardo Reisin, MD; Egon Riss, MD (deceased); Zwi Schlesinger, MD; Izhar Zahavi, MD; Monty Zion, MD

**Participating Centers, Principal Investigators, and Physicians:**

Assaf Harofeh Hospital, Zerifin. Principal investigator: Zwi Schlesinger, MD; Physician: Moshe Algim, MD

Barzilai Medical Center, Ashkelon. Principal investigator: Leonardo Reisin, MD; Physician: Newton Yalom, MD

Beilinson Medical Center, Petach Tikvah. Principal investigator: Yaacov Friedman, MD

Carmel Hospital and Medical “Lin” Haifa. Principal investigator: Efrain Mayer, MD; Physician: Ephraim Mayer, MD

Central Emek Hospital, Afula. Principal investigator: Jacob Barzilay, MD; Physician: Lev Bloch, MD

Hasharon Hospital, Petach Tikvah. Principal investigator: Izhar Zahavi, MD. Physician: Menachem Katz, MD

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Kaplan Hospital, Rehovot. Principal investigator: Nissim Kauli, MD; Physician: Emanuel Liebman, MD

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**Fig 2. Incidence of stroke/transient ischemic attack by number of risk factors.**

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Wolfson Medical Center, Holon. Principal investigator: Yehzekiel Kishon, MD. Physician: Ron Narinsky, MD

Coordinating Center: Neufeld Cardiac Research Institute, Sheba Medical Center, Tel Hashomer. Teemol, Tel Hashomer, MD (Director); Uri Goldbourt, PhD; Henrietta Reicher-Reiss, MD; Lori Mandelzweig, MPH

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References

Frequency and prognosis of stroke/TIA among 4808 survivors of acute myocardial infarction. The SPRINT Study Group.
D Tanne, U Goldbourt, M Zion, H Reicher-Reiss, E Kaplinsky and S Behar

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