Risk-Benefit Stratification as a Guide to Lidocaine
Prophylaxis of Primary Ventricular Fibrillation in Acute
Myocardial Infarction: An Analytic Review

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Early investigators suggested that ventricular fibrillation without heart failure in acute myocardial infarction was reliably preceded by warning arrhythmias, and that suppression of such arrhythmias with intravenous lidocaine could avoid the need for resuscitation. While the efficacy and safety of lidocaine have been substantiated, the reliability of warning arrhythmias as predictors for primary ventricular fibrillation has not. We present data showing that the risk of primary ventricular fibrillation is most dependent on the patient's age and the interval since the onset of his symptoms, rather than on the presence of warning arrhythmias. We have estimated that lidocaine prophylaxis would have to be given to about 12 patients in the highest risk group (patients under age 50 and within six hours of the onset of symptoms), compared to about 400 patients in the lowest risk group (patients above age 70 and more than 24 hours since the onset of symptoms), to prevent one episode of primary ventricular fibrillation in each group. We propose that these risk stratifications, as adapted to the conditions in specific hospitals, provide the most rational approach to lidocaine prophylaxis of primary ventricular fibrillation.

The leading American textbooks of medicine recommend routine prophylactic intravenous lidocaine to prevent ventricular fibrillation in any patient whose acute myocardial infarction is complicated by ventricular premature contractions which occur at a rate of more than about three per minute or occur in certain patterns [1, 2]. More recently, several authors have suggested that all patients with known or suspected acute myocardial infarction receive lidocaine prophylaxis for about 48 hours [3–5].

In the following analysis of these and other recommendations for the prophylaxis of primary ventricular fibrillation in acute myocardial infarction, we will describe the issues involved and review the pertinent literature. We will show that much of the present confusion is related to the small sizes of the individual studies, to the variable drug regimens employed, to the failure of many studies to consider factors which might greatly alter an individual's risk, and to the frequent fear that withholding prophylaxis from a control group might be unethical. Then, using data relevant to specific subgroups, we will suggest what we believe is the most rational approach to the prophylaxis of primary ventricular fibrillation in acute myocardial infarction.

BACKGROUND INFORMATION AND DESCRIPTION OF THE ISSUES

In an attempt to lower the previously standard 35 percent hospital mortality among patients admitted with acute myocardial infarction [6, 7], Day [8, 9] and...
Meltzer [10] in the United States, Brown [11] in Canada, and Robinson and Sloman [12,13] in Australia nearly simultaneously in the early 1960s conceived the idea of an intensive coronary care area. Because each of these authors' small series included case reports of patients who were resuscitated from previously fatal arrhythmias, coronary care units (CCUs) quickly gained wide popularity. The subgroup of patients most likely to be saved by the new CCUs were those with acute myocardial infarctions (MIs) who developed ventricular fibrillation (VF) in the absence of congestive heart failure or shock. Such VF, which is termed primary VF in acute MI and which must be differentiated from "primary VF" without MI [14] and from secondary VF complicating heart failure or shock in acute MI, will be the subject of this review.

Although the first CCUs were designed mainly for patients with pre-existing electrocardiographic evidence of ongoing acute MI, by the middle 1960s many had already decided that the facilities should also be made available to patients whose histories suggested acute MI but in whom the diagnosis could not be verified at the time of admission; these latter patients are commonly termed "rule-out" MIs. By the late 1960s only about 50 percent of patients admitted to CCUs in New England actually developed documented MIs [15], and by the middle 1970s only 37 percent of patients admitted to a large community CCU [16] and only 30 percent of those admitted to a leading university CCU [17] had documented MIs. Pooled series [18–23] show that about 40–45 percent of acute MIs will not be complicated by pre-existing or secondary heart failure to shock; assuming approximately 775,000 patients are admitted annually to United States hospitals with acute MIs [15,24], approximately 330,000 patients with acute MIs are at risk for primary VF each year.

**REVIEW OF THE LITERATURE**

As originally conceived, CCUs were to function mainly as resuscitative facilities for patients with arrhythmias such as primary VF. However, soon after the advent of the CCU, Lown [22] and Julian [25] reported that sudden death from primary VF in acute MI was commonly preceded by "warning" arrhythmias: more than five premature ventricular contractions (VPCs) per minute, multiformed or multifocal VPCs, VPCs occurring on the T wave of the preceding beat, or VPCs appearing in salvos of two or more. Because of this relationship, Lown proposed that all patients with definite or "rule-out" infarction who manifested "warning" arrhythmias be treated with prophylactic lidocaine, and with added procainamide if needed, until the warning arrhythmias disappeared, usually by the third day. Lown never mentioned exactly how many of his reported patients received prophylaxis because of their warning arrhythmias, but none of his 77 patients with heart failure nor 73 patients without heart failure developed ventricular fibrillation after arrival in the CCU. Because previous studies which did not use prophylactic anti-arrhythmic therapy had reported a 5–10 percent incidence of ventricular fibrillation in acute MI [9,13,25,26] the Lown criteria for, and the regimen of, prophylaxis became widely accepted, especially in the United States.

However, subsequent studies have virtually unanimously disputed the importance of "warning" arrhythmias. First, continuous electrocardiographic recordings available for retrospective analysis will show "warning" arrhythmias in 80–90 percent of patients with acute MI, even though CCU personnel will have noted such arrhythmias in only about 20 percent of patients [27,28]. It would seem unreasonable to base the use of prophylactic medications on the one in five chance that an actual arrhythmia will be observed. Also, if such arrhythmias occur in 80 percent of
patients, they are hardly selective for the small proportion who will develop primary VF. In fact, both Lie [29] and El-Sherif [30] have shown that “warning” arrhythmias occur just as frequently among patients who do not develop primary VF as among those who do. Thus it is not surprising that pooled series have observed “warning” arrhythmias in only about one-third of patients who develop primary VF after arrival in the emergency room [20,23,31–34].

A far better correlate of primary VF is the interval since the onset of the patient’s symptoms. Adgey’s series [35], which included the pre-hospital phase of acute MI, showed that 50 percent of primary VF episodes occur within one hour of the onset of symptoms and that the incidence declines exponentially thereafter. Those studies which begin with the patient’s arrival in the emergency room show nearly 70 percent of primary VF occurring within four to six hours after the onset of symptoms and over 80 percent occurring by 12 to 24 hours (Table 1). Only Dhurandhar [32] failed to show at least 83 percent of primary VF within the first 24 hours after onset of symptoms, but his study is limited by the long mean duration of symptoms prior to hospital entry (nine hours) which probably also explains why his reported incidence of primary VF (2.4 percent) is lower than the four to ten percent noted by other contemporary series. Although five of Dhurandhar’s 20 episodes of primary VF occurred more than 48 hours after the original onset of symptoms, other studies [33,36,38] note that such late VF is usually related to complications of the original MI, to extension of the MI, or to reinfarction. In light of the strong correlation between interval since onset of symptoms and incidence of primary VF, Lown’s [22] success in a group of patients in which only 61 percent had been admitted within 12 hours of the onset of symptoms is not so surprising and cannot be definitely attributed to his prophylactic regimen.

A second apparently independent correlate of primary VF is the age of the patient. Lie [34] noted a decrease in the incidence of primary VF with advancing age, and both Lie and Lawrie [20] have found that primary VF is about twice as common in patients under age 50 as in those aged 50 to 69 (Table 2). Julian [25] also reported that ventricular fibrillation in the absence of severe heart failure or shock was twice as common in patients below age 51 as in those aged 51–61. Similarly, Valentine [39] reported pre-hospital sudden death or VF in acute MI to be three times more likely below age 50 as in ages 51–69. In patients over age 70, primary VF [34] or VF in the absence of severe heart failure [25] occurs far less often than in younger patients.

### TABLE 1

| Author       | < 4-6 < 12 < 24 |
|--------------|-----------------|
| Lawrie [20]  | 17* 22 23       |
| Church [23]  | 16 24          |
| Dhurandhar [32] | 24/36 26/36 30/36 |
| Lie [36]     | 24/36          |
| Oliver [37]  | 10/15          |
| Pentecost [38] | 10/10 |
| TOTAL        | 51/75 64/79 75/90 |

|               | 68% 81% 83% |

*Patients who developed primary VF by the end of this time period.
†Total number of patients who developed primary VF in the series.
patients, are points:first, laxis lidocaine levels, translates patients minimum(2.4 ug/ml) to incidence VF [29,30,44].

VPCs and developed warning arrhythmias. A universal prophylaxis of primary VF to be considered an independent risk factor for primary VF. Despite early emphasis on the importance of VPCs which interrupt the T wave of the preceding beat [13,43], recent studies have shown that later VPCs also commonly trigger ventricular tachycardia and primary VF [29,30,44].

**Efficacy of Anti-Arrhythmic Prophylaxis of Primary VF in Acute MI**

The numerous randomized controlled trials of lidocaine prophylaxis of primary VF in acute MI are shown in Table 3. Because of the influence of Lown's original report, the five series in Section A of Table 3 treated "control" patients if they developed warning arrhythmias. Some of these series in Section A showed fewer VPCs and fewer salvos or runs of VPCs in the universally treated groups [45,48], but universal prophylaxis of patients without heart failure or shock was no better at preventing primary VF than was lidocaine reserved for patients with warning arrhythmias. Also, the overall 4.4 percent incidence of primary VF in the treated groups of Section A of Table 3 is not substantially different than the 5.6 percent incidence of primary VF in the control patients in other studies where lidocaine was not given at all (Section B of Table 3). These controlled studies seem to make two points: first, the potential hazards of withholding lidocaine until warning arrhythmias are observed is substantiated; and second, low-dose lidocaine is ineffective prophylaxis for primary VF. This latter point is not surprising in light of recent data in patients without heart failure: lidocaine rates of 35 to 88 ug/kg/min (which translates to rates of 2.5 to 6.2 mg/min for a 70 kg patient) were required to reach minimum (2.4 ug/ml) and maximum (6.0 ug/ml) therapeutic steady-state lidocaine levels, respectively [52]. In summary, among all the randomized trials in hospitalized patients, only Lie's series [31] (Section B of Table 3), which used the highest dose of lidocaine in patients who were all under 70 years of age and had all been admitted

| Age of Patient | Below 50 years | 50-69 years | Above 70 years |
|----------------|----------------|-------------|---------------|
| Lawrie [20]    | 10*/70†        | 10/128      |               |
| Lie [34]**     | 4/44           | 13/245      | 1/111 (0.9%)  |
| Cumulative Totals | 14/114 (12.3%) | 23/373 (6.2%) |               |

*Number of patients who developed primary VF.
†Number of patients in this age range.
**Chi-square of linear trend = 5.86 (0.01 < p < 0.05).
within six hours of the onset of symptoms, showed a significant reduction in primary VF.

The largest reported experience with prophylactic lidocaine therapy is not in the context of a randomized trial but rather from the clinical observations of Wyman [53], who had originally noted VF in the absence of pulmonary edema or shock in 8 of 139 (6.5%) patients treated with prophylactic oral quinidine or procainamide. Over the next seven years, Wyman adopted a policy of lidocaine prophylaxis (which, in the 19 percent of patients with persistent VPCs, was augmented by intravenous procainamide) and reduced the incidence of VF to none of the 145 patients in whom the criteria for prophylaxis were warning arrhythmias, one of the 270 in whom the criterion for prophylaxis was a single VPC, and two of the 611 patients in whom the sole criterion for prophylaxis was the suspicion of an acute MI. In a subsequent report [16], Wyman's prophylaxis regimen (a 75 mg bolus of lidocaine followed by a 2 mg per minute drip, with additional lidocaine or procainamide for persistent VPCs) resulted in only six episodes of primary VF among 1,000 acute MI patients, 31 percent of whom were seen within one hour of onset of symptoms. Another six patients developed primary VF in the emergency room before prophylaxis could be started. Wyman's impressively low 0.3 percent [53] to 0.6 percent [16] incidence of primary VF after initiation of lidocaine prophylaxis is similar to the results of Pitt [48] and Lie [31] who used high-dose lidocaine without procainamide and without concern for warning arrhythmias.

There are only two major reports of lidocaine in the pre-hospital phase of acute

### Table 3
Controlled Trials of Lidocaine Prophylaxis of Primary VF after Hospital Arrival

| Mean No. of Hours since Onset of Symptoms | Dose | No. of Controls Treated with Lidocaine for warning VPC's | Incidence of Primary VF in Control Group | Incidence of Primary VF in Treated Group |
|------------------------------------------|------|--------------------------------------------------------|----------------------------------------|----------------------------------------|
| A. Control Group Treated if Warning VPC's |      |                                                        |                                        |                                        |
| Mogensen [45]                            | 75 mg IV | 2 mg/minute | 24/37 | 1/37 | 0/42 (N.S.)* |
| Darby [46]                               | 200 mg IM | 2 mg/minute | 16/100 | 3/100 | 4/103 (N.S.) |
| Church [47]                              | 50-75 mg IV | 2 mg/minute | 10/44 | 3/44 | 4/42 (N.S.) |
| Pitt [48]                                | Variable | 2.5 mg/minute | 34/114 | 0/114 | 0/108 (N.S.) |
| Bennett [49]                             | 60 mg IV | 0.5–1 mg/minute | 36/125 | 7/125 | 16/251 (N.S.) |
| **TOTALS**                               |      |                                                        | 120/420 | 14/420 | 24/546 |

| B. Controls Not Treated for Warning VPC's |      |                                                        |                                        |                                        |
| Blefield [50]                            | 100 mg IV | 1.0–2.8 mg/minute | 0 | 2/49 | 0/41 (N.S.) |
| Chopra [51]                              | 50–150 mg IV | 1–2 mg/minute | 0 | 0/43 | 1/39 (N.S.) |
| Lie [31]                                 | 100 mg IV | 3 mg/minute | 0 | 9/105 | 0/107 (p < 0.002) |
| **TOTALS**                               |      |                                                        | 11/197 | 5.6% | 1/187 (0.5%) |

*Not statistically significant.
MI. Adgey’s uncontrolled observations showed that only 60 percent of patients responded to a 100 mg intravenous bolus with a decrease in frequency of VPCs but did not comment on prevention of primary VF [35]. In a controlled trial, Valentine [39] reported a significant reduction in “early deaths” (which he defined as documented VF or “sudden death” regardless of whether resuscitation was successful, or an actual death from power failure, within two hours of initial medical attention) in patients given 300 mg intramuscular prophylactic lidocaine. However, closer analysis of his results reveals that the differences in arrhythmic “sudden death” were not quite statistically significant (0.05 < p < 0.10) and that all three patients with documented primary VF were successfully resuscitated. Still, the potential benefits of lidocaine were especially impressive among those less than 50 years of age: five of 24 controls developed “sudden death” compared to none of 37 treated patients.

In summary, lidocaine in large doses can potentially reduce the incidence of primary VF from 5–10 percent to about 0.5 percent. Wyman [16,53] has been very successful with his carefully regulated regimen, but a program of high-dose universal lidocaine prophylaxis without additional procainamide or other drug alterations for persistent VPCs seems easier and equally efficacious for the prevention of primary VF [31,48].

*Risks and Side Effects of Lidocaine Prophylaxis of Primary VF in Acute MI*

None of the major studies of lidocaine prophylaxis in patients with acute MI not complicated by heart failure or shock has reported any important cardiac side effects or any lidocaine-related deaths. However, standard intravenous infusions of 2.5 to 3.0 mg per minute for 48 hours will cause transient neurological side effects in about 16 percent of patients under the age of 70 [31,48]; most of these side effects will be minor (drowsiness, facial numbness, speech disturbances, and dizziness), but seizures or coma may also occur [48,53]. If, on the other hand, the infusion is regulated by a constant infusion pump, seizures and coma are exceedingly rare in patients without heart failure or hepatic dysfunction [31,53]. Lie [31] noted that his minor side effects were three times more frequent in patients 60–69 years old compared to those below age 60; no good data are available specifically for patients over age 70.

Recent data [54] have shown that lidocaine elimination is impaired during prolonged intravenous infusion. After 24 hours of continuous infusion in patients without heart failure, lidocaine blood levels can be maintained with doses only about one-half those required during the first 24 hours.

*Immediate and Post-Discharge Prognosis After Primary VF in Acute MI*

Early reports, which did not usually distinguish between primary and secondary VF, quoted successful immediate resuscitation in only 25–30 percent of patients with VF [25]. In 1973, Wyman [55] reported a 53 percent survival if VF developed in the absence of severe pump failure. If, however, we continue to limit our concern to true primary VF, Table 4 shows a unanimously high rate of successful resuscitation. The success rate, which averaged 94 percent, was just as high in United States community hospitals without housestaffs but with well-trained nursing staffs [16,23] as in teaching hospitals. The series also show that 87 percent of patients with primary VF survive to leave the hospital; at least half of those who die in the hospital after the originally successful resuscitation do so despite vigorous anti-arrhythmic therapy.

After hospital discharge, pooled series [13,21,56–58] show excellent six-month survival (98 percent) and reasonable one-year survival (average 87 percent). Only Conley [21] has described a suggestive, but not statistically significant, decrease in
one-year survival in patients who have been resuscitated from primary VF compared to those acute MI patients without heart failure or shock who did not develop primary VF. Even if one-year mortality is higher in the patients who had primary VF, it is not at all clear that this increased outpatient mortality is a result of the original in-hospital episode of primary VF.

RISK STRATIFICATION

Using the data we have summarized, we can estimate the risk of primary VF in acute MI if we know two independent factors: the patient's age and the interval since the onset of his symptoms (see Appendix). Based on these data, Table 5 shows the approximate number of patients in various subgroups who would have to receive lidocaine prophylaxis in each time period to prevent one episode of primary VF during that specified time period. Based on the rates we have reviewed for resuscitation and survival after primary VF, each number in Table 5 could be multiplied by

| Age below 50 years | Time since onset of symptoms |  |  |
|--------------------|-----------------------------|-----------------|-----------------|
|                    | Indication for prophylaxis  | < 6h            | 6-24h           | > 24h           |
| All MI             | 12                          | 54              | 48              |
| All MI or rule-out MI | 30                          | 135             | 120             |
| Age 50-69 years    | Indication for prophylaxis  | < 6h            | 6-24h           | > 24h           |
| All MI             | 24                          | 110             | 96              |
| All MI or rule-out MI | 60                          | 275             | 240             |
| Age 70 or more years | Indication for prophylaxis | < 24h            | > 24h           |
| All MI             | 100                         | 400             |
| All MI or rule-out MI | 250                         | 1000            |

*Based on our assumptions, only about one patient in 8.3 with primary VF will die in the hospital as a direct or indirect result of the arrhythmia. (See text and Table 4.)
8.3 to estimate how many patients must receive lidocaine prophylaxis to prevent each death from primary VF. Note that the increased "benefit" of prophylaxis after 24 hours compared to during the interval between six and 24 hours is partly artifactual—because of the larger number of hours in the time period—and partly related to reinfarction or extension of infarction [33,36,38]. Actually, the benefit per hour of prophylaxis decreases progressively and dramatically with time; when an individual patient enters a lower risk subgroup, his expected benefits from prophylaxis decline.

Although our risk estimates are carefully based on the principles which should guide the retrospective analysis of pooled data [59], several potentially confounding factors must be considered. First, data on patients who never received lidocaine for any indication are available largely from The Netherlands. Their precise applicability to the United States is unproved. Unfortunately, to prove our estimates prospectively in the high-risk subgroups may be considered unethical, and to prove the estimates in the low-risk subgroups would be logistically impractical. (For example, if our estimates are correct, to show a statistically significant decrease in primary VF and then in death from primary VF among patients over age 70 who receive routine lidocaine prophylaxis for the first 24 hours after the onset of symptoms, randomized controlled trials might require 4,400 and 20,000 patients, respectively.) Second, data on the importance of the interval since the onset of symptoms are less plentiful in patients over age 70 than in younger patients because: (a) in the era before the common use of lidocaine, patients over age 70 were not admitted to many CCUs; and (b) recent randomized trials, whose control groups represent the only reliable published sources of patients never receiving lidocaine, have excluded patients over age 70. According to Lie (personal communication), about 80 percent of primary VF in patients over age 70 occurs within 24 hours of the onset of symptoms. We have used Lie's experience to calculate our risk estimates for patients over age 70, but these estimates are surely less precise than those made for younger patients. Third, our estimate of zero serious side effects of lidocaine presumes constant infusion systems; standard intravenous drips will be associated with such frequent serious side effects (up to four percent) as to outweigh the potential benefits in patients at very low risk for primary VF. Fourth, while well-trained nurses will do as well as housestaff in resuscitating patients from primary VF, if staff is marginal during part of the day, the risk of death from an episode of primary VF might be substantially higher than quoted in our estimates. Fifth, even when quickly converted, primary VF may well have deleterious psychological consequences. We must again emphasize that while these risk estimates may apply to over one million patients admitted yearly for MI or rule-out MI in the absence of heart failure, data are insufficient to stratify risk of VF in those with heart failure.

RECOMMENDATIONS

Although the impact of routine aggressive lidocaine prophylaxis for subgroups of patients at substantial risk for primary VF may not be obvious in any one individual hospital, lidocaine prophylaxis could prevent about 15,000 episodes of primary VF each year nationwide. Using our risk estimates as a guide to therapy would avoid needlessly subjecting many times that number of very low-risk patients to potential lidocaine toxicities.

We cannot recommend any precise risk of primary VF which should be used as a cutoff point for prophylaxis until we have better estimates of the side effects of lidocaine in larger numbers of patients, better knowledge of the success rate for
resuscitation in specific hospitals, and better ways to stratify the chances that infarction will be documented in an individual patient with a “rule-out” MI. For the present we recommend:

1. Routine lidocaine prophylaxis for primary VF should be given as soon as possible when a patient who is below age 70 and who is within six hours of the *onset of symptoms* is diagnosed in an emergency room as having an acute MI or probably even a high likelihood of an acute MI. Assuming normal hepatic function, the dose should be at least a 75–100 mg bolus followed by about a 35 µg/kg/min infusion.

2. Routine lidocaine prophylaxis of primary VF does not seem warranted in patients who are over age 70 and (a) are more than 24 hours after the onset of symptoms or (b) have a low probability of an actual MI.

3. For patients in other subgroups, we can only say that our estimates of the risk of primary VF in acute MI combined with whatever cutoff point seems most appropriate in an individual hospital are a better guide for deciding which patients should receive prophylaxis than the small series originally reported by Lown [22], the standard recommendations of the leading medical textbooks [1,2], or the recent recommendation that *every hospital* should give routine lidocaine prophylaxis for a full 48 hours *after hospital admission* to all patients [3,4] or just to patients below 70 years of age [5].

4. “Warning” arrhythmias do not reliably predict which patients are at high risk for primary VF and their presence or absence should not be used as the guide for instituting prophylaxis. After about 48 hours since the onset of symptoms, or in the presence of heart failure, hypotension, recurrent pain, or arrhythmia-associated symptoms, “warning” VPCs may be important correlates of *secondary* VF or hemodynamic compromise and should be suppressed.

5. If lidocaine is continued for more than 24 hours, the infusion rate should be decreased by about 50 percent because of the reduced clearance of lidocaine after prolonged infusions. Otherwise, the risks of lidocaine toxicity will be increasing substantially with time while the risks of primary VF are declining.

Lidocaine is a usually safe drug and is of proved efficacy in reducing the incidence of primary VF in the CCU setting. We believe our estimates emphasize (a) how certain patient subgroups are most likely to benefit from lidocaine prophylaxis; and (b) how other subgroups at minimal risk for primary VF may have more complications than benefits from lidocaine prophylaxis.

**APPENDIX**

*Assumptions for Calculations of Potential Risks and Benefits of Lidocaine Prophylaxis*

1. The basic risk of primary VF is 8.6 percent in patients who receive no antiarrhythmic prophylaxis, are less than 70 years of age, and are admitted within six hours of the onset of symptoms of a new myocardial infarction without “congestive heart failure, cardiogenic shock, complete atrioventricular block, persistent brady-cardia (rate less than 50 beats per minute), persistent ventricular tachycardia or ventricular fibrillation on admission” [31].

2. The age-related ratios for primary VF are as reported in Table 2: two to one for under age 50 compared to ages 50–69 [24,34], and 6.5 to one for all below age 70 compared to age 70 and higher [34]. Considering the age distribution of hospitalized patients with acute MI [60] and the probably increased prevalence of heart failure in
elderly patients with infarction [20,61–63], our estimated risks for primary VF are: 13 percent for patients under age 50, 6.5 percent for those aged 50–69, and 1.3 percent for patients aged 70 and above. These estimates for each age group are about as high as reported in any large series of patients who reach the hospital.

3. As noted in Table 1, for patients below age 70 about 68 percent of primary VF will occur by six hours after the onset of symptoms, another 15 percent will occur by 24 hours, and nearly all of the remainder will occur by 48 hours. For patients over age 70, far fewer specific data are available, but an approximately equivalent 80 percent of primary VF will appear within 24 hours of the onset of symptoms (Lie, personal communication).

4. Lidocaine given in the only clearly effective doses, 75–100 mg bolus followed by a 2.5–3.0 mg her minute infusion, will reduce the rate of primary VF from 8.6 percent to 0.5 percent in patients below age 70 and by a similar ratio in other types of patients [16,31,48].

5. No patients will die as a result of lidocaine prophylaxis in the absence of heart failure or shock.

6. About 60 percent of the patients who were originally thought to be at potential risk for primary VF in acute MI will not actually have infarction [15,17], the risk of in-hospital primary VF in these patients is assumed to be zero.

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