Pathophysiologic Observations in Prehospital Ventricular Fibrillation and Sudden Cardiac Death

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SUMMARY

In order to better understand the problem of prehospital sudden cardiac death (SCD) two groups of individuals were studied. One group was monitored by rescue squads during attempted rescue. These subjects were defibrillated from prehospital ventricular fibrillation (VF) and hospitalized if they survived or autopsied if they could not be resuscitated. The second group were SCDs which were witnessed and described by observers. Detailed past histories of both groups were collected, and either clinical or autopsy diagnoses were obtained.

On the day of death or VF, one quarter reported new symptoms (primarily chest pain and dyspnea) preceding collapse by more than 30 minutes, one quarter reported symptoms lasting from 1 to 30 minutes, and one half collapsed instantaneously or within 1 minute of acute symptoms. A history of old myocardial infarction (MI) was present in 41% and of angina pectoris in 54%, and 27% reported new or changing symptoms within four weeks.

In defibrillated survivors, "would-be SCDs," electrocardiographic (ECG) changes of acute myocardial infarction (AMI) or ischemia were nearly three times more frequent than changes detected histologically in SCDs, and in the former involved predominantly the anterior wall in contrast to the inferior wall in most autopsied deaths. This disparity implicates acute myocardial lesions, particularly of the anterior wall, in the majority of SCDs.

Acute coronary lesions were found in 58% of SCDs autopsied. Most of these were ruptured plaque; although almost one fifth of all autopsied SCDs had thromboses without intimal rupture. Severe chronic multivessel stenosis was present in most subjects, although 15% had only disease of a single vessel and in these, the left anterior descending (LAD) or left main coronary artery were involved in three quarters.

The SCD population may be subgrouped into those with recent MI, those with only myocardial ischemia, and those with no detectable myocardial change. When rescuers were able to monitor prehospital SCDs, VF was found in the majority; however, 28% did have other terminal rhythms.

Additional Indexing Words:
Sudden death  Cardiac arrest  Pathology  Resuscitation  Epidemiology

MORE THAN 300,000 persons die annually in the United States outside of the hospital, presumably of heart disease, with varying degrees of rapidity and in a more or less unexpected fashion.

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There have been many studies dealing primarily with historical, epidemiologic, and pathologic aspects of sudden cardiac death (SCD). Our understanding of this problem, however, has been limited by the sudden and unexpected nature of these deaths which generally precludes clinical observations; by the shortcomings of conventional histologic techniques which do not detect early acute MI or myocardial ischemia; and by the deficiencies of retrospective second person histories which are replete with errors of omission and transmission.

In the present investigation, by working with trained fire rescue squads who reach most emergencies within minutes of summons, we were able to obtain details of the acute circumstances surrounding SCDs as well as terminal electrocardiographic (ECG) rhythms. Cardiopulmonary resuscitation (CPR) and
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defibrillation were performed on these subjects, and survivors, “would-be SCDs,” were hospitalized, thus permitting evolution and study of physiologic processes which would otherwise have terminated with death. Patients who could not be resuscitated were studied at autopsy, and the morphologic findings in these persons were compared with clinical data obtained from survivors. Correlation of autopsy, historical, and clinical findings provided insight into possible mechanisms involved in SCD as well as possible approaches to management.

Methods and Materials

Study Population

Between April 1970 and March 1973, 576 SCDs and subjects with prehospital VF were studied. Four hundred twenty-six subjects were monitored acutely by rescue workers and had CPR. From this group, 150 subjects were selected because they satisfied the study criteria which included normal amputatory function in their usual environment, absence of a noncardiac cause of death, availability of information concerning past, recent, and acute history, and either hospital follow-up in defibrillated survivors or autopsy study in unsuccessful resuscitations. Within this group of 150 subjects, 80 were defibrillated and had diagnostic evaluation in the hospital, and 70 could not be resuscitated and were autopsied (52 of these had terminal VF and 18 had other terminal rhythms).

One hundred fifty additional SCDs, who were witnessed by observers during the terminal episode but were not reached by rescue workers, had historical and autopsy study.

In all instances, histories were obtained by trained interviewers using detailed questionnaires,* and when available, from hospital and physician records. All autopsies were performed by one of two pathologists using detailed research protocols.*

Rescue, Resuscitation, and Follow-up Studies

Firemen strategically located throughout the city reach 80% of emergency medical calls within 4 min of summons.** Forty-six men were trained to administer CPR, to telemeter ECG rhythms† to physicians in the Jackson Memorial Hospital recovery room, and then under physician’s direction to defibrillate.1 When indicated, intravenous drugs including sodium bicarbonate, epinephrine, lidocaine, atropine, and calcium chloride were given. When cardiovascular success was achieved, the patient was stabilized and transported to the hospital. When unsuccessful, CPR was maintained during transport to the emergency room where further treatment, evaluation, or pronouncement of death were made.

Successful resuscitation was defined as admission to the hospital of a patient who had documented prehospital VF and would otherwise have been an SCD. ECG and clinical diagnoses in these patients were made using standard criteria.** AMI was determined by diagnostic ECG Q waves not previously present. All patients were defibrillated; therefore serum enzyme data were not used. Ischemia was diagnosed when only ischemic ST-T wave changes without evolution of Q waves were present on serial ECGs.

Pathology

The major coronary vessels and their branches were dissected free, fixed in formaldehyde, decalcified, and cut in cross-sections at 1 mm intervals. Acute lesions and severely stenotic segments were embedded in paraffin and alternate sections were stained with hematoxylin and eosin (H and E) and, in some cases, Weigert Van Gieson stains. The myocardium was sectioned from apex to base at 0.5 cm intervals. All overt lesions as well as routine sections from the mid septum and mid anterior, lateral, and inferior walls were fixed in formaldehyde, embedded, stained with H and E, and studied under a light microscope. Acute coronary artery lesions were classified as thrombosis, plaque rupture, or intramural hemorrhage (IMH).

Thrombosis was subgrouped as fresh when the red blood cell, platelet, and fibrin mass was loosely attached to the intimal lining but showed no organization; or organizing when the thrombus was adherent to the intima and evidence of fibroblast proliferation was present.

Plaque rupture was defined as disruption of the intimal wall overlying the plaque with extrusion of plaque substance into the coronary lumen. Included in this group were cases in which rupture was complicated by overlying thrombus or dissecting IMH.

IMH was defined as hemorrhage into a plaque with persistence of an intact intimal lining.

AMI was categorized as: very fresh when the myocardial changes consisted of increased eosinophilia blurred cross-striations and hyalination and vacuolization of myocardial fibers, but nuclear details were still present (less than one day old); fresh when the above changes were accompanied by loss of cellular detail and early polymorphonuclear granulocyte infiltration (one to three days old); recent when necrosis was well delineated and associated with heavy polymorphonuclear granulocyte infiltration and early absorption of necrotic debris (three to seven days old); and organizing when capillary and fibroblast proliferation were present (one to two weeks old).

Severe chronic stenosis was defined as narrowing of more than 75% of the coronary lumen area estimated from standard planimetric templates.* Acute and chronic coronary lesions located in the first 3 cm of the involved vessel were considered proximal. Old MI was defined as fibrous scar 1 cm or greater in diameter. These were either solid or patchy.

Results

Total Subjects Monitored

In the 34-month study period, fire rescue squads monitored 426 prehospital SCDs or subjects with VF. VF was the initial rhythm recorded in 306 subjects (72%), and other terminal rhythms were monitored on arrival in 120 (28%). These other rhythms included idioventricular rhythm in 34, asystole in 33, junctional

* Copies available from the Myocardial Infarction Branch of the National Heart and Lung Institute, Bethesda, Maryland.

† Using GE Portable mobile transceiver and Biocom modulator.

† Using PhysioControl Lifepak cardiac monitors and defibrillators.

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bradycardia in 28, sinus bradycardia in 10, complete atrioventricular block in 9, and ventricular tachycardia in 6.

Characterization of Acutely Monitored Subjects with Available History and Diagnostic Follow-up

Details concerning these 150 subjects (80 hospitalized defibrillations and 70 autopsied SCDs) are presented in table 1. No significant differences were found between the 52 patients with monitored VF and the 18 with other terminal rhythms; therefore these are discussed together. The mean age for women significantly exceeded that for men — 67 compared to 58 years. This was largely a male and a Caucasian population — 81% and 84%, respectively, and significant past cardiovascular history was present in many, including old MI in 41%, angina pectoris in 54%, and hypertension in 36%.

During the four weeks preceding cardiac arrest new or changing symptoms of chest pain and/or dyspnea were reported in 40 subjects (27%) — in 21 within seven days. Forty-one subjects consulted a physician during these four weeks. Only 18 (12%), however, related their visit to new or changing cardiovascular symptoms, and the remaining visits were routine.

On the day of cardiac arrest, 36 subjects (24%) reported new chest pain and/or dyspnea, which preceded arrest by more than 30 min with a mean of 3.8 hr and a median of 2 hr. These symptoms, however, did not alter usual activity or lead to intervention. In 35 (23%), only very acute symptoms of chest pain or dyspnea were present, which preceded arrest by from 1 to 30 min, with a mean of 10.5 min and a median of 10.0 min. In 79 subjects (53%), collapse occurred instantaneously or within 1 min of acute symptoms. No difference was found in the frequency or type of preceding symptoms regardless of the resuscitation outcome.

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Forty-five percent of these events occurred at home and 12% while at work. Activity immediately preceding arrest was strenuous or stressing in 17% and mild or moderate in the remainder.

Clinical Diagnoses in Defibrillated Hospitalized Patients

Of the 306 subjects with prehospital VF, 80 (26%) were successfully defibrillated and hospitalized and had clinical evaluation and diagnosis. Detailed description of these patients is included in table 1 and shows that they are generally comparable to the monitored SCDs as well as to the witnessed but not monitored SCDs. Clinical diagnoses in these patients are given in table 2. By ECG criteria, changes of AMI were present in 39%, and ischemia without definite AMI was found in 34%. These changes were present on the admission ECG in all but two patients. In 18% of patients, serial ECGs failed to demonstrate any acute myocardial change, and in 8% complete left bundle branch block (CLB) masked ECG diagnosis of acute myocardial change.

In the 58 patients (73%) with acute myocardial lesions, either AMI or ischemia, these involved the anterior wall in 60%, the inferior wall in 24%, and both walls in 16%. In those with only ischemia, changes involved the anterior wall in 70%, the inferior wall in 11%, and both in 19%.

Histologic Characterization of Autopsied SCDs

Description of 220 autopsied SCDs is included in table 1, and detailed pathologic characterization is presented in table 3. There were no significant differences in autopsy findings between the 70 terminally monitored SCDs and the 150 witnessed but not monitored SCDs, and although analyzed separately in tables 1 and 3, findings in these groups are combined in this section. Pathologic findings in the 18 subjects with terminal rhythms other than VF did not differ significantly from the 52 with monitored VF, and these also are discussed together in this section.

Acute Changes

AMI was detected in 59 autopsied subjects (27%). These involved the anterior wall in 22%, the inferior wall in 66%, and both anterior and inferior walls in 12%.

In only 10 subjects with AMI (17%) were these changes less than one day old. Five patients had a ruptured ventricular wall and one had a ruptured papillary muscle.

Acute coronary lesions were present in 128 of those autopsied (58%). In 108 subjects a single coronary artery was involved, and in 20 more than one vessel had acute changes. Acute coronary lesions involved the right coronary artery in 48% of subjects, the LAD in 33%, the left main in 3%, and left circumflex in 16%. Acute coronary lesions were most often ruptured plaques — 72 subjects (56% of those with acute coronary changes). Thrombosis without discernible intimal rupture was found in 42 subjects — 32% of those with acute coronary lesions and 19% of all SCDs autopsied, and, in these, were fresh or recent in 32 and organizing in 10. IMH was found in 14 subjects (10%). Acute coronary lesions occurred at the site of severe old stenosis in all but four subjects.

In 81 autopsied subjects (37%), no acute vascular or myocardial lesion was found. Acute coronary or myocardial changes in patients with symptoms prior to SCD were slightly different from those with no known symptoms. In those with preceding symptoms, 33% had detectable AMI, 66% had acute coronary lesions, and 25% had no detectable acute myocardial or vascular changes. In those with no reported prior symptoms, 16% had detectable AMI, 48% had acute vascular lesions, and 51% had no acute changes. Preceding symptoms did not differ with different types of acute coronary lesions nor did the degree of activity immediately preceding collapse.

Chronic Changes

Chronic coronary artery stenosis of more than 75% of the lumen area was present in 207 subjects (94%). In 13 subjects (6%) no severe chronic stenosis was present, and in these, 1 had myocarditis, 4 had cardiomyopathy, 3 had coronary emboli, 1 had acute coronary arteritis, and 4 had severe valvular disease.

In 29 subjects (14%), only one major vessel was stenosed — 2 had total old occlusion. In these subjects, the LAD coronary artery was involved in 19, the left main in 2, the right in 6, and the left circumflex in

Table 2

<table>
<thead>
<tr>
<th>Characterization of Defibrillated Survivors</th>
<th>Patients (%)</th>
<th>Patients (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute myocardial infarction</td>
<td>31 (39%)</td>
<td>16 (52%)</td>
</tr>
<tr>
<td>Anterior wall</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anterior and inferior wall</td>
<td>4 (13%)</td>
<td>11 (35%)</td>
</tr>
<tr>
<td>Inferior wall</td>
<td>11 (35%)</td>
<td></td>
</tr>
<tr>
<td>Ischemia without infarction</td>
<td>27 (34%)</td>
<td>19 (70%)</td>
</tr>
<tr>
<td>Anterior wall</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anterior and inferior wall</td>
<td>5 (19%)</td>
<td>3 (11%)</td>
</tr>
<tr>
<td>Inferior wall</td>
<td>3 (11%)</td>
<td></td>
</tr>
<tr>
<td>No acute ECG change</td>
<td>15 (19%)</td>
<td>7 (8%)</td>
</tr>
<tr>
<td>Complete left bundle branch block*</td>
<td>7 (8%)</td>
<td></td>
</tr>
</tbody>
</table>

*Possibly masking an acute myocardial change.
Table 3

Pathologic Characterization of SCD

<table>
<thead>
<tr>
<th></th>
<th>Monitored (70)*</th>
<th>Witnessed (150)</th>
<th>Total (220)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Average heart weight</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>61 cases 452 g</td>
<td>129 cases 464 g</td>
<td>190 cases 460 g</td>
</tr>
<tr>
<td>Female</td>
<td>9 cases 442 g</td>
<td>21 cases 345 g</td>
<td>30 cases 374 g</td>
</tr>
<tr>
<td><strong>Coronary pattern</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dominant right</td>
<td>86%</td>
<td>88%</td>
<td>86%</td>
</tr>
<tr>
<td>Dominant left</td>
<td>14%</td>
<td>9%</td>
<td>10%</td>
</tr>
<tr>
<td>Balanced</td>
<td>5%</td>
<td>3%</td>
<td>4%</td>
</tr>
<tr>
<td><strong>No coronary stenosis</strong></td>
<td>10%</td>
<td>4%</td>
<td>6%</td>
</tr>
<tr>
<td>Stenosis over 75%</td>
<td>90%</td>
<td>96%</td>
<td>94%</td>
</tr>
<tr>
<td>One vessel</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Two vessel</td>
<td>13%</td>
<td>15%</td>
<td>14%</td>
</tr>
<tr>
<td>Three or four vessel</td>
<td>22%</td>
<td>27%</td>
<td>26%</td>
</tr>
<tr>
<td>Old myocardial infarction</td>
<td>49%</td>
<td>42%</td>
<td>44%</td>
</tr>
<tr>
<td>Anterior wall</td>
<td>18%</td>
<td>21%</td>
<td>20%</td>
</tr>
<tr>
<td>Anterior and inferior wall</td>
<td>24%</td>
<td>32%</td>
<td>29%</td>
</tr>
<tr>
<td>Inferior wall</td>
<td>58%</td>
<td>47%</td>
<td>51%</td>
</tr>
<tr>
<td><strong>Acute coronary occlusion</strong></td>
<td>59%</td>
<td>58%</td>
<td>58%</td>
</tr>
<tr>
<td>Single vessel</td>
<td>85%</td>
<td>82%</td>
<td>84%</td>
</tr>
<tr>
<td>Multiple vessel</td>
<td>15%</td>
<td>18%</td>
<td>16%</td>
</tr>
<tr>
<td>Ruptured plaque</td>
<td>69%</td>
<td>48%</td>
<td>56%</td>
</tr>
<tr>
<td>Thrombosis</td>
<td>22%</td>
<td>38%</td>
<td>32%</td>
</tr>
<tr>
<td>Intramural hemorrhage</td>
<td>9%</td>
<td>10%</td>
<td>10%</td>
</tr>
<tr>
<td>Embolus</td>
<td></td>
<td>4%</td>
<td>2%</td>
</tr>
<tr>
<td><strong>Acute myocardial infarction</strong></td>
<td>27%</td>
<td>27%</td>
<td>27%</td>
</tr>
<tr>
<td>Anterior wall</td>
<td>11%</td>
<td>28%</td>
<td>22%</td>
</tr>
<tr>
<td>Anterior and inferior wall</td>
<td>11%</td>
<td>12%</td>
<td>12%</td>
</tr>
<tr>
<td>Inferior wall</td>
<td>78%</td>
<td>60%</td>
<td>66%</td>
</tr>
<tr>
<td>Very fresh (1 day)</td>
<td>16%</td>
<td>18%</td>
<td>17%</td>
</tr>
<tr>
<td>Fresh (1–3 days)</td>
<td>36%</td>
<td>39%</td>
<td>39%</td>
</tr>
<tr>
<td>Recent (3–7 days)</td>
<td>16%</td>
<td>18%</td>
<td>17%</td>
</tr>
<tr>
<td>Organizing (1 week)</td>
<td>32%</td>
<td>25%</td>
<td>27%</td>
</tr>
</tbody>
</table>

*Comprised of 52 with VF and 18 with other terminal rhythms.

2. These lesions were proximal in 23 (19 LAD and 4 right). Seventeen (62%) of these subjects had a demonstrable acute occlusion, and five (17%) also had histologic changes of AMI. Hearts with stenosis of a single vessel weighed an average of 371 g.

In 53 subjects (26%), two major vessels had severe old stenosis — total occlusion in 14. Acute coronary lesions were found in 28 (53%) of these and AMI in 11 (21%). These heart weights averaged 429 g.

In 125 subjects (60%), severe old stenosis involved three or four vessels — total occlusion in 56. Acute coronary lesions were found in 82 (65%) in this group and AMI in 45 (34%). The weight of these hearts averaged 459 g.

An old MI was present in 97 subjects (44%), and these scars were distributed equally between the anterior and inferior walls.

Discussion

Although much attention has been given to the problem of prehospital SCD,1-4, 46-57 several important areas remain poorly defined. These include: (1) determination of the detailed events preceding SCD, including possible warning symptoms and their temporal relationship to SCD; (2) elucidation of the physiologic mechanisms present at the time of collapse and SCD; and (3) delineation of the pathophysiologic changes involved in SCD, including the role of acute coronary and myocardial lesions.

The present study, as well as others, shows that the majority of prehospital SCDs have past histories of cardiovascular disease, particularly old MI, angina pectoris, and hypertension, and two thirds have chest pain and dyspnea within four weeks of death. However, these symptoms are new or different...
from prior experience in only one quarter of subjects; therefore, in only a minority could they warn a knowledgeable person to seek appropriate medical help. As shown in the present and other studies, many SCDs consult physicians shortly before death; in fact, in 17% of our subjects a physician was actually consulted for new or changing chest pain and/or dyspnea. Those with preceding symptoms had more frequent acute myocardial and coronary changes than those without symptoms, and in addition, in the large majority of those with histologic changes of AMI, these changes were between one day and several weeks of age. By history as well as autopsy exam, perhaps as many as one quarter of SCDs actually have recent AMI. An educational program directed to the community, the physician, and particularly to the high-risk patient could bring some of those who actually die with recent myocardial infarction to earlier medical attention.

One quarter of SCD victims reported new or changing chest pain or dyspnea beginning on the day of SCD or VF and preceding it by a mean of 3½ hr. Again, educational programs could reduce delays in seeking help and could lead to earlier hospitalization and evaluation in many.

One quarter of SCDs studied had only very acute symptoms which preceded arrest by less than 30 min. It is possible that lay educational programs stressing rapid entry into emergency medical systems could bring earlier care to such subjects. It is conceivable as well that self- or lay-administered intramuscular drugs, as recently discussed, could also benefit some in this group.

One half of SCDs occur with essentially no warning. In these, collapse is instantaneous or within 1 min of symptoms, and on arrival of rescue, VF is present in the majority. These victims can be helped acutely only by trained rescue units. The percentage surviving prehospital VF is still small, but it is likely that with educational efforts directed at more rapid recognition of arrest, earlier CPR by lay bystanders, and more rapid summons of rescue, survival will improve. Our study population was selected according to both data availability and the presence of a follow-up diagnosis; for this reason the above presence, frequency, and duration of symptoms preceding SCDs may differ from a true cross-section of the entire SCD population.

Details of the acute circumstances and events surrounding SCDs have been reported by others and support our failure to find specific precipitating factors.

As recently reviewed, the physiologic antecedents of prehospital VF may be varied and are still largely conjectural, and information concerning rhythms which precede those monitored by rescue workers is not available. On arrival of rescue, however, which was within minutes of witnessed collapse, VF was present in almost three quarters, while mechanisms other than VF were present in 28%. While defibrillation offers help for the former, many in the latter group require a different and broader therapeutic approach.

Much is already known of the pathology of SCD, and although the pathology of VF has received little mention, the present findings show no significant pathologic differences between monitored and unmonitored SCDs regardless of the terminal rhythm, although too few subjects with rhythms other than VF were studied to describe this latter group definitively.

The reported frequencies of acute coronary lesions in SCD vary from 13 to 55% and thus are comparable to our finding of 58%. In all studies, the frequency is affected greatly by study methodology. Descriptions of types of acute coronary lesions in SCD have been reported. The present finding of acute thrombosis without evident disruption of the intimal lining in one third of those with acute occlusion and 19% of all SCDs suggests a possible prophylactic role for platelet active substances including aspirin, clofibrate, or dipyridamole and the need for prospective evaluation. While it is possible that the present methodology of 1 mm sectioning of acute coronary segments may miss occasional intimal disruptions and therefore may overestimate the frequency of thrombosis in SCD, it is unlikely that large numbers of significant intimal breaks are missed. The more frequent finding of ruptured plaque seems beyond present medical management short of long-term prevention of coronary atherosclerosis which is the most common denominator in SCD pathology. There has been controversy in the literature concerning the frequency of thrombosis as well as plaque rupture in SCD with various groups reporting markedly different findings. Our data indicate that both lesions are found in SCDs.

Single vessel lesions in autopsied SCDs were rare (13%). In these, however, the LAD or left main were involved in three quarters, thus strongly implicating these vessels in SCD in hearts with single vessel disease. In hearts with multivessel disease, however, with extensive collateralization and prior infarction, there was no preferential location of acute or chronic vascular lesions.

By conventional histologic techniques, the incidence of AMI in prehospital SCD has been reported in from 10 to 47% of SCDs and again is critically dependent on examining...
methodology. Our autopsy finding of 27% is well within this range. Standard histologic techniques underestimate the true frequency of AMI because death occurs too rapidly to allow evolution of detectable lesions which generally require from 8 to 12 hr.

The ECG diagnosis of myocardial change in defibrillated survivors, "would be SCDs," provides a more accurate estimate of the incidence of acute myocardial involvement in SCD than does standard histology, because defibrillation reverses the otherwise terminal course of VF and permits observation of myocardial changes that would otherwise be undetectable. In these patients acute myocardial change, whether AMI or ischemia, was present in almost three quarters and similar figures have been reported in similar patients by others.62 While it is possible that in some of these patients acute myocardial changes evolved secondary to arrest and resuscitation, it seems likely that this estimate is actually quite accurate as very similar frequencies of acute myocardial changes have been reported in autopsied SCDs using highly sensitive histochemical techniques63 as well as potassium-sodium ratios64, 65 which are able to detect very early myocardial changes.

Little attention has been given to the location of acute myocardial changes in SCD or in VF, yet the location of these lesions provides insight into the initiation of VF in SCD. The majority of myocardial changes detected in defibrillated survivors involved the anterior wall in contrast to the predominantly inferior wall distribution detected historically in SCDs. This disparity suggests that anterior wall lesions precipitate VF and SCD too rapidly to allow histologic detection, and only if defibrillation permits survival do the greater number of acute myocardial changes and the anterior wall predominance of these lesions in VF and SCD become evident. This relationship of anterior wall lesions to rapid VF has also been described by others64 and suggests the need for evaluation of intensive VF prophyllaxis in patients with compromise of the anterior wall vasculature, particularly when associated with new or changing symptoms.

The relative frequency of acute coronary and myocardial changes in mildly compared to severely diseased coronary systems also permits speculation concerning the propensity of these subjects to develop rapid VF and SCD. The frequency of acute coronary occlusion remained nearly constant regardless of the extent of pre-existing coronary disease. In contrast, the frequency of histologically detected AMI in patients with stenosis of three and four vessels was twice that of those with disease of a single vessel (34% compared to 17%). This disparity suggests that hearts with less extensive disease tend to fibrillate more rapidly and cause SCD without detectable H and E changes, whereas hearts with extensively diseased coronary systems do not fibrillate as acutely and permit survival at least long enough for evolution of histologically evident AMI. Others have also found that subjects with more frequent past history of infarction survive their subsequent acute attacks longer.66

In summary, SCDs can be subdivided. The first group consists of those with recent myocardial infarction. They have frequent prodromal symptoms and could be helped by educational programs. Patients in the second group have only myocardial ischemia which is not detected by conventional histologic techniques in actual SCDs, but which is evident on ECG evaluation in defibrillated survivors. In these the anterior wall is most often involved. The third group contains those subjects with no detectable acute myocardial change. The majority of these latter have no warning symptoms and could only be helped by rescue efforts. Finally, when SCDs are monitored acutely, VF is present in most within minutes of collapse.

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