Accuracy and Impact of Presumed Cause in Patients With Cardiac Arrest

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Background—International guidelines recommend differentiation between cardiac and noncardiac causes of cardiac arrest. The aim of this study was to find the rate of agreement between primarily postulated and definitive causes of cardiac arrest.

Methods and Results—We retrospectively analyzed the primarily presumed cause of cardiac arrest as determined by the emergency room physician on admission in all patients admitted to the emergency department of one urban tertiary care hospital. This was compared with the definitive cause as established by clinical evidence or autopsy. Within 4 years, the initially presumed cause was unclear in 24 (4%) of 593 patients. In the remaining 569 patients, the presumed cause was correct in 509 (89%) and wrong in 60 (11%) cases. Cardiac origin was presumed in 421 (71%) and the definitive cause in 408 (69%) cases. Noncardiac origin was presumed in 148 (25%) and the definitive cause in 185 (31%) patients. Presumed cardiac cause was sensitive (96%) but less specific (77%). Noncardiac causes such as pulmonary embolism, cerebral disorders, or exsanguination were those most frequently overlooked. Asystole occurred significantly more often in patients in whom presumed cause remained undetermined or differed from the definitive cause.

Conclusions—Cause of cardiac arrest is not as easily recognized as anticipated, especially when the initial rhythm is different from ventricular fibrillation. This might affect comparability of study results, therapeutic strategies, prognosis, and outcome. Patients in whom the presumed cause was confirmed as being correct had significantly better survival and neurological outcome. (Circulation. 1998;98:766-771.)

Key Words: resuscitation ■ epidemiology ■ heart arrest ■ pathology

To ensure comparability of data regarding out-of-hospital cardiac arrest, a uniform reporting style of gathering and presenting such material, the Utstein guidelines have been published. Since 1990 most scientific publications in the field of resuscitation adhere to this de facto standard. In this protocol the differentiation between cardiac and noncardiac causes is obligatory.

The guidelines were developed with the fact in mind that most out-of-hospital cardiac arrests are of cardiac origin. On the other hand, cardiac arrest of noncardiac cause was assumed to be “often obvious and easy to determine,” for example, drowning, drug overdose, and so on. Accordingly, data from those patients who presumably have cardiac arrest from any other cause should not be used for comparison with those whose arrest was of cardiac origin.

Experience shows that the first presumed cause of cardiac arrest is sometimes wrong. This may be explained by the fact that at the time of emergency cardiac care, very little information regarding the patient’s medical history is available. Therefore, any assumption of the cause of cardiac arrest should rather be regarded as a working hypothesis.

To determine the accuracy of the presumed cause in patients with cardiac arrest, we analyzed in a retrospective study the primarily presumed cause of cardiac arrest. This was determined by the emergency room physician on admission in all patients brought to the emergency department of one urban tertiary care hospital within July 1991 and July 1995. We then tried to analyze the cases in which the presumed cause was wrong and to assess the reasons for such misinterpretations. We also tried to find out whether this makes a difference in mortality rate and cerebral outcome and attempted to find common indicators that might function as a warning signal in such cases.

Methods

Between July 1991 and July 1995, all patients admitted to the Department of Emergency Medicine of the University Hospital of Vienna after either in-hospital or out-of-hospital cardiac arrest were documented on arrival according to a specific protocol (Utstein Style). Patients with in-hospital cardiac arrest include mainly patients from our department. In addition, because beds in the intensive care units (ICU) are limited, almost all those patients with cardiac arrest in one of the regular wards or outpatient departments were transferred to the ICU immediately.
are first brought to the emergency department for stabilization before
being admitted to an ICU.

The study procedures followed were in accordance with the ethical
standards of the responsible committee on human experimentation
and with the Helsinki Declaration of 1975, as revised in 1983.

According to the criteria of the American Heart Association,
cardiopulmonary resuscitation was defined as sudden collapse followed by loss of
consciousness and absence of both spontaneous respiration and pulse that required cardiopulmonary resuscitation.\textsuperscript{3,4} Acute care including
basic and advanced life support performed by the Vienna Ambulance Service and in-hospital emergency medical personnel was in accordance with international guidelines.\textsuperscript{3,4} The Vienna Emergency Medical System is based mainly on the municipal
ambulance service founded 1881. Medical emergencies are reported over one emergency telephone number (144) to the central dispatch center, received by a medical technician, processed by computer, and passed on to the dispatcher, who assigns a mobile intensive care unit staffed by a physician and two emergency medical technicians. Patients with cardiac arrest are usually successfully resuscitated or pronounced dead by the ambulance physician. Only patients with special problems such as hypothermia are transported to the hospital under active cardiopulmonary resuscitation.

At the emergency department, a physician stated the presumed
cause of cardiac arrest mainly on the basis of the initial perceptions
of the ambulance physician. In addition, if available, the patient
history, including previous physician reports, bystander information,
preclinical run sheets, and ECG was used. He had to select one of the
following possible causes: cardiac, respiratory, cerebral, near
shock, hypothermia, drug overdose, metabolic, trauma, sepsis, exanguination, others, or unknown cause of cardiac arrest.

Respiratory cause includes pulmonary embolism as well as as-
phyxia by upper airway obstruction, status asthmaticus, and pneu-
monia. Cerebral cause includes cerebrovascular accident and intra-
cerebral or subarachnoidal hemorrhage. Atraumatic exanguination
includes gastrointestinal bleeding as well as ruptured aortic aneur-
sym. No patients with trauma are managed in our department
because there is an independent Department of Traumatology for
those cases.

Definitive cause of cardiac arrest in nonsurvivors was determined
from autopsy in almost all cases. Cardiac dissection was performed
according to standard procedures. The coronary artery tree was examined by transverse cuts at 0.25-cm intervals to localize signif-
ificant stenosis of coronary arteries as well as thrombotic events. Ischemic damage of the myocardium was evaluated by macroscopic appearance of coagulation necrosis and by staining with nitroblue
tetrazolium. As required by law, persons without relevant medical
history before cardiac arrest were examined by the Department of
Forensic Pathology. In the survivors, we examined hospital records
including the patient’s history, clinical examination, and additional
investigations after admission, for example, laboratory examinations,
repeated 12-lead ECGs, radiographs and computed tomography
scans, abdominal ultrasound, transhoracic and transesophageal
echocardiography, selective coronary angiography, and electrophys-
iological stimulation study. Acute myocardial infarction was diag-
nosed by 12-lead ECGs showing ST-segment elevation $>0.2$ mV in
two precordial leads or $>0.1$ mV in 2 limb leads and subsequent
development of Q waves within the hospital stay. All surviving
patients with cardiac cause and good neurological outcome under-
went coronary angiography. Additionally, patients in whom a primary
rhythm disorder was suspected underwent electrophysiological examination.

We assigned the patients to one of two groups: The first group
(group 1) consisted of the patients in whom the presumed cause was
later confirmed as being correct. The second group (group 2) consisted of patients in whom the presumed cause was not deter-
mined by the emergency physician or differed from the definitive
cause. The patients in these two groups were compared for common

denominators such as sex; age; location of cardiac arrest (out-of-
hospital versus in-hospital); initial ECG rhythm observed by any
rescue worker, distinguishing between ventricular fibrillation, pulse-
less ventricular tachycardia, asystole, or pulseless electric activity/
electromechanical dissociation (PEA/EMD); lack or presence of
bystander cardiopulmonary resuscitation; and estimated time until
restoration of spontaneous circulation. For the time interval from
cardiopulmonary resuscitation to start of basic and/or advanced life support, we
presumed sufficient systemic blood flow to be absent (no flow). The
time interval from start of life support until return of spontaneous
circulation we presumed to be representative for reduced systemic
blood flow (low flow).

Mortality and cerebral function were assessed prospectively on
arrival and at regular intervals within 6 months after return of
spontaneous circulation in turns of the cerebral performance catego-
ries (CPC 1 to 5).\textsuperscript{5} Definitions are CPC 1, conscious and alert with
normal function or only slight disability; CPC 2, conscious and alert
with moderate disability; CPC 3, conscious with severe disability;
CPC 4, comatose or in a persistent vegetative state; and CPC 5, brain
death. The best CPC score achieved within 6 months was used for
the analysis. A CPC score of 1 or 2 was considered as favorable and
a CPC score of 3, 4, or 5 was considered as unfavorable functional
neurological outcome. The investigator assessing the CPC score was
blinded to data concerning resuscitation as well as to other medical
data.

According to the Utstein Style, data are expressed as median and
interquartile range (IQR). Percentages were determined for dichot-
omous variables. We used the Mann-Whitney $U$ test for the com-
parison of groups and the $\chi^2$ test for comparison of proportions. We
reviewed the accuracy of presumed cause of cardiac arrest by
comparing the number of cases in which presumed cause was correct
or wrong, calculating sensitivity and specificity of each factor.\textsuperscript{6} A
value of $P<0.05$ was considered statistically significant.

\textbf{Results}

Of the 612 patients admitted between July 1991 and July
1995, 19 had to be excluded because we were unable to
establish any cause of cardiac arrest (Figure). Of the remain-
ing 593 patients, 193 (33\%) were female, the median age was
62 years (IQR 51 to 71), and cardiac arrest occurred out-of-
hospital in 424 (72%) patients. Within the observation period of 6 months after cardiac arrest, 435 (74%) of 593 patients died. Autopsy was performed on 398 patients (91%), 40 of those at the Department of Forensic Pathology and the rest at the Department for Clinical Pathology.

The emergency physician stated the reason for the cardiac arrest to be undetermined in 24 (4%) of 593 cases (Figure). We were able to establish a definitive diagnosis in all of these patients. The cause was of cardiac origin in 11 patients, cerebral in 4, and respiratory in another 4 patients. In 3 patients the underlying cause was metabolic, in 1 patient it was sepsis, and in 1 it was exsanguination.

In the remaining 569 (96%) of 593 patients, a presumed cause of cardiac arrest could be stated by the emergency physician. The presumed causes of cardiac arrest and their specific subcategories are listed together with the definitive cause in Table 1. Noncardiac causes were primarily underestimated. The presumed cause was correct in 509 (89%) cases and wrong in 60 (11%) cases. Data for subgroups and sensitivity and specificity of presumed cause in patients with cardiac arrest of cardiac or noncardiac origin are shown in Table 2.

Cardiac arrest was described to be of cardiac origin in 421 of these patients. This assumption was correct in 382 and wrong in 39 patients. Noncardiac origin was suspected in 148 patients. This was correct in 127 and wrong in 21 patients (Figure and Table 2).

The definitive cause was of cardiac origin in 408 (69%) and noncardiac origin in 185 (31%) patients (Table 1). Of those 408 patients, acute myocardial infarction was the most common cardiac cause of cardiac arrest [255 (63%)]. Of the remaining 153 patients (37%), sudden cardiac death was the result of a primary electric event in 116 (28%) patients and the result of hypoxia caused by cardiac pulmonary edema in 37 (9%) patients. In this group of 153 patients without acute myocardial infarction, coronary artery disease was found in 50 (12%), dilated cardiomyopathy in 33 (8%), ischemic cardiomyopathy in 28 (7%), and hypertensive cardiomyopathy in 28 (7%), and hypertensive cardiomyopa-thy in 11 (3%) patients. Other cardiac causes were established in 31 (8%) cases, including 4 with idiopathic ventricular fibrillation and 2 with complete heart block degenerating into ventricular fibrillation.

We grouped the patients who had a cardiac arrest of noncardiac origin (n=185) into subcategories according to the most frequent cause, as presented in Table 1. Of 59 patients with cardiac arrest of respiratory origin, 27 had cardiac arrest after pulmonary embolism (23 of them had deep vein thrombosis) and 12 after status asthmaticus. Of 27 patients with cardiac arrest caused by exsanguination, 17 had ruptured aortic aneurysm (13 abdominal and 4 thoracic).

Presumed cardiac cause was highly sensitive (95%) but less specific (77%). In comparison, specificity for the different noncardiac causes was 98% and better. Most often, presumed diagnoses failed to consider exsanguination, cerebral disorder, respiratory disease, sepsis, and metabolic disorder as cause of cardiac arrest. In Table 3, we show the distribution of erroneously presumed causes within the definitive causes. We found that in 39 cases in which a cardiac origin had been primarily suspected, 14 patients had cardiac arrest from respiratory origin (10 pulmonary embolism), 10 because of exsanguination after a ruptured aortic aneurysm, 17 had ruptured aortic aneurysm (13 abdominal and 4 thoracic).

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We then compared group 1 (n=509), which consisted of the patients in whom the presumed cause was later confirmed as being correct, with group 2 (n=84), which consisted of 24 patients with undetermined and 60 patients with erroneous presumed first diagnosis regarding various parameters at the time of resuscitation. We did not observe a statistically significant difference between the two groups regarding the location of cardiac arrest (out-of-hospital versus in-hospital), whether or not the cardiac arrest was witnessed, the presence or the lack of bystander CPR, and the sex of the patients, as shown in Table 4. When comparing the most frequently documented initial rhythms, we found a significant difference between the two groups for ventricular fibrillation and asystole as described in Table 4. In group 2, asystole was found as the initial rhythm in 50% of the cases compared with 20%
in group 1. The times of no flow and low flow were not different in the two groups. For all groups, the median no-flow time was 1 minute (IQR 0 to 7 minutes) and the low-flow time was 10 minutes (IQR 3 to 20 minutes).

The rate of no restoration of spontaneous circulation was significantly higher in group 2 (34%) than in group 1 (20%), as seen in Table 5 (P < 0.003). Of all patients who achieved restoration of spontaneous circulation initially, 306 (52%) died within 6 months, with a median survival of 1 day (IQR 1 to 8) [group 1, 2 days (IQR 1 to 8) and group 2, 7 days (IQR 2 to 35)]. Out of all patients, 158 (26%) were discharged alive. When comparing the two groups, group 1 had a significantly better outcome (P < 0.001). Of all surviving patients, 138 patients (87%) had a good neurological outcome with a CPC score of 1 or 2. Those patients in whom the presumed cause was later confirmed as being correct had a significantly better neurological outcome than patients with undetermined or erroneous presumed first diagnosis (< 0.001) (Table 5).

### Discussion

Comparing the initial assumption regarding the cause of cardiac arrest with the definitive diagnosis, the first presumed cause of cardiac arrest is disproved in 11% of the studied cases. In another 4% of the patients the emergency physician could not reach a conclusion regarding the cause of cardiac arrest. In our study, cardiac arrest of cardiac origin is most common (69%) and is recognized with a sensitivity of 95%. This might be explained by the fact that in most patients with ventricular fibrillation as the first documented rhythm, a cardiac cause is the most likely cause of cardiac arrest. The relatively low specificity of 77% reflects those patients who were erroneously assumed to have suffered cardiac arrest of cardiac origin, although it was actually of noncardiac origin. When we examined the real cause for this group of patients, we found that especially pulmonary embolism, ruptured aortic aneurysm, and neurological disorders such as intracerebral and subarachnoidal hemorrhage were erroneously classified as cardiac arrest of cardiac origin. Silvast and Kuisma and Alsspåå came to similar proportions in their studies. The reason might be that although these diseases are not uncommon, the patients rarely have cardiac arrest as first manifestation. Because of this, there might be a lack of experience in interpreting the possible prearrest clinical symptoms corre-

<table>
<thead>
<tr>
<th>TABLE 3. Distribution of Erroneous Presumed Cause Within Definitive Cause of Cardiac Arrest</th>
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<tbody>
<tr>
<td>Definitive Cause</td>
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<tr>
<td>-----------------</td>
</tr>
<tr>
<td>Cardiac</td>
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<tr>
<td>Respiratory (PE)</td>
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<tr>
<td>Cerebral</td>
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<tr>
<td>Drug overdose</td>
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<tr>
<td>Exsanguination</td>
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<td>Metabolic</td>
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<td>Sepsis</td>
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<tr>
<td>Near drowning</td>
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<tr>
<td>Others</td>
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PE indicates pulmonary embolism.

<table>
<thead>
<tr>
<th>TABLE 4. Factors at Time of Resuscitation</th>
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<tr>
<td>Factors</td>
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<tr>
<td>---------</td>
</tr>
<tr>
<td>Male</td>
</tr>
<tr>
<td>Female</td>
</tr>
<tr>
<td>Out-of-hospital arrest</td>
</tr>
<tr>
<td>In-hospital arrest</td>
</tr>
<tr>
<td>Witnessed arrest</td>
</tr>
<tr>
<td>Nonwitnessed arrest</td>
</tr>
<tr>
<td>Bystander CPR</td>
</tr>
<tr>
<td>No bystander CPR</td>
</tr>
<tr>
<td>Ventricular fibrillation</td>
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<tr>
<td>Asystole</td>
</tr>
<tr>
<td>PEA/EMD</td>
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<tr>
<td>Ventricular tachycardia</td>
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</tbody>
</table>

Values are n (%).
*Patients in whom presumed cause was later confirmed as being correct.
†Patients with undetermined or erroneous presumed first diagnosis.

<table>
<thead>
<tr>
<th>TABLE 5. Outcome of Cardiac Arrest Within 6 Months</th>
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<tbody>
<tr>
<td>Outcome</td>
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<tr>
<td>---------</td>
</tr>
<tr>
<td>Not resuscitated</td>
</tr>
<tr>
<td>Dead within 6 months</td>
</tr>
<tr>
<td>Alive</td>
</tr>
<tr>
<td>Cerebral performance category 1/2 (of alive patients)</td>
</tr>
</tbody>
</table>

Values are n (%).
*Patients in whom presumed cause was later confirmed as being correct.
†Patients with undetermined or erroneous presumed first diagnosis.
directly. In addition, these patients may have symptoms quite similar to cardiac arrest of cardiac origin.

Silfvast\(^7\) has disproved cardiac origin in 25 (31\%) of 80 patients who had cardiac arrest of presumed cardiac origin. A previous study by Kuisma and Alsspa¨a¨\(^7\) showed that 36\% of both traumatic and nontraumatic out-of-hospital cardiac arrests assumed to be of cardiac origin were diagnosed to be really of noncardiac origin after in-hospital investigations or autopsy. Of our patients, only 11\% were misdiagnosed initially. This difference from the above-mentioned studies could be explained by including in our study patients with all causes of cardiac arrest and primary in-hospital or out-of-hospital successful restoration of spontaneous circulation in almost all cases. When no return of spontaneous circulation could be achieved before hospitalization, establishing a correct diagnosis is in itself more difficult because these patients usually do not enter the hospital at all.

We have shown that in half of the cases in which the emergency physician was wrong in his assumption about the cause of cardiac arrest, the initial ECG rhythm was asystole. This problem is enhanced when there are no relatives who can be questioned about the prior medical history of those patients. Because asystole is the common terminal rhythm of all causes of cardiac arrest and is often the result of the delay before resuscitation, it is evident that a diagnosis is inherently more difficult. There is also no way of knowing whether in the case of asystole the rhythm initially was ventricular fibrillation that deteriorated over time or whether it was an EMD/PEA that finally converted from mechanical to electrical asystole. In addition, it might be that because initial resuscitation often fails and no restoration of spontaneous circulation can be achieved,\(^8\) the emergency physician has less time to reevaluate the diagnosis and some diagnostic procedures cannot be performed at all. This further enhances the likelihood of a mistaken first assessment as to the cause of cardiac arrest. We therefore suggest that asystole as first rhythm should lead to a high degree of suspicion as to whether the patient has really arrested because of cardiac cause, or whether there is any clinical evidence that a noncardiac reason might be present.

The rate of correct diagnosis was much higher when the initial rhythm was ventricular fibrillation or EMD/PEA. It is known that ventricular fibrillation is the most common rhythm in cardiac arrest of cardiac origin,\(^9\) whereas EMD/PEA is associated with pulmonary embolism\(^10\) and ruptured or dissecting aortic aneurysm with cardiac tamponade.\(^2\)

When looking for other factors influencing resuscitation such as age, sex, absence or presence of basic life support, location (in-hospital versus out-of-hospital) of cardiac arrest, as well as the times of no flow and low flow, we did not find significant differences between the two groups of patients when the presumed cause was later confirmed as being correct and patients with undetermined and erroneous presumed first diagnosis. Thus it can be presumed that none of these factors had any influence on sensitivity or specificity for the initial diagnosis.

Despite massive efforts, the rate of survival after cardiac arrest has not improved significantly in the last years. Studies have analyzed a multitude of factors influencing the outcome of resuscitation.\(^11,12\) For obvious reasons, the outcome after cardiac arrest as shown in our data does not adhere to the Utstein template. Therefore it should not be compared uncritically with other published data. The neurological outcome depends largely on the arrest time. We thought that the poor outcome in the group with wrong presumed cause might be explained partly by the fact that 50\% of those patients were asystolic and did not achieve restoration of spontaneous circulation as often as the patients in the other group. However, in a post hoc analysis, we noticed two things. The outcome is worse when the first presumed diagnosis is wrong, irrespective of the initial rhythm. If misclassification occurs, mortality rate is almost doubled. In addition, our data once more confirm the well-known fact that patients with ventricular fibrillation have a much better chance to survive than those with asystole or EMD/PEA (Table 6).

The question remains whether reducing the cardiopulmonary resuscitation time by establishing the correct cause of cardiac arrest might improve the primary resuscitation rate and the long-time prognosis of those patients. Therefore we suggest that not only improved diagnostic procedures but also more concern by the treating physicians with regard to the possible definitive cause of cardiac arrest may help to identify the underlying cause in at least some of these patients, thereby allowing earlier origin-dependent advanced treatment.

Unfortunately, there are no reports comparing the efficacy of distinct treatment on the basis of the underlying pathology in cardiac arrest. The knowledge of the origin of the cardiac arrest might directly influence the cause-specific treatment and, because of this, indirectly influence the outcome. For example, fulminant pulmonary embolism associated with cardiac arrest has an extremely high mortality rate.\(^13\) However, previous studies\(^14\) demonstrate that a prompt treatment

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**TABLE 6. Outcome of Cardiac Arrest Grouped According to Initial ECG and Misclassification of Cause**

<table>
<thead>
<tr>
<th></th>
<th>Ventricular Fibrillation (n=305)</th>
<th>EMD/PEA (n=136)</th>
<th>Asystole (n=128)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Correct</td>
<td>Wrong</td>
<td>Correct</td>
</tr>
<tr>
<td>Survivors, %</td>
<td>272</td>
<td>11</td>
<td>63</td>
</tr>
<tr>
<td>Presumed cardiac</td>
<td>159 (58)</td>
<td>2 (18)</td>
<td>8 (13)</td>
</tr>
<tr>
<td>Presumed noncardiac</td>
<td>17</td>
<td>5</td>
<td>56</td>
</tr>
<tr>
<td>Survivors, %</td>
<td>6 (35)</td>
<td>1 (20)</td>
<td>8 (14)</td>
</tr>
</tbody>
</table>

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\(^1\) F. Silfvast: Circulation 103, 2047-2051 (1996).


\(^3\) M. Silfvast: Circulation 103, 2047-2051 (1996).


of systemic thrombolysis or embolectomy, even under ongoing resuscitation, may decrease mortality rates. While preoperative cardiac arrest in patients with ruptured or dissecting aortic aneurysm is associated with a high mortality rate, prompt treatment results in significantly better survival. Early sonographic diagnosis of ruptured aortic aneurysm is important, and the patient may be saved even if this necessitates cardiopulmonary resuscitation while on the way to the operating theater.

There is enough evidence that the number of patients discharged from the hospital after cardiac arrest depends in large part on the way that judicious therapeutic decisions are made on the basis of presumed cause. The importance of prehospital medical history-taking, examination, and resuscitation by ambulance physicians in this respect cannot be overestimated. Our data corroborate the necessity of using all available means of proving or—even more important—excluding a specific diagnosis by the emergency physician, allowing specific treatment to be initiated. He or she must take into account the prearrest information as well as the present status and the results of clinical monitoring, laboratory examinations, bedside echocardiography, and abdominal ultrasound. These should be performed as soon as feasible to establish a correct diagnosis, possibly even in ongoing resuscitation situations. This implies that after the receipt of the message that the emergency department is about to receive a patient after cardiac arrest, the necessary equipment already will be at the bedside, ready for use on arrival of the patient.

In conclusion, in a significant number of cases particularly of noncardiac cause, origin of cardiac arrest is not as easily recognized as initially anticipated. Asystole as first-recorded ECG rhythm and noncardiac causes such as pulmonary embolism, ruptured aortic aneurysm, and cerebral disorders frequently led to erroneous diagnosis. This might affect comparability of study results, therapeutic strategies, prognosis, and outcome of patients after cardiac arrest. Those patients in whom the presumed cause was later confirmed as being correct had a significantly better survival rate and neurological outcome than patients with undetermined or erroneous presumed first diagnosis. We therefore believe that it is of paramount importance to actively procure all relevant information from bystanders, relatives, and emergency medical service personnel, even before arrival of the patient in the emergency department. In addition, early availability of crucial medical information and past medical history should be provided by an information forwarding service through the dispatch center. In the emergency department itself, all diagnostic equipment should be ready on arrival of the patient, and diagnostic procedures should be performed as soon as feasible.

Acknowledgment

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References

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