The Accuracy of an Out-of-Hospital 12-Lead ECG for the Detection of ST-Elevation Myocardial Infarction Immediately After Resuscitation

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Study objective: Severe myocardial ischemia is the leading cause of arrhythmic sudden cardiac death. It is unclear, however, in which percentage of patients sudden cardiac death is triggered by ST-elevation myocardial infarction (STEMI) and whether the diagnosis of STEMI can be reliably established immediately after resuscitation from out-of-hospital sudden cardiac death.

Methods: A 12-lead ECG was registered after return of spontaneous circulation after cardiac arrest. After hospital admission, further ECG, creatine kinase MB, and troponin measures; results of coronary angiograms; and autopsies were evaluated to confirm the definitive diagnosis of STEMI.

Results: Seventy-seven patients were included in our study (67% men, age 64 [14 to 93] years). STEMI was diagnosed in 44 patients. The diagnosis of myocardial infarction was confirmed in 84% of the 77 patients who survived to hospital admission. The sensitivity of the out-of-hospital ECG was 88% (95% confidence interval [CI] 74% to 96%), the specificity 69% (95% CI 51% to 83%), the positive predictive value 77% (95% CI 62% to 87%), and the negative predictive value 83% (95% CI 64% to 87%). The accuracy of the out-of-hospital ECG and that registered on admission was the same.

Conclusion: The diagnosis of STEMI can be established in the field immediately after return of spontaneous circulation in most patients. This may enable an early decision about reperfusion therapy, ie, immediate out-of-hospital thrombolysis or targeted transfer for percutaneous coronary intervention. [Ann Emerg Med. 2008;52: 658-664.]

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INTRODUCTION

Background

It is estimated that about one third of all patients experiencing an acute myocardial infarction die out of hospital in the field.[1-3] Often, the so-called sudden cardiac death is the first and only symptom of coronary heart disease. Substantial progress has been made in the treatment of acute myocardial infarction in recent years. Different reperfusion strategies, newly developed antiplatelet agents, antithrombins, and improved preventive therapies have reduced inhospital mortality of myocardial infarction. However, there has been almost no reduction of the out-of-hospital case-fatality rate.[4,5]

The prognosis of out-of-hospital cardiac arrest is poor, with a survival rate of less than 10%.[6] Epidemiologic studies on the cause of arrest in patients with sudden cardiac death are difficult because autopsy rates are low in most countries and nearly zero in some countries such as Germany. Ventricular fibrillation, the most common arrhythmia underlying sudden cardiac death in adults, is triggered by cardiac ischemia.[7] Correspondingly, in most patients, sudden cardiac death is caused by severe coronary heart disease.[8-10] However, it is unclear in which percentage of patients sudden cardiac death is actually caused by acute ST-elevation myocardial infarction (STEMI).

Importance

The sensitivity of an out-of-hospital 12-lead ECG for the routine diagnosis of STEMI is about 70%, with an approximately 90% specificity in patients.[11-18] Prolonged ischemia, medication, and electrical countershocks during resuscitation may induce ECG alterations of variable extent, which may reduce its diagnostic reliability.[19,20] ST elevation (or a presumably new left bundle-branch block in symptomatic patients) in 2 or more contiguous leads in the ECG is by definition the principal diagnostic criterion of STEMI.

Editor's Capsule Summary

What is already known on this topic

Cardiac arrest is often caused by myocardial ischemia.

What question this study addressed

Whether or not the ECG obtained immediately postreturn of spontaneous circulation is useful to identify ST-elevation myocardial infarction (STEMI).

What this study adds to our knowledge

The first ECG after return of spontaneous circulation in 77 cardiac arrest patients is a reasonably accurate way to identify STEMI (sensitivity 88%; specificity 69%).

How this might change clinical practice

This result could mean that patients with ECGs suggestive of STEMI after return of spontaneous circulation could receive earlier and more aggressive therapies in the field or the emergency department.

According to the guidelines, reperfusion therapy in STEMI should be initiated as soon as possible after verification of the diagnosis.[21,22]

Goals of This Investigation

To our knowledge, it has not yet been investigated whether it is possible to establish the diagnosis of STEMI with sufficient reliability on the basis of an ECG recorded immediately after the return of spontaneous circulation after out-of-hospital resuscitation from sudden death of assumed cardiac origin. We sought to determine the test characteristics of an out-of-hospital ECG registered immediately after return of spontaneous circulation.

MATERIALS AND METHODS

Study Design

For the purpose of this prospective study, sudden cardiac death was defined as sudden and unexpected circulatory arrest of assumed cardiac origin, irrespective of the duration of preceding symptoms. Patients in whom resuscitation was attempted for clearly noncardiac reasons (eg, trauma, suicide, intoxication) were excluded from the study. If a noncardiac cause for resuscitation was recognized retrospectively, the patient was also excluded from the analysis.

Setting

Consecutive patients were included by the emergency physician–manned mobile ICU and the rescue helicopter stationed at our hospital. Emergency physicians in Germany are mostly specialists in internal medicine or anesthesia who are also trained in intensive care medicine. In the emergency medical services (EMS) of the city of Berlin, in case of a suspected life-threatening condition an emergency medical technician trained in basic life support and use of an automatic external defibrillator is alarmed in parallel with a mobile ICU. The emergency medical technician equipped with defibrillator usually arrives first at the scene after a median alarm-scene interval of 7 minutes. The mobile ICU arrives after a median alarm-scene interval of 11 minutes. A detailed description of the 2-tiered Berlin EMS has been given elsewhere.[23]

Selection of Participants

To be included into the study, the patients had to have spontaneous circulation at least for a few minutes after resuscitation so that a 12-lead ECG could be recorded as soon as possible by means of a CorPuls 08/16 device (Kaufering, Germany). A form was used to record the circumstances of the arrest, the victim's medical history, and character and duration of any specific preceding symptoms (if any). If eyewitnesses were present, the time of collapse was recorded; otherwise, it was estimated according to all other available information. Moreover, because the ECG machines do not offer automated ECG analysis, the emergency physician had to interpret the ECG with regard to the diagnosis of STEMI (and its localization). A second 12-lead ECG was recorded immediately at arrival in those patients who survived to hospital admission.

Methods of Measurement

Corresponding to the guidelines the clinical diagnosis was STEMI if the ECG revealed an ST elevation of greater than or equal to 0.1 mV in at least 2 contiguous limb leads or greater than or equal to 0.2 mV in 2 or more adjacent precordial leads as the basis for decisionmaking in the acute situation. STEMI was not diagnosed if a left bundle-branch block was present. In case of an ST-segment elevation, the sum of ST elevation for leads affected was determined according to the method of Schröder et al.[24] Differences in the ST elevation between the out-of-hospital and the first hospital ECG were calculated and given as percentage resolution of ST elevation.[25]

Outcome Measures

Clinical courses during inpatient treatment, including additional ECGs and results of laboratory tests, were recorded to verify or exclude the diagnosis of myocardial infarction. In agreement with actual definitions, myocardial infarction was defined as follows: an increase of at least 2.5 times the upper limit of normal of the myocardial fraction of the creatine kinase MB level or an abnormal troponin value (T or I, above the undetermined range) according to local definitions, if coronary angiography resulted in the diagnosis of acute myocardial infarction (subtotal or total occlusion of the related artery) or if new ECG signs typical of a developing transmural infarction (new Q waves, R loss) were found, or if available autopsy findings were evaluated in nonsurvivors.[26]

Table 1. Characteristics of the 91 patients.

Characteristics	Included	Excluded
No.	77	14
Women, No. (%)	26 (33)	4 (29)
Age, y, median (IQR)	67(14, 93)	69 (41, 92)
Symptoms reported, No.*	41	10
Symptoms unknown, No.	36	4
Duration of symptoms, min, median (IQR)	10 (5, 38)	9 (4, 41)
Arrest at home, No. (%)	43 (56)	9 (64)
Eyewitnessed arrest, No. (%)	63 (81)	12 (82)
Bystander resuscitation, No. (%)	19 (25)	5 (35)

IQR, Interquartile range.
*Symptoms reported=any symptoms preceding the arrest reported by eyewitnesses.

Primary Data Analysis

All data were analyzed with the statistics program Jump 6.0.2 (SAS Institute, Inc., Cary, NC). The statistical significance of differences was calculated by the Mann-Whitney U test for continuous variables and the χ^2 test for ordinal variables.

Sensitivity Analyses

The sensitivity and specificity, as well as positive predictive value and negative predictive value, were calculated for ECG diagnoses. The 95% confidence intervals were calculated if appropriate. Unless otherwise specified in the text, continuous variables were described by giving the median and the 25th and 75th percentiles.

RESULTS

From June 2002 to August 2004, we observed a total of 808 patients with an out-of-hospital cardiac arrest. Fifteen patients were primarily excluded from the study because of noncardiac causes of arrest (4 drowning, 4 intoxication, 7 trauma). One patient was retrospectively excluded because of diagnosis of intoxication. None of the patients had signs of myocardial infarction during the hospital course. In 362 patients, the resuscitation attempt was unsuccessful, and in 340 patients no resuscitation attempt was undertaken because of definitive signs of death (rigor mortis, postmortem lividity). Ninety-one patients were primarily successfully resuscitated from out-of-hospital sudden cardiac death. Whereas a 12-lead ECG immediately after return of spontaneous circulation could be recorded for all 91 patients, an ECG at hospital admission was available for only 77 patients. Thirteen patients died before admission and 1 survivor had no ECG at admission. The patients' characteristics and circumstances of resuscitation of the included 77 patients and the excluded patients are shown in Table 1. Table 2 shows findings of the out-of-hospital ECG and the ECG at admission.

The median time from symptom onset to collapse was 10 minutes (5 to 38 minutes), as far as information that was available from bystanders. Intervals from collapse to start of cardiopulmonary resuscitation (CPR) varied; in case of bystander resuscitation, the median interval was 2 minutes (1 to 5 minutes), and first-tier emergency medical technician equipped with defibrillator-initiated CPR, within a median of 9 minutes (7 to 11 minutes) after collapse. The second-tier (emergency physician–staffed mobile ICU) arrived 14 minutes (10 to 18 minutes) after collapse. Thus, the median interval from collapse to stable return of spontaneous circulation was 20 minutes (14 to 30 minutes), ie, 16 minutes (7 to 21 minutes) after arrival of the first-tier emergency medical technician equipped with defibrillator (Figure). In 69 patients, first registered underlying arrhythmia was stored in the memory of automated external defibrillators and diagnosed by the emergency physician on scene in the remaining 11 patients (Table 3).

The first 12-lead ECG was recorded at the scene at a median of 7 minutes (2 to 12 minutes) after return of spontaneous circulation, corresponding to a time delay of 32 minutes (25 to

Table 2. Results of the ECG analysis in 77 patients.

Analysis	Out-of-Hospital	ECG at Hospital ECG Admission
Time, min, median (IQR)	32 (25-39)	72 (61-97)
Rhythm, No.		
Sinus rhythm	46	60
Junctional rhythm	14	3
Atrial fibrillation	17	14
RBBB, No.	19	15
LBBB, No.	6	5
STEMI, No.		
AWI	27	28
Non-AWI	15	12
No infarction/uncertain, No.	35	37

Time, Time from collapse to ECG; *RBBB,* right bundle-branch block; *LBBB,* left bundle-branch block; *AWI,* anterior wall infarction.

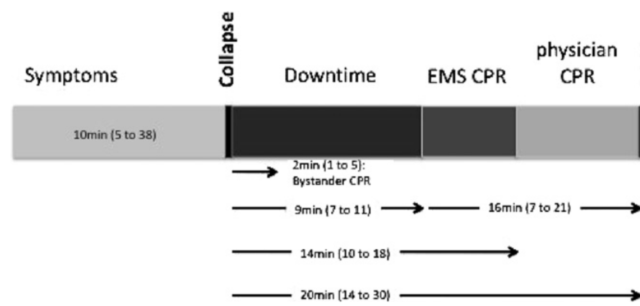

Figure. Time scale of the resuscitated patients (n=77). *ROSC,* Return of spontaneous circulation.

Table 3. First registered dysrhythmia of included patients.

Documented in the automated external defibrillator, No. (%) (n=69)	
Defibrillatable	42 (61)
Not defibrillatable	27 (39)
At arrival of the emergency physician, No. (%) (n=77)	
Ventricular fibrillation	36 (47)
Asystole	20 (26)
Pulseless electrical activity	21 (27)

Table 4. Definitive confirmation of STEMI diagnosis at discharge or death.

Multiple criteria, No. (n=44)	
ECG course typical of myocardial infarction	13
Pathologic increase in creatine kinase MB/troponin (I or T)	35
IRA occlusion according to ECG finding	20
Autopsy result	4
Single criteria, No. (n=4)	
ECG course typical of myocardial infarction	1
Pathological increase in creatine kinase MB/troponin (I or T)	3
IRA occlusion according to ECG finding	0
Autopsy result	0

IRA, Infarction-related artery.

Table 5. Out-of-hospital ECG diagnosis compared with diagnosis on hospital discharge.

	Myocardial Infarction Diagnosis on Hospital Discharge		
Out-of-hospital diagnosis	**Yes**	**No**	
STEMI diagnosis on out-of-hospital ECG	37	5	42
No STEMI diagnosis on out-of-hospital ECG	11	24	29
	48	29	77

Table 6. ECG diagnosis at hospital admission compared with diagnosis on hospital discharge.

	Myocardial Infarction Diagnosis on Hospital Discharge		
Diagnosis at hospital admission	**Yes**	**No**	
STEMI diagnosis on hospital admission ECG	37	3	40
No STEMI diagnosis on hospital admission ECG	11	24	35
	48	27	77

39 minutes) after collapse. The first ECG after hospital admission was recorded at a median of 72 minutes (61 to 97 minutes) after arrest, ie, 47 minutes (35 to 69 minutes) after return of spontaneous circulation. Forty-two of the 77 patients who survived to hospital admission had signs of acute STEMI in the out-of-hospital ECG, and 40 patients in the ECG registered at admission. In 20 patients with signs of an STEMI in the out-of-hospital ECG, aspirin (500 mg intravenously) and heparin (5000 IE) were administered. Additionally, in 17 patients with signs of a STEMI in the first ECG, intravenous thrombolysis was initiated.

Information on the clinical course and discharge diagnosis was available for all 77 patients. In 44 patients, definitive diagnosis of acute myocardial infarction was established (33 with anterior and 11 with nonanterior myocardial infarction) according to multiple diagnostic criteria and in 4 patients relying on only 1 positive criterion (Table 4). In 46 patients, coronary angiography was performed during the clinical course because of suspected acute myocardial infarction. In 31 of these patients (69%), an acute myocardial infarction was confirmed and a percutaneous coronary intervention (PCI) was performed. Of the 17 patients who received thrombolysis before hospital admission, 9 were treated by additional PCI. In all 9 patients, an acute myocardial infarction was confirmed angiographically. In the patients for whom the indication for out-of-hospital thrombolysis had been established because of the ECG tracings, no adverse effects (ie, bleeding, stroke) were observed. Relatively rare diseases, which may mimic a STEMI in the ECG (eg, aortic dissection), were not observed in the group of patients treated by thrombolysis.

A total of 35 patients (39%) survived to hospital discharge, 14 of the 52 patients (27%) who initially had asystole or pulseless electrical activity and 21 of the 39 patients (54%) who initially had ventricular fibrillation.

The initial emergency physicians' diagnosis on the scene was STEMI in 42 of the 77 patients, the diagnosis being confirmed by the clinical course in 37 of them and not verified in 5 patients (pulmonary artery embolism: 1 patient; dilative cardiomyopathy/acute myocarditis: 2 patients; chronic occlusion of a coronary artery: 2 patients). In 35 patients, the ECG registered on the scene did not fulfill the criteria for a STEMI diagnosis. Twenty-four of these 35 patients did not develop clinical signs of myocardial infarction, whereas the remaining 11 patients had a definitive discharge diagnosis of myocardial infarction (Table 5). Thus, for the diagnosis of STEMI of an on-scene ECG registered a few minutes after return of spontaneous circulation, the sensitivity was 88% (74% to 96%), the specificity 69% (51% to 83%), the positive predictive value 77% (62% to 87%), and the negative predictive value 83% (83% to 93%). If only angiographically proven ECG cases were included (n=46), the sensitivity was 90% (83% to 96%) and the specificity 83% (74% to 95%).

After hospital admission, ECG diagnosis of STEMI was established in 40 of the 77 primary survivors, for whom an ECG was registered immediately. The ECGs of 37 patients evidenced no characteristic signs of STEMI. The tentative diagnosis of STEMI in the ECG at hospital admission was confirmed in 37 and rejected in 3 patients (Table 6). Another 11 patients had a discharge diagnosis of acute myocardial infarction that had not been diagnosed by the ECG at admission. Thus, for myocardial infarction diagnosis of the first in-hospital ECG after out-of-hospital resuscitation the sensitivity is 93% (79% to 98%), the specificity 70% (53% to 84%), the positive predictive value 77% (62% to 87%), and the negative predictive value 90% (72% to 97%). If only angiographically cases were included, the value for sensitivity was 91%, and for specificity 63%, respectively. The accuracy of

STEMI diagnosis is 79% for the out-of-hospital ECG and 81% for the ECG at admission, ie, the predictive value of the out-of-hospital ECG equals that of the first ECG after admission with regard to the final diagnosis of acute myocardial infarction, with a concordance of κ=0.68. However, the on-scene ECG was registered 40 minutes (28 to 69 minutes) earlier, which provides the opportunity for timely therapeutic decisions in the majority of patients. For 3 of the 11 surviving patients without signs of STEMI, in neither the out-of-hospital ECG nor in the ECG at admission was the final definitive diagnosis of acute myocardial infarction, according to our definition, based solely on a typical course of specific biomarkers of myocardial cell death. Thus, these patients formally had a non–ST-elevation infarction (one of them having a left bundle-branch block in the first ECG after return of spontaneous circulation), because they at no time displayed distinct ECG changes for an unambiguous diagnosis of acute myocardial infarction by ECG alone (Table 4). Consequently, these patients could not have been identified with the aid of an ECG. If these 3 patients are excluded from the evaluation, the calculated specificity of the out-of-hospital ECG and the ECG at admission is 83%, and the sensitivity is 82% for both. Exclusion of patients with left bundle-branch block in the first ECG after return of spontaneous circulation did not change the results because in only 1 other patient with left bundle-branch block was the final diagnosis of myocardial infarction documented by angiography.

The ST elevation was 1.25 mV (0.65 to 2.2 mV) in the out-of-hospital ECG and 0.83 mV (0.4 to 1.5 mV) in the first intrahospital ECG (*P*=.001). The corresponding ST resolution thus was 80% (23% to 100%), indicating a partial ST-segment resolution of the patients presenting with ST-segment elevation in the first out-of-hospital ECG after return of spontaneous circulation.

LIMITATIONS

Our study has several limitations. First, the registration of out-of-hospital ECG and interpretation was done by experienced physicians (two thirds internists and one third anesthesiologists). It remains unclear whether other providers may achieve similar or identical results. An alternative to ECG interpretation could be radio or telephone transmission to a hospital-based physician who may also decide on specific therapeutic procedures.

Second, a selection bias of patients cannot be excluded. Compared to routine conditions in the Berlin EMS, the bystander resuscitation rate is much lower (14%) than that observed in our study.[27] Also, the location of arrest is different from the routine experience. Generally, the location of arrest in Berlin is the patient's home in about 75% of the victims, whereas it is only 52% in the present study. Finally, the duration of symptoms in the present study was shorter than routinely observed.[27] All these factors have a favorable influence on the probability of survival and may have also influenced the significance of the ECG registered immediately after return of spontaneous circulation. Although complete data sets are present for all 77 included patients, the final diagnosis could not be verified by, for example, autopsy in all patients who died throughout the clinical course.

DISCUSSION

In this study, we wanted to evaluate the significance of a standard 12-lead ECG registered immediately after successful out-of-hospital resuscitation in relation to the final diagnosis of acute ST-elevation myocardial infarction. This study was not designed or powered to differentiate between different patient groups prone to the underlying qualifying event for the out-of-hospital sudden cardiac death (eg, non ST-elevation myocardial infarction or STEMI). Previous studies have demonstrated a high reliability for an out-of-hospital ECG diagnosis of STEMI under a wide variety of situations involving cardiologists, emergency physicians, computerized ECG interpretation, radio transmission of ECGs to a hospital-based physician, and even evaluation by trained nurses or paramedics.[11,12,14-18,28,29] However, these ECGs have been registered against the background of more or less typical clinical signs of angina pectoris in most cases, ie, in a group of preselected patients with a high probability of an acute coronary syndrome. This study included unselected cases after resuscitated circulatory arrest of assumed cardiac origin. Defibrillation with or without additional resuscitation measures can lead to significant ECG changes. These changes include transient ST elevations suggestive of STEMI.[20] Moreover, the influence of vasoactive or antiarrhythmic medications, as well as prolonged resuscitation measures with minimal cardiac perfusion, may also alter the ECG. Also, prolonged resuscitation measures and countershocks could induce an increase in troponins and creatine kinase. Even if the diagnosis of a myocardial infarction was based on troponin or creatine kinase values in only 3 patients alone in our series, it cannot completely be ruled out that these changes occurred because of the resuscitation measures by itself. However, according to a recent European Society of Cardiology/American College of Cardiology/American Heart Association/World Heart Federation consensus document, the circulatory arrest is by itself a sign of high probability of a myocardial infarction.[26] It is obvious, even if it is unlikely, that it is impossible to rule out that a STEMI develops in the short time between the onset of arrest and registration of the first out-of-hospital ECG. Irrespective of these considerations, our results indicate that the diagnostic reliability of an ECG registered shortly after return of spontaneous circulation in patients with out-of-hospital resuscitation is comparable to or even higher than the data described for patients with severe chest pain and suspected myocardial infarction without previous resuscitation.[14] This might be partially explained by a selection bias because cardiac arrest is apparently the consequence of severe cardiac ischemia at least in the majority of the survivors. In our series, it is obvious that the development of myocardial infarction in the short window after arresting and before registration of the first ECG cannot completely be ruled out.

With regard to the diagnosis of STEMI, it is much more likely that major changes would develop during the interval from registration of the first out-of-hospital ECG until registration of the ECG at admission. In only 4 patients, however, the out-of-hospital ECG diagnosis of STEMI could not be confirmed in the ECG at admission, whereas in 2 other patients characteristic signs of a STEMI present in the ECG at admission were existing in the out-of-hospital ECG. Thus, there is an astonishing concordance of the in-field ECG and the ECG at hospital admission registered about 40 minutes later. ST elevation was more pronounced in the first out-of-hospital ECG compared with the ECG after hospital admission, which may be due to the administration of aspirin, heparin, and particularly thrombolysis that could have led to some extent to partial reperfusion that leads to ST-segment resolution of about 80%.[25] Consequently, an early out-of-hospital ECG allows a reliable fast-track decision on the reperfusion strategy, including immediate out-of-hospital thrombolysis, which is supported by anecdotal reports.[30] Where percutaneous intervention is planned, out-of-hospital diagnosis of STEMI will lead to substantial time savings compared with PCI by advanced announcement of the patient, which was indeed the case in those hospitals with PCI facilities.[31]

There was about a 40-minute time difference between the out-of-hospital diagnosis to the diagnosis by the first inhospital ECG, even in an urban environment. This delay to definitive diagnosis may be of utmost importance because, as far as it could be clarified by interviewing bystanders, the patients had symptoms suggestive of cardiac ischemia for a median of about 10 minutes (5 to 38 minutes) only before collapse. For patients with such a short duration of symptoms, even relatively small delays may play a decisive role with regard to efficacy of reperfusion treatment.[8,32,33]

In many patients, sudden cardiac death is the first and only symptom of coronary heart disease. Although the absolute risk is relatively low in a population without known cardiac risk factors, this group includes the largest absolute number of victims of sudden cardiac death.[7] If an acute myocardial infarction is the cause of cardiac arrest, early reperfusion therapy is of utmost importance.[8,33] Thus, the results of our investigation underscore the potential of early diagnosis because STEMI was the cause of arrest in two thirds of the successfully resuscitated patients. Our results, however, do not reveal anything about the distribution of underlying diseases or the cause of arrest in general in patients with sudden cardiac death. Accordingly, this study was not designed to evaluate the prevalence of acute myocardial infarction in all sudden cardiac death victims.

In patients resuscitated successfully from out-of-hospital arrest of assumed cardiac origin, the underlying disease is an acute STEMI in a large percentage. These patients can be identified with an out-of-hospital ECG shortly after return of spontaneous circulation, with a high diagnostic accuracy. Future studies are needed to investigate whether or not out-of-hospital fibrinolysis or immediate PCI on hospital arrival will improve patient survival.

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