Heart Rate After Resuscitation From Out-of-Hospital Cardiac Arrest due to Acute Coronary Syndrome Is an Independent Predictor of Clinical Outcome

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Background: Heart rate (HR) is a useful predictor of cardiovascular disease, especially in acute coronary syndrome (ACS). However, it is unclear whether there is an association between HR and clinical outcomes after resuscitation from out-of-hospital cardiac arrest (OHCA) due to ACS. The aim of this study was to investigate the impact of HR on clinical outcome in individuals resuscitated from OHCA due to ACS.

Methods and Results: Data from 3,687 OHCA patients between October 2002 and October 2014 were retrospectively analyzed. We divided 154 patients diagnosed with ACS into 2 groups: those with tachycardia (HR >100 beats/min, n=71) and those without tachycardia (HR ≤100 beats/min, n=83) after resuscitation. The primary endpoint was 1-year mortality and the secondary endpoint was neurological injury at discharge according to cerebral performance category score. Overall, mean HR was 95.6 beats/min. There were several significant differences in patient characteristics, indicating poor general condition of patients with tachycardia. Mortality at 1-year was 41.6%, and neurological injury at discharge was observed in 44.1% of individuals. In the multivariate analysis, tachycardia after resuscitation was an independent predictor of both 1-year mortality (hazard ratio, 2.66; 95% CI: 1.20–5.85; P=0.03) and neurological injury at discharge (odds ratio, 2.65; 95% CI: 1.27–5.55; P=0.04).

Conclusions: In patients who recovered from OHCA due to ACS, tachycardia after resuscitation predicted poor clinical outcome.

Key Words: Cardiac arrest; Cardiopulmonary resuscitation; Myocardial infarction
Apart from epinephrine for resuscitation. Tachycardia was defined as >100 beats/min. When patients had arrhythmia such as atrial fibrillation or atrioventricular block, the mean of the HR during the ECG recording was taken as the HR.

This study was approved by the ethics committee of Toho University Omori Medical Center (no. M19030).

Endpoints
In the present study, the primary endpoint was defined as 1-year all-cause mortality. We also analyzed neurological outcome as evaluated on cerebral performance category (CPC) score at discharge as the secondary endpoint. A good neurological outcome was defined as CPC score 1 (normal) or 2 (mild or moderate impairment). A poor neurological outcome was defined as CPC score 3 (severe neurological disability and dependency), 4 (coma or vegetative state), or 5 (death).

Data Collection
The data before arrival at hospital were collected from the emergency medical technician reports. These included whether the cardiac arrest was witnessed, details of bystander-delivered cardiopulmonary resuscitation (CPR), the initial observed rhythm based on ECG monitoring, time to ROSC and whether pre-hospital ROSC occurred, and the dose of epinephrine given. All data after arrival at hospital were also collected from the hospital charts. These included the patient’s history, characteristics, vital signs, true for predicting worse outcome of resuscitation in patients with ACS.

Methods
Subjects
Of 3,687 OHCA patients transported to the tertiary emergency hospital (Toho University Omori Medical Center, Tokyo, Japan) between October 2002 and October 2014, we retrospectively analyzed 154 consecutive patients presenting with OHCA with ACS who were successfully resuscitated and underwent immediate successful PCI with stenting after arrival at hospital. Patients who were <18 years old or who had unsuccessful PCI were excluded.

According to the current guidelines, ST elevation myocardial infarction (STEMI) was defined as an ST elevation ≥0.1 mV in 2 contiguous standard leads or ≥0.2 mV in precordial leads on post-resuscitation electrocardiogram (ECG). Patients without STEMI but with elevated cardiac enzymes, both creatine phosphokinase-MB and troponin-I, were diagnosed as having non-STEMI (NSTEMI).

Study Design
Patients were divided into the non-tachycardia group and the tachycardia group according to HR on 12-lead ECG after resuscitation in the emergency room. All ECG were recorded at hospital arrival before any drug treatment apart from epinephrine for resuscitation. Tachycardia was defined as >100 beats/min. When patients had arrhythmia such as atrial fibrillation or atrioventricular block, the mean of the HR during the ECG recording was taken as the HR.

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Heart Rate After Resuscitation

Results

Patient Characteristics
Mean HR for the non-tachycardia and tachycardia groups was 76.6 beats/min and 118.0 beats/min, respectively (P<0.001). Mean time to record HR on 12-lead ECG after ROSC was 19.5±11.3 min.

Table 1 lists the baseline patient characteristics between the non-tachycardia and tachycardia groups. Overall, mean age was 64.2 years, 85.1% of the patients were male, and 119 patients (77.3%) were resuscitated from a shockable rhythm, that is, VT or VF. Although the tachycardia group was older compared with the non-tachycardia group, clinical risk factors such as hypertension, diabetes mellitus, dyslipidemia, hemodialysis, and a history of PCI were not significantly different between the 2 groups. Compared with the non-tachycardia group, the tachycardia group had a longer time to ROSC and a higher frequency of epinephrine use. Nevertheless, the total dose of epinephrine prior to resuscitation was not significantly different between the 2 groups. In the analysis of patients with epinephrine use before ROSC, there was no correlation between HR after resuscitation and epinephrine dose (Figure 1; r=-0.094, P=0.42).

With regard to physical examination, patients without tachycardia had a 2-fold higher prevalence of normal light reflex at arrival compared with those presenting with tachycardia. Other characteristics were not significantly different between the 2 groups.

Examination Data
Examination findings and treatment information after hospital arrival are listed in Table 2. In each group, approximately three-quarters of the patients had sinus rhythm after resuscitation (P=0.99). Patients in the tachycardia group had significantly higher lactate compared with the non-tachycardia group. Renal function, hemoglobin, and maximum creatinine kinase were not significantly different between the 2 groups. Other parameters including UCG and ICA also did not differ.
**Table 2. Examination Parameters and Treatment After Hospital Arrival**

| Parameter                          | Non-tachycardia (n=83) | Tachycardia (n=71) | P-value |
|------------------------------------|------------------------|--------------------|---------|
| **ECG parameters**                 |                        |                    |         |
| HR (beats/min)                     | 76.6±14.3              | 118.0±15.6         | <0.001  |
| Wide QRS (≥120 ms)                 | 20 (24.1)              | 13 (18.3)          | 0.43    |
| **Arrhythmia**                     |                        |                    |         |
| Sinus rhythm                       | 63 (75.9)              | 54 (76.1)          | 0.99    |
| AF                                 | 15 (18.1)              | 16 (22.5)          | 0.55    |
| CAVB                               | 4 (4.8)                | 0 (0)              | 0.13    |
| CLBBB                              | 4 (4.8)                | 2 (2.8)            | 0.69    |
| **Laboratory parameters**          |                        |                    |         |
| pH                                 | 7.21±0.19              | 7.29±0.19          | 0.46    |
| Lactate (mmol/L)                   | 8.64±5.29              | 10.97±4.60         | 0.02    |
| Na (mEq/L)                         | 138.6±4.2              | 138.3±4.0          | 0.69    |
| K (mEq/L)                          | 4.18±0.86              | 4.27±0.84          | 0.57    |
| Ca (mEq/L)                         | 9.15±0.58              | 9.01±0.65          | 0.20    |
| BUN (mg/dL)                        | 20.0±13.6              | 22.5±19.0          | 0.39    |
| Cr (mg/dL)                         | 1.34±1.75              | 1.69±2.12          | 0.32    |
| eGFR (mL/min/1.73 m²)              | 67.1±28.3              | 66.9±35.4          | 0.96    |
| Hb (g/dL)                          | 13.7±2.0               | 14.1±1.9           | 0.53    |
| Max. CK (IU/L)                     | 4,489±5,357            | 5,065±6,314        | 0.58    |
| BNP (pg/mL)                        | 227.7±346.8            | 365.6±541.8        | 0.11    |
| **UCG parameters**                 |                        |                    |         |
| LVEF (%)                           | 47.1±18.7              | 41.6±16.8          | 0.09    |
| LVDd (mm)                          | 5.18±1.15              | 5.14±0.96          | 0.85    |
| LVDs (mm)                          | 3.79±1.19              | 3.87±1.01          | 0.73    |
| **ICA culprit lesion**             |                        |                    |         |
| LMT                                | 6 (7.2)                | 4 (5.6)            | 0.75    |
| RCA                                | 30 (36.1)              | 19 (26.8)          | 0.23    |
| LAD                                | 36 (43.4)              | 36 (50.7)          | 0.42    |
| LCX                                | 11 (13.3)              | 12 (16.9)          | 0.65    |
| **No. vessel disease**             |                        |                    |         |
| 1                                  | 34 (41.0)              | 31 (43.7)          | 0.75    |
| 2                                  | 26 (31.3)              | 17 (23.9)          | 0.37    |
| 3                                  | 23 (27.7)              | 23 (32.4)          | 0.60    |
| **Treatment**                      |                        |                    |         |
| Administration                     |                        |                    |         |
| Dopamine                           | 68 (81.9)              | 56 (78.9)          | 0.69    |
| Dobutamine                         | 33 (39.7)              | 29 (40.8)          | 0.99    |
| Norepinephrine                     | 58 (69.9)              | 59 (83.1)          | 0.06    |
| PDE-3 inhibitor                    | 1 (1.2)                | 2 (2.8)            | 0.60    |
| Amiodarone                         | 36 (40.9)              | 29 (40.8)          | 0.87    |
| Nifekalant                         | 2 (2.4)                | 1 (1.4)            | 0.99    |
| Lidocaine                          | 25 (30.1)              | 24 (33.8)          | 0.73    |
| Nicorandil                         | 56 (67.5)              | 45 (63.4)          | 0.61    |
| Carperitide                        | 9 (10.8)               | 2 (2.8)            | 0.07    |
| Ventilator                         | 82 (98.8)              | 66 (93.0)          | 0.10    |
| IABP                               | 58 (71.1)              | 52 (73.2)          | 0.72    |
| PCPS                               | 23 (27.7)              | 21 (29.6)          | 0.86    |
| Hypothermia therapy                | 52 (62.6)              | 42 (59.2)          | 0.74    |

Data given as mean±SD or n (%). AF, atrial fibrillation; BNP, brain natriuretic peptide; BUN, blood urea nitrogen; CAVB, complete atioventricular block; CK, creatinine kinase; CLBBB, complete left bundle branch block; Cr, creatinine; ECG, electrocardiogram; eGFR, estimated glomerular filtration rate; Hb, hemoglobin; HR, heart rate; IABP, intra-aortic balloon pumping; ICA, invasive coronary angiography; LAD, left anterior descending artery; LCX, left circumflex artery; LMT, left main trunk; LVDd, left ventricular end-diastolic diameter; LVDs, left ventricular end-systolic diameter; LVEF, left ventricular ejection fraction; PCPS, percutaneous cardiopulmonary support; PDE, phosphodiesterase; RCA, right coronary artery; UCG, ultrasound cardiography.
Figure 2. Kaplan-Meier survival curves for (A) out-of-hospital cardiac arrest due to acute coronary syndrome (including both in-hospital and after-discharge death), and (B) with (n=75) or (C) without epinephrine during resuscitation (n=79), according to tachycardia status.
between the 2 groups.

With regard to treatment after admission, all 154 patients were treated with catecholamine drugs, although there were no significant differences in individual catecholamine drugs between the 2 groups. The proportion of patients receiving additional treatment, such as other drugs, ventilator support, intra-aortic balloon pumping (IABP), percutaneous cardiopulmonary support (PCPS), and hypothermia therapy, was not significantly different between the groups.

Clinical Outcomes
The number of patients who had died at 1 year after OHCA was 41/71 (57.7%) in the tachycardia group and 23/83 (27.7%) in the non-tachycardia group (P<0.001). On Kaplan-Meier analysis the tachycardia group had a higher 1-year all-cause mortality than the non-tachycardia group (Figure 2A).

Of the 154 patients in this study, the number of patients with poor neurological outcome (CPC score 3–5) at discharge was 68 (44.1%); CPC score 3, 13 patients (8.4%); CPC score 4, 5 patients (3.2%); and CPC score 5, 50 patients (32.5%). The proportion of these patients with poor neurological outcome at discharge in the tachycardia and non-tachycardia groups was 44/71 (62.0%) and 24/83 (28.9%), respectively (P<0.001).

Absolute HR and HR >100 beats/min were the independent predictors of both endpoints (1-year all-cause mortality and poor neurological outcome). In adjusted multivariate Cox proportional and logistic regression models with regard to both endpoints, tachycardia was independently associated with a worse 1-year mortality, and with a poor neurological outcome at discharge (Table 3).

In terms of the utility of epinephrine during resuscitation, the 1-year all-cause mortality in the tachycardia group was increased compared with the non-tachycardia group, although there were no significant differences in individual catecholamine drugs between the 2 groups. The proportion of patients receiving additional treatment, such as other drugs, ventilator support, IABP, hypothermia therapy, was not significantly different between the groups.

Table 3. Multivariate Indicators of 1-Year All-Cause Mortality and Poor Neurological Outcome at Discharge

| Model | All-cause mortality | Poor neurological outcome |
|-------|---------------------|--------------------------|
|       | Hazard ratio (95% CI) | P-value | C-statistics | Odds ratio (95% CI) | P-value | C-statistics |
| Model 1: HR >100 beats/min + demographics | 2.27 (1.30–3.95) | 0.003 | 0.708 | 2.48 (1.45–4.18) | 0.001 | 0.730 |
| Model 2: model 1 + incident†† | 2.41 (1.14–5.12) | 0.009 | 0.799 | 2.09 (1.01–4.32) | 0.02 | 0.815 |
| Model 3: model 2 + epinephrine dose | 2.26 (1.22–4.19) | 0.03 | 0.810 | 2.21 (1.19–4.11) | 0.03 | 0.822 |
| Model 4-1: model 3 + LVEF | 2.66 (1.20–5.85) | 0.03 | 0.832 | 2.65 (1.27–5.55) | 0.04 | 0.823 |
| Model 4-2: model 3 + SBP | 2.64 (1.16–4.12) | 0.03 | 0.822 | 2.12 (1.07–4.43) | 0.02 | 0.831 |
| Model 4-3: model 3 + max. CK | 2.28 (1.04–5.00) | 0.04 | 0.830 | 2.44 (1.13–4.89) | 0.03 | 0.832 |
| Model 4-4: model 3 + no. vessel disease††† | 2.31 (1.23–4.35) | 0.03 | 0.827 | 2.40 (1.36–4.66) | 0.03 | 0.828 |

†Age, gender; ††incident-related characteristics (bystander-delivered CPR, initial rhythm VT/VF, time to ROSC); †††vessel disease: ≥75% stenosis on emergency coronary angiography. Abbreviations as in Tables 1, 2.

Discussion
In the present study, we have shown that increased HR was strongly associated with both a higher 1-year mortality and a poor neurological outcome at discharge in patients resuscitated from OHCA due to ACS. Although associations between increased HR and poor clinical outcomes after ACS without cardiac arrest have been previously shown,8–11 to our knowledge, this is the first study to suggest the importance of HR as an independent predictor of outcome in patients with ACS regardless of the antecedent cardiac arrest.

HR and the Impaired Autonomic Nervous System
The increase in HR after resuscitation is mainly caused by a stimulated (and not suppressed) sympathetic nervous system that results from hemodynamic instability and impaired vagal nerve function due to severe anoxic brain injury.24–26 In the current study, the tachycardia group had higher lactate and a lower prevalence of normal light reflex, along with a lower incidence of bystander-delivered CPR and a longer time to ROSC. Furthermore, the early establishment of severe neurological injury that was noted in the tachycardia group might reflect the poor neurological outcomes at discharge, as suggested in the multivariate analysis models.

HR and Cardiac Function
Compared with non-cardiac causes, survivors of OHCA due to ACS frequently have severe cardiac injury and dysfunction, and thus cardiac reperfusion after resuscitation has a particular role in improving clinical outcomes.7 Increased HR after resuscitation, however, leads to insufficient coronary blood flow due to a reduction in diastolic time and higher oxygen consumption because of excessive cardiac work.27,28 Furthermore, inappropriate tachycardia leads to low cardiac output, despite high cardiac oxygen demand, which is directly associated with more myocardial damage and multiple organ failure.27,28 Thus, increased HR might be directly associated with poor clinical outcome, as observed in the current study.

In spite of previous evidence from individuals with CVD, HR is conventionally judged to be less important than other physical or examination findings as a predictor or an intervention point in the setting of post-cardiac arrest syndrome (PCAS).7,29 Given, however, that HR independently pre-
dicted clinical outcome even after resuscitation was taken into account, tachycardia may have particular importance as a marker or a therapeutic target for PCAS accompanied by ACS.

**Increased HR and Epinephrine**

Although a higher dose of epinephrine may cause increased HR after resuscitation because of its β-adrenergic effects, there is no clear evidence for a relationship between usage or dose of epinephrine and increased HR after ROSC. In the present study, the use of epinephrine was more commonly observed in the tachycardia group, but it is unknown whether epinephrine use affected HR because this group also had a longer time to CPR, which may have also affected HR. Increased HR after resuscitation is mainly caused by hemodynamic instability and cerebral injury, not by use or dose of epinephrine. These earlier findings support our observation that tachycardia was an independent predictor of mortality and neurological outcome after adjusting for epinephrine dose. Furthermore, no correlation was shown between HR after resuscitation and epinephrine dose. On subanalysis, the effect of HR on clinical outcome was similar regardless of the status of epinephrine during resuscitation. Of interest, 1-year all-cause mortality was different between the tachycardia and non-tachycardia group but the difference was smaller in patients treated without epinephrine during resuscitation compared with those treated with epinephrine during resuscitation. This suggests that HR could be a more reliable and better prognostic marker in patients with poor general condition after ROSC such as those treated with epinephrine during resuscitation. Based on the present study, epinephrine usage or dose may have a lower impact on HR during or after resuscitation than the general or cerebral conditions after ROSC.

**Study Limitations**

There were several limitations in the present study. First, this was a retrospective observational study, and thus a causal relationship between tachycardia after resuscitation and increased mortality cannot be proven. Furthermore, this was a single-center study, and the number of enrolled patients was small. Additional studies are needed to confirm the present findings. Given the nature of the current study, >30% of the total patients had in-hospital death. In this regard, we were not able to investigate the impact of oral medication after PCI on clinical outcomes due to the limited number of patients who were successfully discharged but eventually died at 1 year (n=14). The relationship between oral medication and outcome in patients who are successfully discharged and rehabilitated needs to be investigated in the future. Although we collected the data by following a uniform procedure, we cannot exclude the possibility of other uncontrolled confounders. In particular, the time to ROSC was recorded based on the description of any witnesses. The actual onset time of OHCA for patients without a witness was not available. Furthermore, data on previous β-blocker therapy and Killip class at hospital arrival, which may affect HR and mortality after resuscitation, were not available for all patients. Finally, we analyzed HR after resuscitation based on that captured on ECG in the emergency room. Associations between HR during PCI or after patient admission were not evaluated.

**Conclusions**

HR after resuscitation was independently associated with 1-year all-cause mortality and neurological outcomes in patients with OHCA due to ACS. Despite successful resuscitation and PCI, increased HR after ROSC predicted poor clinical outcome.

**Disclosures**

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