Intensive Care Med (1991) 17:419–420

Intensive Care Medicine

Hyperthermia following cardiopulmonary resuscitation *

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Received: 8 October 1990; accepted: 2 August 1991

Abstract. To clarify the clinical nature of post-resuscitation hyperthermia, we reviewed the charts of 18 patients who had cardiac arrest on arrival and regained cardiovascular stability for a study period of sufficient length. Patients with trauma, burns, poisoning and cerebrovascular accidents were excluded. We analyzed the hyperthermia (above 38 °C) occurring in the initial 48 h after resuscitation. After resuscitation, most patients showed a rapid rise in body temperature. Patients with later brain death showed significantly earlier appearance of hyperthermia (6.2 h after cardiac resuscitation; median) and a higher peak temperature (39.8 °C; median) compared with patients showing prolonged coma (12.7 h and 38.3 °C, respectively). Hyperthermia above 39 °C was associated with subsequent brain death. The incidence of factors influencing body temperature did not differ between the brain death and prolonged coma groups. Patients achieving full recovery did not show hyperthermia. In conclusion, hyperthermia is an early indicator of brain damage after resuscitation.

Key words: Resuscitation – Hyperthermia – Ischemic brain damage

Hyperthermia is frequently observed in the early post-resuscitation period. Although analysis of this hyperthermia is necessary because it can affect the prognosis of patients through the circulatory and metabolic changes [1, 2], little attention has been paid to it [3, 4]. Mason et al. [5] have shown that post-resuscitation body temperature instability indicates a poor prognosis, but their report was limited to children after near-drowning. The purpose of this study was to clarify the clinical nature of early hyperthermia in post-resuscitation patients of various ages and with different etiologies.

* No financial support was given to this study

Patients and methods

We reviewed all records of 333 patients who were in a state of cardiorespiratory arrest on arrival between January 1980 and December 1990. Criteria for selection of patients were restoration of spontaneous circulation (ROSC) and survival for a period long enough to allow determination of the neurological outcome. Patients with trauma, burns, poisoning and cerebrovascular accidents were excluded.

We analyzed the hyperthermia and related factors which might have affected the body temperature during the initial 48 h after ROSC. In terms of neurological outcome, we divided the patients into 3 groups: a full recovery group, a prolonged (longer than 4 weeks) coma group, and a brain death group, and compared the results between the groups.

Cardiopulmonary resuscitation was performed according to the standard, closed-chest method [6]. ROSC was defined as return of the heart beat with an obtainable peripheral arterial pressure. Following ROSC, ordinary general supportive care was employed. Catecholamines, barbiturates and antipyretic therapies were used as indicated. Body temperature was measured using a rectal thermometer at various intervals from 30 min to 4 h as deemed necessary. Hyperthermia was defined as a rectal temperature above 38 °C.

Results

Eighteen patients fulfilled the criteria. Their characteristics are shown in Table 1. The causes of cardiac arrest were suffocation in 7 patients, myocardial infarction in 3, asthma attack in 3, hanging in 2, arrhythmia in 2 and near-drowning in 1. No patient developed clinically evident infection or was declared brain-dead within 48 h after ROSC.

Following ROSC, most patients showed a rapid rise in the body temperature, reaching a peak after 19.5 h (median). The development of hyperthermia is shown in Table 2. In the brain death group, the development of hyperthermia was significantly faster and the peak temperature significantly higher than in the prolonged coma group. All 7 patients with a peak body temperature above 39 °C developed brain death later. The patients in the full recovery group did not show hyperthermia.

Table 1. Patient characteristics and the factors related to body temperature

	Full recovery	Prolonged coma	Brain death
Number of patients	3	7	8
Median age (range); years	44 (27–61)[a]	77 (53–86)[b]	44 (4–56)[c]
Sex (male/female)	2/1	6/1	4/4
Median arrest time[d] (range); min	23 (10–27)	25.5 (9–42)	29.5 (16–60)
Initial hypothermia (<35 °C)	1	6	7
Active rewarming	1	2	3
Catecholamines	1	4	5
Barbiturate coma	0	3	4
Antipyretics; cases (occasions)	0	2 (3)	8 (20)
Physical cooling; cases (occasions)	0	7 (12)	8 (41)

[a] vs [b] $p<0.05$, [b] vs [c] $p<0.001$ (Mann-Whitney U-test)
[d] Including resuscitation time

Table 2. Post-resuscitation hyperthermia

	Full recovery	Prolonged coma	Brain death
Number of patients	3	7	8
Hyperthermia (>38 °C)	0	6	8
h after ROSC[a]	–	12.7 (11.0–17.8)[b]	6.2 (3.1–15.6)[c]
Peak body temperature (°C)	37.2 (37.0–37.4)[d]	38.3 (37.8–38.9)[e]	39.8 (38.8–42.0)[f]

[a] Restoration of spontaneous circulation
[b–f] Numerals indicate median and range of the data. [b] vs [c] $p<0.005$, [d] vs [e] $p<0.01$, [e] vs [f] $p<0.001$ (Mann-Whitney U-test)

Discussion

After cardiopulmonary resuscitation, various pathophysiological changes appear [7]. Hyperthermia should be considered one of these changes. Several mechanisms can be speculated: increased heat production due to ischemia-related factor such as endogenous catecholamines [8]; thermodysregulation of central nervous system origin [9]; decreased heat loss or altered distribution of body heat due to vasoconstriction [10]. True mechanism, however, remains to be defined.

The degree and rate of development of hyperthermia was greater in the poorer outcome groups. This result cannot be attributed to the factors influencing body temperature (Table 1), suggesting the real nature of post-resuscitation hyperthermia. The advanced age of the prolonged coma group might have affected the body temperature, but further investigation will be required to examine this possibility.

In conclusion, hyperthermia is an early indicator of brain damage following cardiopulmonary resuscitation.

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