

An 85-year-old female with a history of diabetes was hospitalized for dizziness. She was bradycardic; otherwise, her vital signs were normal. An ECG (Fig. 1) was recorded by a Schiller system, an electrocardiograph commonly used in Europe. The interpretation software indicated a heart rate of 71/min, twice the true heart rate of 35/min. Computer diagnosis was atrial fibrillation, ventricular premature complexes, and bigeminy. The true rhythm, however, was a slightly irregular junctional bradycardia. On laboratory testing, the patient's serum potassium level was found to be 9.2 mmol/L. After correcting the drug-induced hyperkalemia elicited by the coadministration of an angiotensin-converting enzyme-inhibitor and a potassium-sparing diuretic, the patient became asymptomatic, and the ECG returned to normal.

Our case supports the findings by Littmann et al that double counting of heart rate by ECG interpretation software may be a sign of severe hyperkalemia. Moreover, it also proves that this ECG sign is not specific to the GE-Marquette electrocardiographs and interpretation softwares. In our case with the Schiller system and in several of Dr Littmann's cases with the GE-Marquette system, the interpretation software mistakenly read the tall and peaked T waves as ventricular bigeminy. Clinicians treating critically ill patients need to be educated that whenever the ECG interpretation software double counts the heart rate and falsely diagnoses bigeminy, severe hyperkalemia should be suspected.

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Hypothermia for out-of-hospital cardiac arrest survivors: a single-center experience

To the Editor,

Out of hospital cardiac arrest (OHCA) patients have a poor prognosis, with only 10% of patients surviving. Recent randomized trials have shown that moderate therapeutic hypothermia improves neurologic outcome and survival in selected patients after cardiac arrest [1,2]. Therapeutic

hypothermia is now recommended by the Advanced Life Support Task Force of the International Liaisons Committee on Resuscitation and incorporated in the American and European resuscitation guidelines as part of post resuscitation care [3].

We undertook this study to investigate the mortality and neurologic outcome of mild therapeutic hypothermia in surviving OHCA patients in a single university hospital in the southern part of the Netherlands with a catchments area of approximately 200 000 inhabitants, with a yearly incidence of sudden cardiac arrest of approximately 10 per 10 000 inhabitants [4].

Medical charts of 101 consecutive OHCA patients admitted alive to our intensive coronary care unit were retrospectively analyzed. Forty-three patients receiving hypothermic treatment in 2004 and 2005 (hypothermia group) were compared to 58 historical control patients from 2001 to 2003 not treated with hypothermia (normothermia group). Data on cardiac arrest were recorded and analyzed according to the "Utstein Style" recommended guidelines [5]. Differences between groups were analyzed using χ^2 , Mann-Whitney *U*, or Students *t* test. Our hypothermia protocol is primarily based upon the inclusion and exclusion criteria from 2 recent randomized controlled clinical trials; however, the actual decision of initiation and duration of mild hypothermia was left to the discretion of the treating cardiologist [1,2]. All patients received current standard care including percutaneous coronary intervention; were mechanically ventilated; and received intravenous midazolam and piritramide for sedation and analgesia, respectively, and intravenous pancuronium to prevent shivering. Mild hypothermia was initiated as soon as possible after admission and was induced and maintained at a target temperature of 33°C using a closed-loop endovascular system (Alsios CoolGard, Irvine, Calif). Patients were allowed to passively rewarm. The mean arterial pressure was maintained at ≥ 90 mmHg with inotropic support when necessary. The institution's ethics committee approved the study. Neurologic death was regarded when patients had absent somatosensory evoked potentials after 72 hours and died after active care was withdrawn. Cardiac death was regarded as death due to persistent cardiogenic shock despite (non)invasive measures. Discharged survivors or one of their relatives were contacted by telephone at least 6 months after discharge, and neurologic outcome was evaluated using predefined questionnaires (Glasgow Outcome Score [GOS] [6]). Unfavorable outcome was defined as death, severe disability, or vegetative state (GOS 1-3). Discharge to home or a rehabilitation facility with GOS ≥ 4 was defined as favorable neurologic outcome.

Baseline characteristics were comparable between both groups; however, patients in the normothermia group tended to be older, were more likely to have bystander cardiopulmonary resuscitation (CPR), and had shorter arrival of paramedics and return of spontaneous circulation (ROSC) times (Table 1). Hypothermia was initiated at a median of 4 hours (1.8-6.4 hours) after ROSC. The target temperature

Table 1 Baseline characteristics and outcome

	Hypothermia (n = 43)	Normothermia (n = 58)	P
<i>Baseline characteristics</i>			
Age (y)			
Mean \pm SD	56.2 (12.8)	63 (10.9)	.12
Female sex (%)	14 (33)	18 (31)	.87
Initial rhythm			
VT/VF (%)	37 (86)	54 (93)	.24
Asystole (%)	3 (7)	4 (7)	
PEA (%)	3 (7)	0	
Bystander-performed CPR (%)	22 (51)	42 (72)	.05
Time collapse—arrival of paramedics (min) (median [range])	10 (2-25)	8 (1-30)	.6
Time to ROSC (min) (median, [range])	27 (11-75)	22 (8-60)	.50
<i>Outcome</i>			
Unfavorable outcome (%)	22 (51)	28 (48)	.56
GOS (%)			
1	22 (51)	27 (47)	
2	0	0	
3	0	1	
4	11	12	
5	9	17	
Lost to follow-up	1	1	

PEA indicates pulseless electrical activity; GOS 1, dead; GOS 2, vegetative state; GOS 3, severe disability; GOS 4, moderate disability; GOS 5, good recovery; VT/VF, ventricular tachycardia and ventricular fibrillation.

was reached in all patients at a median of 3 hours (0.1-7 hours) and was maintained for 16.2 ± 6.3 hours. Two patients were lost to follow-up.

Twenty-two (51%) of 43 patients treated with hypothermia vs 28 (48%) of 58 normothermic patients had an unfavorable outcome. Mortality was 51% in the hypothermia group vs 47% in the normothermia group. The cause of death was not significantly different between both groups with all deaths occurring in hospital. Uncomplicated hyperglycemia, which was controllable with insulin, and the need for inotropic support were significantly more prevalent in the hypothermia group. Only 9 (21%) of 43 hypothermic patients completely fulfilled our protocol criteria. These patients were compared with 8 (14%) of 58 patients from the normothermia group who would have been eligible for hypothermia when available at that time. Again, no significant difference in mortality was found (66.7% vs. 62.5%).

Thus, we were unable to demonstrate a significant benefit of moderate hypothermia for OHCA patients in our hospital. Physicians should all be aware that the available evidence is based on randomized clinical trials with strict inclusion and exclusion criteria, excluding approximately 92% of patients initially screened. In addition, the variation in survival rates among communities can be attributed to

differences in the chain of survival concept. Relatively high bystander CPR rates have previously been reported in the Netherlands and may make it more difficult to demonstrate additional improvement in outcome after mild hypothermia [7]. Our protocol was frequently violated, and patients were cooled more liberally but, on the other hand, more closely followed the recommended International Liaisons Committee on Resuscitation guideline indications [3]. Our study partly demonstrates the difficulty in generalizing randomized trial results to everyday practice. Further research is therefore needed, and prospective randomized trials should be done to determine the broadest application of moderate therapeutic hypothermia.

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Shaken baby syndrome vs inflicted brain injury

To the Editor,

I read with interest the article by Healey and Schradling [1], describing an alleged case of shaken baby syndrome (SBS) with unilateral retinal hemorrhages and no associated intracranial hemorrhage (ICH). It is applaudable that the authors were alerted to the potential that the child was likely the victim of abuse. The authors' conclusion that the child was a victim of SBS however is not supported by their findings and must be viewed cautiously. Although the issue I raise may be viewed as semantic, it is crucial that physicians be careful not to make assertions that cannot withstand scrutiny.

The term *SBS* is often erroneously used synonymously for *inflicted traumatic brain injury (ITBI)* in infants. One must remember however that shaking is only one of many mechanisms of injury that can lead to brain injury and retinal hemorrhages.

Although I agree that the reported case likely constitutes a case of ITBI, it is not necessarily a case of SBS. The authors seem to have fallen victim to the use of the term *SBS* as a synonym for *child abuse* in their efforts to convey that unilateral retinal hemorrhages may point to a diagnosis of SBS even in the absence of ICH. I find it important to point out that the simple presence of retinal hemorrhages in an infant with evidence of brain injury does not automatically imply a diagnosis of SBS, as seems to be suggested by the authors. The determination of the cause of injury should be based on a more thorough investigation including skeletal survey, nuclear bone scintigraphy, elimination of differential diagnostic possibilities, and interprofessional collaboration.

A number of features go against shaking alone as a mechanism of injury in the reported case. The presence of bruising on the head of the child represents definitive

evidence of an impact to the head, a feature not seen in shaking alone. The retinal hemorrhages, although concerning, are not sufficiently described to allow their attribution solely to shaking, stressing the need to consider other causes, such as an impact to the head, in the differential diagnosis. In addition, although the absence of ICHs in shaking injuries has been reported in the literature [2,3], such a presentation is unusual. Overall, the sum of all of these factors points to an impact to the head as a distinct possibility to account for this child's findings; and although shaking could have occurred in addition to blunt trauma, the term *SBS* should not have been used in this case.

Inflicted traumatic brain injury is a more generic, less mechanism-specific term for cases of inflicted head injury. The use of this term reflects a broader, more inclusive approach to the diagnosis of abusive head injuries in children that acknowledges the fact that ITBI and its associated findings (retinal hemorrhages, fractures, bruising) may be the result of shaking, impact, or both.

Physicians who face suspected cases of abusive head injuries can best serve their patients by using the term *ITBI* instead of *SBS*. Discussions of specific mechanisms of injury are best addressed by physicians experienced in neurotrauma or child abuse pediatrics.

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