Hypertonic saline during CPR: Feasibility and safety of a new protocol of fluid management during resuscitation

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KEYWORDS
Cardiopulmonary resuscitation;
Out-of-hospital cardiac arrest;
Hypertonic saline;
Resuscitation success

Summary
Background and purpose: In experimental studies infusion of hypertonic saline during cardiopulmonary resuscitation (CPR) increased resuscitation success rate and improved myocardial and cerebral reperfusion during CPR. We tested the feasibility and the safety of this new therapeutic measure in a randomised, preclinical pilot study.

Methods: The study was performed in the EMS system of Bonn after approval of the local ethical committee. Study inclusion criteria were out-of-hospital cardiac arrest (CA) of non-traumatic origin, age ≥18 years, application of adrenaline (epinephrine) during CPR, duration of CA ≤15 min, and estimated body weight ≤125 kg. Patients randomly received 2 ml/kg/10 min HHS (7.2% NaCl with 6% hydroxy ethyl starch 200,000/0.5 [HES]) or HES alone. Haemoglobin, blood gases, plasma sodium and potassium concentrations were measured before and 10 min after infusion, and after admission to hospital. Feasibility and safety of the new fluid management was evaluated by looking for side effects and determination of resuscitation success and admission rates.

Results: Sixty-six patients were included. After infusion of HHS, plasma sodium concentration increased to 168 ± 29 mmol/l at 10 min after application but already decreased to near normal (147 ± 5.5 mmol/l) at admission to hospital. Patients receiving HHS showed a trend to higher resuscitation success and hospital admission rates (ROSC: HHS 66.7%, HES 51.5%, \(p = 0.21\); admission: HHS 57.6%, HES 39.4%, \(p = 0.14\)). The benefit of HHS was more pronounced if duration of untreated CA was...
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>6 min or if initial rhythm was asystole or pulseless electrical activity (PEA). Negative side-effects were not observed after HHS.

Conclusions: HHS after CA is feasible and safe and might improve short term survival after CPR. However, whether giving HHS could be a useful measure to increase resuscitation success after out-of-hospital CA requires a larger preclinical trial.

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Introduction

If defibrillation fails or if the initial ECG rhythm is not ventricular fibrillation (VF) resuscitation success after cardiac arrest (CA) depends crucially on reperfusion of the heart. Myocardial reperfusion during cardiopulmonary resuscitation (CPR) can be improved by an increase in systemic vascular resistance, i.e. by the application of the vasopressors like adrenaline (epinephrine) or vasopressin. However, this concept does not cover the fact that other pathophysiological changes such as haemoconcentration, endothelial cell swelling and rolling, and adhesion of leucocytes also compromise myocardial and cerebral reperfusion. Several experimental studies after CA and CPR demonstrated haemoconcentration due to capillary leakage and plasma loss of up to 20 ml/kg. One therapeutic option to compensate this plasma loss could be the infusion of a large volume of an isotonic solution. This approach, however, decreases cerebral and myocardial perfusion pressure during CPR.7–9

A well-known measure to increase blood volume and to improve haemodynamics and nutritive organ blood flow in haemorrhagic shock and brain trauma is to use a small volume of a hypertonic solution (“small volume resuscitation”).10–15 In the last decade the use of hypertonic solutions during CPR was investigated systematically in several experimental studies. The rationale to test this therapeutic option during CPR were the known effects of hypertonic solutions from hemorrhagic shock models, i.e. an increase of the intravascular volume, and a decrease of the ischaemic endothelial cell swelling and leukocyte activation. In experimental CA/CPR models the use of hypertonic saline significantly increased myocardial blood flow (MBF), myocardial perfusion pressure (MPP), and cardiac index (CI) during CPR. In addition, microcirculatory reperfusion of the brain was improved and cerebral blood flow (CBF) during CPR was sustained or even improved in HHS treated animals.17–20

In these experimental studies infusion of hypertonic solution significantly increased resuscitation success and survival rate. Furthermore, Krieter et al. found that hypertonic saline application after resuscitation from CA reduced astroglial S-100β protein release, which might indicate a possible reduction in neuronal injury after CPR.22

In the treatment of haemorrhagic shock a dose of 4 ml/kg bodyweight is recommended. After CA similar effects on myocardial reperfusion and success rate were found after infusion of 2 or 4 ml/kg hypertonic saline.20 A maximum plasma loss of 16 ml/kg was found by Lin and a solution of 7.2% NaCl showed a plasma expansion capacity of about eight-fold,10,23 a dose of 2 ml/kg bodyweight was used in our preclinical pilot study.

In human CA, however, comorbidity and haemodynamics during external chest compressions and after CPR may be different compared to animal experiments. We, therefore, studied the feasibility and safety of an intravenous infusion of hypertonic saline/hydroxyethyl starch (HHS) during preclinical CPR after out-of-hospital CA.

Materials and methods

The study was conducted by the Department of Anaesthesiology and Intensive Care Medicine of the University Bonn, Germany. It was in accordance with the declaration of Helsinki and in accordance with all current German regulations and standards for investigations upon human subjects after approval of the local ethical committee. Information and consent of the patients or relatives in advance of the study was not practicable, but the patients’ families and surviving patients were informed immediately about the trial. The study protocol specified that if there were any objections, the patient would be withdrawn from the study, but there were no objections. After successful CPR and admission to hospital additionally an extensive informed consent document explaining application of hypertonic saline and research data collection was handed over to the patient or his family or legal representative. Before using any data we documented the agreement of the patient or his representative by informed consent.

Patients

Starting in May 2001, patients who experienced CA were screened for inclusion. Included were all
patients with out-of-hospital cardiac arrest of non-traumatic origin, with the need of adrenaline during CPR, an age ≥ 18 years, duration of cardiac arrest ≤ 15 min and body weight ≤ 125 kg. Excluded were patients with ROSC before starting infusion of the study drug, known malignancy, pregnancy, and renal or heart failure (NYHA IV). Duration of CA was defined as the interval from the moment of collapse (presumed time of CA) until the beginning of basic life support by EMS personnel. Following the “Utstein Style” definitions cardiac aetiology was presumed if a non-cardiac external aetiology (i.e. trauma, asphyxia, drug overdose, drowning, electric shock, et cetera) and a non-cardiac internal aetiology could not be identified by the emergency physicians at scene (i.e. lung disease, cerebrovascular disease, gastrointestinal haemorrhage, pulmonary embolism, epilepsy, diabetes mellitus, renal disease, et cetera). Up to March 2002 66 patients were included.

All patients were resuscitated by the emergency physicians of the Department of Anaesthesiology and Intensive Care Medicine of the University of Bonn.

General management

The acute medical care, including basic and advanced life support was carried out by the EMS physicians and paramedics in accordance with the German- and ILCOR-guidelines 2000.2,24 When CA was confirmed an ECG was recorded and CPR was started. After cannulation of an external jugular vein the patients randomly received 2 ml/kg/10 min of either hypertonic saline with hydroxy ethyl starch (HHS; 7.2% NaCl with 6% hydroxy ethyl starch 200,000/0.5 [HES]) or HES in a blinded manner. After successful resuscitation the patient was admitted to an emergency hospital in Bonn. The intensive care after admittance followed standard protocols concerning ventilation/oxygenation, control of blood pressure and plasma glucose.

Blood analysis

Before and 10 min after infusion of the study solution 5 ml of venous blood was taken to measure haemoglobin, \(pO_2\), pH, \(HCO_3^{-}\), BE, plasma sodium and potassium concentrations. The blood was cooled within a transportable cool box and analysed later in the admitting hospital. Directly after admission to hospital 1 ml arterial blood was taken to measure blood gases, haemoglobin, glucose, plasma sodium and potassium concentrations.

Data evaluation

Time of emergency call reception and ambulance stop on arrival at the scene was documented precisely by the fire departments dispatch centre. The intervals between ambulance stop at the scene and patient contact and all important facts during CPR were taken from the defibrillator protocols. All other preclinical data concerning the patient were obtained by evaluating a standard protocol and interviewing the emergency physician after CPR. In case of a successful resuscitation one doctoral candidate was responsible for the collection of all clinical data in a defined Utstein styled study protocol.

Statistical analysis

This clinical investigation was designed as a pilot study, because to date there is no clinical experience published investigating the effects of hypertonic saline solutions during resuscitation from cardiac arrest. This first data will help to design an enlarged study powered to a primary endpoint of survival to hospital admission. All numerical data are expressed as means ± standard deviation (S.D.). Differences between groups and time points were analysed for significance by the \(x^2\)-test, Student’s \(t\)-test and by using a multivariate analysis of variance (MANOVA) with a repeated measures factor and a between group factor for treatment. For post-hoc analysis the Tukey HSD test was employed. Statistical significance was assumed for \(p < 0.05\). All data were computed with Microsoft® Excel 2000 for Windows, Statistica® (StatSoft, Inc.) and SPSS® for Windows (SPSS, Inc.). Randomisation was performed using a random number generator (Microsoft® Excel 2000).

Results

Patients

Within an observation period of 11 months 66 patients with out-of-hospital CA of non-traumatic origin were included into the study protocol. Four patients did not match the study criteria: in three cases duration of cardiac arrest was >15 min and one patient developed ROSC without adrenaline. The median age of the patients was equal in both groups (HHS 64 years, HES 65 years). In the HHS group 76% of patients were male and 24% female, in the HES group 70% of patients were male and 30% female (\(p = 0.58\)), (Table 1).
Both groups showed a similar distribution of determinants and external circumstances influencing resuscitation success (Table 1). After CA the infusion therapy with either 2 ml/kg HHS or HES was initiated within 13.2 ± 1.5 min in HHS group and within 14.0 ± 4.4 min in HES group (p = 0.48). The collapse was bystander witnessed in 75.8% of cases in HHS group and in 69.7% in HES group (p = 0.58). Bystander CPR was performed in 18.2% of the cases in the HHS group and in 15.2% of the cases in the HES group (p = 0.74). The average time from CA until the beginning of basic life support by the EMS personnel was comparable in both groups (HHS 8.0 ± 4.1 min; HES 7.4 ± 3.1 min; p = 0.51). In addition, the dose of adrenalin during CPR were similar in both groups (HHS 13.6 ± 10.0 mg; HES 13.0 ± 7.4 mg; p = 0.77). Twelve patients (36.4%) in HHS group and 11 patients (33.3%) in HES group received sodium-bicarbonate (8.4%) in a dosage of 50–100 mval (p = 0.80).

**Blood analyses**

After the establishment of a venous line, 10 min after infusion of the study solution and at arrival at the hospital blood samples were taken from each patient. Plasma sodium and potassium concentrations, haemoglobin concentration and blood gases were analysed (Tables 2 and 3). Before application of the study solution the mean plasma sodium concentration was in the normal range in both groups. After infusion of the study solution we found a significant increase in plasma sodium concentration in the HHS group (Table 2). But this peak was only short lasting and plasma sodium concentration returned to normal until admission to hospital (Table 3). In the HES group the plasma sodium concentration stayed within the normal range at all times (Figure 1). Starting from a discreet haemodilution at the beginning of CPR after infusion of the study solution in both groups a haemodilution was found, but in HHS group haemodilution was more pronounced and lasted longer (Figure 2). During CPR an increase of pO2 was apparent in both groups, but this increase was significant only in the HHS group (25.6–45.9 mmHg; p = 0.02). But at admission to hospital both groups showed similar pO2 values during ventilation with pure oxygen (214.8 mmHg in HHS group versus 173.6 mmHg in HES group; p = 0.62) indicating a minor pulmonary dysfunction similarly in both groups. No differences were found for the other determined values like pH, pO2, K+, HCO3 and BE (Tables 2 and 3).

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**Table 1**  Factors at resuscitation

<table>
<thead>
<tr>
<th></th>
<th>Overall (±SD)</th>
<th>HHS (±SD)</th>
<th>HES (±SD)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients age (years)</td>
<td>64.1 (±13.7)</td>
<td>64.7 (±14.1)</td>
<td>63.5 (±13.5)</td>
<td>0.73</td>
</tr>
<tr>
<td>Gender of patients (%)</td>
<td>m = 73/f = 27</td>
<td>m = 76/f = 24</td>
<td>m = 70/f = 30</td>
<td>0.58</td>
</tr>
<tr>
<td>Down time of circulation (min)</td>
<td>7.7 (±3.6)</td>
<td>8.0 (±4.1)</td>
<td>7.4 (±3.1)</td>
<td>0.51</td>
</tr>
<tr>
<td>Rate of bystander witnessed CA (%)</td>
<td>72.7</td>
<td>75.8</td>
<td>69.7</td>
<td>0.58</td>
</tr>
<tr>
<td>Rate of bystander-CPR (%)</td>
<td>16.7</td>
<td>18.2</td>
<td>15.2</td>
<td>0.74</td>
</tr>
<tr>
<td>Infusion after CA (min)</td>
<td>13.6 (±4.7)</td>
<td>13.2 (±5.1)</td>
<td>14.0 (±4.4)</td>
<td>0.48</td>
</tr>
<tr>
<td>Dosage of epinephrine (mg)</td>
<td>13.3 (±8.8)</td>
<td>13.6 (±10.0)</td>
<td>13.0 (±7.4)</td>
<td>0.77</td>
</tr>
<tr>
<td>Rate of natriumbicarbonate (%)</td>
<td>34.9</td>
<td>36.4</td>
<td>33.3</td>
<td>0.80</td>
</tr>
<tr>
<td>Rate of defibrillation by paramedic (%)</td>
<td>19.7</td>
<td>21.2</td>
<td>21.2</td>
<td>0.76</td>
</tr>
<tr>
<td>Rate of VF (%)</td>
<td>42.4</td>
<td>42.4</td>
<td>42.4</td>
<td>1.00</td>
</tr>
</tbody>
</table>

Mean ± standard deviation or percentage; n = 33 in each group; p value calculated by t-test or χ²-test.

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**Table 2**  Blood variables before and 10 min after infusion of study solution

<table>
<thead>
<tr>
<th></th>
<th>HHS before infusion</th>
<th>HHS after infusion</th>
<th>HES before infusion</th>
<th>HES after infusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na⁺ (mmol/l)</td>
<td>140.9 ± 8.2</td>
<td>168.1 ± 29.2</td>
<td>138.5 ± 6.9</td>
<td>139.0 ± 15.6</td>
</tr>
<tr>
<td>K⁺ (mmol/l)</td>
<td>5.2 ± 1.5</td>
<td>4.5 ± 1.6</td>
<td>5.3 ± 1.8</td>
<td>4.6 ± 1.7</td>
</tr>
<tr>
<td>pH</td>
<td>7.04 ± 0.11</td>
<td>7.00 ± 0.12</td>
<td>7.07 ± 0.18</td>
<td>7.00 ± 0.19</td>
</tr>
<tr>
<td>Hb (g/dl)</td>
<td>15.7 ± 3.8</td>
<td>12.7 ± 3.4</td>
<td>14.4 ± 3.6</td>
<td>12.5 ± 3.1</td>
</tr>
<tr>
<td>pO₂ venous (mmHg)</td>
<td>25.6 ± 10.3</td>
<td>45.9 ± 35.3</td>
<td>33.8 ± 25.0</td>
<td>49.5 ± 37.2</td>
</tr>
<tr>
<td>HCO₃ (mmol/l)</td>
<td>20.6 ± 3.4</td>
<td>17.1 ± 3.9</td>
<td>21.7 ± 5.8</td>
<td>19.3 ± 10.7</td>
</tr>
<tr>
<td>BE (mmol/l)</td>
<td>−12.3 ± 4.7</td>
<td>−15.5 ± 5.4</td>
<td>−11.1 ± 6.6</td>
<td>−15.2 ± 7.3</td>
</tr>
</tbody>
</table>

Blood analysis of patients receiving either HHS or HES during CPR; n = 33 in each group; MANOVA: *p < 0.05 (after vs. before infusion); †p < 0.05 (HHS vs. HES).
Table 3  Blood variables at admission to hospital

<table>
<thead>
<tr>
<th>Variable</th>
<th>HHS</th>
<th>HES</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na⁺ (mmol/l)</td>
<td>147.4 ± 5.5</td>
<td>140.1 ± 3.2</td>
<td>0.002</td>
</tr>
<tr>
<td>K⁺ (mmol/l)</td>
<td>3.8 ± 0.5</td>
<td>3.5 ± 0.3</td>
<td>0.26</td>
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<tr>
<td>pH</td>
<td>7.22 ± 0.13</td>
<td>7.25 ± 0.08</td>
<td>0.50</td>
</tr>
<tr>
<td>Hb (g/dl)</td>
<td>13.8 ± 2.1</td>
<td>13.9 ± 1.9</td>
<td>0.97</td>
</tr>
<tr>
<td>pO₂ (mmHg)</td>
<td>214.8 ± 193.1</td>
<td>173.6 ± 132.4</td>
<td>0.62</td>
</tr>
<tr>
<td>HCO₃ (mmol/l)</td>
<td>17.9 ± 4.2</td>
<td>19.2 ± 3.2</td>
<td>0.50</td>
</tr>
<tr>
<td>BE (mmol/l)</td>
<td>-9.5 ± 5.5</td>
<td>-7.5 ± 3.1</td>
<td>0.38</td>
</tr>
<tr>
<td>Hct (%)</td>
<td>41.7 ± 5.8</td>
<td>42.4 ± 5.4</td>
<td>0.78</td>
</tr>
<tr>
<td>pCO₂ (mmHg)</td>
<td>45.1 ± 7.4</td>
<td>45.1 ± 12.4</td>
<td>1.00</td>
</tr>
<tr>
<td>SO₂</td>
<td>96.6 ± 4.7</td>
<td>98.4 ± 2.0</td>
<td>0.63</td>
</tr>
<tr>
<td>Glucose (mg/dl)</td>
<td>298.2 ± 88.1</td>
<td>239.6 ± 70.0</td>
<td>0.14</td>
</tr>
</tbody>
</table>

Blood analysis of patients receiving either HHS or HES during CPR. n = 33 in each group; p value calculated by t-test.

Side effects

Severe side effects of hypertonic solution during CPR or during recirculation were not observed throughout the whole observation period. The increase in the plasma sodium concentration was only short lasting and moderate. Furthermore, no allergic reaction or pulmonary or cardiac dysfunction was observed after application of HHS.

CPR success rate

In both groups a remarkably high ROSC rate was observed. ROSC could be achieved in 22 of 33 patients receiving 2 ml/kg HHS (66.7%) and in 17 of 33 patients receiving 2 ml/kg HES (51.5%; p = 0.21) (Figure 3). The admission to hospital rate tended to be higher in the HHS group: 57.6% successful resuscitations in HHS group against 39.4% in the HES group (p = 0.14) (Figure 4). A pronounced benefit of HHS treatment was found in non VF patients and in patients with untreated cardiac arrest for longer than 6 min (44% admission to hospital rate in HHS group versus 21% in HES group for non-VF patients; p = 0.13; 57% admission to hospital rate in HHS group versus 29% in HES group in patients with untreated CA > 6 min; p = 0.06) (Figure 5).
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Discussion

To the best of our knowledge, this is the first human study investigating the infusion of hypertonic saline solution after out-of-hospital CA to test the safety and the feasibility of this new protocol of fluid management during CPR. We found that the infusion of 2 ml/kg/10 min HHS (7.2% NaCl with 6% hydroxyethyl starch 200,000/0.5) during CPR is safe and feasible. It is a simple and practicable method to improve reperfusion of the heart and the brain that can be applied within the usual resuscitation algorithm.

Negative side effects of hypertonic solution were not observed during CPR or the recirculation period and further clinical course. Anaphylactic reactions or negative effects on pulmonary function were not found. Even plasma sodium concentration was elevated only moderately and returned to normal within 30 min.

The volume effect of the investigated fluid management with hypertonic saline solution during CPR could not exceed 16 ml/kg bodyweight due to pharmacological reasons, started during haemoconcentration and is only short lasting, because it results from a fluid shift following the osmotic gradient. For induction of therapeutic hypothermia several authors published clinical data after the infusion of 2000—4000 ml cold saline solution. They did not find compromise in the cardiac function by this volume load. The volume effect observed after 2 ml/kg hypertonic saline therefore should not be a problem after resuscitation from cardiac arrest, but must be further investigated using echocardiography or transpulmonary indicator dilution methods. Nevertheless, due to the small number of patients treated, uncommon but serious adverse side effects may not be detected in this pilot study.

In addition, our preliminary data demonstrate that infusion of hypertonic saline during CPR might increase CPR-success rate, which indicates the safety of the used protocol. Both ROSC rate and admission to hospital rate (ROSC: 66.7% versus 51.5%; \(p = 0.21\); 57.6% versus 39.4%; \(p = 0.14\) tended to be higher in HHS group.

The feasibility and safety of HHS treatment was tested in this study in all patients suffering from CA irrespective of initial ECG-rhythm excluding only patients aged <18 years, weight >125 kg, duration of cardiac arrest >15 min and patients with traumatic origin of CA. Patients with ROSC without administration of any vasopressors were not included in the

![Figure 4](image1.png)

Figure 4 Admission to hospital rate (%) of patients receiving either HHS or HES during CPR; \(n = 33\) in each group; \(p\) calculated with \(\chi^2\)-test.

![Figure 5](image2.png)

Figure 5 Admission to hospital rate (%) in different subgroups. Patients receiving either HHS or HES during CPR; \(n = 33\) in each group; \(p\) calculated with \(\chi^2\)-test.
study because our intention was to investigate the improvement of resuscitation success rate by raising myocardial reperfusion during CPR.

In 1980 it was demonstrated that so called 'small volume resuscitation' was able to restore normovolaemia and to normalise nutritional blood flow after severe haemorrhagic shock. The application of this therapeutic measure to CPR is based on the findings that after CA the plasma volume shrinks to 62% leading to substantial hypovolaemia and haemoconcentration. Additionally it is known that reperfusion of the heart and the brain, which is disturbed by several factors, is the basic requirement for resuscitation success. The improvement of haemodynamics after CA by small volume resuscitation is based on different functional principles. The organ blood flow, and especially myocardial and cerebral blood flow during CPR increase because the osmotic gradient generated by 7.2% saline reduces endothelial cell swelling caused by hypoxic cell injury and reduce the leukocyte adherence at the endothelial wall. Both effects attenuate postischaemic microcirculatory disturbances like the cerebral no-reflow phenomenon after CA. Another beneficial factor is the reduction of postischaemic brain edema caused by the increase in brain tissue osmolality during circulatory arrest due to accumulation of osmotically active metabolites. Krieter et al. demonstrated that hypertonic solutions improve microvascular conductivity after ischemia and that an increase of troponin-I and astroglial S-100 β protein as indicators of myocardial and cerebral damage after ROSC was reduced significantly by using HHS. Those effects might support better myocardial and cerebral function after successful resuscitation. In different experimental models it was demonstrated that hypertonic solutions improves cerebral reperfusion during as well as after CPR. Therefore, infusion of hypertonic solution might be also a therapeutic option for patients with ROSC early after defibrillation without a long period of mechanical resuscitation.

In conclusion, our preclinical pilot trial demonstrated that infusion of a hypertonic solution during CPR is feasible and seemed to be safe. This new measure might be a therapeutic option for most of the patients suffering from sudden CA and not only for those patients with ventricular fibrillation. Several experimental studies clearly demonstrated that hypertonic saline will improve vital organ blood flow without negative side effects during CPR. The use of hypertonic solutions during CPR in human might also have a positive effect on resuscitation success and neurological recovery after CA. However, based on this data a randomised clinical trial powered to the primary endpoints of ROSC and survival to hospital admittance is planned. Further clinical studies investigating the impact of hypertonic saline solutions during CPR on long term survival and neurological outcome should be undertaken soon.

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