ELSEVIER

Resuscitation 66 (2005) 271–283

RESUSCITATION

www.elsevier.com/locate/resuscitation

Recommended guidelines for reviewing, reporting, and conducting research on post-resuscitation care: The Utstein style☆

Audun Langhelle[a,*], Jerry Nolan[b], Johan Herlitz[c], Maaret Castren[d], Volker Wenzel[e], Eldar Soreide[f], Johan Engdahl[g], Petter Andreas Steen[h],

on behalf of the participants at the 2003 Utstein Consensus Symposium[1]

[a] *Norwegian Air Ambulance, Box 39, N-1441 Drøbak, Norway*
[b] *Department of Anaesthesia and Critical Care Medicine, Royal United Hospital, Combe Park, Bath BA1 3NG, UK*
[c] *Division of Cardiology, Sahlgrenska University Hospital, S-413 45 Gothenburg, Sweden*
[d] *Uusimaa EMS, Kauniala hospital, Kylpylätie 19, 02700 Kauniainen, Finland*
[e] *Department of Anesthesiology and Critical Care Medicine, Innsbruck Medical University, Anichstrasse 35, 6020 Innsbruck, Austria*
[f] *Department of Anaesthesia and Intensive Care, Division of Acute Care Medicine, Stavanger University Hospital, N-4068 Stavanger, Norway*
[g] *Clinic of Medicine, Sahlgrenska Sahlgrenska University Hospital, S-413 45 Gothenburg, Sweden*
[h] *Department of Anaesthesiology and Emergency Medical Services, Ullevaal University Hospital, N-0407 Oslo, Norway*

Received 4 February 2005; accepted 9 June 2005

Abstract

The aim of this report is to establish recommendations for reviewing, reporting, and conducting research during the post-resuscitation period in hospital. It defines data that are needed for research and more specialised registries and therefore supplements the recently updated Utstein template for resuscitation registries. The updated Utstein template and the out-of-hospital ''Chain of Survival'' describe factors of importance for successful resuscitation up until return of spontaneous circulation (ROSC).

Several factors in the in-hospital phase after ROSC are also likely to affect the ultimate outcome of the patient. Large differences in survival to hospital discharge for patients admitted alive are reported between hospitals. Therapeutic hypothermia has been demonstrated to improve the outcome, and other factors such as blood glucose, haemodynamics, ventilatory support, etc., might also influence the result. No generally accepted, scientifically based protocol exists for the post-resuscitation period in hospital, other than general brain-oriented intensive care. There is little published information on this in-hospital phase.

This statement is the result of a scientific consensus development process started as a symposium by a task force at the Utstein Abbey, Norway, in September 2003. Suggested data are defined as core and supplementary and include the following categories: pre-arrest co-morbidity and functional status, cause of death, patients' quality of life, in-hospital system factors, investigations and treatment, and physiological data at various time points during the first three days after admission.

It is hoped that the publication of these recommendations will encourage research into the in-hospital post-resuscitation phase, which we propose should be included in the chain-of-survival as a fifth ring. Following these recommendations should enable better understanding of the impact of different in-hospital treatment strategies on outcome.

Keywords: Cardiac arrest; Cardiopulmonary resuscitation (CPR); Outcome; Post-resuscitation period; Quality of life; Utstein template

☆ A Spanish translated version of the abstract of this article appears as Appendix in the online version at 10.1016/j.resuscitation.2005.06.005.

* Corresponding author. Tel.: +47 64904450; fax: +47 64904451.
E-mail address: audun.langhelle@snla.no (A. Langhelle).

[1] Conference participants: Angela Bång, RN; Dag Jacobsen, MD; Freddy Lippert, MD; Hans Friberg, MD; Hans Rosén, MD; Kjetil Sunde, MD; Kristian Lexow, MD; Risto Roine, MD; Tom Silfvast, MD.

0300-9572/$ – see front matter © 2005 Elsevier Ireland Ltd. All rights reserved.
doi:10.1016/j.resuscitation.2005.06.005

1. Background

International consensus on how to report data from in- and out-of-hospital cardiac arrest (OHCA), the Utstein template, [1–3] has enabled better and more consistent evaluation of factors associated with survival [4–7] and comparisons between different services [8–10].

Only pre-hospital factors plus the ultimate outcome (survival to hospital discharge and later) were included in the initial Utstein template for OHCA. This is consistent with the four rings of the ''Chain of Survival'' [11] (1. early access; 2. early basic CPR; 3. early defibrillation; and 4. early advanced life support (ALS)), which are incorporated in international guidelines for CPR [12].

Several factors are likely to influence outcome after return of spontaneous circulation (ROSC) following either in- or OHCA. Pathophysiological factors associated with this post-ischaemic phase such as cerebral hypoperfusion, eicosanoid and free radical production have been studied extensively over the last decades [13]. Two randomised, controlled studies have demonstrated that therapeutic mild hypothermia improves neurological outcome of comatose survivors after ventricular fibrillation (VF) [14,15]; this treatment has been the subject of a recent advisory statement by the International Liaison Committee on Resuscitation (ILCOR) [16]. The influence of blood glucose after cardiac arrest has been studied recently [17,18], and tight control of blood glucose has been reported to improve outcome in critically ill patients [19].

A Norwegian multicentre study revealed striking discrepancies in survival between four hospital regions that could not easily be explained by prehospital factors, and in-hospital factors like lower body temperature, lower blood glucose and absence of seizure were associated with survival [10]. There was a significant difference in survival after OHCA between two hospitals in Gothenburg, Sweden that received their patients from the same pre-hospital system [20].

The importance of these factors prompts us to propose the addition of a fifth ring, post-resuscitation care (Fig. 1), to the ''Chain of Survival''. The idea is not new; the hospital ring was included by Niemann [21] and more recently by Engdahl et al. [20]. Except for the few studies above, there is a paucity of data reported from this in-hospital phase, and no generally accepted, scientifically based protocol exists, other than general brain-oriented intensive care. We must learn how these patients are treated and what factors in this phase affect the outcome.

The considerable variation across the world in survival of patients admitted alive to hospital after OHCA indicates the importance of collecting data on in-hospital treatment. In Sweden, this survival ranged from 13 to 60% [22]. By paying greater attention to post-resuscitation care and identifying the interventions that improve outcome it is hoped that this variability can be reduced. The Hawthorne effect implies that merely focusing on the post-resuscitation care phase should improve outcome by approximately 30% [23,24]. Considerable resources are used in this post-ROSC phase and it is appropriate to question whether they are being used effectively. In a recent study, half the total health care costs for out-of-hospital cardiac arrest patients were incurred in the intensive care units [25].

Physicians' knowledge of post-resuscitation care is often inadequate: some believe that a patient admitted with fixed, dilated pupils has irreversible brain damage and assume medical treatment to be futile, although current knowledge indicates that the negative predictive value of any clinical sign is too low during the first 48 h [26]. Clinicians need better guidelines, including prognostication, which are based on scientific evidence, and not merely general advice on brain oriented intensive care.

Thus, in conjunction with the 6th Scandinavian Congress on Resuscitation, a group of experts gathered at Utstein Abbey (Norway) in September 2003 and, by consensus, established recommendations for reviewing, reporting, and conducting research on post-resuscitation care. This document is meant as a supplement to the recently updated Utstein template for resuscitation registries [2]. It defines data that are needed for research and more specialised registries. It is therefore, more detailed with more data defined as core elements than the updated Utstein template, which is directed more towards routine registries. The development of a template (available in electronic format, Appendix C) for collecting and recoding data in the post-resuscitation phase will enable comparisons between trials and institutions. This will help to determine the impact of different treatment strategies on outcome.

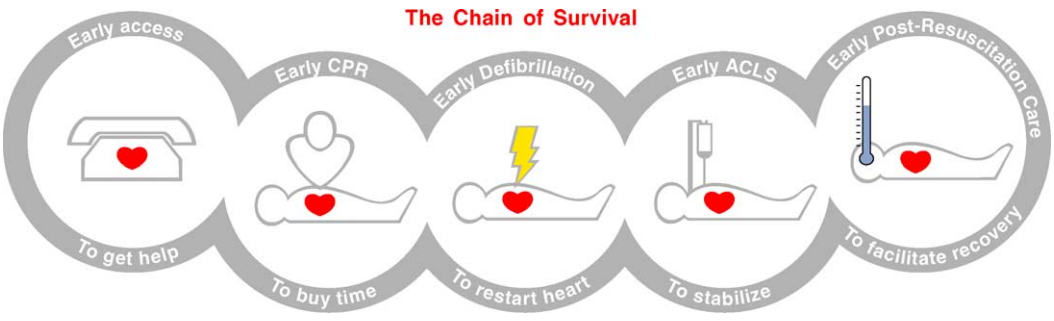

Fig. 1. Updated ''Chain of Survival'' with a 5th ring (Post-resuscitation Care).

Table 1
Premorbid status, core data (C) and supplementary data (S)

	Core/ supplementary
Best CPC and OPC before cardiac arrest	C
Previous cardiac arrest	S
Acute myocardial infarction	S
Angina pectoris	S
Congestive heart failure, classified according to NYHA classification (scale)	S
Supraventricular and ventricular arrhythmias	S
Stroke	S
Hypertension	S
Diabetes mellitus (denote IDDM or NIDDM)	S
Lung disease	S
Neurological disease (previous stroke to be reported separately)	S
Renal disease	S
Liver cirrhosis	S
Current smoking	S
Obesity (BMI > 30 kg m^{-2})	S
Alcohol/drug abuse	S
DNAR order	S
Healthy	S

CPC: cerebral performance category; OPC: overall performance category. NYHA: New York heart association; IDDM: insulin dependent diabetes mellitus; NIDDM: noninsulin-dependent diabetes mellitus; BMI: Body Mass Index; and DNAR: do-not-attempt-resuscitation. Supplementary data: denote yes/no unless otherwise stated. A disease should be denoted if it is currently pharmacologically or previously surgically treated or subjected to continuous supervision. If the patient was previously healthy, this should be denoted separately.

Table 2
Outcome variables, core data (C) and supplementary data (S)

Data	Core/ supplementary
In hospital	
Date of death	C
Hours after admission if <24 h	C
After discharge	
Date of death	C
During hospital stay	
Best achieved CPC	C
Best achieved OPC	C
At discharge	
CPC	C
OPC	C
Discharge destination (home, other prearrest residence, rehabilitation facility, extended care facility (nursing home), and other duration of hospitalization.	C
Neurological evaluation(including GCS)	S
On admission	
48 h after admission	
96 h after admission	
CPC/OPC 1 year after index event	S
Quality of life	S

CPC: cerebral performance category; OPC: overall performance category; and GCS: glasgow coma score.

2. Prehospital phase and outcome (Tables 1 and 2)

These guidelines should be integrated with the Utstein guidelines for OHCA [1], which have been updated recently [2]. We have made no attempt to redefine or add pre-hospital factors, except that we suggest pre-arrest co-morbidity as Supplementary data, and pre-arrest categorisation of functional status similar to the categories used for outcome evaluation as core data.

Data on the influence of co-morbidity on survival are scarce. A history of congestive heart failure pre-arrest was found to be a strong negative predictor in patients with VF [27]. An index of co-morbidity strongly associated with outcome has been established ($P = .004$) [28], but when analyzing a comprehensive set of predictors of survival after out-of-hospital VF, including this index of co-morbidity, only 25% of the variability was accounted for [28]. There are data on the influence of co-morbid conditions in the in-hospital setting of cardiac arrest [29,30].

To enable linkage of data in larger databases, researchers are encouraged to collect co-morbidity data according to Table 1; these include cardiovascular diseases and risk factors along with relevant other chronic diseases. The date and time of death (before or after discharge), the presence of a do-not-attempt-resuscitation (DNAR) order, and time of discharge and destination should be recorded. The principle cause of death should be reported as cardiac, neurological, trauma, or other and, ideally, defined by an International Classification of Diseases (ICD)-10 code [31]. Two thirds of in-hospital deaths after OHCA are associated with brain damage [32,33].

In addition to the outcome measures that are recommended in the standard Utstein guidelines, we encourage researchers to also attempt to record patients' quality of life (QOL) [34]; this is an increasingly used and accepted measure of ICU outcome and effectiveness of care. One of three QOL scales can be used: the EuroQual-5D (EQ-5D), is a short generic health related QOL-measure consisting of five questions, created in 1990 by the international Euro Qol Group [35]. The RAND 36 is generic and health related and consists of 36 questions with two to six alternatives [36]. The RAND 36-questionnaire may be most suitable, as it gives more precise information and detects minor differences; however, the EQ-5D is easier to use [37]. A more precise instrument is 15D [38], which is particularly valid for deriving quality-adjusted life years (QALYs) gained; thus, it enables decisions on allocation of resources. It is a generic, self-administered instrument and its health state descriptive system consists of 15 dimensions. For each dimension, the respondent selects one of the five levels that best describes his/her state of health. The single index score (15D score) on a 0–1 scale, representing the overall health related QOL, is calculated from the health state descriptive system by using a set of population-based preference or utility weights.

A 1 year follow up may be too soon for reliable assessment of neurological recovery; patients often need a longer time

to become accustomed to new, often more restricted circumstances [39]. The optimal time for follow up is unknown.

3. Hospital variables

In-hospital systems and treatment variables are likely to affect outcome after out-of-hospital cardiac arrest. Researchers are encouraged to report data describing the hospital and its available personnel, equipment and routines along with the demographics of the catchment area (Table 3). The existence of a written instruction or policy for patient allocation (e.g. age) should be recorded if the EMS transports patients to more than one hospital. Any policy for the distribution of patients in-hospital should also be recorded. In many systems, the in-hospital pathways for these patients vary greatly, and may be based more often on inaccurate prognostication by attending physicians [29], or lack of intensive care resources, rather than a systematic approach. Some patients might be admitted to the ICU for mechanical ventilation and therapeutic hypothermia, etc., while others get admitted to a less intensive coronary care or intermediate care unit. Some are taken to the catheter laboratory for acute percutaneous coronary intervention (PCI), while in other hospitals this service may not be available. Some patients are left intubated, breathing spontaneously; others are extubated in the emergency department and either remain there or go to a standard ward. Although very likely to affect patient outcome, the basis for decisions on patient destination or the withdrawal or non-initiation of active therapies are often not recorded. These data should be recorded. We encourage researchers to undertake detailed studies on the decision processes, paying particular attention to ethical, logistical, and financial issues and the problems of self-fulfilling prophesies associated with futility decisions.

In keeping with previous Utstein recommendations, data from the in-hospital phase are divided into core and supplementary [40]. Core data are deemed essential and enable comparative audit; Supplementary data will be valuable for higher level research studies. The data delineated in this post resuscitation template should be collected in addition to those obtained at the time of cardiac arrest and indicated in the revised Utstein template [2]. The post resuscitation data are divided into physiological systems, consistent with the approach normally taken by critical care physicians. Recommendations have also been made on the frequency of recording. For simplicity, the time periods are divided into arrival at hospital (defined as the first hour after arrival), 1–24, 24–48, and 48–72 h (Table 4). Some data are collected only once (e.g. tracheotomy). Factors on hospital arrival associated with increased hospital mortality are adverse neurological status [41,42], hypotension [43], cardiogenic shock after defibrillation [44], and a requirement for mechanical ventilation [41].

In institutions with sophisticated information technology systems, much data can be captured electronically. With more modest systems, data must be recorded and entered into a database manually. The ultimate goal is data collection on a national and international basis enabling comparisons using large numbers of patients. Such a system has been developed very successfully by the Intensive Care National Audit and Research Centre (ICNARC) in the United Kingdom (UK) [45]. This organisation now has a database of approximately 150,000 patients admitted to critical care units in the UK.

3.1. Airway

Core data on hospital arrival and in each time period should be recorded: intubated/not intubated, alternative airway device (e.g. laryngeal mask airway, laryngeal tube, Combitube), and misplaced tracheal tube (oesophageal, supraglottic, right bronchial stem). The need for tracheotomy and days on ventilator (defined as hours on ventilator divided by 24) are core data items.

The time and location (prehospital, emergency department (ED), coronary care unit (CCU)/intensive care unit (ICU)/high dependency unit (HDU), ward) of patient at extubation are supplementary and core, respectively.

3.2. Breathing

Core data on hospital arrival and in each time period should be recorded: the existence and highest rate of spontaneous breathing, with or without assisted ventilation; the fraction of inspired oxygen (FiO_2, a table is provided for estimating the FiO_2 in patients breathing oxygen from a standard mask); documented episodes of oxygen desaturation ($SaO_2 < 85\%$); arterial blood gases (highest and lowest values for pH, $PaCO_2$ and PaO_2 and lowest values for base excess). Data such as these are already collected by many critical care units to enable calculation of sickness severity scores [e.g. acute physiology and chronic health evaluation (APACHE II) and Simplified Acute Physiology Score (SAPS II)] [46,47]. APACHE II score, APACHE II mortality risk and SAPS II are valuable Supplementary data that should be collected whenever possible. The highest lactate in each of the time periods is Supplementary data because it is not measured in all institutions. Prolonged hypoxaemia is likely to contribute to poor neurological outcome after cardiac arrest, and the mode of ventilation influences outcome in some critically ill patients and should be documented (Supplementary) at each time period [48]. The tidal volume (ml kg^{-1} ideal body weight) and PEEP strategy used in the ICU should be documented; it is not realistic to expect these data for individual patients. The presence of a pneumothorax, haemothorax, pulmonary oedema, or evidence of aspiration, such as infiltrates on the chest radiograph and gastric contents in the trachea, on the first post resuscitation chest radiograph are core items.

3.3. Circulation

Core data on hospital arrival and in each time period should be recorded: mean arterial blood pressure; cardiac rhythm and

Table 3
Checklist of information to include in reports on hospital treatment of survivors from out-of-hospital cardiac arrest

Data	Core (C)/supplementary (S)
Hospital factors	
Care level of the hospital (primary, secondary, or tertiary)	C
Somatic beds (N)	C
Total beds (N) allocated to Intensive care units (ICU) Coronary care units (CCU) Other monitored wards General wards	C
Number of hospitals in the EMS system if more than one.	C
If more than one hospital are served by the EMS system, any system or policy for patient allocation should be described.	C
The percentage of the population served by the EMS system, geographic area served (in square kilometers), and percentage of the total population more than 65 years old.	C
Educational level: average level of education, percentage of persons who continue their education past the compulsory school level, or both	S
Socioeconomic status: percentage of persons below the poverty level (the definition of this poverty level must be stated for each community)	S
The percentage of the population in each of the following age groups should be stated: 0–12 months, 1–4 years, 5–14 years, 15–24 years, 25–34 years, 35–44 years, 45–54 years, 55–64 years, 65–74 years, 75–84 years, and more than 85 years.	S
Total number of annual deaths in the community	S
Percentage of deaths attributable to ischemic or coronary heart disease (International Classification of Diseases, tenth edition [31] codes I20–I25)	S
Personnel factors	
Denote the following disciplines if they are represented in the hospital: anesthesiology, emergency medicine, cardiology, neurology, rehabilitation medicine and thoracic surgery.	C
Description of the number of and general training level of the personnel that primarily receives the patient in the emergency department and if that level varies over the day. If key personnel are available only on call within the hospital or from home, this should be stated.	C
If an emergency team is alerted when the patient arrives, the composition of this team and its routines should be described.	C
Denote the CPR and ACLS training level of the ED personnel.	C
Routines	
The use of prehospital thrombolysis and transmission of 12–14 lead ECG from EMS vehicle to hospital should be described.	C
If the hospital has guidelines for postresuscitation care, these should be briefly described including DNAR policy.	C
Description of how and at what stage the EMS alert the hospital/emergency department (ED) prior to arrival with a resuscitated cardiac arrest patient	C
Description of how the ED is activated by the above mentioned alert.	C
Denote who is formally responsible for the care of the patient and if this is the same person who receives the patient in the ED	C
It should be described whether patients with presumed cardiac etiology are reviewed by a cardiologist and at what stage.	S
The use of neurological prognostication should be described.	S
If there is a policy for patient allocation to the ICU or CCU, it should be stated.	S
Equipment	
Describe the ED by stating total number of beds and number of monitored beds. The ED is supposed to have facilities for monitoring of cardiac rhythm, capnography, blood gas, electrolytes and glucose measurement and invasive blood pressure. If some of these facilities are lacking in the ED, this should be described.	S
Describe the intensive and coronary care units with regard to number of beds, monitoring facilities, staff density and number of beds with mechanical ventilation.	S
If there are facilities for PCI and/or invasive electrophysiological investigation, this should be stated. The availability of acute PCI and hours of availability should also be stated.	S

EMS: emergency medical service; DNAR: do-not-attempt-resuscitation; and PCI: percutaneous coronary intervention.

arrhythmias requiring treatment: highest and lowest values for heart rate (core on arrival and supplementary at subsequent intervals). The presence of a central venous catheter is a core item and documentation of the highest and lowest central venous pressure at each time period is Supplementary data. The presence of cardiac output (CO) monitoring and the method used is a core item. Highest and lowest values for Cardiac Index are Supplementary data. The role of the pulmonary artery catheter in the treatment of critically ill patients in general, and in the post resuscitation phase specifically, is controversial [49–52]. For CO monitoring, pulse contour continuous cardiac output (PiCCO) or lithium dilution measurement of cardiac output (LiDCO) are options [53,54] The occurrence of myocardial infarction (as defined by the European Society of Cardiology in Appendix A) and reinfarction are core data [55] as well as the way they are treated [56]. The use of fluid is core data, while the specific type of fluid (crystalloids, colloids) and fluid balance (intravenous and enteral

Table 4
Template for collecting and recoding data in the post-resuscitation phase

System	Description	0–1 h	1–24 h	24–48 h	48–72 h	Single item
Airway						
Intubated	Yes/no	C	C	C	C	
Duration (days) on ventilator	Hours/24					C
Time of extubation	Date/hour					S
Location of patient at extubation	Describe					S
Other airway device	Describe	C				
Tracheostomy	Yes/no					C
Misplaced tracheal tube	Yes/no	C				
Breathing						
Spontaneous breathing	Yes/no	C	C	C	C	
Spontaneous respiratory rate	Highest value	C	C	C	C	
Mode of ventilation	Describe mode	S	S	S	S	
Neuromuscular blockers	Yes/no	C	C	C	C	
Arterial blood gases						
FiO_2	Highest value	C	C	C	C	
PEEP	Value	C	C	C	C	
CPAP	Value	C	C	C	C	
pH	Highest/lowest values	C	C	C	C	
PO_2	Highest/lowest values	C	C	C	C	
PCO_2	Highest/lowest values	C	C	C	C	
HCO_3	Lowest value	C	C	C	C	
BE	Lowest value	C	C	C	C	
Lactate	Highest value	S	S	S	S	
SpO_2	Episodes <85%	C	C	C	C	
Supplemental oxygen after discharge from ICU	Yes/no					C
Blood gas measurements	Number	S	S	S	S	
Capnography	Yes/no					S
Chest radiograph						
Pneumothorax	Yes/no	C				
Haemothorax	Yes/no	C				
Pulmonary oedema	Yes/no	C				
Evidence of aspiration						
Infiltrates on CXR, gastric contents in trachea	Yes/no	C				
Complications						
Accidental extubation	Yes/no					C
Need for reintubation	Yes/no					C
Circulation						
Rhythm	Describe	C				
Arrhythmias requiring treatment	Describe (ICD, pacemaker, drug)	C	C	C	C	
Rate	Highest/lowest values	C	C	C	C	
Blood pressure						
MAP (measured or calculated)	Highest/lowest values	C	C	C	C	
Episode(s) of hypotension (>10 min of MAP <60 mmHg)	Yes/no	C	C	C	C	
CVP monitoring	Yes/no					C
CVP	Highest/lowest values		S	S	S	
Cardiac output monitoring	Yes/no/method					C
Cardiac Index	Highest/lowest values		S	S	S	
Invasive blood pressure	Yes/no					S
Pulmonary artery catheter	Yes/no					S
Myocardial infarction/reinfarction	Yes/no					C
S-cardiac troponin T	Value					C
ST-T wave changes on ECG	Yes/no					C
New Q-waves on ECG	Yes/no					C

Table 4 (*Continued*)

System	Description	0–1 h	1–24 h	24–48 h	48–72 h	Single item
Volume	Yes/no					C
Type	List					S
Inotropes	Yes/no					C
Type	List					S
Vasopressor	Yes/no					C
Type	List					S
IABP	Yes/no					C
Echocardiography						
Ejection fraction (%)	Value closest to hospital discharge or death					C
Clinical evidence of cardiac failure	Yes/no					C
Holter monitoring	Yes (time)/no					S
Electrophysiological testing	Yes (time)/no					S
Thrombolysis	Yes (time)/no					C
Coronary angiography	Yes (time)/no					C
PCI	Yes (time)/no					C
CABG	Yes (time)/no					C
Anticoagulants	Yes (time)/no					C
Antiplatelets	Yes (time)/no					C
IABP	Yes/no					C
	Time/duration					S
ECMO	Yes/no					C
	Time/duration					S
CVVHF	Yes/no					C
	Time/duration					S
Neurological						
RLS						
Extend to 96 hours	Best response	C	C	C	C	
GCS						
Extend to 96 hours	Best response (eyes, motor and verbal	C	C	C	C	
Pupillary light response	Yes/no	C	C	C	C	
Corneal reflex	Yes/no	S	S	S	S	
Following eye movements	Yes/no	S	S	S	S	
Involuntary movements	Yes/no	C	C	C	C	
Seizures	Yes/no	S	S	S	S	
Myoclonus	Yes/no	S	S	S	S	
Extension	Yes/no	S	S	S	S	
S 100	Highest value	S	S	S	S	
EEG						
Grading scale			S	S	S	
SSEPs						
Describe type	N 70 Peak		S	S	S	
NSE	Highest value		S	S	S	
Best CPC during hospital						C
Best OPC during hospital						C
Time to follow verbal commands						
Recovery from coma	hours/days					C
Anticonvulsants	Yes/no					C
Type	Describe					S
Sedation						C
Drugs	List					S
Duration	Hours/days					C
Continuous monitoring	Yes/no					S

Table 4 (*Continued*)

System	Description	0–1 h	1–24 h	24–48 h	48–72 h	Single item
Analgesia						C
Drugs	List					S
Paralysis						C
Drug	List					S
Duration	Hours/days					C
Level of anxiety						
SAS, MAAS, VICS, RASS, BIS, other	Highest/lowest value					S
Stroke (during hospital stay)	Yes/no					C
Onset of symptoms						
Time	Date/hour					S
Type	Ischaemic, intracranial haemorrhage, iatrogenic unknown					S
Brain imaging	Yes/no Date					S
Transcranial doppler	Yes/no					S
Renal						
Renal replacement therapy	Yes/no					C
S-creatinine	Highest value					S
Urine output	24 hour value		S	S	S	
Diuretics	Yes/no		S	S	S	
Fluid balance	Value		S	S	S	
Metabolism						
Blood glucose	Highest/lowest value	C	C	C	C	
Insulin	Yes/no		C	C	C	
B-glucose measurements	Number	S	S	S	S	
Body temperature						
Core temp	Highest/lowest value	C	C	C	C	
Therapeutic hypothermia	Yes/no					C
Cooling technique	Describe					C
Start time	Day/hour/min					C
Target temperature	°C					C
Time to reach target temp	Hours					C
Time at target temperature	Hours					C
Rewarming time	Hours					C
Use of muclerelaxants						C
Additional anti-pyrexial therapy			S	S	S	
Nutrition first 72 hours						
Attempt enteral feed	Yes/no					C
Parenteral nutrition						C
Infection						
Sepsis						
Defined in Appendix B	Yes/no					C
Severe sepsis						
Defined in Appendix B	Yes/no					C
Septic Shock						
Defined in Appendix B	Yes/no					C
C-reactive protein	Highest at any time					C
Pneumonia						
Defined in Appendix B	Yes/no					C
Other						
S-haemoglobin	Highest/lowest	C	C	C	C	
S-sodium	Highest/lowest	C	C	C	C	
S-potassium	Highest/lowest	C	C	C	C	

Table 4 (*Continued*)

System	Description	0–1 h	1–24 h	24–48 h	48–72 h	Single item
S-magnesium	Lowest					S
S-phosphate	Lowest					S
Thrombo-prophylaxis	Yes/no					C
Stress ulceration prophylaxis	Yes/no					C
APACHE II Score						S
APACHE II Mortality risk						S
SAPS II						S
Cause of cardiac arrest						
Cardiac/non-cardiac etiology	Select from list					C
Time points						
Admission to ED	Day/hour/min					C
Admission to ICU	Day/hour/min					C
Discharge from ICU	Month/day					C
Total days of hospitalisation						
Discharge from hospital	Month/day					C
Assumed cause of death	Select from list					C
Autopsy	Yes/no					C

C: core data; S: supplementary data; CXR: chest X-ray; ICU: intensive care unit; PEEP: positive end expiratory pressure; CPAP: continuous positive air pressure; IABP: intra-arterial balloon pump; PCI: percutaneous coronary intervention; CABG: coronary artery bypass graft; MAP: mean arterial pressure; CVP: central venous pressure; ICD: internal converting defibrillator; ECMO: extra corporeal membrane oxygenation; CVVHF: continuous venovenous haemofitration; RLS: Reaction Level Scale; GCS: Glasgow Coma Scale; EEG: electroencephalogram; SSEP: somatosensory evoked potential; NSE: neuron-specific enolase; SAS: Sedation-Agitation Scale; MAAS: Motor Activity Assessment Scale; VICS: Vancouver Interaction And Calmness Scale; RASS: Richmond Agitation-Sedation Scale; BIS: Bispectral Index; CPC: cerebral performance category; OPC: overall performance category; APACHE: acute physiology and chronic health evaluation; SAPS: simplified acute physiology score; and ED: emergency department.

intake versus urine output and other losses) during the hospital stay should be documented as Supplementary data. Use and type of inotropes and vasopressors and the use of an intra-aortic balloon pump (IABP) are core data. The value of the ejection fraction recorded by echocardiography first 24 h and at the closest time to discharge should be recorded (if available), and clinical evidence of heart failure are core items.

3.4. Neurological

Attempting to predict the final neurological outcome of the patient who has been resuscitated after cardiac arrest is fraught with difficulties [26,57–62]. Although previous publications suggest that prognostication with a fairly high accuracy can be achieved 48–72 h after arrest [26], these prognostic indicators must be modified if therapeutic hypothermia has been used [63]. Accurate collection and documentation of neurological data in the post resuscitation phase is vitally important if reliable prognostic indicators are to be developed. The best Glasgow Coma Scale (GCS) score or Reaction Level Scale (RLS) [64,65] score should be recorded for each time period (core). As the score is valid only in the absence of sedation, the GCS (documenting the separate components) or RLS must be recorded before giving sedative drugs. Documentation of the GCS and RLS should be extended to 96 h after cardiac arrest.

The best cerebral performance category (CPC) achieved during the patient's hospital stay and at hospital discharge must be documented (core). The presence of pupil response to light is documented for each time period (core), as are the presence of a corneal reflex (supplementary) and following eye movements (supplementary). The time taken to follow verbal commands (i.e. patient looking at you when spoken to or obeying orders regarding limb movements) is a core item that represents the time taken to recover from coma. The presence of any involuntary movements is recorded for each time period (core). These are further defined by type (supplementary): seizures, myoclonus and extension movements. The types of anticonvulsants, sedative drugs and neuromuscular blocking drugs used should be documented (supplementary) along with the duration of sedative infusions and neuromuscular blockade (core). The use of continuous sedation monitoring and level of anxiety [66–72] should also be documented (supplementary). Stroke may be a complication of treatment and should be recorded as a core item [73]; cause and time of onset are supplementary items.

Electroencephalogram (EEG) and peak somatosensory evoked potentials (SSEPs) data are supplementary [59,74–76]. Protein S-100 (a glial protein) and serum neuron-specific enolase (NSE) have some prognostic value, but are supplementary as most institutions do not measure them [77,78]. The value of these investigations is probably also modified or negated by therapeutic hypothermia [79].

3.5. Renal

The treatment of impending renal failure may influence the outcome significantly in the post-resuscitation phase. The need to start renal replacement therapy at any time is a core item, while urine output, daily fluid balance (intravenous and enteral intake versus diuresis and other losses), the highest value for creatinine and use of diuretics are supplementary.

3.6. Metabolic

Treatment of a variety of metabolic disturbances that may occur after cardiac arrest might impact significantly on neurological outcome. Hyperthermia worsens neurological outcome [80], while mild therapeutic hypothermia improves neurological outcome in patients remaining comatose after initial resuscitation from ventricular fibrillation (VF) cardiac arrest, and possibly other resuscitated patients [14–16,81]. Optimal target temperature, duration of cooling and rate of rewarming have yet to be determined [82]. This makes collection of data on this intervention particularly important: start time, target temperature, time to reach target temperature, time at target temperature, rewarming time, cooling technique (external cooling (specify) versus invasive) and use of neuromuscular blockers are all core data. Lowest and highest values for core temperature should be recorded in each time period (core). Bladder temperature is easy and is probably best; alternatives are rectal, tympanic (infrared or thermistor), or nasopharyngeal. Use of additional antipyretic therapy (chlorpromazine, paracetamol, others) is Supplementary data.

Tight control of blood glucose using insulin reduces mortality in critically ill patients [19,83,84]. High blood glucose concentrations in the post-resuscitation are associated with mortality and poor neurological recovery [10,18,85–88]. Hyperglycaemia after stroke is associated with infarct expansion and worse neurological outcome [89]. In an animal model, insulin attenuated ischaemic brain injury, independent of its hypoglycaemic effect [90]. Control of blood glucose is likely to have a significant impact on neurological outcome after cardiac arrest. For this reason, documentation of highest and lowest values of blood glucose and the need for insulin in each time period are core data. The strategy used by the unit for blood glucose control should be described [91].

Attention to nutrition is an essential component of the treatment of critically ill patients [92]. Documentation of an attempt to feed enterally and/or the use of parenteral nutrition in the first 72 h should be recorded (core).

3.7. Infection

The incidence of infection will be affected by the quality of treatment and infection control as well by patient factors. Infection can impact negatively on outcome. The presence of sepsis or pneumonia should be documented (core). The definitions are those proposed by the American College of Chest Physicians/Society of Critical Care Medicine Consensus Conference in 1992 [93] (Appendix B). The use of therapeutic hypothermia may increase the risk of pneumonia [94]. Strategies to prevent hospital-associated pneumonia (HAP) and ventilator-associated pneumonia (VAP) are essential [95]. The highest value of C-reactive protein should also be documented in every time period (core). The use of antibiotics are core data, the duration and type are supplemental.

3.8. Other data

Highest and lowest values for haemoglobin, sodium, and potassium should be recorded for each time period (core). Lowest values of magnesium and potassium during the patient stay in the ICU or first 72 h are Supplementary data.

The use of drugs for thromboprophylaxis and stress ulceration prophylaxis should also be documented (core).

4. Summary

Considerable efforts have been made the last few decades to describe factors associated with survival after out-of-hospital cardiac arrest (OHCA). International consensus on how to report pre-hospital and outcome data in OHCA, the Utstein template, has helped in these efforts and enabled comparison of different services. As several factors likely to influence outcome are not included in this template, particularly after return of spontaneous circulation and admission to hospital, we propose an extended list of data for reviewing, reporting, and conducting research in the post-resuscitation period. The complete list is too long for routine data collection and more suitable for research purposes. Following these recommendations should enable better understanding of the impact of different in-hospital treatment strategies on outcome. We recommend that the in-hospital post-resuscitation phase is included in the chain-of-survival as a fifth ring.

Two European registries, The Hypothermia After Cardiac Arrest Registry, (http://www.erchacar.org/index.html) and Northern Hypothermia Network, (www.scctg.org) both register patients in the post-resuscitation period after cardiac arrest, whether or not they are cooled. Through international collaboration and such registration many of the problems discussed in this article may be addressed in the future.

Acknowledgement

The symposium was supported by grants from the Laerdal Foundation of Acute Medicine.

Appendix A. European Society of Cardiology (ESC) definition of acute myocardial infarction (AMI) [55]

The diagnosis of AMI in connection with OHCA has impact on prognosis and initial treatment. A working definition for acute evolving myocardial infarction in the presence of clinically appropriate symptoms is:

1. Patients with ST-segment elevation, i.e. new ST-segment elevation at the J point with the cut-off points $\geq$0.2 mV in V_1 through V_3 and $\geq$0.1 mV in other leads, or
2. Patients without ST segment elevation, i.e. ST-segment depression or T wave abnormalities.

Clinically established myocardial infarction may be defined by any Q wave in leads V_1 through V_3, or Q wave $\geq$0.03 s in leads I, II, aVL, aVF, V_4, V_5 or V_6.

Myocardial infarction can be recognised when blood levels of biochemical markers are increased in the clinical setting of acute myocardial ischaemia. Current knowledge on biochemical markers of myocardial damage after cardiac arrest is limited and there are no accepted consensus [96–99]. The preferred biomarker for myocardial damage is cardiac troponin T.

Appendix B. Definitions for sepsis [93]

Infection is the inflammatory response to the presence of microorganisms or the invasion of normally sterile host tissue by those organisms.

(a) *Bacteraemia* is the presence of viable bacteria in the blood.
(b) *The Systemic Inflammatory Response Syndrome* (*SIRS*) is diagnosed when the patient exhibits two of the following four abnormalities:
   - Temperature > 38 °C or <36 °C.
   - Heart rate > 90 beats min^{-1}.
   - Respiratory rate >20 breaths min^{-1} or $PaCO_2$ < 4.3 kPa.
   - White blood cell count > 12 000 cells mm^{-3} or <4000 cells mm^{-3} or >10% immature cells (band forms).
(c) *Sepsis* is SIRS resulting from infection.
(d) *Severe sepsis* is sepsis associated with organ dysfunction, hypoperfusion or hypotension. Hypoperfusion and perfusion abnormalities may include, but are not limited to, lactic acidosis, oliguria or an acute alteration in mental status.
(e) *Septic shock* is sepsis with hypotension (systolic blood pressure <90 mmHg or a reduction of >40 mmHg from baseline) and perfusion abnormalities (see severe sepsis above) or the requirement for vasoactive drugs despite adequate fluid resuscitation in the absence of other causes for hypotension.

The diagnosis of nosocomial *pneumonia* is based on the following criteria:

1. New or progressive consolidation on the chest radiograph.
2. Fever
3. Leucocytosis
4. Purulent tracheobronchial secretions

Appendix C. Template

Template for collecting and recoding data in the post-resuscitation phase associated with this article can be found, in the online version, at 10.1016/j.resuscitation.2005.06.005.

References

[1] Cummins RO, Chamberlain DA, Abramson NS, et al. Recommended guidelines for uniform reporting of data from out-of-hospital cardiac arrest: the Utstein style. Task force of the American Heart Association, the European Resuscitation Council, the Heart and Stroke Foundation of Canada, and the Australian Resuscitation Council. Ann Emerg Med 1991;20:861–74.
[2] Jacobs I, Nadkarni V, Bahr J, et al. Cardiac arrest and cardiopulmonary resuscitation outcome reports: update and simplification of the Utstein templates for resuscitation registries: a statement for healthcare professionals from a task force of the International Liaison Committee on Resuscitation (American Heart Association, European Resuscitation Council, Australian Resuscitation Council, New Zealand Resuscitation Council, Heart and Stroke Foundation of Canada, InterAmerican Heart Foundation, Resuscitation Councils of Southern Africa). Circulation 2004;110:3385–97.
[3] Cummins RO, Chamberlain D, Hazinski MF, et al. Recommended guidelines for reviewing, reporting, and conducting research on in-hospital resuscitation: the in-hospital 'Utstein style'. A statement for healthcare professionals from the American Heart Association, the European Resuscitation Council, the Heart and Stroke Foundation of Canada, the Australian Resuscitation Council, and the Resuscitation Councils of Southern Africa. Resuscitation 1997;34:151–83.
[4] Valenzuela TD, Roe DJ, Cretin S, Spaite DW, Larsen MP. Estimating effectiveness of cardiac arrest interventions: a logistic regression survival model. Circulation 1997;96:3308–13.
[5] Larsen MP, Eisenberg MS, Cummins RO, Hallstrom AP. Predicting survival from out-of-hospital cardiac arrest: a graphic model. Ann Emerg Med 1993;22:1652–8.
[6] Montgomery WH, Brown DD, Hazinski MF, Clawsen J, Newell LD, Flint L. Citizen response to cardiopulmonary emergencies. Ann Emerg Med 1993;22:428–34.
[7] Callaham M, Madsen CD. Relationship of timeliness of paramedic advanced life support interventions to outcome in out-of-hospital cardiac arrest treated by first responders with defibrillators. Ann Emerg Med 1996;27:638–48.
[8] Eisenberg MS, Horwood BT, Cummins RO, Reynolds-Haertle R, Hearne TR. Cardiac arrest and resuscitation: a tale of 29 cities. Ann Emerg Med 1990;19:179–86.
[9] Herlitz J, Bahr J, Fischer M, Kuisma M, Lexow K, Thorgeirsson G. Resuscitation in Europe: a tale of five European regions. Resuscitation 1999;41:121–31.
[10] Langhelle A, Tyvold SS, Lexow K, Hapnes S, Sunde K, Steen PA. In-hospital factors associated with improved outcome after out-of-hospital cardiac arrest. A comparison between four regions in Norway. Resuscitation 2003;56:247–63.
[11] Cummins RO, Ornato JP, Thies WH, Pepe PE. Improving survival from sudden cardiac arrest: the ''Chain of Survival'' concept. A statement for health professionals from the Advanced Cardiac Life Support Subcommittee and the Emergency Cardiac Care Committee, American Heart Association. Circulation 1991;83:1832–47.
[12] International Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care - An International Consensus on Science. Resuscitation 2000; 46:1–448.
[13] Siesjo BK, Siesjo P. Mechanisms of secondary brain injury. Eur J Anaesthesiol 1996;13:247–68.
[14] Bernard SA, Gray TW, Buist MD, et al. Treatment of comatose survivors of out-of-hospital cardiac arrest with induced hypothermia. N Engl J Med 2002;346:557–63.
[15] Mild therapeutic hypothermia to improve the neurologic outcome after cardiac arrest. N Engl J Med 2002; 346:549–556.
[16] Nolan JP, Morley PT, Hoek TL, Hickey RW. Therapeutic hypothermia after cardiac arrest. An advisory statement by the Advancement Life support Task Force of the International Liaison committee on Resuscitation. Resuscitation 2003;57:231–5.

[17] Longstreth Jr WT, Diehr P, Inui TS. Prediction of awakening after out-of-hospital cardiac arrest. N Engl J Med 1983;308:1378–82.
[18] Skrifvars MB, Pettila V, Rosenberg PH, Castren M. A multiple logistic regression analysis of in-hospital factors related to survival at six months in patients resuscitated from out-of-hospital ventricular fibrillation. Resuscitation 2003;59:319–28.
[19] Krinsley JS. Effect of an intensive glucose management protocol on the mortality of critically ill adult patients. Mayo Clin Proc 2004;79:992–1000.
[20] Engdahl J, Abrahamsson P, Bang A, Lindqvist J, Karlsson T, Herlitz J. Is hospital care of major importance for outcome after out-of-hospital cardiac arrest? Experience acquired from patients with out-of-hospital cardiac arrest resuscitated by the same Emergency Medical Service and admitted to one of two hospitals over a 16-year period in the municipality of Goteborg. Resuscitation 2000;43:201–11.
[21] Niemann JT. Perfusing during cardiopulmonary resuscitation. In: Harwood AL, editor. Cardiopulmonary resuscitation. Baltimore: Williams & Wilkins; 1982. p. 34–54.
[22] Överlevnad. In: Herlitz, J, Holmberg, S, editors. Nationellt register för hjärtstopp utanfor sjukhus. Årsrapport 2002. Göteborg: OFTA, Grafiska AB; 2002.
[23] Roethlisberger FJ. Management and the worker. An account of a research program conducted by the Western Electric Company, Hawthorne Works, Chicago/by FJ Roethlisberger and William J Dickson; with the assistance and collaboration of Harold A Wright. Cambridge, MA: Harvard University Press; 1939.
[24] Campbell JP, Maxey VA, Watson WA. Hawthorne effect: implications for prehospital research. Ann Emerg Med 1995;26:590–4.
[25] Naess AC, Steen PA. Long term survival and costs per life year gained after out-of-hospital cardiac arrest. Resuscitation 2004;60:57–64.
[26] Edgren E, Hedstrand U, Kelsey S, Sutton-Tyrrell K, Safar P, BRCT I Study Group. Assessment of neurological prognosis in comatose survivors of cardiac arrest. Lancet 1994;343:1055–9.
[27] Hallstrom AP, Cobb LA, Swain M, Mensinger K. Predictors of hospital mortality after out-of-hospital cardiopulmonary resuscitation. Crit Care Med 1985;13:927–9.
[28] Hallstrom AP, Cobb LA, Yu BH. Influence of comorbidity on the outcome of patients treated for out-of-hospital ventricular fibrillation. Circulation 1996;93:2019–22.
[29] Schultz SC, Cullinane DC, Pasquale MD, Magnant C, Evans SR. Predicting in-hospital mortality during cardiopulmonary resuscitation. Resuscitation 1996;33:13–7.
[30] de Vos R, Koster RW, de Haan RJ, Oosting H, van der Wouw PA, Lampe-Schoenmaeckers AJ. In-hospital cardiopulmonary resuscitation: prearrest morbidity and outcome. Arch Intern Med 1999;159:845–50.
[31] International Statistical Classification of Diseases and Related Health Problems, 1989 Revision ed. Geneva: World Health Organization, 1992.
[32] Herlitz J, Ekstrom L, Wennerblom B, Axelsson A, Bang A, Holmberg S. Risk indicators for, and symptoms associated with, death among patients hospitalized after out-of-hospital cardiac arrest. Coron Artery Dis 1994;5:407–14.
[33] Laver S, Farrow C, Turner D, Nolan J. Mode of death after admission to an intensive care unit following cardiac arrest. Intensive Care Med 2004;30:2126–8.
[34] Gill TM, Feinstein AR. A critical appraisal of the quality of quality-of-life measurements. JAMA 1994;272:619–26.
[35] EuroQol—a new facility for the measurement of health-related quality of life. The EuroQol Group. Health Policy 1990; 16:199–208.
[36] Hays RD, Sherbourne CD, Mazel RM. The RAND 36-Item Health Survey 1.0. Health Econ 1993;2:217–27.
[37] Pettila V, Kaarlola A, Makelainen A. Health-related quality of life of multiple organ dysfunction patients one year after intensive care. Intensive Care Med 2000;26:1473–9.
[38] Sintonen H. The 15D instrument of health-related quality of life: properties and applications. Ann Med 2001;33:328–36.
[39] Kaarlola A, Pettila V, Kekki P. Quality of life six years after intensive care. Intensive Care Med 2003;29:1294–9.
[40] Cummins RO. The Utstein style for uniform reporting of data from out-of-hospital cardiac arrest. Ann Emerg Med 1993;22:37–40.
[41] Grubb NR, Elton RA, Fox KA. In-hospital mortality after out-of-hospital cardiac arrest. Lancet 1995;346:417–21.
[42] Thompson RJ, McCullough PA, Kahn JK, O'Neill WW. Prediction of death and neurologic outcome in the emergency department in out-of-hospital cardiac arrest survivors. Am J Cardiol 1998;81:17–21.
[43] Herlitz J, Bang A, Gunnarsson J, et al. Factors associated with survival to hospital discharge among patients hospitalised alive after out of hospital cardiac arrest: change in outcome over 20 years in the community of Goteborg, Sweden. Heart 2003;89:25–30.
[44] Dickey W, Adgey AA. Mortality within hospital after resuscitation from ventricular fibrillation outside hospital. Br Heart J 1992;67:334–8.
[45] Young JD, Goldfrad C, Rowan K. Development and testing of a hierarchical method to code the reason for admission to intensive care units: the ICNARC Coding Method. Intensive Care National Audit & Research Centre. Br J Anaesth 2001;87:543–8.
[46] Knaus WA, Draper EA, Wagner DP, Zimmerman JE. APACHE II: a severity of disease classification system. Crit Care Med 1985;13:818–29.
[47] Le Gall JR, Lemeshow S, Saulnier F. A new Simplified Acute Physiology Score (SAPS II) based on a European/North American multicenter study. JAMA 1993;270:2957–63.
[48] Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. The Acute Respiratory Distress Syndrome Network. N Engl J Med 2000; 342:1301–1308.
[49] Connors Jr AF, Speroff T, Dawson NV, et al. The effectiveness of right heart catheterization in the initial care of critically ill patients. SUPPORT Investigators. JAMA 1996;276:889–97.
[50] Richard C, Warszawski J, Anguel N, et al. Early use of the pulmonary artery catheter and outcomes in patients with shock and acute respiratory distress syndrome: a randomized controlled trial. JAMA 2003;290:2713–20.
[51] Angus D, Black N. Wider lessons of the pulmonary artery catheter trial. BMJ 2001;322:446.
[52] Young JD. Right heart catheterization in intensive care. Br J Anaesth 2001;86:327–9.
[53] Wiesenack C, Prasser C, Keyl C, Rodig G. Assessment of intrathoracic blood volume as an indicator of cardiac preload: single transpulmonary thermodilution technique versus assessment of pressure preload parameters derived from a pulmonary artery catheter. J Cardiothorac Vasc Anesth 2001;15:584–8.
[54] Jonas MM, Tanser SJ. Lithium dilution measurement of cardiac output and arterial pulse waveform analysis: an indicator dilution calibrated beat-by-beat system for continuous estimation of cardiac output. Curr Opin Crit Care 2002;8:257–61.
[55] Van de WF, Ardissino D, Betriu A, et al. Management of acute myocardial infarction in patients presenting with ST-segment elevation. The Task Force on the Management of Acute Myocardial Infarction of the European Society of Cardiology. Eur Heart J 2003;24:28–66.
[56] Andersen HR, Nielsen TT, Rasmussen K, et al. A comparison of coronary angioplasty with fibrinolytic therapy in acute myocardial infarction. N Engl J Med 2003;349:733–42.
[57] Grubb NR. Managing out-of-hospital cardiac arrest survivors: 1. Neurological perspective. Heart 2001;85:6–8.
[58] Levy DE, Caronna JJ, Singer BH, Lapinski RH, Frydman H, Plum F. Predicting outcome from hypoxic-ischemic coma. JAMA 1985;253:1420–6.
[59] Zandbergen EG, de Haan RJ, Stoutenbeek CP, Koelman JH, Hijdra A. Systematic review of early prediction of poor

outcome in anoxic-ischaemic coma. Lancet 1998;352:1808–12.
[60] Zandbergen EG, de Haan RJ, Koelman JH, Hijdra A. Prediction of poor outcome in anoxic-ischemic coma. J Clin Neurophysiol 2000;17:498–501.
[61] Zandbergen EG, de Haan RJ, Hijdra A. Systematic review of prediction of poor outcome in anoxic-ischaemic coma with biochemical markers of brain damage. Intensive Care Med 2001;27:1661–7.
[62] Zandbergen EG, de Haan RJ, Reitsma JB, Hijdra A. Survival and recovery of consciousness in anoxic-ischemic coma after cardiopulmonary resuscitation. Intensive Care Med 2003;29:1911–5.
[63] Booth CM, Boone RH, Tomlinson G, Detsky AS. Is this patient dead, vegetative, or severely neurologically impaired? Assessing outcome for comatose survivors of cardiac arrest. JAMA 2004;291:870–9.
[64] Walther SM, Jonasson U, Gill H. Comparison of the Glasgow Coma Scale and the Reaction Level Scale for assessment of cerebral responsiveness in the critically ill. Intensive Care Med 2003;29:933–8.
[65] Starmark JE, Stalhammar D, Holmgren E. The Reaction Level Scale (RLS85). Manual and guidelines. Acta Neurochir (Wien) 1988;91:12–20.
[66] Devlin JW, Boleski G, Mlynarek M, et al. Motor Activity Assessment Scale: a valid and Reliable Sedation Scale for use with mechanically ventilated patients in an adult surgical intensive care unit. Crit Care Med 1999;27:1271–5.
[67] de Lemos J, Tweeddale M, Chittock D. Measuring quality of sedation in adult mechanically ventilated critically ill patients. the Vancouver Interaction and Calmness Scale. Sedation Focus Group. J Clin Epidemiol 2000;53:908–19.
[68] Ely EW, Truman B, Shintani A, et al. Monitoring sedation status over time in ICU patients: reliability and validity of the Richmond Agitation-Sedation Scale (RASS). JAMA 2003;289:2983–91.
[69] Sessler CN, Gosnell MS, Grap MJ, et al. The Richmond Agitation-Sedation Scale: validity and reliability in adult intensive care unit patients. Am J Respir Crit Care Med 2002;166:1338–44.
[70] Simmons LE, Riker RR, Prato BS, Fraser GL. Assessing sedation during intensive care unit mechanical ventilation with the Bispectral Index and the Sedation-Agitation Scale. Crit Care Med 1999;27:1499–504.
[71] Riker RR, Picard JT, Fraser GL. Prospective evaluation of the Sedation-Agitation Scale for adult critically ill patients. Crit Care Med 1999;27:1325–9.
[72] Brattebo G, Hofoss D, Flaatten H, Muri AK, Gjerde S, Plsek PE. Effect of a scoring system and protocol for sedation on duration of patients' need for ventilator support in a surgical intensive care unit. BMJ 2002;324:1386–9.
[73] Dalby M, Bouzamondo A, Lechat P, Montalescot G. Transfer for primary angioplasty versus immediate thrombolysis in acute myocardial infarction: a meta-analysis. Circulation 2003;108:1809–14.
[74] Gendo A, Kramer L, Hafner M, et al. Time-dependency of sensory evoked potentials in comatose cardiac arrest survivors. Intensive Care Med 2001;27:1305–11.
[75] Chen R, Bolton CF, Young B. Prediction of outcome in patients with anoxic coma: a clinical and electrophysiologic study. Crit Care Med 1996;24:672–8.
[76] Madl C, Holzer M. Brain function after resuscitation from cardiac arrest. Curr Opin Crit Care 2004;10:213–7.
[77] Rosen H, Rosengren L, Herlitz J, Blomstrand C. Increased serum levels of the S-100 protein are associated with hypoxic brain damage after cardiac arrest. Stroke 1998;29:473–7.
[78] Rosen H, Sunnerhagen KS, Herlitz J, Blomstrand C, Rosengren L. Serum levels of the brain-derived proteins S-100 and NSE predict long-term outcome after cardiac arrest. Resuscitation 2001;49:183–91.
[79] Tiainen M, Roine RO, Pettila V, Takkunen O. Serum neuron-specific enolase and S-100B protein in cardiac arrest patients treated with hypothermia. Stroke 2003;34:2881–6.
[80] Zeiner A, Holzer M, Sterz F, et al. Hyperthermia after cardiac arrest is associated with an unfavorable neurologic outcome. Arch Intern Med 2001;161:2007–12.
[81] Sterz F, Holzer M, Roine R, et al. Hypothermia after cardiac arrest: a treatment that works. Curr Opin Crit Care 2003;9:205–10.
[82] Polderman KH. Application of therapeutic hypothermia in the ICU: opportunities and pitfalls of a promising treatment modality. Part 1: Indications and evidence. Intensive Care Med 2004;30:556–75.
[83] van den Berghe G, Wouters P, Weekers F, et al. Intensive insulin therapy in the critically ill patients. N Engl J Med 2001;345:1359–67.
[84] van den Berghe G, Wouters PJ, Bouillon R, et al. Outcome benefit of intensive insulin therapy in the critically ill: Insulin dose versus glycemic control. Crit Care Med 2003;31:359–66.
[85] Calle PA, Buylaert WA, Vanhaute OA, The Cerebral Resuscitation Study Group. Glycemia in the post-resuscitation period. Resuscitation 1989;(17 Suppl.):S181–8.
[86] Longstreth Jr WT, Inui TS. High blood glucose level on hospital admission and poor neurological recovery after cardiac arrest. Ann Neurol 1984;15:59–63.
[87] Longstreth Jr WT, Diehr P, Cobb LA, Hanson RW, Blair AD. Neurologic outcome and blood glucose levels during out-of-hospital cardiopulmonary resuscitation. Neurology 1986;36:1186–91.
[88] Müllner M, Sterz F, Binder M, Schreiber W, Deimel A, Laggner AN. Blood glucose concentration after cardiopulmonary resuscitation influences functional neurological recovery in human cardiac arrest survivors. J Cereb Blood Flow Metab 1997;17:430–6.
[89] Baird TA, Parsons MW, Phanh T, et al. Persistent poststroke hyperglycemia is independently associated with infarct expansion and worse clinical outcome. Stroke 2003;34:2208–14.
[90] Voll CL, Auer RN. Insulin attenuates ischemic brain damage independent of its hypoglycemic effect. J Cereb Blood Flow Metab 1991;11:1006–14.
[91] Laver S, Preston S, Turner D, McKinstry C, Padkin A. Implementing intensive insulin therapy: development and audit of the Bath insulin protocol. Anaesth Intensive Care 2004;32:311–6.
[92] Heyland DK, Dhaliwal R, Drover JW, Gramlich L, Dodek P. Canadian clinical practice guidelines for nutrition support in mechanically ventilated, critically ill adult patients. JPEN J Parenter Enteral Nutr 2003;27:355–73.
[93] Bone RC, Balk RA, Cerra FB, et al. Definitions for sepsis and organ failure and guidelines for the use of innovative therapies in sepsis. The ACCP/SCCM Consensus Conference Committee. American College of Chest Physicians/Society of Critical Care Medicine. Chest 1992;101:1644–55.
[94] Polderman KH. Application of therapeutic hypothermia in the intensive care unit. Opportunities and pitfalls of a promising treatment modality–Part 2: Practical aspects and side effects. Intensive Care Med 2004;30:757–69.
[95] Kollef MH. Prevention of hospital-associated pneumonia and ventilator-associated pneumonia. Crit Care Med 2004;32:1396–405.
[96] Grubb NR, Fox KA, Cawood P. Resuscitation from out-of-hospital cardiac arrest: implications for cardiac enzyme estimation. Resuscitation 1996;33:35–41.
[97] Mullner M, Hirschl MM, Herkner H, et al. Creatine kinase-mb fraction and cardiac troponin T to diagnose acute myocardial infarction after cardiopulmonary resuscitation. J Am Coll Cardiol 1996;28:1220–5.
[98] Mullner M, Oschatz E, Sterz F, et al. The influence of chest compressions and external defibrillation on the release of creatine kinase-MB and cardiac troponin T in patients resuscitated from out-of-hospital cardiac arrest. Resuscitation 1998;38:99–105.
[99] Lai CS, Hostler D, D'Cruz BJ, Callaway CW. Prevalence of troponin-T elevation during out-of-hospital cardiac arrest. Am J Cardiol 2004;93:754–6.