

Part 4: Advanced Life Support

The topics reviewed by the International Liaison Committee on Resuscitation (ILCOR) Advanced Life Support Task Force are grouped as follows: (1) causes and prevention, (2) airway and ventilation, (3) drugs and fluids given during cardiac arrest, (4) techniques and devices to monitor and assist the circulation, (5) periarrest arrhythmias, (6) cardiac arrest in special circumstances, (7) postresuscitation care, and (8) prognostication. Defibrillation topics are discussed in Part 3.

The most important developments in advanced life support (ALS) since the last ILCOR review in 2000 include

- The emergence of medical emergency teams (METs) as a means of preventing in-hospital cardiac arrest
- Additional clinical data on the use of vasopressin in cardiac arrest
- Several new devices to assist circulation during CPR
- The use of therapeutic hypothermia to improve neurologic outcome after ventricular fibrillation (VF) cardiac arrest
- The potential importance of glucose control after cardiac arrest

For many topics there were insufficient data with which to make firm treatment recommendations. The following interventions in particular need further research:

- The impact of METs on the incidence of cardiac arrest
- Outcome data to define the most appropriate advanced airway adjunct
- Evidence to identify the most effective vasopressor or if any vasopressor is better than placebo for cardiac arrest
- Randomized controlled trials on several new devices to assist circulation during CPR
- Randomized controlled trial data on several postresuscitation care therapies, such as control of ventilation, sedation, and glucose
- The precise role of, and method for implementing, therapeutic hypothermia: patient selection, external versus internal cooling, optimum target temperature, and duration of therapy

Causes and Prevention

Rescuers may be able to identify some noncardiac causes of arrest and tailor the sequence of attempted resuscitation. Most patients sustaining in-hospital cardiac arrest display signs of deterioration for several hours before the arrest. Early identification of these high-risk patients and the immediate arrival of a MET (also known as Rapid Response Team in the United

States) to care for them may help prevent cardiac arrest. Hospitals in many countries are introducing early warning systems such as METs.

Identification of the Etiology of Cardiac Arrest^{W119A,W120,W121}

Consensus on Science

Very few data address the etiology of cardiac arrest directly. One prospective study (LOE 3)¹ and one retrospective study (LOE 4)² suggested that rescuers can identify some noncardiac causes of some arrests.

Treatment Recommendation

The physical circumstances, history, or precipitating events may enable the rescuer to determine a noncardiac cause of the cardiorespiratory arrest. Under these circumstances the rescuer should undertake interventions based on the presumed noncardiac etiology.

Impact of Medical Emergency Teams^{W128A,W128B,W129A,W129B,W130A,W130B,W195A,W195B,W195C,W195D,W195E}

The METs studied were composed generally of a doctor and nurse with critical-care training who were available at all times, responded immediately when called, and had specific, well-defined calling criteria. The MET system normally includes a strategy for educating ward staff about early recognition of critical illness. Variations of the MET system include critical-care outreach teams and patient-at-risk teams; all such variants use early warning scoring (EWS) systems to indicate patients who may be critically ill or at risk of cardiac arrest.

Consensus on Science

Two supportive before-and-after single-center studies (LOE 3)^{3,4} documented significant reductions in cardiac arrest rates and improved outcomes following cardiac arrest (eg, survival and length of stay in the intensive care unit [ICU]) after introduction of a MET. One cluster randomized controlled trial documented no difference in the composite primary outcome (cardiac arrest, unexpected death, unplanned ICU admission) between 12 hospitals in which a MET system was introduced and 11 hospitals that continued to function as normal (LOE 2).⁵ In this study, however, the MET system increased significantly the rate of emergency team calling. Two neutral studies documented a trend toward reduction in the rates of adult in-hospital cardiac arrest and overall mortality (LOE 3)⁶ and a reduction in unplanned admissions to the ICU (LOE 3).⁷ A before-and-after study documented reductions in cardiac arrest and death in children after

From the 2005 International Consensus Conference on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations, hosted by the American Heart Association in Dallas, Texas, January 23–30, 2005.

This article has been copublished in *Resuscitation*.

(*Circulation*. 2005;112:III-25-III-54.)

© 2005 International Liaison Committee on Resuscitation, American Heart Association, and European Resuscitation Council.

This special supplement to *Circulation* is freely available at <http://www.circulationaha.org> DOI: 10.1161/CIRCULATIONAHA.105.166474

introduction of a MET service into a children's hospital,⁸ but these did not reach statistical significance.

Two before-and-after studies (LOE 3)^{9,10} showed reduced mortality among unplanned ICU admissions after the introduction of an EWS system. Another before-and-after in-hospital study (LOE 3)¹¹ failed to show any significant reduction in the incidence of cardiac arrest or unplanned ICU admissions when an EWS system was used to identify and treat adult patients at risk of deterioration.

Treatment Recommendation

Introduction of a MET system for adult hospital in-patients should be considered, with special attention to details of implementation (eg, composition and availability of the team, calling criteria, education and awareness of hospital staff, and method of activation of the team). Introduction of an EWS system for adult in-hospital patients may be considered.

Airway and Ventilation

Consensus conference topics related to the management of airway and ventilation are categorized as (1) basic airway devices, (2) advanced airway devices, (3) confirmation of advanced airway placement, (4) strategies to secure advanced airways, and (5) strategies for ventilation.

Basic Airway Devices

Nasopharyngeal Airway^{W45, W46A}

Consensus on Science

Despite frequent successful use of nasopharyngeal airways by anesthesiologists, there are no published data on the use of these airway adjuncts during CPR. One study in anesthetized patients showed that nurses inserting nasopharyngeal airways were no more likely than anesthesiologists to cause nasopharyngeal trauma (LOE 7).¹² One LOE 5 study¹³ showed that the traditional methods of sizing a nasopharyngeal airway (measurement against the patient's little finger or anterior nares) do not correlate with the airway anatomy and are unreliable. In one report insertion of a nasopharyngeal airway caused some airway bleeding in 30% of cases (LOE 7).¹⁴ Two case reports involve inadvertent intracranial placement of a nasopharyngeal airway in patients with basal skull fractures (LOE 7).^{15,16}

Treatment Recommendation

In the presence of a known or suspected basal skull fracture, an oral airway is preferred, but if this is not possible and the airway is obstructed, gentle insertion of a nasopharyngeal airway may be lifesaving (ie, the benefits may far outweigh the risks).

Advanced Airway Devices

The tracheal tube has generally been considered the optimal method of managing the airway during cardiac arrest. There is evidence that without adequate training and experience, the incidence of complications, such as unrecognized esophageal intubation, is unacceptably high. Alternatives to the tracheal tube that have been studied during CPR include the bag-valve mask and advanced airway devices such as the laryngeal mask airway (LMA) and esophageal-tracheal combitube

(Combitube). There are no data to support the routine use of any specific approach to airway management during cardiac arrest. The best technique depends on the precise circumstances of the cardiac arrest and the competence of the rescuer.

Tracheal Intubation Versus Ventilation With Bag-Valve Mask^{W57}

Consensus on Science

There were no randomized trials that assessed the effect of airway and ventilation management with bag-valve mask (BVM) alone versus airway management that includes tracheal intubation in adult victims of cardiac arrest.

The only published randomized controlled trial identified (LOE 7)¹⁷ that compared tracheal intubation with BVM ventilation was performed in children who required airway management out-of-hospital. In this study there was no difference in survival-to-discharge rates, but it is unclear how applicable this pediatric study is to adult resuscitation. The study had some important limitations, including the provision of only 6 hours of additional training for intubation, limited opportunity to perform intubations, and short transport times. Two studies compared outcomes from out-of-hospital cardiac arrest in adults treated by either emergency medical technicians or paramedics (LOE 3¹⁸; LOE 4¹⁹). The skills provided by the paramedics, including intubation and intravenous (IV) cannulation^{18,19} and drug administration,¹⁹ made no difference in survival to hospital discharge.

The reported incidence of unrecognized misplaced tracheal tube is 6% (LOE 5)²⁰⁻²² to 14% (LOE 5).²³ An additional problem common to any advanced airway is that intubation attempts generally require interruptions in chest compressions.

Treatment Recommendation

There is insufficient evidence to support or refute the use of any specific technique to maintain an airway and provide ventilation in adults with cardiopulmonary arrest. Either bag-valve mask alone or in combination with tracheal intubation is acceptable for ventilation during CPR by prehospital providers. Rescuers must weigh the risks and benefits of intubation versus the need to provide effective chest compressions. The intubation attempt will require interruption of chest compressions, but once an advanced airway is in place, ventilation will not require interruption (or even pausing) of chest compressions. To avoid substantial interruptions in chest compressions, providers may defer an intubation attempt until return of spontaneous circulation (ROSC). To ensure competence, healthcare systems that utilize advanced airways should address factors such as adequacy of training and experience and quality assurance. Providers must confirm tube placement and ensure that the tube is adequately secured (see below).

Tracheal Intubation Versus the Combitube/Laryngeal Mask Airway^{W42A, W42B, W43A, W43B, W44A, W44B}

Consensus on Science

In some communities tracheal intubation is not permitted or practitioners have inadequate opportunity to maintain their

intubation skills. Under these circumstances several studies indicate a high incidence of unrecognized esophageal intubation misplacement and unrecognized dislodgment. Prolonged attempts at tracheal intubation are harmful: the cessation of chest compressions during this time will compromise coronary and cerebral perfusion. Several alternative airway devices have been considered or studied for airway management during CPR; the Combitube and the LMA are the only alternative devices to be studied specifically during CPR. None of the studies of the LMA and Combitube during CPR has been adequately powered to study survival as a primary end point; instead, most researchers have studied insertion and ventilation success rates.

Combitube. Five randomized controlled trials conducted on adult patients undergoing resuscitation (LOE 2)^{24–28} and 3 additional randomized controlled trials involving patients undergoing anesthesia (LOE 7)^{29–31} documented successful Combitube insertion and acceptable ventilation when compared with tracheal intubation. Benefits were documented for both experienced and inexperienced healthcare professionals with patients in hospital as well as in out-of-hospital settings.

Six additional studies support the use of the Combitube during CPR (LOE 3³²; LOE 4³³; LOE 5^{34–37}). Successful ventilation was achieved with the Combitube during CPR in 78.9% to 98% of patients (LOE 2^{26,27,38}; LOE 3³²; LOE 4³³; LOE 5^{34,35}).

LMA. Seven randomized controlled trials involving anesthetized patients (LOE 7)^{39–45} that compared the LMA with tracheal intubation and another 7 randomized control trials (LOE 7)^{46–52} that compared the LMA with other airways or ventilation techniques were reviewed. These studies suggested that experienced and inexperienced personnel can insert the device or successfully ventilate the patient's lungs in a high proportion of cases compared with the tracheal tube or other airway management and ventilation devices.

One randomized crossover study (LOE 2)³⁸ in adults undergoing resuscitation in the prehospital setting compared the Combitube with the LMA and showed that LMA insertion and successful ventilation could be achieved in a high proportion of patients.

Nonrandomized studies (LOE 3^{53–55}; LOE 4³³; LOE 5^{56–61}) have also shown high insertion success rates by inexperienced providers both in and out of the hospital. Complication rates in nonrandomized studies (LOE 3⁵⁸; LOE 4⁵³; LOE 5⁵⁶) have been extremely low.

Successful ventilation was achieved with the LMA during CPR in 71.5% to 98% of cases (LOE 2³⁸; LOE 3⁵⁴; LOE 4³³; LOE 5^{56,58–60}).

Additional airway devices. Use of the laryngeal tube during CPR was described in just a few cases included in 2 LOE 5 studies^{62,63} and 1 LOE 8 paper.⁶⁴ There were no studies comparing the laryngeal tube with the tracheal tube in any patient population, although 4 randomized controlled trials compared the laryngeal tube favorably with the LMA in anesthetized patients (LOE 7).^{65–68}

Other devices include the ProSeal LMA, intubating LMA, airway management device, and pharyngeal airway express.

There are no published data on the use of these devices during CPR.

Treatment Recommendation

It is acceptable for healthcare professionals to use the Combitube or the LMA as alternatives to the tracheal tube for airway management in cardiac arrest.

Confirming Advanced Airway Placement

Unrecognized esophageal intubation is the most serious complication of attempted tracheal intubation. Routine confirmation of correct placement of the tracheal tube should reduce this risk. There are inadequate data to identify the optimal method of confirming tube placement during cardiac arrest. All devices should be considered adjuncts to other confirmatory techniques. There is no data quantifying the capability of these devices to monitor tube position after initial placement.

Exhaled CO₂^{W47,W50}

Consensus on Science

Evidence from 1 meta-analysis in adults (LOE 1),⁶⁹ 1 prospective controlled cohort study (LOE 3),⁷⁰ case series (LOE 5),^{71–79} and animal models (LOE 6)^{80,81} indicate that exhaled CO₂ detectors (waveform, colorimetry, or digital) may be useful as adjuncts to confirm tracheal tube placement during cardiac arrest. Of the 14 references included in this statement, 10 referred to colorimetric assessment,^{69,71–76,79,81,82} 4 to digital,^{69–71,77} and 4 to waveform.^{69,70,78,80} There are insufficient data from cardiac arrests to enable any firm recommendations for any particular technique. The range of results obtained from the reviewed papers is as follows:

- Percentage of tracheal placements detected: 33% to 100%
- Percentage of esophageal placements detected: 97% to 100%
- Probability of tracheal placement if test result is positive (exhaled CO₂ is detected): 100%
- Probability of esophageal placement if test result is negative (exhaled CO₂ is not detected): 20% to 100%

One adult case series (LOE 5)⁸² shows that in the presence of a perfusing rhythm, exhaled CO₂ detection can be used to monitor tracheal tube position during transport.

No studies directly evaluated exhaled CO₂ to confirm placement of the Combitube or LMA during cardiac arrest in humans.

Treatment Recommendation

Healthcare providers should recognize that evaluation of exhaled CO₂ is not infallible for confirming correct placement of a tracheal tube, particularly in patients in cardiac arrest. Exhaled CO₂ should be considered as just one of several independent methods for confirming tracheal tube placement. Continuous capnometry may be useful for early detection of tracheal tube dislodgment during transport.

Esophageal Detector Device^{W48A,W51A,W51B}

Consensus on Science

Eight studies of at least fair quality evaluated the accuracy of the syringe or self-inflating bulb type of esophageal detector

device (EDD) (LOE 3^{21,77,83}; LOE 5⁸⁴; LOE 7 [noncardiac arrest setting]^{85–88}), but many suffer from few subjects and lack of a control group.

The EDD was highly sensitive for detection of misplaced tracheal tubes in the esophagus (LOE 5⁸⁴; LOE 7^{85–88}). In 2 studies (LOE 3)^{77,83} of patients in cardiac arrest, the EDD had poor sensitivity for confirming tracheal placement of a tracheal tube. In these studies up to 30% of correctly placed tubes may have been removed because the EDD suggested esophageal placement of a tube (LOE 3).⁷⁸

The EDD had poor sensitivity and specificity in the operating room in 20 children <1 year of age (LOE 2).⁸⁹

Treatment Recommendation

The use of the EDD should be considered as just one of several independent methods for tracheal tube confirmation.

Strategies to Secure Advanced Airways

Accidental dislodgment of a tracheal tube can occur at any time but may be more likely during resuscitation and during transport. The most effective method for securing the tracheal tube has yet to be determined.

Securing the Tracheal Tube^{W49A,W49B}

Consensus on Science

There are no studies comparing different strategies for securing the tracheal tube during CPR. Two studies in the intensive care setting (LOE 7)^{90,91} indicated that commercial devices for securing tracheal tubes, backboards, cervical collars, and other strategies provide an equivalent method for preventing accidental tube displacement when compared with the traditional method of securing the tube with tape.

Treatment Recommendation

Either commercially made tracheal tube holders or conventional tapes or ties should be used to secure the tracheal tube.

Strategies for Ventilation

Very few studies address specific aspects of ventilation during ALS. Three recent observational studies report the ventilation rates delivered by healthcare personnel during cardiac arrest (LOE 5)^{92–94}: 2 studies^{92,93} show ventilation rates that are much higher than those recommended by the 2000 *International Guidelines for CPR and ECC*. Automatic transport ventilators (ATVs) might enable delivery of appropriate ventilatory rates, but no data demonstrate clear benefit over bag-valve mask devices.

Disconnection From Ventilation During Cardiac Arrest^{W54A,W54B}

Consensus on Science

Eighteen LOE 5 articles involving 31 cases^{95–112} reported unexpected return of circulation (and in some cases prolonged neurologically intact survival) after cessation of resuscitation attempts. One case series suggested that this occurred in patients with obstructive airway disease (LOE 5).¹⁰⁰ Four studies reported unexpected return of circulation in 6 cases in which resuscitation had ceased and ventilation was shown on repeated occasions (or was highly likely) to result in gas trapping and consequent hemodynamic compro-

mise (LOE 5).^{100,108–110} The authors of all these studies suggested that a period of disconnection from ventilation during resuscitation from PEA may be useful to exclude gas trapping.

Automatic Transport Ventilators^{W55,W152A}

Consensus on Science

Research of simulated cardiac arrest with manikins showed a significant decrease in gastric inflation with manually triggered, flow-limited, oxygen-powered resuscitators and masks compared with bag-valve masks (LOE 6).¹¹³ Anesthetized patients with unprotected airways but not in cardiac arrest who were ventilated by firefighters had less gastric inflation with manually triggered, flow-limited, oxygen-powered resuscitators and masks than with bag-valve masks (LOE 5).¹¹⁴ A prospective cohort study of intubated patients, most of whom were in cardiac arrest, in an out-of-hospital urban setting showed no significant difference in arterial blood gas values between those ventilated with an ATV and those ventilated with a bag-valve device (LOE 4).¹¹⁵ Two laboratory studies showed that ATVs may provide safe and effective management of mask ventilation during CPR of adult patients with an unprotected airway (LOE 6).^{116,117}

Treatment Recommendation

The use of a manually triggered, flow-limited resuscitator or an ATV by professional healthcare providers is reasonable for ventilation of adults with an advanced airway in place during cardiac arrest. The use of ATVs for adults without an advanced airway in place is discussed in Part 2: “Adult Basic Life Support.”

Drugs and Fluids for Cardiac Arrest

Questions related to the use of drugs during cardiac arrest that were discussed during the 2005 Consensus Conference are categorized as (1) vasopressors, (2) antiarrhythmics, (3) other drugs and fluids, and (4) alternative routes of delivery.

Vasopressors

Despite the widespread use of epinephrine/adrenaline during resuscitation and several studies involving vasopressin, there is no placebo-controlled study that shows that the routine use of any vasopressor at any stage during human cardiac arrest increases survival to hospital discharge. Current evidence is insufficient to support or refute the routine use of any particular drug or sequence of drugs. Despite the lack of human data, it is reasonable to continue to use vasopressors on a routine basis.

Epinephrine and Vasopressin^{W83B,W83E,W83F,W83G,W83H,W84A,W84B,W84D,W85A,W85B,W85C,W112}

Consensus on Science

Despite promising lower-level data (LOE 2¹¹⁸; LOE 5^{119–121}) and multiple well-performed animal studies [LOE 6]), 2 large randomized controlled human trials of adults in cardiac arrest (LOE 1)^{122,123} were unable to show an increase in the rates of ROSC or survival for vasopressin (40 U, with the dose repeated in 1 study) when compared with epinephrine (1 mg, repeated) as the initial vasopressor. In 1 large multicenter trial

involving out-of-hospital cardiac arrest with all rhythms (LOE 1),¹²³ on post hoc analysis the subset of patients with asystole had significant improvement in rate of survival to discharge but not neurologically intact survival when vasopressin 40 U (dose repeated once if necessary) was used as the initial vasopressor compared with epinephrine (1 mg, repeated if necessary). A meta-analysis of 5 randomized trials (LOE 1)¹²⁴ showed no statistically significant differences between vasopressin and epinephrine for ROSC, death within 24 hours, or death before hospital discharge. The subgroup analysis based on initial cardiac rhythm did not show any statistically significant differences in the rate of death before hospital discharge (LOE 1).¹²⁴

Treatment Recommendation

Despite the absence of placebo-controlled trials, epinephrine has been the standard vasopressor in cardiac arrest. There is insufficient evidence to support or refute the use of vasopressin as an alternative to, or in combination with, epinephrine in any cardiac arrest rhythm.

Alpha-methyl Norepinephrine^{W83B}

Consensus on Science

Preliminary animal studies (LOE 6)^{125–127} have suggested some potential short-term benefits with the use of alpha-methyl norepinephrine in animal models of VF. At this stage no published human studies have been identified.

Endothelin^{W83D,W83I}

Consensus on Science

Evidence from 5 studies of cardiac arrest in animals (LOE 6)^{128–132} documented consistent improvement in coronary perfusion pressure with endothelin-1, but this did not translate into improved myocardial blood flow. No published human studies were available.

Antiarrhythmics

There is no evidence that giving any antiarrhythmic drug routinely during human cardiac arrest increases rate of survival to hospital discharge. In comparison with placebo and lidocaine, the use of amiodarone in shock-refractory VF improves the short-term outcome of survival to hospital admission. Despite the lack of human long-term outcome data, it is reasonable to continue to use antiarrhythmic drugs on a routine basis.

Amiodarone^{W83A,W83I}

Consensus on Science

In 2 blinded randomized controlled clinical trials in adults (LOE 1),^{133,134} administration of amiodarone (300 mg¹³³; 5 mg/kg¹³⁴) by paramedics to patients with refractory VF/pulseless ventricular tachycardia (VT) in the out-of-hospital setting improved survival to hospital admission when compared with administration of placebo¹³³ or lidocaine (1.5 mg/kg).¹³⁴ Additional studies (LOE 7)^{135–139} document consistent improvement in defibrillation response when amiodarone is given to humans or animals with VF or hemodynamically unstable VT.

Treatment Recommendation

In light of the short-term survival benefits, amiodarone should be considered for refractory VF/VT.

Other Drugs and Fluids

There is no evidence that routinely giving other drugs (eg, buffers, aminophylline, atropine, calcium, magnesium) during human cardiac arrest increases survival to hospital discharge. There are several reports on the successful use of fibrinolytics during cardiac arrest, particularly when the arrest was caused by pulmonary embolism.

Aminophylline^{W98A,W98B}

Consensus on Science

One case series (LOE 5)¹⁴⁰ and 3 small randomized trials (LOE 2)^{141–143} indicate that aminophylline does not increase ROSC when given for bradysystolic cardiac arrest. No studies have shown an effect of aminophylline on rates of survival to hospital discharge. There is no evidence of harm from giving aminophylline in bradysystolic cardiac arrest (LOE 2^{141–143}; LOE 5¹⁴⁰).

Atropine^{W97A,W97B}

Consensus on Science

Five prospective controlled nonrandomized cohort studies in adults (LOE 3)^{19,144–147} and 1 LOE 4 study¹⁴⁸ showed that treatment with atropine was not associated with any consistent benefits after in-hospital or out-of-hospital cardiac arrest.

Buffers^{W34,W100A,W100B}

Consensus on Science

There were no published LOE 1, 2, or 3 studies on the use of sodium bicarbonate during CPR. One LOE 2 study¹⁴⁹ showed no advantage of Tribonate over placebo (neutral), and 5 retrospective analyses of uncontrolled clinical use of sodium bicarbonate were inconclusive (LOE 4).^{150–154} One LOE 4 study¹⁵⁵ suggested that emergency medical services (EMS) systems using sodium bicarbonate earlier and more frequently had significantly higher rates of ROSC and hospital discharge and better long-term neurologic outcome.

Results of animal studies are conflicting and inconclusive. Sodium bicarbonate was effective for treating the cardiovascular toxicity (hypotension, cardiac arrhythmias) caused by tricyclic antidepressants and other fast sodium channel blockers (see “Drug Overdose and Poisoning,” below). Only 1 LOE 5 publication¹⁵⁶ reported the successful treatment of VF cardiac arrest caused by tricyclic poisoning using sodium bicarbonate.

Treatment Recommendation

Giving sodium bicarbonate routinely during cardiac arrest and CPR (especially in out-of-hospital cardiac arrest) or after ROSC is not recommended. Sodium bicarbonate may be considered for life-threatening hyperkalemia or cardiac arrest associated with hyperkalemia, preexisting metabolic acidosis, or tricyclic antidepressant overdose.

Magnesium^{W83K,W101A,W101B}

Consensus on Science

Studies in adults in- and out-of-hospital (LOE 2^{157–160}; LOE 3¹⁶¹; LOE 7¹⁶²) and animal studies (LOE 6)^{163–166} indicated

no increase in the rate of ROSC when magnesium was given during CPR. Results from 1 small case series of 5 patients (LOE 5)¹⁶⁷ indicated benefit from giving magnesium in shock-resistant and epinephrine/lidocaine-resistant VF.

Treatment Recommendation

Magnesium should be given for hypomagnesemia and torsades de pointes, but there is insufficient data to recommend for or against its routine use in cardiac arrest.

Fibrinolysis During CPR^{W96A,W96B,W96C}

Consensus on Science

Adults have been successfully resuscitated following administration of fibrinolytics after initial failure of standard CPR techniques, particularly when the condition leading to the arrest was acute pulmonary embolism or other presumed cardiac cause (LOE 3¹⁶⁸; LOE 4^{169–171}; LOE 5^{172–176}). One large clinical trial (LOE 2)¹⁷⁷ failed to show any significant treatment effect from administration of fibrinolytics to out-of-hospital patients with undifferentiated pulseless electrical activity (PEA) cardiac arrest unresponsive to initial interventions. Four clinical studies (LOE 3¹⁶⁸; LOE 4^{169–171}) and 5 case series (LOE 5)^{172–176} indicated that there is no increase in bleeding complications with fibrinolysis during CPR for nontraumatic cardiac arrest. Two animal studies (LOE 6)^{178,179} showed positive effects on cerebral reperfusion with fibrinolysis during CPR.

Treatment Recommendation

Fibrinolysis should be considered in adult patients with cardiac arrest with proven or suspected pulmonary embolism. There is insufficient data to support or refute the routine use of fibrinolysis in cardiac arrest from other causes.

Fluids^{W105}

Consensus on Science

There were no published human studies of routine fluid use compared with no fluids during normovolemic cardiac arrest. Four animal studies (LOE 6)^{180–183} of experimental VF neither support nor refute the use of IV fluids routinely. Fluids should be infused if hypovolemia is suspected.

Alternative Routes for Drug Delivery

If IV access cannot be established, intraosseous (IO) delivery of resuscitation drugs will achieve adequate plasma concentrations. Resuscitation drugs can also be given via the tracheal tube, but the plasma concentrations achieved are variable and substantially lower than those achieved when the same drug is given by the IV or IO route.

Intraosseous Route^{W29}

Consensus on Science

Two prospective trials in adults and children (LOE 3)^{184,185} and 6 other studies (LOE 4¹⁸⁶; LOE 5^{187–189}; LOE 7^{190,191}) documented that IO access is safe and effective for fluid resuscitation, drug delivery, and laboratory evaluation, and is attainable in all age groups.

Drugs Given via the Tracheal Tube^{W32,W108}

Consensus on Science

Atropine and epinephrine. In 1 historic nonrandomized cohort study (LOE 4)¹⁹² in adults, the rate of ROSC (27% vs 15%, $P=0.01$) and rate of survival to hospital admission (20% vs 9%, $P=0.01$) was significantly higher in the IV drug (atropine and adrenaline) group compared with the tracheal drug group. No patient who received tracheal drugs survived to hospital discharge compared with 5% of those who received IV drugs.

Epinephrine. During CPR the equipotent epinephrine dose given endobronchially was approximately 3 to 10 times higher than the IV dose (LOE 5¹⁹³; LOE 6¹⁹⁴). Endobronchial epinephrine (2 to 3 mg) diluted in 5 to 10 mL 0.9% NaCl achieved therapeutic plasma concentrations (LOE 5).¹⁹³ Endobronchial epinephrine achieved higher plasma concentrations when diluted with water rather than 0.9% saline (LOE 6).¹⁹⁵

During CPR lung perfusion is only 10% to 30% of the normal value, resulting in a pulmonary epinephrine depot. When cardiac output is restored after a high dose of endobronchial epinephrine, prolonged reabsorption of epinephrine from the lungs into the pulmonary circulation may occur (LOE 6),¹⁹⁴ causing arterial hypertension, malignant arrhythmias, and recurrence of VF.

Lidocaine. All studies were performed in hemodynamically stable (nonarrest) patients. Therapeutic plasma concentrations of lidocaine were achieved in these patients (LOE 5)^{196,197} after tracheal tube instillation but in only 40% of similar patients after instillation via an LMA (LOE 5).^{197,198} In anesthetized healthy adults, endobronchial delivery delayed the increase in lidocaine plasma concentrations (LOE 2).¹⁹⁹ In some (LOE 5),^{198,200} but not all of these studies (LOE 2¹⁹⁹; LOE 5¹⁹⁶), deep endobronchial delivery of lidocaine via a catheter achieved lower blood concentrations than when lidocaine was injected directly into the tracheal tube. Endobronchial lidocaine achieved higher plasma concentrations and caused less reduction in PaO₂ when diluted with water instead of 0.9% sodium chloride (LOE 5).²⁰¹

Vasopressin. Endobronchial vasopressin was more effective in increasing diastolic blood pressure than equivalent doses of endobronchial epinephrine (LOE 6).²⁰² In a small animal study, endobronchial vasopressin was more effective than placebo in increasing coronary perfusion pressure during CPR and improving survival rates (LOE 6).²⁰³

Treatment Recommendation

If IV access is delayed or cannot be achieved, IO access should be considered. Give drugs via the tracheal tube if intravascular (IV or IO) access is delayed or cannot be achieved. There are no benefits from endobronchial injection compared with injection of the drug directly into the tracheal tube. Dilution with water instead of 0.9% saline may achieve better drug absorption.

Monitoring and Assisting the Circulation

Specific questions related to the use of techniques and devices to (1) monitor the performance of CPR during cardiac arrest or (2) assist the circulation (alternatives to standard

CPR) during cardiac arrest were discussed during the 2005 Consensus Conference. They are listed below.

Monitoring CPR Performance

End-tidal CO₂ can be used as an indicator of ROSC. Arterial blood gas analysis may help to guide therapy. Measurement of coronary artery perfusion might be helpful, but because it is technically difficult to measure, it is not available routinely.

End-Tidal CO₂ Monitoring to Guide Therapy During Cardiac Arrest^{W92A, W92B}

Consensus on Science

No studies have addressed this topic directly. The studies published over the past 5 years were consistent with the older literature, which showed that higher end-tidal CO₂ values during CPR correlate with ROSC (LOE 5).^{204–207}

In experimental models, end-tidal CO₂ concentration during ongoing CPR correlated with cardiac output, coronary perfusion pressure, and successful resuscitation from cardiac arrest (LOE 6).^{208–214} Eight case series have shown that patients who were successfully resuscitated from cardiac arrest had significantly higher end-tidal CO₂ levels than patients who could not be resuscitated (LOE 5).^{73,204–207,215–217} Capnometry can also be used as an early indicator of ROSC (LOE 5^{218,219}; LOE 6²²⁰).

In case series totaling 744 patients, intubated adults in cardiac arrest receiving CPR who had a *maximum* end-tidal CO₂ of <10 mm Hg had a poor prognosis even if CPR was optimal (LOE 5).^{204,205,217,221–223} This prognostic indicator may be unreliable immediately after starting CPR because 2 studies (LOE 5)^{217,223} showed no difference in ROSC and survival in those with an *initial* end-tidal CO₂ of <10 mm Hg. Two additional studies (LOE 5)^{221,222} reported that 5 patients achieved ROSC despite an *initial* end-tidal CO₂ of <10 mm Hg (1 patient survived).

Treatment Recommendation

End-tidal CO₂ monitoring is a safe and effective noninvasive indicator of cardiac output during CPR and may be an early indicator of ROSC in intubated patients.

Arterial Blood Gas Monitoring During Cardiac Arrest^{W93A, W93B}

Consensus on Science

There was evidence from 1 LOE 5 study²²⁴ and 10 LOE 7 studies^{225–234} that arterial blood gas values are an inaccurate indicator of the magnitude of tissue acidosis during cardiac arrest and CPR in both the in-hospital and out-of-hospital settings. The same studies indicate that both arterial and mixed venous blood gases are required to establish the degree of acidosis.

Arterial blood gas analysis alone can disclose the degree of hypoxemia (LOE 5²³⁵; LOE 6^{236,237}; LOE 7^{225,227,231,238–240}). Arterial blood gas analysis can also highlight the extent of metabolic acidosis (LOE 5²⁴¹; LOE 6²³⁶; LOE 7^{225,227,230,231,238,239}).

Arterial CO₂ is an indicator of adequacy of ventilation during CPR (LOE 2²⁴²; LOE 5²³⁵; LOE 6²³⁶; LOE 7^{92,227,239,243}). If ventilation is constant, an increase in PaCO₂ is a potential marker

of improved perfusion during CPR (LOE 5²⁴⁴; LOE 6^{209,245}; LOE 7²⁴⁶).

Treatment Recommendation

Arterial blood gas monitoring during cardiac arrest enables estimation of the degree of hypoxemia and the adequacy of ventilation during CPR but is not a reliable indicator of the extent of tissue acidosis.

Coronary Perfusion Pressure to Guide Resuscitation^{W95A, W95C}

Consensus on Science

Coronary perfusion pressure (CPP) (aortic relaxation [diastolic] minus the right atrial relaxation phase blood pressure during CPR) correlated with both myocardial blood flow and ROSC (LOE 3)^{247,248}: a value ≥ 15 mm Hg is predictive of ROSC. Increased CPP correlated with improved 24-hour survival in animal studies (LOE 6)²⁴⁹ and is associated with improved myocardial blood flow and ROSC in studies of epinephrine, vasopressin, and angiotensin II (LOE 6).^{249–251}

Treatment Recommendation

Coronary perfusion pressure can guide therapy during cardiac arrest. In an intensive care facility the availability of direct arterial and central venous pressure monitoring makes calculation of CPP potentially useful. Outside the intensive care facility the technical difficulties of invasive monitoring of central arterial and venous pressure make it difficult to calculate CPP routinely during cardiac arrest.

Techniques and Devices to Assist Circulation During Cardiac Arrest

Several techniques or adjuncts to standard CPR have been investigated, and the relevant data was reviewed extensively. One multicenter human study (LOE 2)⁹⁴ showed poor quality and frequent interruptions in chest compressions delivered during prehospital CPR. In the hands of some groups, novel techniques and adjuncts may be better than standard CPR. The success of any technique depends on the education and training of the rescuers or the resources available (including personnel). Because information about these techniques and devices is often limited, conflicting, or supportive only for short-term outcomes, no recommendations can be made to support or refute their routine use.

Transcutaneous Pacing for Asystole^{W104}

Consensus on Science

Three randomized controlled trials (LOE 2)^{252–254} and additional studies (LOE 3²⁵⁵; LOE 5^{256–259}; LOE 6²⁶⁰; LOE 7²⁶¹) indicate no improvement in the rate of admission to hospital or survival to hospital discharge when pacing was attempted by paramedics or physicians in asystolic patients in the prehospital or the hospital (emergency department) setting.

Treatment Recommendation

Pacing is not recommended for patients in asystolic cardiac arrest.

CPR Prompt Devices^{W190A, W190B}

Consensus on Science

Two studies in adults (LOE 5)^{93,94} show that unprompted CPR was frequently of poor quality in the out-of-hospital and

in-hospital settings. One study in adults (LOE 3),²⁶² one study in children (LOE 3),²⁶³ and animal (LOE 6)^{264,265} and manikin studies (LOE 6)^{266–272} show consistent improvement in end tidal CO₂ or quality of CPR performed, or both, when feedback was provided with a variety of formats to guide CPR. In one manikin study (LOE 6),²⁷⁰ 95% of rescuers reported discomfort in the heels of their hands and wrists when using a CPR prompt applied between their hands and the victim's chest, but no long-term injuries were noted. A crossover study of paramedic students previously trained in CPR showed that audio feedback significantly improved the proportion of correct inflations, correct compression depth, and duration of compressions (LOE 6).²⁶⁸ A similar study of nursing students showed improved inflations and depth of compression (LOE 6).²⁷²

Treatment Recommendation

CPR prompt devices may improve CPR performance. See also Part 8: "Interdisciplinary Topics."

Interposed Abdominal Compression CPR^{W73A,W73B}

Consensus on Science

Two randomized controlled trials (LOE 1²⁷³; LOE 2²⁷⁴) of in-hospital cardiac arrests showed improved ROSC and survival of event when interposed abdominal compression CPR (IAC-CPR) performed by rescuers trained in the technique was compared with standard CPR. One of these studies (LOE 1)²⁷³ also reported improved rates of survival to hospital discharge. This data and that from a crossover study (LOE 3)²⁷⁵ were combined in 2 meta-analyses (LOE 1).^{276,277} One randomized controlled trial (LOE 2)²⁷⁸ of out-of-hospital cardiac arrests did not show any survival advantage when IAC-CPR was undertaken by rescuers trained in the technique compared with standard CPR. Some harm was reported in 1 child (LOE 5).²⁷⁹ Although only a small proportion of patients had postmortem examinations, there was no evidence of significant harm.

High-Frequency CPR^{W74,W163H}

Consensus on Science

One clinical trial of 9 patients (LOE 4)²⁸⁰ showed that high-frequency CPR (120 compressions per minute) improved hemodynamics over standard CPR. Three laboratory studies (LOE 6)^{281–283} showed that high-frequency CPR (120 to 150 compressions per minute) improved hemodynamics without increasing trauma. In one additional laboratory study (LOE 6),²⁸⁴ high-frequency CPR did not improve hemodynamics over standard CPR.

Active Compression-Decompression CPR^{W75A,W75B,W163J}

Consensus on Science

Despite initial promising studies suggesting short-term survival benefits (LOE 2)^{285,286} and even intact neurologic survival (LOE 1),²⁸⁷ a Cochrane meta-analysis (LOE 1)²⁸⁸ of 10 trials (involving 4162 patients) compared active compression-decompression (ACD) CPR with standard CPR in the out-of-hospital setting and did not show a significant increase in rates of immediate survival or hospital discharge. One meta-analysis (LOE 1)²⁸⁸ of 2 trials (826 patients)

comparing ACD-CPR with standard CPR after in-hospital cardiac arrest did not detect a significant increase in rates of immediate survival or hospital discharge. Although one small study (LOE 4)²⁸⁹ showed harm with an increased incidence of sternal fractures in the ACD-CPR group when compared with standard CPR alone, the large meta-analysis²⁸⁸ did not find any increase in complications when ACD-CPR was compared with standard CPR.

Load Distributing Band CPR^{W76A,W76B,W163F}

Consensus on Science

The load distributing band (LDB) is a circumferential chest compression device composed of a pneumatically actuated constricting band and backboard. A case control study of 162 adults (LOE 4)²⁹⁰ documented improvement in survival to the emergency department when LDB-CPR was administered by adequately trained rescue personnel to patients with cardiac arrest in the prehospital setting. The use of LDB-CPR improved hemodynamics in 1 in-hospital study of end-stage patients (LOE 3)²⁹¹ and 2 laboratory studies (LOE 6).^{292,293}

Mechanical (Piston) CPR^{W77A,W77B,W163B,W163E}

Consensus on Science

One prospective randomized study and 2 prospective randomized crossover studies in adults (LOE 2)^{294–296} indicated improvement in end-tidal CO₂ and mean arterial pressure when automatic mechanical (piston) CPR was undertaken by medical and paramedical personnel in the hospital or prehospital setting. In several studies in animals (LOE 6),^{297–300} mechanical (piston) CPR improved end-tidal CO₂, cardiac output, cerebral blood flow, mean arterial pressure, and short-term neurologic outcome.

Lund University Cardiac Arrest System CPR^{W77B,W163D}

Consensus on Science

The Lund University Cardiac Arrest System (LUCAS) is a gas-driven sternal compression device that incorporates a suction cup for active decompression. There were no published randomized human studies comparing LUCAS-CPR with standard CPR. A single study of pigs with VF showed that LUCAS-CPR improved hemodynamic and short-term survival rates compared with standard CPR (LOE 6).²⁹⁹ The LUCAS was also used in 20 patients, but incomplete outcome data was reported (LOE 6).²⁹⁹

Phased Thoracic-Abdominal

Compression-Decompression CPR^{W78B,W163C,W168}

Consensus on Science

Phased thoracic-abdominal compression-decompression (PTACD) CPR combines the concepts of IAC-CPR and ACD-CPR. One modeling study (LOE 7)³⁰¹ and one laboratory study (LOE 6)³⁰² showed that PTACD-CPR improved hemodynamics. One clinical, randomized study in adults (LOE 2)³⁰¹ and additional experimental studies (LOE 6^{302,303}; LOE 7³⁰⁴) documented no improvement in survival rates for patients with cardiac arrest when PTACD-CPR was used for assistance of circulation during ALS in the prehospital or in-hospital setting. PTACD-CPR did not substantially delay

starting CPR and had no significant known disadvantages nor caused harm when used correctly.

Minimally Invasive Direct Cardiac Massage^{W79A,W79B}

Consensus on Science

Minimally invasive direct cardiac massage (MIDCM) involves insertion of a plunger-like device through a small incision in the chest wall to enable direct compression of the heart. MIDCM improved ROSC and coronary perfusion pressure compared with standard CPR in one laboratory study (LOE 6)³⁰⁵ and generated systemic blood flow and myocardial and cerebral flow similar to that produced with open-chest cardiac massage in 2 laboratory studies (LOE 6).^{306,307} The MIDCM device was placed in patients in the field and generated improved blood pressure over standard CPR in one clinical study (LOE 3).³⁰⁸ But in this study, use of the MIDCM device caused cardiac rupture in 1 patient. MIDCM increased the defibrillation threshold for standard external defibrillation but reduced the defibrillation threshold if the MIDCM device was used as one of the electrodes in one laboratory study (LOE 6).³⁰⁹

Impedance Threshold Device^{W80,W163A,W163I}

Consensus on Science

The impedance threshold device (ITD) is a valve that limits air entry into the lungs during chest recoil between chest compressions. It is designed to reduce intrathoracic pressure and enhance venous return to the heart. A randomized study of 230 adults documented increased admissions to the ICU and 24-hour survival rates (LOE 2)³¹⁰ when an ITD was used with standard CPR in patients with cardiac arrest (PEA only) in the prehospital setting. The addition of the ITD improved the hemodynamics during standard CPR in 5 laboratory studies (LOE 6)^{311–315} and 1 clinical study (LOE 2).³¹⁶

A randomized study of 400 adults showed increased ROSC and 24-hour survival rates (LOE 1)³¹⁷ when an ITD was used with ACD-CPR in patients with cardiac arrest in the prehospital setting. The addition of the ITD improved the hemodynamics during ACD-CPR in 1 laboratory study (LOE 6)³¹⁸ and 1 clinical study (LOE 2).³¹⁹ One laboratory study failed to show an improvement in hemodynamics with the use of the ITD during ACD-CPR (LOE 6).³¹⁴ Compared with standard CPR, ROSC and 24-hour survival were increased when the ITD was used with ACD in a randomized study of 210 prehospital patients (LOE 1),³²⁰ and hemodynamics were improved in 2 laboratory studies (LOE 6).^{321,322}

Extracorporeal Techniques and Invasive Perfusion Devices^{W28,W82}

Consensus on Science

The only adult data comes from 3 case series (LOE 5).^{323–325} One of these³²³ indicated that extracorporeal CPR (ECPR) was more successful in postcardiotomy patients than those in cardiac arrest from other causes. The other 2 studies^{324,325} suggested that ECPR is not beneficial for patients presenting to the emergency department in cardiac arrest with the exception of cardiac arrest associated with hypothermia or drug intoxication.

Open-Chest CPR^{W81B}

Consensus on Science

No prospective randomized studies of open-chest CPR for resuscitation have been published. Four relevant human studies were reviewed, 2 after cardiac surgery (LOE 4³²⁶; LOE 5³²⁷) and 2 after out-of-hospital cardiac arrest (LOE 4³²⁸; LOE 5³²⁹). The observed benefits of open-chest cardiac massage included improved coronary perfusion pressure³²⁹ and increased ROSC.³²⁸ Evidence from animal studies (LOE 6)^{330–344} indicates that open-chest CPR produces greater survival rates, perfusion pressures, and organ blood flow than closed-chest CPR.

Treatment Recommendation

Open-chest CPR should be considered for patients with cardiac arrest in the early postoperative phase after cardiothoracic surgery or when the chest or abdomen is already open.

Periarrest Arrhythmias

Narrow-Complex Tachycardia

There are 4 options for the treatment of narrow-complex tachycardia in the periarrest setting: electrical conversion, physical maneuvers, pharmacologic conversion, or rate control. The choice depends on the stability of the patient and the rhythm. In a hemodynamically unstable patient, narrow-complex tachycardia is best treated with electrical cardioversion.

Drug Therapy for Atrial Fibrillation^{W86}

Consensus on Science

One randomized controlled trial in adults and 3 additional studies documented improvement in rate control when magnesium (LOE 3),³⁴⁵ diltiazem (LOE 2),³⁴⁶ or β -blockers (LOE 2)^{347,348} were given by physicians, nurses, and paramedics in both the out-of-hospital (LOE 3)³⁴⁹ and hospital settings to patients with atrial fibrillation with a rapid ventricular response.³⁴⁹

Two randomized controlled trials in adults (LOE 2)^{350,351} and additional studies documented improvement in rhythm when ibutilide, digoxin, clonidine, magnesium, or amiodarone were given by physicians or nurses to patients with atrial fibrillation in the hospital setting.

Treatment Recommendation

Magnesium, diltiazem, or β -blockers may be used for rate control in patients with atrial fibrillation with a rapid ventricular response. Amiodarone, ibutilide, propafenone, flecainide, digoxin, clonidine, or magnesium may be used for rhythm control in patients with atrial fibrillation.

Drug Therapy for Regular Narrow-Complex Tachycardia^{W87}

Consensus on Science

In one randomized study in the ED, 41 of 148 (28%) patients with paroxysmal supraventricular tachycardia (PSVT) were converted to sinus rhythm with carotid sinus massage or a Valsalva maneuver (LOE 2).³⁵² One study (LOE 4)³⁵³ showed that stable paroxysmal supraventricular tachycardia (PSVT)

in younger patients may be treated first with vagal maneuvers but will be unsuccessful 80% of the time.

Five prospective controlled nonrandomized cohort studies (LOE 2³⁵⁴; LOE 3^{355–358}) indicated that adenosine is safe and effective in converting PSVT in the hospital and out-of-hospital settings. Two randomized clinical trials (LOE 2)^{355,359} documented no statistical significance in PSVT conversion rate between adenosine and calcium channel blockers, but the effect of adenosine is more rapid, and side effects are less severe than with verapamil. One randomized clinical trial in the ED (LOE 2)³⁶⁰ documented no difference in the PSVT conversion rate between infusions of verapamil (99%) and diltiazem (96%). One randomized clinical trial in the ED (LOE 1)³⁶¹ documented significantly better PSVT conversion rates with diltiazem (100%) in comparison with esmolol (25%). One electrophysiologic study (LOE 6)³⁶² documented that amiodarone achieved 100% efficacy in the inhibition of induced sustained reentrant PSVT.

Treatment Recommendation

Stable narrow-complex tachycardia (excluding atrial fibrillation or atrial flutter) should be treated first with vagal maneuvers (avoiding carotid sinus massage in the elderly); these will terminate about 20% of PSVTs. If vagal maneuvers are not used or if they fail, give adenosine.

A calcium channel blocker (verapamil or diltiazem) infusion or amiodarone may be used as a second-line treatment for the 10% to 15% of patients who do not respond to adenosine. In unstable PSVT electrical cardioversion is the treatment of choice; IV rapid bolus adenosine can be tried if electrical cardioversion is not immediately available.

Broad-Complex Tachycardia

The stability of the patient determines the choice of treatment for wide-complex (broad-complex) tachycardia. In unstable wide-complex tachycardia electrical cardioversion is the treatment of choice.

Drug Therapy for Stable Ventricular Tachycardia^{W35,W88}

Consensus on Science

Three observational studies (LOE 5)^{363–365} indicated that amiodarone is effective for the termination of shock-resistant or drug-refractory VT. One randomized parallel study (LOE 2)¹³⁸ indicated that aqueous amiodarone is more effective than lidocaine in the treatment of shock-resistant VT. One randomized trial (LOE 2)³⁶⁶ indicated that procainamide is superior to lidocaine in terminating spontaneously occurring VT. Three retrospective analyses (LOE 5)^{367–369} indicated a low rate of termination of VT with lidocaine in patients with and without acute myocardial infarction. One randomized controlled trial (LOE 1)³⁷⁰ indicated that sotalol is significantly more effective than lidocaine for terminating acute sustained VT. One meta-analysis (LOE 1)³⁶⁷ showed that the overall risk of torsades de pointes in patients treated with a single infusion of IV sotalol is approximately 0.1%.

Treatment Recommendation

Amiodarone, procainamide, and sotalol are effective in terminating stable sustained VT.

Drug Therapy for Polymorphic Ventricular Tachycardia^{W89}

Consensus on Science

One observational study (LOE 5)³⁷¹ showed that IV magnesium will not terminate polymorphic VT (excluding torsades de pointes) in patients with a normal QT interval. Lidocaine is not effective, but amiodarone may be (LOE 4).³⁷²

Treatment Recommendation

For hemodynamically stable polymorphic VT, where electrical therapy is not desirable or is ineffective, treatment with amiodarone may be effective.

Therapy for Torsades de Pointes^{W90}

Consensus on Science

Two observational studies (LOE 5)^{371,373} showed that IV magnesium can effectively terminate torsades de pointes in patients with prolonged QT interval. One adult case series (LOE 5)³⁷⁴ showed that isoproterenol or ventricular pacing can be effective in terminating torsades de pointes associated with bradycardia and drug-induced QT prolongation.

Treatment Recommendation

Magnesium, isoproterenol, and ventricular pacing can be used to treat torsades de pointes.

Bradycardia

In the periarrest setting the rescuer should seek and treat reversible causes of bradycardia. In the absence of reversible causes, atropine remains the first-line drug for acute symptomatic bradycardia. Failure to respond to atropine will usually necessitate transcutaneous pacing, although second-line drug therapy with dopamine, epinephrine, isoproterenol, or theophylline may be successful. Fist pacing may be attempted pending the arrival of an electrical pacing unit.

Drug Therapy for Symptomatic Bradycardia^{W91}

Consensus on Science

In 1 randomized clinical trial in adults (LOE 2)³⁷⁵ and 1 historic cohort study in adults and additional reports (LOE 4),^{376–379} IV atropine improved heart rate, symptoms, and signs associated with bradycardia. An initial dose of 0.5 mg, repeated as needed to a total of 1.5 mg, was effective in both in-hospital and out-of-hospital treatment of symptomatic bradycardia.

In 2 prospective controlled nonrandomized cohort studies in hospitalized adults (LOE 4),^{376,380} administration of IV theophylline improved heart rate, symptoms, and signs associated with bradycardia that did not respond to atropine.

One case series (LOE 5)³⁷⁹ documented improvement in heart rate, symptoms, and signs associated with bradycardia when IV glucagon (3 mg initially, followed by infusion at 3 mg/h if necessary) was given to hospital patients with drug-induced symptomatic bradycardia not responding to atropine.

One study in 10 healthy volunteers indicated that a 3-mg dose of atropine produces the maximum achievable increase in resting heart rate (LOE 7).³⁸¹ One study indicated that atropine may paradoxically cause high-degree AV block in patients after cardiac transplantation (LOE 5).³⁸²

Treatment Recommendation

For symptomatic bradycardia, give atropine 0.5 to 1 mg IV, repeated every 3 to 5 minutes, to a total of 3 mg. Be prepared to initiate transcutaneous pacing quickly in patients who do not respond to atropine (or second-line drugs if these do not delay definitive management). Pacing is also recommended for severely symptomatic patients, especially when the block is at or below the His-Purkinje level. Second-line drugs for symptomatic bradycardia include dopamine, epinephrine, isoproterenol, and theophylline. Consider IV glucagon if β -blockers or calcium channel blockers are a potential cause of the bradycardia. Atropine should not be used in patients with cardiac transplants.

Fist Pacing in Cardiac Arrest^{W58}*Consensus on Science*

Three case series indicated that fist pacing can be effective. Two of the largest studies have included 100 (LOE 5)³⁸³ and 50 (LOE 5)³⁸⁴ patients. One study (LOE 5)³⁸⁵ compared fist pacing with 2 electrical modes in the same patient and found all 3 techniques equally effective. Selected case series indicate that the most effective technique is to deliver serial rhythmic blows (fist pacing) with the closed fist over the left lower edge of the sternum to pace the heart at a physiological rate of 50 to 70 beats per minute (bpm) (LOE 5).^{383,384} There are no prehospital case reports of fist pacing. In virtually all published cases of fist pacing, complete heart block was the underlying bradyarrhythmia.

Treatment Recommendation

Fist pacing may be considered in hemodynamically unstable bradyarrhythmias until an electrical pacemaker (transcutaneous or transvenous) is available.

Cardiac Arrest in Special Circumstances

In some circumstances modification of the standard resuscitation technique is required to maximize the victim's chance of survival. In many of these special circumstances recognition of the critically ill patient may enable early treatment to prevent cardiac arrest. The special circumstances reviewed during the consensus process can be categorized as environmental (hypothermia, submersion, electrocution), pregnancy, asthma, and drug overdose/poisoning.

Environmental**Hypothermia**^{W131,W162A}*Consensus on Science*

Hypothermic patients with pulse. One randomized controlled trial (LOE 1)³⁸⁶ showed active surface heating to be more effective than metallic foil insulation in an experimental model of accidental hypothermia. Two studies (LOE 4)^{387,388} documented successful active rewarming with external surface, forced air, and warm infusions.

Hypothermic patients with cardiac arrest. Two studies (LOE 4)^{389,390} documented successful resuscitation with prolonged CPR and successful recovery using invasive rewarming (extracorporeal circulation or cardiopulmonary bypass). Successful resuscitation from hypothermic cardiac arrest was

reported using active noninvasive rewarming (forced air, warm infusions) (LOE 4).³⁸⁹ Better outcomes were documented for nonasphyxial versus presumed asphyxial hypothermic arrest (LOE 4).³⁸⁹ For victims of avalanche, a small air pocket may prevent an asphyxial component of the arrest (LOE 5).³⁹¹

Treatment Recommendation

For hypothermic patients with a perfusing rhythm and without a preceding cardiac arrest, consider active (noninvasive) external warming (with heating blankets, forced air, and warmed infusion). Severely hypothermic patients in cardiac arrest may benefit from invasive warming (cardiopulmonary bypass or extracorporeal circulation).

Drowning^{W132,W160A,W160B}

For additional information see "Drowning" in Part 2: "Adult Basic Life Support."

Consensus on Science

One study indicated that victims of drowning are at risk for cervical spine injury only if they have clinical signs of severe injury (LOE 4).³⁹² Three single case reports (LOE 5)³⁹³⁻³⁹⁵ documented the use of exogenous surfactant for fresh water-induced severe respiratory distress syndrome; 2 victims survived. A case report described the use of noninvasive positive-pressure ventilation in 2 victims of submersion (LOE 5).³⁹⁶

There was no evidence to support or refute the use of steroids (LOE 5),³⁹⁷ nitric oxide (LOE 5),³⁹⁸ extracorporeal membrane oxygenation (ECMO) rewarming after ROSC (LOE 5),³⁸⁹ therapeutic hypothermia after ROSC (LOE 5),³⁹⁹ or vasopressin (LOE 5)⁴⁰⁰ after submersion. Case reports documented the use of ECMO in young children with severe hypothermia after submersion (LOE 5).^{401,402}

Treatment Recommendation

Victims of submersion should be removed from the water and resuscitated by the fastest means available. Only victims with risk factors (history of diving, water slide use, trauma, alcohol) or clinical signs of injury or focal neurologic signs should be treated as having a potential spinal cord injury, with stabilization of the cervical and thoracic spine.

Electrocution^{W135}*Consensus on Science*

Case reports (LOE 5)⁴⁰³⁻⁴¹² indicated that early BLS and ALS may be lifesaving and may decrease short- and long-term cardiac and neurologic sequelae for victims of electrocution and lightning injuries.

Case studies of victims of lightning and electric injuries emphasize the possible coexistence of multiple injuries and the importance of ensuring initial responder safety. Survivors may have permanent neurologic and cardiac sequelae.

Pregnancy**Etiology of Cardiac Arrest in Pregnancy**^{W119C,W134}*Consensus on Science*

One large case series (LOE 5)⁴¹³ suggested that systematic consideration of the reversible causes of cardiac arrest may

enable skilled rescuers to identify the etiology of cardiac arrest in pregnancy in the hospital setting.

Evidence extrapolated from peri-arrest resuscitation scenarios (LOE 7)^{414,415} indicated that ultrasound assessment undertaken by trained rescuers may help to identify intra-abdominal hemorrhage as a cause of cardiac arrest in pregnancy in the hospital setting.

Treatment Recommendation

Rescuers should try to identify common and reversible causes of cardiac arrest in pregnancy during resuscitation attempts. The use of abdominal ultrasound by a skilled operator should be considered in detecting pregnancy and possible causes of cardiac arrest in pregnancy, but this should not delay other treatments.

Resuscitation Technique for Pregnancy^{W134}

Consensus on Science

A case series (LOE 5)⁴¹⁶ and numerous case reports (LOE 7⁴¹⁷; LOE 8^{418–421}) documented an improvement in rates of maternal and neonatal survival to discharge when delivery of the fetus was performed within 5 minutes of cardiac arrest in pregnancy if initial resuscitative efforts by skilled rescuers in the hospital setting failed.

Extrapolation from anesthesia (LOE 7)⁴²² and a manikin study (LOE 6⁴²³) suggests that a left lateral tilt of 15 degrees will relieve aortocaval compression in the majority of pregnant women and enable effective chest compressions by rescuers in any setting.

A human volunteer study (LOE 7)⁴²⁴ showed that there was no change in transthoracic impedance during pregnancy. The standard recommended energy levels for adults should be used by rescuers when attempting defibrillation in cardiac arrest during pregnancy in any setting.

Treatment Recommendation

If initial resuscitative efforts fail, caesarean delivery of the fetus (hysterotomy) should be performed within 5 minutes of onset of cardiac arrest in pregnancy to improve maternal or fetal survival. A left lateral tilt of 15 degrees is required to relieve inferior vena caval compression in the majority of pregnant women. The energy levels used for defibrillation in adults are appropriate for use in pregnancy.

Asthma

Defibrillation in Asthma^{W119B,W133}

Consensus on Science

One volunteer study in healthy adults (LOE 7)⁴²⁵ documented an increased transthoracic impedance with increasing positive end-expiratory pressure (PEEP) and suggested that increased shock energy may be required if initial defibrillation attempts fail for patients with asthma-induced cardiac arrest in any clinical setting.

Treatment Recommendation

If initial attempts at defibrillation fail for the patient with asthma and VF, higher shock energies should be considered.

Ventilation in Asthma^{W119B}

Consensus on Science

Evidence extrapolated from a systematic review of patients with noncardiac arrest (LOE 7)⁴²⁶ suggested decreased dynamic hyperinflation (auto-PEEP) when helium/oxygen mixtures were used to ventilate the lungs of asthmatic patients during in-hospital cardiac arrest.

Evidence extrapolated from 3 noncardiac arrest case series (LOE 7)^{427–429} suggested that asthmatic patients were at risk of gas trapping during cardiac arrest, especially if they were ventilated with higher tidal volumes and rates than recommended. Two small case series (LOE 5)^{430,431} and anecdotal reports (LOE 8)⁴³² failed to show a consistent benefit from compression of the chest wall, followed by a period of apnea to relieve gas trapping, for patients with asthma-induced cardiac arrest in any clinical setting (see also “Disconnection From Ventilation During Cardiac Arrest,” above).

Evidence extrapolated from a noncardiac arrest case series (LOE 7)⁴²⁸ suggested improved ventilation of the lungs and decreased gastric inflation if the trachea is intubated early by trained rescuers for patients with asthma-induced cardiac arrest in any setting. Evidence from 2 noncardiac arrest case reports (LOE 7⁴³³; LOE 8⁴³⁴) neither supported nor refuted the use of open-chest ventilation and cardiac compressions in asthma-induced cardiac arrest.

Treatment Recommendation

There are insufficient data to support or refute the use of helium-oxygen mixtures in asthma-related cardiac arrest. Compression of the chest wall or a period of apnea may relieve gas trapping if dynamic hyperinflation occurs. In asthma-related cardiac arrest the patient’s trachea should be intubated early to facilitate ventilation and minimize the risk of gastric inflation.

Drug Overdose and Poisoning^{W198}

Sodium Bicarbonate for Poisoning and Electrolyte

Disturbances^{W197A,W197B,W197C,W197D,W197E}

Consensus on Science

Evidence from the use of bicarbonate in calcium channel blocker overdose in 2 children (LOE 5)⁴³⁵ with fatal overdoses of nifedipine neither supported nor refuted the value of bicarbonate in calcium channel blocker overdose.

There were no controlled human studies of sodium bicarbonate therapy for arrhythmias or hypotension related to tricyclic antidepressant overdose. However, evidence from case reports (LOE 5);^{436,437} animal studies (LOE 6),^{438–447} and in vitro studies (LOE 6^{445,448,449}; LOE 7^{450,451}) supported the use of sodium bicarbonate to treat tricyclic antidepressant-induced arrhythmias or hypotension.

Treatment Recommendation

Sodium bicarbonate is recommended for the treatment of tricyclic antidepressant-induced arrhythmia or hypotension. Although no study has investigated the optimal target pH with bicarbonate therapy, a pH of 7.45 to 7.55 has been commonly accepted and seems reasonable.

Ventilation Before Naloxone in Opioid Overdose^{W18, W106}*Consensus on Science*

Evidence from case series (LOE 5)^{452–454} in adults and extrapolation from LOE 7^{455,456} and LOE 8⁴⁵⁷ studies indicate fewer adverse events when ventilation is provided before administration of naloxone by EMS personnel to patients with opioid-induced respiratory depression in the prehospital setting.

Postresuscitation Care

ROSC is just the first step toward the goal of complete recovery from cardiac arrest. Interventions in the postresuscitation period are likely to significantly influence the final outcome, yet there are relatively few data relating to this phase. In the absence of firm guidelines, approaches to postresuscitation care are heterogeneous. Postresuscitation interventions are categorized into the following areas: (1) ventilation, (2) temperature control (therapeutic hypothermia and prevention and treatment of hyperthermia), (3) seizure control and sedation, and (4) other supportive therapies (blood glucose control, coagulation control, prophylactic antiarrhythmic therapy).

Therapeutic hypothermia improves neurologic outcome in some cardiac arrest survivors, and hyperthermia appears harmful. Tight blood glucose control improves outcome in undifferentiated critically ill patients, but the effect of this therapy in the postresuscitation phase is unknown. Prediction of outcome in comatose survivors of cardiac arrest remains problematic: median nerve somatosensory-evoked potentials measured 72 hours after cardiac arrest may be helpful, but analyses of several serum markers were inconclusive.

Ventilation**Control of Arterial Carbon Dioxide**^{W114B}*Consensus on Science*

Five studies in adults (LOE 2^{458,459}; LOE 3⁴⁶⁰; LOE 5⁴⁶¹; LOE 7⁴⁶²) and numerous animal studies (LOE 6)^{463–465} documented harmful effects of hypocapnia (cerebral ischemia) after cardiac arrest. Two studies provide neutral evidence (LOE 5⁴⁶⁶; LOE 6⁴⁶⁷).

Treatment Recommendation

There are no data to support the targeting of a specific PaCO₂ after resuscitation from cardiac arrest. Data extrapolated from patients with brain injury, however, imply that ventilation to normocarbia is appropriate. Routine hyperventilation may be detrimental and should be avoided.

Temperature Control**Therapeutic Hypothermia**^{W109A, W109B}*Consensus on Science*

Two randomized clinical trials (LOE 1⁴⁶⁸; LOE 2⁴⁶⁹) showed improved outcome in adults who remained comatose after initial resuscitation from out-of-hospital VF cardiac arrest and who were cooled within minutes to hours after ROSC. Patients in these studies were cooled to 33°C⁴⁶⁸ or to the range of 32°C to 34°C⁴⁶⁹ for 12 to 24 hours. The Hypother-

mia After Cardiac Arrest (HACA) study⁴⁶⁸ included a small subset of patients with in-hospital cardiac arrest.

One study (LOE 2)⁴⁷⁰ documented improved metabolic end points (lactate and O₂ extraction) when comatose adult patients were cooled after ROSC from out-of-hospital cardiac arrest in which the initial rhythm was PEA/asystole. A small study (LOE 4)⁴⁷¹ showed benefit after therapeutic hypothermia in comatose survivors of non-VF arrest.

External or internal cooling techniques can be used to initiate cooling within minutes to hours (LOE 1⁴⁶⁸; LOE 2^{469,470}; LOE 5^{472–475}). The only studies documenting improved outcome with therapeutic hypothermia after cardiac arrest used external cooling (LOE 1⁴⁶⁸; LOE 2^{469,470}). An infusion of 30 mL/kg of 4°C saline achieved a decrease in core temperature of approximately 1.5°C (LOE 5).^{472,473,475} One study in patients with cardiac arrest (LOE 5)⁴⁷⁴ and 3 other studies (LOE 7)^{476–478} have documented that intravascular cooling enables more precise control of core temperature than external methods.

Studies documenting improved outcome with therapeutic hypothermia after cardiac arrest used continuous temperature monitoring (LOE 1⁴⁶⁸; LOE 2^{469,470}).

Multiple studies in animals (LOE 6)^{479–484} documented the importance of initiating cooling as soon as possible and for adequate duration (eg, 12 to 24 hours). Optimal parameters, including onset, depth, and duration of cooling, are unknown.

Seizures or myoclonus occurs in survivors of cardiac arrest (LOE 5)^{474,485–487}. Shivering will necessitate sedation and intermittent or continuous neuromuscular blockade. Use of continuous neuromuscular blockade could mask seizure activity.

Treatment Recommendation

Unconscious adult patients with spontaneous circulation after out-of-hospital cardiac arrest should be cooled to 32°C to 34°C for 12 to 24 hours when the initial rhythm was VF. Cooling to 32°C to 34°C for 12 to 24 hours may be considered for unconscious adult patients with spontaneous circulation after out-of-hospital cardiac arrest from any other rhythm or cardiac arrest in hospital.

Prevention and Treatment of Hyperthermia^{W110}*Consensus on Science*

A period of postarrest hyperthermia is common in the first 48 hours after cardiac arrest (LOE 4).^{488–490} There were no controlled prospective studies that examined the clinical impact of antipyretics (or physical cooling devices) to prevent hyperthermia after cardiac arrest.

The risk of unfavorable neurologic outcome increased for each degree of body temperature >37°C (LOE 3).⁴⁹¹ Hyperthermia was associated with increased morbidity and mortality in post-stroke patients (LOE 7).⁴⁹² Post-stroke pyrexia was not treated effectively by antipyretics such as acetaminophen or ibuprofen (LOE 7);^{493,494} however, antipyretics or physical cooling methods have been associated with decreased infarct volumes in animal models of global ischemia (LOE 7).^{495,496}

Treatment Recommendation

Hyperthermia should be avoided after cardiac arrest.

Seizure Control and Sedation**Prevention and Control of Seizures**^{W111A,W111B}*Consensus on Science*

There were no studies that directly addressed the use of prophylactic anticonvulsant drugs after cardiac arrest in adults. There are data indicating that seizures can precipitate cardiac arrest (LOE 4^{497,498}; LOE 5^{486,499–501}; LOE 8⁵⁰¹) and respiratory arrest (LOE 5).⁵⁰²

Treatment Recommendation

Seizures increase the oxygen requirements of the brain and can cause life-threatening arrhythmias and respiratory arrest; therefore, seizures following cardiac arrest should be treated promptly and effectively. Maintenance therapy should be started after the first event once potential precipitating causes (eg, intracranial hemorrhage, electrolyte imbalance, etc) are excluded.

Sedation and Pharmacologic Paralysis^{W113}*Consensus on Science*

There were no data to support or refute the use of a defined period of ventilation, sedation, and neuromuscular blockade after cardiac arrest. One observational study in adults (LOE 5)⁵⁰³ documents increased incidence of pneumonia when sedation is prolonged beyond 48 hours after prehospital or in-hospital cardiac arrest.

Other Supportive Therapies**Blood Glucose Control**^{W115A,W115B}*Consensus on Science*

Tight control of blood glucose (range 80 to 110 mg/dL or 4.4 to 6.1 mmol/L) with insulin reduces hospital mortality rates in critically ill adults (LOE 1⁵⁰⁴; LOE 4⁵⁰⁵), but this has not been shown in post-cardiac arrest patients. Several human studies have documented a strong association between high blood glucose after resuscitation from cardiac arrest and poor neurologic outcome (LOE 4⁵⁰⁶; LOE 5^{507–513}). There was good evidence that persistent hyperglycemia after stroke is associated with a worse neurologic outcome (LOE 7).^{514–517}

The optimal blood glucose target in critically ill patients has not been determined. Comatose patients were at particular risk from unrecognized hypoglycemia, and the risk of this complication occurring increases as the target blood glucose concentration is lowered (LOE 8). One study in rats has shown that glucose plus insulin improves cerebral outcome after asphyxial cardiac arrest (LOE 6).⁵¹⁸

Therapeutic hypothermia was associated with hyperglycemia (LOE 2).⁴⁶⁹

Treatment Recommendation

Providers should monitor blood glucose frequently after cardiac arrest and should treat hyperglycemia with insulin but avoid hypoglycemia.

Coagulation Control^{W116}*Consensus on Science*

There are no studies evaluating the role of anticoagulation alone to improve outcome after ROSC. In three nonexper-

mental reports (LOE 4¹⁶⁸; LOE 5⁵¹⁹; LOE 6¹⁷⁹) using fibrinolytics combined with heparin (anticoagulation) after prolonged cardiac arrest in humans, ROSC, but not 24-hour survival rates, was significantly better.

Prophylactic Antiarrhythmic Therapy^{W118A,W118B}*Consensus on Science*

No studies specifically and directly addressed the prophylactic use of antiarrhythmic therapy started immediately after resuscitation from cardiac arrest. Six studies (LOE 5)^{520–525} documented inconsistent improvement in long-term survival when prophylactic antiarrhythmics were given to survivors of cardiac arrest from all causes. Six studies (LOE 1^{526–528}; LOE 2^{529,530}; LOE 3⁵³¹) showed that implantable cardioverter defibrillators (ICDs) improve survival when compared with antiarrhythmics in survivors of cardiac arrest.

Treatment Recommendation

Giving prophylactic antiarrhythmics to patients who have survived cardiac arrest, irrespective of etiology, can neither be recommended nor rejected. It may be reasonable, however, to continue an infusion of an antiarrhythmic drug that successfully restored a stable rhythm during resuscitation.

Prognostication**Prognostication During Cardiac Arrest****Predictive Value of Neurologic Examination**^{W122A,W122B}*Consensus on Science*

Five studies (LOE 4^{532,533}; LOE 5^{534–536}) documented some ability to predict outcome in adults when neurologic examination is undertaken during cardiac arrest, but there is insufficient negative predictive value for this assessment to be used clinically.

Treatment Recommendation

Relying on the neurologic exam during cardiac arrest to predict outcome is not recommended and should not be used.

Prognostication After Resuscitation**Predictive Value of Standard Laboratory Analyses**^{W126}*Consensus on Science*

In 8 human prospective studies (LOE 3^{537,538}; LOE 4^{241,539–543}) of the value of biomarkers in predicting outcome from cardiac arrest, none was clinically useful in ascertaining outcome in the acute setting. One retrospective human study suggested that creatine kinase-MB could be used as an independent predictor of survival (LOE 4),⁵³⁹ but delays in completing the measurement may make this clinically less helpful.

In some studies in animals (LOE 6),^{544–556} lactate and acid base values showed a trend correlating with unfavorable outcomes. None of these studies could conclusively formulate a predictive model identifying a biochemical marker level that gave a reasonable prediction of outcome.

Predictive Value of Neuron-Specific Enolase and Protein S-100b^{W126}*Consensus on Science*

One randomized controlled study (LOE 2),⁵⁵⁷ 4 prospective controlled studies (LOE 3),^{558–561} and 11 case series/cohort

studies (LOE 4^{506,539,562–564}; LOE 5^{512,513,565–568}) indicated that neuron-specific enolase (NSE) and protein S-100b may be useful in predicting the outcome of cardiac arrest. But the 95% confidence interval (CI) in these trials was wide, and in many of the trials, return to consciousness (without comment on level of function) was considered a “good” outcome.

The only meta-analysis to look at this topic estimated that to obtain 95% CI with a 5% false-positive rate would require a study population of approximately 600 patients (LOE 1).⁵⁶⁹ No study this large has been conducted.

Treatment Recommendation

No laboratory analyses (NSE, S-100b, base deficit, glucose, or soluble P-selectin) provide reliable prediction of the outcome after cardiac arrest.

Somatosensory-Evoked Potentials^{W124A,W124B}

Consensus on Science

Eighteen prospective studies (LOE 3)^{568,570–586} and 1 meta-analysis (LOE 1)⁵⁸⁷ indicated that median nerve somatosensory-evoked potentials in normothermic patients comatose for at least 72 hours after cardiac arrest predict poor outcome with 100% specificity. Bilateral absence of the N20 component of the evoked potentials in comatose patients with coma of hypoxic-anoxic origin is uniformly fatal.

Treatment Recommendation

Median nerve somatosensory-evoked potentials measured 72 hours after cardiac arrest can be used to predict a fatal outcome in patients with hypoxic-anoxic coma.

Electroencephalogram

Consensus on Science

The use of the electroencephalogram (EEG), performed at least 24 to 48 hours after arrest, has been evaluated in case series of humans (LOE 5)^{578,585,588–598} and animals (LOE 6).^{599–601} On the modified Hockaday scale, grades I (normal alpha with theta-delta activity), IV (alpha coma, spikes, sharp waves, slow waves with very little background activity), and V (very flat to isoelectric) were most useful prognostically. But the prognosis was unpredictable for those with grade II and III EEGs.

Treatment Recommendation

The use of the EEG performed a minimum of 24 to 48 hours after a cardiac arrest can help define the prognosis in patients with grade I, IV, and V EEGs.

References

- Kuisma M, Alaspaa A. Out-of-hospital cardiac arrests of non-cardiac origin: epidemiology and outcome. *Eur Heart J*. 1997;18:1122–1128.
- Pell JP, Sirel JM, Marsden AK, Ford I, Walker NL, Cobbe SM. Presentation, management, and outcome of out of hospital cardiopulmonary arrest: comparison by underlying aetiology. *Heart (British Cardiac Society)*. 2003;89:839–842.
- Bellomo R, Goldsmith D, Uchino S, Buckmaster J, Hart GK, Opdam H, Silvester W, Doolan L, Gutteridge G. A prospective before-and-after trial of a medical emergency team. *Med J Aust*. 2003;179:283–287.
- Buist MD, Moore GE, Bernard SA, Waxman BP, Anderson JN, Nguyen TV. Effects of a medical emergency team on reduction of incidence of and mortality from unexpected cardiac arrests in hospital: preliminary study. *BMJ*. 2002;324:387–390.
- Hillman K, Chen J, Cretikos M, Bellomo R, Brown D, Doig G, Finfer S, Flabouris A. Introduction of the medical emergency team (MET) system: a cluster-randomised controlled trial. *Lancet*. 2005;365:2091–2097.
- Kenward G, Castle N, Hodgetts T, Shaikh L. Evaluation of a medical emergency team one year after implementation. *Resuscitation*. 2004;61:257–263.
- Bristow PJ, Hillman KM, Chey T, Daffurn K, Jacques TC, Norman SL, Bishop GF, Simmons EG. Rates of in-hospital arrests, deaths and intensive care admissions: the effect of a medical emergency team. *Med J Aust*. 2000;173:236–240.
- Tibballs J, Kinney S, Duke T, Oakley E, Hennessy M. Reduction of paediatric in-patient cardiac arrest and death with a medical emergency team: preliminary results. *Archives for Disease in Children*. In press.
- Goldhill DR, Worthington L, Mulcahy A, Tarling M, Sumner A. The patient-at-risk team: identifying and managing seriously ill ward patients. *Anaesthesia*. 1999;54:853–860.
- Pittard AJ. Out of our reach? Assessing the impact of introducing a critical care outreach service. *Anaesthesia*. 2003;58:882–885.
- Subbe CP, Davies RG, Williams E, Rutherford P, Gemmill L. Effect of introducing the Modified Early Warning score on clinical outcomes, cardio-pulmonary arrests and intensive care utilisation in acute medical admissions. *Anaesthesia*. 2003;58:797–802.
- Chung CH, Sum CW, Li HL, Cheng KS, Tan PC. Comparison of nasal trauma associated with nasopharyngeal airway applied by nurses and experienced anesthesiologists. *Changeng Yi Xue Za Zhi*. 1999;22:593–597.
- Roberts K, Porter K. How do you size a nasopharyngeal airway. *Resuscitation*. 2003;56:19–23.
- Stoneham MD. The nasopharyngeal airway. Assessment of position by fiberoptic laryngoscopy. *Anaesthesia*. 1993;48:575–580.
- Schade K, Borzotta A, Michaels A. Intracranial malposition of nasopharyngeal airway. *J Trauma*. 2000;49:967–968.
- Muzzi DA, Losasso TJ, Cucchiara RF. Complication from a nasopharyngeal airway in a patient with a basilar skull fracture. *Anesthesiology*. 1991;74:366–368.
- Gausche M, Lewis RJ, Stratton SJ, Haynes BE, Gunter CS, Goodrich SM, Poore PD, McCollough MD, Henderson DP, Pratt FD, Seidel JN. Effect of out-of-hospital pediatric endotracheal intubation on survival and neurological outcome: a controlled clinical trial. *JAMA*. 2000;283:783–790.
- Guly UM, Mitchell RG, Cook R, Steedman DJ, Robertson CE. Paramedics and technicians are equally successful at managing cardiac arrest outside hospital. *BMJ*. 1995;310:1091–1094.
- Stiell IG, Wells GA, Field B, Spaite DW, Nesbitt LP, De Maio VJ, Nichol G, Cousineau D, Blackburn J, Munkley D, Luinstra-Toohy L, Campeau T, Dagnone E, Lyver M. Advanced cardiac life support in out-of-hospital cardiac arrest. *N Engl J Med*. 2004;351:647–656.
- Jones JH, Murphy MP, Dickson RL, Somerville GG, Brizendine EJ. Emergency physician-verified out-of-hospital intubation: miss rates by paramedics. *Acad Emerg Med*. 2004;11:707–709.
- Pelucio M, Halligan L, Dhindsa H. Out-of-hospital experience with the syringe esophageal detector device. *Acad Emerg Med*. 1997;4:563–568.
- Sayre MR, Sakles JC, Mistler AF, Evans JL, Kramer AT, Pancioli AM. Field trial of endotracheal intubation by basic EMTs. *Ann Emerg Med*. 1998;31:228–233.
- Katz SH, Falk JL. Misplaced endotracheal tubes by paramedics in an urban emergency medical services system. *Ann Emerg Med*. 2001;37:32–37.
- Atherton GL, Johnson JC. Ability of paramedics to use the Combitube in prehospital cardiac arrest. *Ann Emerg Med*. 1993;22:1263–1268.
- Frass M, Frenzer R, Rauscha F, Schuster E, Glogar D. Ventilation with the esophageal tracheal combitube in cardiopulmonary resuscitation: promptness and effectiveness. *Chest*. 1988;93:781–784.
- Rabitsch W, Schellongowski P, Staudinger T, Hofbauer R, Dufek V, Eder B, Raab H, Thell R, Schuster E, Frass M. Comparison of a conventional tracheal airway with the Combitube in an urban emergency medical services system run by physicians. *Resuscitation*. 2003;57:27–32.
- Rumball C, Macdonald D, Barber P, Wong H, Smecher C. Endotracheal intubation and esophageal tracheal Combitube insertion by regular ambulance attendants: a comparative trial. *Prehosp Emerg Care*. 2004;8:15–22.
- Staudinger T, Brugger S, Roggla M, Rintelen C, Atherton GL, Johnson JC, Frass M. [Comparison of the Combitube with the endotracheal tube

- in cardiopulmonary resuscitation in the prehospital phase]. *Wien Klin Wochenschr.* 1994;106:412–415.
29. Oczenski W, Krenn H, Dahaba AA, Binder M, El-Schahawi-Kienzl I, Kohout S, Schwarz S, Fitzgerald RD. Complications following the use of the Combitube, tracheal tube and laryngeal mask airway. *Anaesthesia.* 1999;54(12):1161–1165.
 30. Hartmann T, Krenn CG, Zoeggeler A, Hoerauf K, Benumof JL, Krafft P. The oesophageal-tracheal Combitube Small Adult. *Anaesthesia.* 2000;55:670–675.
 31. Frass M, Rodler S, Frenzer R, Ilias W, Leithner C, Lackner F. Esophageal tracheal combitube, endotracheal airway, and mask: comparison of ventilatory pressure curves. *J Trauma.* 1989;29:1476–1479.
 32. Staudinger T, Brugger S, Watschinger B, Roggla M, Dielacher C, Lobl T, Fink D, Klausner R, Frass M. Emergency intubation with the Combitube: comparison with the endotracheal airway. *Ann Emerg Med.* 1993;22:1573–1575.
 33. Tanigawa K, Shigematsu A. Choice of airway devices for 12,020 cases of nontraumatic cardiac arrest in Japan. *Prehosp Emerg Care.* 1998;2:96–100.
 34. Lefrançois DP, Dufour DG. Use of the esophageal tracheal combitube by basic emergency medical technicians. *Resuscitation.* 2002;52:77–83.
 35. Ochs M, Vilke GM, Chan TC, Moats T, Buchanan J. Successful prehospital airway management by EMT-Ds using the combitube. *Prehosp Emerg Care.* 2000;4:333–337.
 36. Vezina D, Lessard MR, Bussieres J, Topping C, Trepanier CA. Complications associated with the use of the Esophageal-Tracheal Combitube. *Can J Anaesth.* 1998;45:76–80.
 37. Richards CF. Piriform sinus perforation during Esophageal-Tracheal Combitube placement. *J Emerg Med.* 1998;16:37–39.
 38. Rumball CJ, MacDonald D. The PTL, Combitube, laryngeal mask, and oral airway: a randomized prehospital comparative study of ventilatory device effectiveness and cost-effectiveness in 470 cases of cardiorespiratory arrest. *Prehosp Emerg Care.* 1997;1:1–10.
 39. Davies PR, Tighe SQ, Greenslade GL, Evans GH. Laryngeal mask airway and tracheal tube insertion by unskilled personnel. *Lancet.* 1990;336:977–979.
 40. Flaishon R, Sotman A, Ben-Abraham R, Rudick V, Varssano D, Weinbroum AA. Antichemical protective gear prolongs time to successful airway management: a randomized, crossover study in humans. *Anesthesiology.* 2004;100:260–266.
 41. Ho BY, Skinner HJ, Mahajan RP. Gastro-oesophageal reflux during day case gynaecological laparoscopy under positive pressure ventilation: laryngeal mask vs. tracheal intubation. *Anaesthesia.* 1998;53:921–924.
 42. Reinhart DJ, Simmons G. Comparison of placement of the laryngeal mask airway with endotracheal tube by paramedics and respiratory therapists. *Ann Emerg Med.* 1994;24:260–263.
 43. Rewari W, Kaul HL. Regurgitation and aspiration during gynaecological laparoscopy: comparison between laryngeal mask airway and tracheal intubation. *Journal of Anaesthesiology Clinical Pharmacology.* 1999;15(1):67–70.
 44. Pennant JH, Walker MB. Comparison of the endotracheal tube and laryngeal mask in airway management by paramedical personnel. *Anesth Analg.* 1992;74:531–534.
 45. Maltby JR, Berialt MT, Watson NC, Liepert DJ, Fick GH. LMA-Classic and LMA-ProSeal are effective alternatives to endotracheal intubation for gynecologic laparoscopy. *Can J Anaesth.* 2003;50:71–77.
 46. Dorges V, Ocker H, Wenzel V, Steinfath M, Gerlach K. The laryngeal tube S: a modified simple airway device. *Anesth Analg.* 2003;96(2):618–621.
 47. Alexander R, Hodgson P, Lomax D, Bullen C. A comparison of the laryngeal mask airway and Guedel airway, bag and face mask for manual ventilation following formal training. *Anaesthesia.* 1993;48:231–234.
 48. Burgoyne L, Cyna A. Laryngeal mask vs intubating laryngeal mask: insertion and ventilation by inexperienced resuscitators. *Anaesth Intensive Care.* 2001;29:604–608.
 49. Coulson A, Brimacombe J, Keller C, Wiseman L, Ingham T, Cheung D, Popwycz L, Hall B. A comparison of the ProSeal and classic laryngeal mask airways for airway management by inexperienced personnel after manikin-only training. *Anaesth Intensive Care.* 2003;31:286–289.
 50. Dingley J, Baynham P, Swart M, Vaughan RS. Ease of insertion of the laryngeal mask airway by inexperienced personnel when using an introducer. *Anaesthesia.* 1997;52:756–760.
 51. Roberts I, Allsop P, Dickinson M, Curry P, Eastwick-Field P, Eyre G. Airway management training using the laryngeal mask airway: a comparison of two different training programmes. *Resuscitation.* 1997;33:211–214.
 52. Yardy N, Hancox D, Strang T. A comparison of two airway aids for emergency use by unskilled personnel: the Combitube and laryngeal mask. *Anaesthesia.* 1999;54:181–183.
 53. Stone BJ, Chantler PJ, Baskett PJ. The incidence of regurgitation during cardiopulmonary resuscitation: a comparison between the bag valve mask and laryngeal mask airway. *Resuscitation.* 1998;38:3–6.
 54. Verghese C, Prior-Willeard PF, Baskett PJ. Immediate management of the airway during cardiopulmonary resuscitation in a hospital without a resident anaesthesiologist. *Eur J Emerg Med.* 1994;1:123–125.
 55. Samarkandi AH, Seraj MA, el Dawlatly A, Mastan M, Bakhamees HB. The role of laryngeal mask airway in cardiopulmonary resuscitation. *Resuscitation.* 1994;28:103–106.
 56. Kokkinis K. The use of the laryngeal mask airway in CPR. *Resuscitation.* 1994;27:9–12.
 57. Paterson SJ, Byrne PJ, Molesky MG, Seal RF, Finucane BT. Neonatal resuscitation using the laryngeal mask airway. *Anesthesiology.* 1994;80:1248–1253.
 58. The use of the laryngeal mask airway by nurses during cardiopulmonary resuscitation: results of a multicentre trial. *Anaesthesia.* 1994;49:3–7.
 59. Leach A, Alexander CA, Stone B. The laryngeal mask in cardiopulmonary resuscitation in a district general hospital: a preliminary communication. *Resuscitation.* 1993;25:245–248.
 60. Grantham H, Phillips G, Gilligan JE. The laryngeal mask in prehospital emergency care. 1994;6:193–197.
 61. Martin PD, Cyna AM, Hunter WA, Henry J, Ramayya GP. Training nursing staff in airway management for resuscitation. A clinical comparison of the facemask and laryngeal mask. *Anaesthesia.* 1993;48:33–37.
 62. Asai T, Moriyama S, Nishita Y, Kawachi S. Use of the laryngeal tube during cardiopulmonary resuscitation by paramedical staff. *Anaesthesia.* 2003;58:393–394.
 63. Genzwuerker HV, Dhonau S, Ellinger K. Use of the laryngeal tube for out-of-hospital resuscitation. *Resuscitation.* 2002;52:221–224.
 64. Schmidbauer W, Bubser H. [Use of the laryngeal tube during premedical resuscitation.]. *Notarzt.* 2002;18:266–268.
 65. Cook TM, McCormick B, Asai T. Randomized comparison of laryngeal tube with classic laryngeal mask airway for anaesthesia with controlled ventilation. *Br J Anaesth.* 2003;91(3):373–378.
 66. Asai T, Hidaka I, Kawachi S. Efficacy of the laryngeal tube by inexperienced personnel. *Resuscitation.* 2002;55(2):171–175.
 67. Ocker H, Wenzel V, Schmucker P, Steinfath M, Dorges V. A comparison of the laryngeal tube with the laryngeal mask airway during routine surgical procedures. *Anesth Analg.* 2002;95(4):1094–1097.
 68. Wrobel M, Grundmann U, Wilhelm W, Wagner S, Larsen R. [Laryngeal tube versus laryngeal mask airway in anaesthetised non-paralysed patients: A comparison of handling and postoperative morbidity]. *Anaesthesist.* 2004;53:702–708.
 69. Li J. Capnography alone is imperfect for endotracheal tube placement confirmation during emergency intubation. *J Emerg Med.* 2001;20:223–229.
 70. Grmec S. Comparison of three different methods to confirm tracheal tube placement in emergency intubation. *Intensive Care Med.* 2002;28:701–704.
 71. Anton WR, Gordon RW, Jordan TM, Posner KL, Cheney FW. A disposable end-tidal CO₂ detector to verify endotracheal intubation. *Ann Emerg Med.* 1991;20:271–275.
 72. Bhende MS, Thompson AE, Cook DR, Saville AL. Validity of a disposable end-tidal CO₂ detector in verifying endotracheal tube placement in infants and children. *Ann Emerg Med.* 1992;21:142–145.
 73. Bhende MS, Thompson AE. Evaluation of an end-tidal CO₂ detector during pediatric cardiopulmonary resuscitation. *Pediatrics.* 1995;95:395–399.
 74. Hayden SR, Sciammarella J, Viccellio P, Thode H, Delagi R. Colorimetric end-tidal CO₂ detector for verification of endotracheal tube placement in out-of-hospital cardiac arrest. *Acad Emerg Med.* 1995;2:499–502.
 75. MacLeod BA, Heller MB, Gerard J, Yealy DM, Menegazzi JJ. Verification of endotracheal tube placement with colorimetric end-tidal CO₂ detection. *Ann Emerg Med.* 1991;20:267–270.
 76. Ornato JP, Shipley JB, Racht EM, Slovic CM, Wrenn KD, Pepe PE, Almeida SL, Ginger VF, Fotre TV. Multicenter study of a portable, hand-size, colorimetric end-tidal carbon dioxide detection device. *Ann Emerg Med.* 1992;21:518–523.

77. Takeda T, Tanigawa K, Tanaka H, Hayashi Y, Goto E, Tanaka K. The assessment of three methods to verify tracheal tube placement in the emergency setting. *Resuscitation*. 2003;56:153–157.
78. Tanigawa K, Takeda T, Goto E, Tanaka K. The efficacy of esophageal detector devices in verifying tracheal tube placement: a randomized cross-over study of out-of-hospital cardiac arrest patients. *Anesth Analg*. 2001;92:375–378.
79. Varon AJ, Morrino J, Civetta JM. Clinical utility of a colorimetric end-tidal CO₂ detector in cardiopulmonary resuscitation and emergency intubation. *J Clin Monit*. 1991;7:289–293.
80. Sayah AJ, Peacock WF, Overton DT. End-tidal CO₂ measurement in the detection of esophageal intubation during cardiac arrest. *Ann Emerg Med*. 1990;19:857–860.
81. Sum Ping ST, Mehta MP, Symreng T. Accuracy of the FEF CO₂ detector in the assessment of endotracheal tube placement. *Anesth Analg*. 1992;74:415–419.
82. Campbell RC, Boyd CR, Shields RO, Odum JW, Corse KM. Evaluation of an end-tidal carbon dioxide detector in the aeromedical setting. *J Air Med Transp*. 1990;9:13–15.
83. Tanigawa K, Takeda T, Goto E, Tanaka K. Accuracy and reliability of the self-inflating bulb to verify tracheal intubation in out-of-hospital cardiac arrest patients. *Anesthesiology*. 2000;93:1432–1436.
84. Bozeman WP, Hexter D, Liang HK, Kelen GD. Esophageal detector device versus detection of end-tidal carbon dioxide level in emergency intubation. *Ann Emerg Med*. 1996;27:595–599.
85. Sharieff GQ, Rodarte A, Wilton N, Bleyle D. The self-inflating bulb as an airway adjunct: is it reliable in children weighing less than 20 kilograms? *Acad Emerg Med*. 2003;10:303–308.
86. Wee MY, Walker AK. The oesophageal detector device: an assessment with uncuffed tubes in children. *Anaesthesia*. 1991;46:869–871.
87. Williams KN, Nunn JF. The oesophageal detector device: a prospective trial on 100 patients. *Anaesthesia*. 1989;44:412–424.
88. Zaleski L, Abello D, Gold MI. The esophageal detector device. Does it work? *Anesthesiology*. 1993;79:244–247.
89. Haynes SR, Morton NS. Use of the oesophageal detector device in children under one year of age. *Anaesthesia*. 1990;45:1067–1069.
90. Levy H, Griego L. A comparative study of oral endotracheal tube securing methods. *Chest*. 1993;104:1537–1540.
91. Tasota FJ, Hoffman LA, Zullo TG, Jamison G. Evaluation of two methods used to stabilize oral endotracheal tubes. *Heart Lung*. 1987;16:140–146.
92. Aufderheide TP, Sigurdsson G, Pirralo RG, Yannopoulos D, McKnite S, von Briesen C, Sparks CW, Conrad CJ, Provo TA, Lurie KG. Hyperventilation-induced hypotension during cardiopulmonary resuscitation. *Circulation*. 2004;109:1960–1965.
93. Abella BS, Alvarado JP, Myklebust H, Edelson DP, Barry A, O'Hearn N, Vanden Hoek TL, Becker LB. Quality of cardiopulmonary resuscitation during in-hospital cardiac arrest. *JAMA*. 2005;293:305–310.
94. Wik L, Kramer-Johansen J, Myklebust H, Sorebo H, Svensson L, Fellows B, Steen PA. Quality of cardiopulmonary resuscitation during out-of-hospital cardiac arrest. *JAMA*. 2005;293:299–304.
95. Ben-David B, Stonebraker VC, Hershman R, Frost CL, Williams HK. Survival after failed intraoperative resuscitation: a case of "Lazarus syndrome". *Anesth Analg*. 2001;92:690–692.
96. Bradbury N. Lazarus phenomenon: another case? *Resuscitation*. 1999;41:87.
97. Bray JG Jr. The Lazarus phenomenon revisited. *Anesthesiology*. 1993;78:991.
98. Frolich MA. Spontaneous recovery after discontinuation of intraoperative cardiopulmonary resuscitation: case report. *Anesthesiology*. 1998;89:1252–1253.
99. Fumeaux T, Borgeat A, Cuenoud PF, Erard A, de Werra P. Survival after cardiac arrest and severe acidosis (pH = 6.54). *Intensive Care Med*. 1997;23:594.
100. Lapinsky SE, Leung RS. Auto-PEEP and electromechanical dissociation. *N Engl J Med*. 1996;335:674.
101. Letellier N, Coulomb F, Lebec C, Brunet JM. Recovery after discontinued cardiopulmonary resuscitation. *Lancet*. 1982;1:1019.
102. Linko K, Honkavaara P, Salmenpera M. Recovery after discontinued cardiopulmonary resuscitation. *Lancet*. 1982;1:106–107.
103. MacGillivray RG. Spontaneous recovery after discontinuation of cardiopulmonary resuscitation. *Anesthesiology*. 1999;91:585–586.
104. Maeda H, Fujita MQ, Zhu BL, Yukioka H, Shindo M, Quan L, Ishida K. Death following spontaneous recovery from cardiopulmonary arrest in a hospital mortuary: 'Lazarus phenomenon' in a case of alleged medical negligence. *Forensic Sci Int*. 2002;127:82–87.
105. Maleck WH, Piper SN, Triem J, Boldt J, Zittel FU. Unexpected return of spontaneous circulation after cessation of resuscitation (Lazarus phenomenon). *Resuscitation*. 1998;39:125–128.
106. Martens P, Vandekerckhove Y, Mullie A. Restoration of spontaneous circulation after cessation of cardiopulmonary resuscitation. *Lancet*. 1993;341:841.
107. Quick G, Bastani B. Prolonged asystolic hyperkalemic cardiac arrest with no neurologic sequelae. *Ann Emerg Med*. 1994;24:305–311.
108. Rogers PL, Schlichtig R, Miro A, Pinsky M. Auto-PEEP during CPR. An "occult" cause of electromechanical dissociation? *Chest*. 1991;99:492–493.
109. Rosengarten PL, Tuxen DV, Dziukas L, Scheinkestel C, Merrett K, Bowes G. Circulatory arrest induced by intermittent positive pressure ventilation in a patient with severe asthma. *Anaesth Intensive Care*. 1991;19:118–121.
110. Sprung J, Hunter K, Barnas GM, Bourke DL. Abdominal distention is not always a sign of esophageal intubation: cardiac arrest due to "auto-PEEP". *Anesth Analg*. 1994;78:801–804.
111. Voelckel W, Kroesen G. Unexpected return of cardiac action after termination of cardiopulmonary resuscitation. *Resuscitation*. 1996;32:27–29.
112. Walker A, McClelland H, Brechley J. The Lazarus phenomenon following recreational drug use. *Emerg Med J*. 2001;18:74–75.
113. Stallinger A, Wenzel V, Wagner-Berger H, Schafer A, Voelckel WG, Augenstein S, Dorges V, Idris AH, Lindner KH, Hornmann C. Effects of decreasing inspiratory flow rate during simulated basic life support ventilation of a cardiac arrest patient on lung and stomach tidal volumes. *Resuscitation*. 2002;54:167–173.
114. Noordergraaf GJ, van Dun PJ, Kramer BP, Schors MP, Hornman HP, de Jong W, Noordergraaf A. Can first responders achieve and maintain normocapnia when sequentially ventilating with a bag-valve device and two oxygen-driven resuscitators? A controlled clinical trial in 104 patients. *Eur J Anaesthesiol*. 2004;21:367–372.
115. Johannigman JA, Branson RD, Johnson DJ, Davis K Jr, Hurst JM. Out-of-hospital ventilation: bag–valve device vs transport ventilator. *Acad Emerg Med*. 1995;2:719–724.
116. Updike G, Mosesso VNJ, Auble TE, Delgado E. Comparison of bag-valve-mask, manually triggered ventilator, and automated ventilator devices used while ventilating a nonintubated mannikin model. *Prehosp Emerg Care*. 1998;2:52–55.
117. Johannigman JA, Branson RD, Davis K Jr, Hurst JM. Techniques of emergency ventilation: a model to evaluate tidal volume, airway pressure, and gastric insufflation. *J Trauma*. 1991;31:93–98.
118. Lindner KH, Dirks B, Strohmenger HU, Prengel AW, Lindner IM, Lurie KG. Randomised comparison of epinephrine and vasopressin in patients with out-of-hospital ventricular fibrillation. *Lancet*. 1997;349:535–537.
119. Lindner KH, Prengel AW, Brinkmann A, Strohmenger HU, Lindner IM, Lurie KG. Vasopressin administration in refractory cardiac arrest. *Ann Intern Med*. 1996;124:1061–1064.
120. Mann K, Berg RA, Nadkarni V. Beneficial effects of vasopressin in prolonged pediatric cardiac arrest: a case series. *Resuscitation*. 2002;52:149–156.
121. Morris DC, Dereczyk BE, Grzybowski M, Martin GB, Rivers EP, Wortsman J, Amico JA. Vasopressin can increase coronary perfusion pressure during human cardiopulmonary resuscitation. *Acad Emerg Med*. 1997;4:878–883.
122. Stiell IG, Hebert PC, Wells GA, Vandemheen KL, Tang AS, Higginson LA, Dreyer JF, Clement C, Battram E, Watpool I, Mason S, Klassen T, Weitzman BN. Vasopressin versus epinephrine for in-hospital cardiac arrest: a randomised controlled trial. *Lancet*. 2001;358:105–109.
123. Wenzel V, Krismer AC, Arntz HR, Sitter H, Stadlbauer KH, Lindner KH. A comparison of vasopressin and epinephrine for out-of-hospital cardiopulmonary resuscitation. *N Engl J Med*. 2004;350:105–113.
124. Aung K, Htay T. Vasopressin for cardiac arrest: a systematic review and meta-analysis. *Arch Intern Med*. 2005;165:17–24.
125. Klouche K, Weil MH, Tang W, Povoa H, Kamohara T, Bisera J. A selective alpha(2)-adrenergic agonist for cardiac resuscitation. *J Lab Clin Med*. 2002;140:27–34.
126. Klouche K, Weil MH, Sun S, Tang W, Zhao DH. A comparison of alpha-methylnorepinephrine, vasopressin and epinephrine for cardiac resuscitation. *Resuscitation*. 2003;57:93–100.

127. Sun S, Weil MH, Tang W, Kamohara T, Klouche K. alpha-Methylnor-epinephrine, a selective alpha2-adrenergic agonist for cardiac resuscitation. *J Am Coll Cardiol*. 2001;37:951–956.
128. DeBehnke DJ, Spreng D, Wickman LL, Crowe DT. The effects of endothelin-1 on coronary perfusion pressure during cardiopulmonary resuscitation in a canine model. *Acad Emerg Med*. 1996;3:137–141.
129. DeBehnke DJ, Benson L. Effects of endothelin-1 on resuscitation rate during cardiac arrest. *Resuscitation*. 2000;47:185–189.
130. DeBehnke D. The effects of graded doses of endothelin-1 on coronary perfusion pressure and vital organ blood flow during cardiac arrest. *Acad Emerg Med*. 2000;7:211–221.
131. Hilwig RW, Berg RA, Kern KB, Ewy GA. Endothelin-1 vasoconstriction during swine cardiopulmonary resuscitation improves coronary perfusion pressures but worsens postresuscitation outcome. *Circulation*. 2000;101:2097–2102.
132. Holzer M, Sterz F, Behringer W, Oschatz E, Kofler J, Eisenburger P, Kittler H, Kunschitzky R, Laggner AN. Endothelin-1 elevates regional cerebral perfusion during prolonged ventricular fibrillation cardiac arrest in pigs. *Resuscitation*. 2002;55:317–327.
133. Kudenchuk PJ, Cobb LA, Copass MK, Cummins RO, Doherty AM, Fahrenbruch CE, Hallstrom AP, Murray WA, Olsufka M, Walsh T. Amiodarone for resuscitation after out-of-hospital cardiac arrest due to ventricular fibrillation. *N Engl J Med*. 1999;341:871–878.
134. Dorian P, Cass D, Schwartz B, Cooper R, Gelaznikas R, Barr A. Amiodarone as compared with lidocaine for shock-resistant ventricular fibrillation. *N Engl J Med*. 2002;346:884–890.
135. Skrifvars MB, Kuisma M, Boyd J, Maatta T, Repo J, Rosenberg PH, Castren M. The use of undiluted amiodarone in the management of out-of-hospital cardiac arrest. *Acta Anaesthesiol Scand*. 2004;48:582–587.
136. Petrovic T, Adnet F, Lapandry C. Successful resuscitation of ventricular fibrillation after low-dose amiodarone. *Ann Emerg Med*. 1998;32:518–519.
137. Levine JH, Massumi A, Scheinman MM, Winkle RA, Platia EV, Chilson DA, Gomes A, Woosley RL. Intravenous amiodarone for recurrent sustained hypotensive ventricular tachyarrhythmias. Intravenous Amiodarone Multicenter Trial Group. *J Am Coll Cardiol*. 1996;27:67–75.
138. Somberg JC, Bailin SJ, Haffajee CI, Paladino WP, Kerin NZ, Bridges D, Timar S, Molnar J. Intravenous lidocaine versus intravenous amiodarone (in a new aqueous formulation) for incessant ventricular tachycardia. *Am J Cardiol*. 2002;90:853–859.
139. Somberg JC, Timar S, Bailin SJ, Lakatos F, Haffajee CI, Tarjan J, Paladino WP, Sarosi I, Kerin NZ, Borbola J, Bridges DE, Molnar J. Lack of a hypotensive effect with rapid administration of a new aqueous formulation of intravenous amiodarone. *Am J Cardiol*. 2004;93:576–581.
140. Viskin S, Belhassen B, Roth A, Reicher M, Averbuch M, Sheps D, Shalaby E, Laniado S. Aminophylline for bradycardic cardiac arrest refractory to atropine and epinephrine. *Ann Intern Med*. 1993;118:279–281.
141. Mader TJ, Gibson P. Adenosine receptor antagonism in refractory asystolic cardiac arrest: results of a human pilot study. *Resuscitation*. 1997;35:3–7.
142. Mader TJ, Smithline HA, Gibson P. Aminophylline in undifferentiated out-of-hospital asystolic cardiac arrest. *Resuscitation*. 1999;41:39–45.
143. Mader TJ, Smithline HA, Durkin L, Scriver G. A randomized controlled trial of intravenous aminophylline for atropine-resistant out-of-hospital asystolic cardiac arrest. *Acad Emerg Med*. 2003;10:192–197.
144. Stiell IG, Wells GA, Hebert PC, Laupacis A, Weitzman BN. Association of drug therapy with survival in cardiac arrest: limited role of advanced cardiac life support drugs. *Acad Emerg Med*. 1995;2:264–273.
145. Engdahl J, Bang A, Lindqvist J, Herlitz J. Can we define patients with no and those with some chance of survival when found in asystole out of hospital? *Am J Cardiol*. 2000;86:610–614.
146. Engdahl J, Bang A, Lindqvist J, Herlitz J. Factors affecting short- and long-term prognosis among 1069 patients with out-of-hospital cardiac arrest and pulseless electrical activity. *Resuscitation*. 2001;51:17–25.
147. Dumot JA, Burval DJ, Sprung J, Waters JH, Mraovic B, Karafa MT, Mascha EJ, Bourke DL. Outcome of adult cardiopulmonary resuscitations at a tertiary referral center including results of “limited” resuscitations. *Arch Intern Med*. 2001;161:1751–1758.
148. Tortolani AJ, Risucci DA, Powell SR, Dixon R. In-hospital cardiopulmonary resuscitation during asystole. Therapeutic factors associated with 24-hour survival. *Chest*. 1989;96:622–626.
149. Dybvik T, Strand T, Steen PA. Buffer therapy during out-of-hospital cardiopulmonary resuscitation. *Resuscitation*. 1995;29:89–95.
150. Aufderheide TP, Martin DR, Olson DW, Aprahamian C, Woo JW, Hendley GE, Hargarten KM, Thompson B. Prehospital bicarbonate use in cardiac arrest: a 3-year experience. *Am J Emerg Med*. 1992;10:4–7.
151. Deloos H, Lewi PJ. Are inter-center differences in EMS-management and sodium-bicarbonate administration important for the outcome of CPR? The Cerebral Resuscitation Study Group. *Resuscitation*. 1989;17 Suppl:S199–S206.
152. Roberts D, Landolfo K, Light R, Dobson K. Early predictors of mortality for hospitalized patients suffering cardiopulmonary arrest. *Chest*. 1990;97:413–419.
153. Suljaga-Pechtel K, Goldberg E, Strickon P, Berger M, Skovron ML. Cardiopulmonary resuscitation in a hospitalized population: prospective study of factors associated with outcome. *Resuscitation*. 1984;12:77–95.
154. Weil MH, Trevino RP, Rackow EC. Sodium bicarbonate during CPR. Does it help or hinder? *Chest*. 1985;88:487–494.
155. Bar-Joseph G, Abramson NS, Kelsey SF, Mashiach T, Craig MT, Safar P. Improved resuscitation outcome in emergency medical systems with increased usage of sodium bicarbonate during cardiopulmonary resuscitation. *Acta Anaesthesiol Scand*. 2005;49:6–15.
156. Sandeman DJ, Alahakoon TI, Bentley SC. Tricyclic poisoning—successful management of ventricular fibrillation following massive overdose of imipramine. *Anaesth Intensive Care*. 1997;25:542–545.
157. Thel MC, Armstrong AL, McNulty SE, Califf RM, O’Connor CM. Randomised trial of magnesium in in-hospital cardiac arrest. Duke Internal Medicine Housestaff. *Lancet*. 1997;350:1272–1276.
158. Allegra J, Lavery R, Cody R, Birnbaum G, Brennan J, Hartman A, Horowitz M, Nashed A, Yablonski M. Magnesium sulfate in the treatment of refractory ventricular fibrillation in the prehospital setting. *Resuscitation*. 2001;49:245–249.
159. Fatovich D, Prentice D, Dobb G. Magnesium in in-hospital cardiac arrest. *Lancet*. 1998;351:446.
160. Hassan TB, Jagger C, Barnett DB. A randomised trial to investigate the efficacy of magnesium sulphate for refractory ventricular fibrillation. *Emerg Med J*. 2002;19:57–62.
161. Miller B, Craddock L, Hoffenberg S, Heinz S, Lefkowitz D, Callender ML, Battaglia C, Maines C, Masick D. Pilot study of intravenous magnesium sulfate in refractory cardiac arrest: safety data and recommendations for future studies. *Resuscitation*. 1995;30:3–14.
162. Longstreth WT Jr, Fahrenbruch CE, Olsufka M, Walsh TR, Copass MK, Cobb LA. Randomized clinical trial of magnesium, diazepam, or both after out-of-hospital cardiac arrest. *Neurology*. 2002;59:506–514.
163. Siemkiewicz E. Magnesium sulfate solution dramatically improves immediate recovery of rats from hypoxia. *Resuscitation*. 1997;35:53–59.
164. Brown CG, Griffith RF, Neely D, Hobson J, Miller B. The effect of intravenous magnesium administration on aortic, right atrial and coronary perfusion pressures during CPR in swine. *Resuscitation*. 1993;26:3–12.
165. Seaberg DC, Menegazzi JJ, Check B, MacLeod BA, Yealy DM. Use of a cardiocerebral-protective drug cocktail prior to countershock in a porcine model of prolonged ventricular fibrillation. *Resuscitation*. 2001;51:301–308.
166. Zhang Y, Davies LR, Martin SM, Bawaney IM, Buettner GR, Kerber RE. Magnesium reduces free radical concentration and preserves left ventricular function after direct current shocks. *Resuscitation*. 2003;56:199–206.
167. Baraka A, Ayoub C, Kawkabani N. Magnesium therapy for refractory ventricular fibrillation. *J Cardiothorac Vasc Anesth*. 2000;14:196–199.
168. Bottiger BW, Bode C, Kern S, Gries A, Gust R, Glatzer R, Bauer H, Mutsch J, Martin E. Efficacy and safety of thrombolytic therapy after initially unsuccessful cardiopulmonary resuscitation: a prospective clinical trial. *Lancet*. 2001;357:1583–1585.
169. Lederer W, Lichtenberger C, Pechlaner C, Kroesen G, Baubin M. Recombinant tissue plasminogen activator during cardiopulmonary resuscitation in 108 patients with out-of-hospital cardiac arrest. *Resuscitation*. 2001;50:71–76.
170. Lederer W, Lichtenberger C, Pechlaner C, Kinzl J, Kroesen G, Baubin M. Long-term survival and neurological outcome of patients who received recombinant tissue plasminogen activator during out-of-hospital cardiac arrest. *Resuscitation*. 2004;61:123–129.
171. Janata K, Holzer M, Kurkciyan I, Losert H, Riedmuller E, Pikula B, Laggner AN, Laczika K. Major bleeding complications in cardiopulmonary resuscitation: the place of thrombolytic therapy in cardiac arrest due to massive pulmonary embolism. *Resuscitation*. 2003;57:49–55.

172. Scholz KH, Hilmer T, Schuster S, Wojcik J, Kreuzer H, Tebbe U. Thrombolysis in resuscitated patients with pulmonary embolism. *Dtsch Med Wochenschr.* 1990;115:930–935.
173. Gramann J, Lange-Braun P, Bodemann T, Hochrein H. Der Einsatz von Thrombolytika in der Reanimation als Ultima ratio zur Überwindung des Herztodes. *Intensiv- und Notfallbehandlung.* 1991;16:134–137.
174. Klefisch F, et al. Praktische ultima-ratio thrombolyse bei therapierefraktärer kardiopulmonaler reanimation. *Intensivmedizin.* 1995;32:155–162.
175. Tiffany PA, Schultz M, Stueven H. Bolus thrombolytic infusions during CPR for patients with refractory arrest rhythms: outcome of a case series. *Ann Emerg Med.* 1998;31:124–126.
176. Ruiz-Bailen M, Aguayo-de-Hoyos E, Serrano-Corcoles MC, Diaz-Castellanos MA, Fierro-Roson JL, Ramos-Cuadra JA, Rodriguez-Elvira M, Torres-Ruiz JM. Thrombolysis with recombinant tissue plasminogen activator during cardiopulmonary resuscitation in fulminant pulmonary embolism. A case series. *Resuscitation.* 2001;51:97–101.
177. Abu-Laban RB, Christenson JM, Innes GD, van Beek CA, Wanger KP, McKnight RD, MacPhail IA, Puskaric J, Sadowski RP, Singer J, Schechter MT, Wood VM. Tissue plasminogen activator in cardiac arrest with pulseless electrical activity. *N Engl J Med.* 2002;346:1522–1528.
178. Lin SR. The effect of dextran and streptokinase on cerebral function and blood flow after cardiac arrest. An experimental study on the dog. *Neuroradiology.* 1978;16:340–342.
179. Fischer M, Bottiger BW, Popov-Cenic S, Hossmann KA. Thrombolysis using plasminogen activator and heparin reduces cerebral no-reflow after resuscitation from cardiac arrest: an experimental study in the cat. *Intensive Care Med.* 1996;22:1214–1223.
180. Ditchey RV, Lindenfeld J. Potential adverse effects of volume loading on perfusion of vital organs during closed-chest resuscitation. *Circulation.* 1984;69:181–189.
181. Gentile NT, Martin GB, Appleton TJ, Moeggenberg J, Paradis NA, Nowak RM. Effects of arterial and venous volume infusion on coronary perfusion pressures during canine CPR. *Resuscitation.* 1991;22:55–63.
182. Jameson SJ, Mateer JR, DeBehnke DJ. Early volume expansion during cardiopulmonary resuscitation. *Resuscitation.* 1993;26:243–250.
183. Voorhees WD, Ralston SH, Kougiac C, Schmitz PM. Fluid loading with whole blood or Ringer's lactate solution during CPR in dogs. *Resuscitation.* 1987;15:113–123.
184. Banerjee S, Singhi SC, Singh S, Singh M. The intraosseous route is a suitable alternative to intravenous route for fluid resuscitation in severely dehydrated children. *Indian Pediatr.* 1994;31:1511–1520.
185. Brickman KR, Krupp K, Rega P, Alexander J, Guinness M. Typing and screening of blood from intraosseous access. *Ann Emerg Med.* 1992;21:414–417.
186. Fiser RT, Walker WM, Seibert JJ, McCarthy R, Fiser DH. Tibial length following intraosseous infusion: a prospective, radiographic analysis. *Pediatr Emerg Care.* 1997;13:186–188.
187. Ummenhofer W, Frei FJ, Urwyler A, Drewé J. Are laboratory values in bone marrow aspirate predictable for venous blood in paediatric patients? *Resuscitation.* 1994;27:123–128.
188. Glaeser PW, Hellmich TR, Szwecuga D, Losek JD, Smith DS. Five-year experience in prehospital intraosseous infusions in children and adults. *Ann Emerg Med.* 1993;22:1119–1124.
189. Guy J, Haley K, Zuspan SJ. Use of intraosseous infusion in the pediatric trauma patient. *J Pediatr Surg.* 1993;28:158–161.
190. Macnab A, Christenson J, Findlay J, Horwood B, Johnson D, Jones L, Phillips K, Pollack C Jr, Robinson DJ, Rumball C, Stair T, Tiffany B, Whelan M. A new system for sternal intraosseous infusion in adults. *Prehosp Emerg Care.* 2000;4:173–177.
191. Ellemunter H, Simma B, Trawoger R, Maurer H. Intraosseous lines in preterm and full term neonates. *Arch Dis Child Fetal Neonatal Ed.* 1999;80:F74–F75.
192. Niemann JT, Stratton SJ, Cruz B, Lewis RJ. Endotracheal drug administration during out-of-hospital resuscitation: where are the survivors? *Resuscitation.* 2002;53:153–157.
193. Schuttler J, Bartsch A, Ebeling BJ, Hornchen U, Kulka P, Suhling B, Stoeckel H. [Endobronchial administration of adrenaline in preclinical cardiopulmonary resuscitation]. *Anasth Intensivther Notfallmed.* 1987;22:63–68.
194. Hornchen U, Schuttler J, Stoeckel H, Eichelkraut W, Hahn N. Endobronchial instillation of epinephrine during cardiopulmonary resuscitation. *Crit Care Med.* 1987;15:1037–1039.
195. Naganobu K, Hasebe Y, Uchiyama Y, Hagio M, Ogawa H. A comparison of distilled water and normal saline as diluents for endobronchial administration of epinephrine in the dog. *Anesth Analg.* 2000;91:317–321.
196. Prengel AW, Lindner KH, Hahnel J, Ahnefeld FW. Endotracheal and endobronchial lidocaine administration: effects on plasma lidocaine concentration and blood gases. *Crit Care Med.* 1991;19:911–915.
197. Prengel AW, Rembecki M, Wenzel V, Steinbach G. A comparison of the endotracheal tube and the laryngeal mask airway as a route for endobronchial lidocaine administration. *Anesth Analg.* 2001;92:1505–1509.
198. Prengel AW, Lindner KH, Hahnel JH, Georgieff M. Pharmacokinetics and technique of endotracheal and deep endobronchial lidocaine administration. *Anesth Analg.* 1993;77:985–989.
199. Steinfath M, Scholz J, Schulte am Esch J, Laer S, Reymann A, Scholz H. The technique of endobronchial lidocaine administration does not influence plasma concentration profiles and pharmacokinetic parameters in humans. *Resuscitation.* 1995;29:55–62.
200. Hahnel J, Lindner KH, Ahnefeld FW. Endobronchial administration of emergency drugs. *Resuscitation.* 1989;17:261–272.
201. Hahnel JH, Lindner KH, Schurmann C, Prengel A, Ahnefeld FW. Plasma lidocaine levels and PaO₂ with endobronchial administration: dilution with normal saline or distilled water? *Ann Emerg Med.* 1990;19:1314–1317.
202. Efrati O, Barak A, Ben-Abraham R, Modan-Moses D, Berkovitch M, Manisterski Y, Lotan D, Barzilay Z, Paret G. Should vasopressin replace adrenaline for endotracheal drug administration? *Crit Care Med.* 2003;31:572–576.
203. Wenzel V, Lindner KH, Prengel AW, Lurie KG, Strohmenger HU. Endobronchial vasopressin improves survival during cardiopulmonary resuscitation in pigs. *Anesthesiology.* 1997;86:1375–1381.
204. Grmec S, Klemen P. Does the end-tidal carbon dioxide (EtCO₂) concentration have prognostic value during out-of-hospital cardiac arrest? *Eur J Emerg Med.* 2001;8:263–269.
205. Grmec S, Kupnik D. Does the Mainz Emergency Evaluation Scoring (MEES) in combination with capnometry (MEESc) help in the prognosis of outcome from cardiopulmonary resuscitation in a pre-hospital setting? *Resuscitation.* 2003;58:89–96.
206. Grmec S, Lah K, Tusek-Bunc K. Difference in end-tidal CO₂ between asphyxia cardiac arrest and ventricular fibrillation/pulseless ventricular tachycardia cardiac arrest in the prehospital setting. *Crit Care.* 2003;7:R139–R144.
207. Mauer D, Schneider T, Elich D, Dick W. Carbon dioxide levels during pre-hospital active compression–decompression versus standard cardiopulmonary resuscitation. *Resuscitation.* 1998;39:67–74.
208. Berg RA, Henry C, Otto CW, Sanders AB, Kern KB, Hilwig RW, Ewy GA. Initial end-tidal CO₂ is markedly elevated during cardiopulmonary resuscitation after asphyxial cardiac arrest. *Pediatr Emerg Care.* 1996;12:245–248.
209. Gazmuri RJ, Von Planta M, Weil MH, Rackow EC. Arterial PCO₂ as an indicator of systemic perfusion during cardiopulmonary resuscitation. *Crit Care Med.* 1989;17:237–240.
210. Gudipati C, Weil M, Bisera J, Deshmukh H, Rackow E. Expired carbon dioxide: a noninvasive monitor of cardiopulmonary resuscitation. *Circulation.* 1988;77:234–239.
211. Kern KB, Sanders AB, Voorhees WD, Babbs CF, Tacker WA, Ewy GA. Changes in expired end-tidal carbon dioxide during cardiopulmonary resuscitation in dogs: a prognostic guide for resuscitation efforts. *J Am Coll Cardiol.* 1989;13:1184–1189.
212. Ornato JP, Garnett AR, Glauser FL. Relationship between cardiac output and the end-tidal carbon dioxide tension. *Ann Emerg Med.* 1990;19:1104–1106.
213. Sanders A, Atlas M, Ewy G, Kern K, Bragg S. Expired pCO₂ as an index of coronary perfusion pressure. *Am J Emerg Med.* 1985;3:147–149.
214. Sanders ABea. Expired PCO₂ as a prognostic indicator of successful resuscitation from cardiac arrest. *Ann Emerg Med.* 1985;14:948–952.
215. Callahan M, Barton C. Prediction of outcome of cardiopulmonary resuscitation from end-tidal carbon dioxide concentration. *Crit Care Med.* 1990;18:358–362.
216. Sanders AB, Kern KB, Otto CW, Milander MM, Ewy GA. End-tidal carbon dioxide monitoring during cardiopulmonary resuscitation: a prognostic indicator for survival. *JAMA.* 1989;262:1347–1351.
217. Wayne MA, Levine RL, Miller CC. Use of end-tidal carbon dioxide to predict outcome in prehospital cardiac arrest. *Ann Emerg Med.* 1995;25:762–767.

218. Entholzner E, Felber A, Mielke L, Hargasser S, Breinbauer B, Hundelshausen VB, Hipp R. Assessment of end-tidal CO₂ measurement in reanimation. *Anesthesiologie Intensivmedizin Notfallmedizin Schmerztherapie*. 1992;27:473–476.
219. Garnett AR, Ornato JP, Gonzalez ER, Johnson EB. End-tidal carbon dioxide monitoring during cardiopulmonary resuscitation. *JAMA*. 1987;257:512–515.
220. Bhende MS, Karasic DG, Karasic RB. End-tidal carbon dioxide changes during cardiopulmonary resuscitation after experimental asphyxial cardiac arrest. *Am J Emerg Med*. 1996;14:349–350.
221. Ahrens T, Schallom L, Bettorf K, Ellner S, Hurt G, O'Mara V, Ludwig J, George W, Marino T, Shannon W. End-tidal carbon dioxide measurements as a prognostic indicator of outcome in cardiac arrest. *Am J Crit Care*. 2001;10:391–398.
222. Cantineau JP, Lambert Y, Merckx P, Reynaud P, Porte F, Bertrand C, Duvaldestin P. End-tidal carbon dioxide during cardiopulmonary resuscitation in humans presenting mostly with asystole: a predictor of outcome. *Crit Care Med*. 1996;24:791–796.
223. Levine RL, Wayne MA, Miller CC. End-tidal carbon dioxide and outcome of out-of-hospital cardiac arrest. *N Engl J Med*. 1997;337:301–306.
224. Weil MH, Rackow EC, Trevino R, Grundler W, Falk JL, Griffel MI. Difference in acid-base state between venous and arterial blood during cardiopulmonary resuscitation. *N Engl J Med*. 1986;315:153–156.
225. Kette F, Weil MH, Gazmuri RJ, Bisera J, Rackow EC. Intramyocardial hypercarbic acidosis during cardiac arrest and resuscitation. *Crit Care Med*. 1993;21:901–906.
226. Adroque HJ, Rashad MN, Gorin AB, Yacoub J, Madias NE. Arteriovenous acid-base disparity in circulatory failure: studies on mechanism. *Am J Physiol*. 1989;257:F1087–F1093.
227. Tucker KJ, Idris AH, Wenzel V, Orban DJ. Changes in arterial and mixed venous blood gases during untreated ventricular fibrillation and cardiopulmonary resuscitation. *Resuscitation*. 1994;28:137–141.
228. Tang W, Weil MH, Sun S, Kette D, Gazmuri RJ, O'Connell F, Bisera J. Cardiopulmonary resuscitation by precordial compression but without mechanical ventilation. *Am J Respir Crit Care Med*. 1994;150:1709–1713.
229. Gudipati CV, Weil MH, Gazmuri RJ, Deshmukh HG, Bisera J, Rackow EC. Increases in coronary vein CO₂ during cardiac resuscitation. *J Appl Physiol*. 1990;68:1405–1408.
230. Capparelli EV, Chow MS, Kluger J, Fieldman A. Differences in systemic and myocardial blood acid-base status during cardiopulmonary resuscitation. *Crit Care Med*. 1989;17:442–446.
231. von Planta M, Weil MH, Gazmuri RJ, Bisera J, Rackow EC. Myocardial acidosis associated with CO₂ production during cardiac arrest and resuscitation. *Circulation*. 1989;80:684–692.
232. Grundler W, Weil MH, Rackow EC. Arteriovenous carbon dioxide and pH gradients during cardiac arrest. *Circulation*. 1986;74:1071–1074.
233. Sanders AB, Ewy GA, Taft TV. Resuscitation and arterial blood gas abnormalities during prolonged cardiopulmonary resuscitation. *Ann Emerg Med*. 1984;13(pt 1):676–679.
234. Nowak RM, Martin GB, Carden DL, Tomlanovich MC. Selective venous hypercarbia during human CPR: implications regarding blood flow. *Ann Emerg Med*. 1987;16:527–530.
235. Ornato JP, Gonzalez ER, Coyne MR, Beck CL, Collins MS. Arterial pH in out-of-hospital cardiac arrest: response time as a determinant of acidosis. *Am J Emerg Med*. 1985;3:498–502.
236. Idris AH, Wenzel V, Becker LB, Banner MJ, Orban DJ. Does hypoxia or hypercarbia independently affect resuscitation from cardiac arrest? *Chest*. 1995;108:522–528.
237. DeBehnke DJ, Hilander SJ, Dobler DW, Wickman LL, Swart GL. The hemodynamic and arterial blood gas response to asphyxiation: a canine model of pulseless electrical activity. *Resuscitation*. 1995;30:169–175.
238. Idris AH, Becker LB, Fuerst RS, Wenzel V, Rush WJ, Melker RJ, Orban DJ. Effect of ventilation on resuscitation in an animal model of cardiac arrest. *Circulation*. 1994;90:3063–3069.
239. Idris AH, Banner MJ, Wenzel V, Fuerst RS, Becker LB, Melker RJ. Ventilation caused by external chest compression is unable to sustain effective gas exchange during CPR: a comparison with mechanical ventilation. *Resuscitation*. 1994;28:143–150.
240. Engoren M, Plewa MC, Buderer NF, Hymel G, Brookfield L. Effects of simulated mouth-to-mouth ventilation during external cardiac compression or active compression-decompression in a swine model of witnessed cardiac arrest. *Ann Emerg Med*. 1997;29:607–615.
241. Prause G, Ratenhofer-Comenda B, Smolle-Juttner F, Heydar-Fadai J, Wildner G, Spornbauer P, Smolle J, Hetz H. Comparison of lactate or BE during out-of-hospital cardiac arrest to determine metabolic acidosis. *Resuscitation*. 2001;51:297–300.
242. Langhelle A, Sunde K, Wik L, Steen PA. Arterial blood-gases with 500-versus 1000-ml tidal volumes during out-of-hospital CPR. *Resuscitation*. 2000;45:27–33.
243. Idris AH, Staples ED, O'Brien DJ, Melker RJ, Rush WJ, Del Duca KD, Falk JL. Effect of ventilation on acid-base balance and oxygenation in low blood-flow states. *Crit Care Med*. 1994;22:1827–1834.
244. Barton C, Callahan M. Lack of correlation between end-tidal carbon dioxide concentrations and Paco₂ in cardiac arrest. *Crit Care Med*. 1991;19:108–110.
245. Angelos MG, DeBehnke DJ, Leasure JE. Arterial blood gases during cardiac arrest: markers of blood flow in a canine model. *Resuscitation*. 1992;23:101–111.
246. Wiklund L, Soderberg D, Henneberg S, Rubertsson S, Stjernstrom H, Groth T. Kinetics of carbon dioxide during cardiopulmonary resuscitation. *Crit Care Med*. 1986;14:1015–1022.
247. Paradis NA, Martin GB, Rivers EP, Goetting MG, Appleton TJ, Feingold M, Nowak RM. Coronary perfusion pressure and the return of spontaneous circulation in human cardiopulmonary resuscitation. *JAMA*. 1990;263:1106–1113.
248. Halperin HR, Tsitlik JE, Gelfand M, Weisfeldt ML, Gruben KG, Levin HR, Rayburn BK, Chandra NC, Scott CJ, Kreps BJ, et al. A preliminary study of cardiopulmonary resuscitation by circumferential compression of the chest with use of a pneumatic vest. *N Engl J Med*. 1993;329:762–768.
249. Kern KB, Ewy GA, Voorhees WD, Babbs CF, Tacker WA. Myocardial perfusion pressure: a predictor of 24-hour survival during prolonged cardiac arrest in dogs. *Resuscitation*. 1988;16:241–250.
250. Lindner KH, Prengel AW, Pfenninger EG, Lindner IM, Strohmenger HU, Georgieff M, Lurie KG. Vasopressin improves vital organ blood flow during closed-chest cardiopulmonary resuscitation in pigs. *Circulation*. 1995;91:215–221.
251. Little CM, Angelos MG, Paradis NA. Compared to angiotensin II, epinephrine is associated with high myocardial blood flow following return of spontaneous circulation after cardiac arrest. *Resuscitation*. 2003;59:353–359.
252. Hedges JR, Syverud SA, Dalsey WC, Feero S, Easter R, Shultz B. Prehospital trial of emergency transcutaneous cardiac pacing. *Circulation*. 1987;76:1337–1343.
253. Barthell E, Troiano P, Olson D, Stueven HA, Hendley G. Prehospital external cardiac pacing: a prospective, controlled clinical trial. *Ann Emerg Med*. 1988;17:1221–1226.
254. Cummins RO, Graves JR, Larsen MP, Hallstrom AP, Hearne TR, Ciliberti J, Nicola RM, Horan S. Out-of-hospital transcutaneous pacing by emergency medical technicians in patients with asystolic cardiac arrest. *N Engl J Med*. 1993;328:1377–1382.
255. Quan L, Graves JR, Kinder DR, Horan S, Cummins RO. Transcutaneous cardiac pacing in the treatment of out-of-hospital pediatric cardiac arrests. *Ann Emerg Med*. 1992;21:905–909.
256. Dalsey WC, Syverud SA, Hedges JR. Emergency department use of transcutaneous pacing for cardiac arrests. *Crit Care Med*. 1985;13:399–401.
257. Knowlton AA, Falk RH. External cardiac pacing during in-hospital cardiac arrest. *Am J Cardiol*. 1986;57:1295–1298.
258. Ornato JP, Carveth WL, Windle JR. Pacemaker insertion for prehospital bradyasystolic cardiac arrest. *Ann Emerg Med*. 1984;13:101–103.
259. White JD. Transthoracic pacing in cardiac asystole. *Am J Emerg Med*. 1983;1:264–266.
260. Niemann JT, Adomian GE, Garner D, Rosborough JP. Endocardial and transcutaneous cardiac pacing, calcium chloride, and epinephrine in postcountershock asystole and bradycardias. *Crit Care Med*. 1985;13:699–704.
261. Ornato JP, Peberdy MA. The mystery of bradyasystole during cardiac arrest. *Ann Emerg Med*. 1996;27:576–587.
262. Kern KB, Sanders AB, Raife J, Milander MM, Otto CW, Ewy GA. A study of chest compression rates during cardiopulmonary resuscitation in humans: the importance of rate-directed chest compressions. *Arch Intern Med*. 1992;152:145–149.
263. Berg RA, Sanders AB, Milander M, Tellez D, Liu P, Beyda D. Efficacy of audio-prompted rate guidance in improving rescuator performance of cardiopulmonary resuscitation on children. *Acad Emerg Med*. 1994;1:35–40.

264. Barsan WG, Levy RC. Experimental design for study of cardiopulmonary resuscitation in dogs. *Ann Emerg Med.* 1981;10:135-137.
265. Milander MM, Hiscok PS, Sanders AB, Kern KB, Berg RA, Ewy GA. Chest compression and ventilation rates during cardiopulmonary resuscitation: the effects of audible tone guidance. *Acad Emerg Med.* 1995; 2:708-713.
266. Chiang WC, Chen WJ, Chen SY, Ko PC, Lin CH, Tsai MS, Chang WT, Chen SC, Tsan CY, Ma MH. Better adherence to the guidelines during cardiopulmonary resuscitation through the provision of audio-prompts. *Resuscitation.* 2005;64:297-301.
267. Boyle AJ, Wilson AM, Connelly K, McGuigan L, Wilson J, Whitbourn R. Improvement in timing and effectiveness of external cardiac compressions with a new non-invasive device: the CPR-Ezy. *Resuscitation.* 2002;54:63-67.
268. Wik L, Thowsen J, Steen PA. An automated voice advisory manikin system for training in basic life support without an instructor. A novel approach to CPR training. *Resuscitation.* 2001;50:167-172.
269. Wik L, Myklebust H, Auestad BH, Steen PA. Retention of basic life support skills 6 months after training with an automated voice advisory manikin system without instructor involvement. *Resuscitation.* 2002;52: 273-279.
270. Elding C, Baskett P, Hughes A. The study of the effectiveness of chest compressions using the CPR-plus. *Resuscitation.* 1998;36:169-173.
271. Thomas SH, Stone CK, Austin PE, March JA, Brinkley S. Utilization of a pressure-sensing monitor to improve in-flight chest compressions. *Am J Emerg Med.* 1995;13:155-157.
272. Handley AJ, Handley SA. Improving CPR performance using an audible feedback system suitable for incorporation into an automated external defibrillator. *Resuscitation.* 2003;57:57-62.
273. Sack JB, Kesselbrenner MB, Bregman D. Survival from in-hospital cardiac arrest with interposed abdominal counterpulsation during cardiopulmonary resuscitation. *JAMA.* 1992;267:379-385.
274. Sack JB, Kesselbrenner MB. Hemodynamics, survival benefits, and complications of interposed abdominal compression during cardiopulmonary resuscitation. *Acad Emerg Med.* 1994;1:490-497.
275. Ward KR, Sullivan RJ, Zelenak RR, Summer WR. A comparison of interposed abdominal compression CPR and standard CPR by monitoring end-tidal Pco₂. *Ann Emerg Med.* 1989;18:831-837.
276. Babbs CF. Interposed abdominal compression CPR: a comprehensive evidence based review. *Resuscitation.* 2003;59:71-82.
277. Babbs CF. Meta-analysis of 2-treatment clinical trials including both continuous and dichotomous results. *Med Decis Making.* 2004;24: 299-312.
278. Mateer JR, Stueven HA, Thompson BM, Aprahamian C, Darin JC. Pre-hospital IAC-CPR versus standard CPR: paramedic resuscitation of cardiac arrests. *Am J Emerg Med.* 1985;3:143-146.
279. Waldman PJ, Walters BL, Grunau CF. Pancreatic injury associated with interposed abdominal compressions in pediatric cardiopulmonary resuscitation. *Am J Emerg Med.* 1984;2:510-512.
280. Swenson RD, Weaver WD, Niskanen RA, Martin J, Dahlberg S. Hemodynamics in humans during conventional and experimental methods of cardiopulmonary resuscitation. *Circulation.* 1988;78:630-639.
281. Swart GL, Mateer JR, DeBehnke DJ, Jameson SJ, Osborn JL. The effect of compression duration on hemodynamics during mechanical high-impulse CPR. *Acad Emerg Med.* 1994;1:430-437.
282. Maier GW, Tyson GS Jr, Olsen CO, Kernstein KH, Davis JW, Conn EH, Sabiston DC Jr, Rankin JS. The physiology of external cardiac massage: high-impulse cardiopulmonary resuscitation. *Circulation.* 1984;70: 86-101.
283. Kern KB, Carter AB, Showen RL, Voorhees WD, 3rd, Babbs CF, Tacker WA, Ewy GA. CPR-induced trauma: comparison of three manual methods in an experimental model. *Ann Emerg Med.* 1986;15: 674-679.
284. Tucker KJ, Khan J, Idris A, Savitt MA. The biphasic mechanism of blood flow during cardiopulmonary resuscitation: a physiologic comparison of active compression-decompression and high-impulse manual external cardiac massage. *Ann Emerg Med.* 1994;24:895-906.
285. Cohen TJ, Goldner BG, Maccaro PC, Ardito AP, Trazzera S, Cohen MB, Dibs SR. A comparison of active compression-decompression cardiopulmonary resuscitation with standard cardiopulmonary resuscitation for cardiac arrests occurring in the hospital. *N Engl J Med.* 1993;329:1918-1921.
286. Tucker KJ, Redberg RF, Schiller NB, Cohen TJ. Active compression-decompression resuscitation: analysis of transmitral flow and left ventricular volume by transesophageal echocardiography in humans. Car-
diopulmonary Resuscitation Working Group. *J Am Coll Cardiol.* 1993; 22:1485-1493.
287. Plaisance P, Lurie KG, Vicaut E, Adnet F, Petit JL, Epain D, Ecollan P, Gruat R, Cavagna P, Biens J, Payen D. A comparison of standard cardiopulmonary resuscitation and active compression-decompression resuscitation for out-of-hospital cardiac arrest. French Active Compression-Decompression Cardiopulmonary Resuscitation Study Group. *N Engl J Med.* 1999;341:569-575.
288. Lafuente-Lafuente C, Melero-Bascones M. Active chest compression-decompression for cardiopulmonary resuscitation. *Cochrane Database Syst Rev.* 2004:CD002751.
289. Baubin M, Sumann G, Rabl W, Eibl G, Wenzel V, Mair P. Increased frequency of thorax injuries with ACD-CPR. *Resuscitation.* 1999;41: 33-38.
290. Casner M, Anderson, D. et al. Preliminary report of the impact of a new CPR assist device on the rate of return of spontaneous circulation in out of hospital cardiac arrest. *Prehosp Emerg Care.* 2005;9:61-67.
291. Timerman S, Cardoso LF, Ramires JA, Halperin H. Improved hemodynamic performance with a novel chest compression device during treatment of in-hospital cardiac arrest. *Resuscitation.* 2004;61:273-280.
292. Halperin H, Berger R, Chandra N, Ireland M, Leng C, Lardo A, Paradis N. Cardiopulmonary resuscitation with a hydraulic-pneumatic band. *Crit Care Med.* 2000;28:N203-N206.
293. Halperin HR, Paradis N, Ornato JP, Zviman M, Lacorte J, Lardo A, Kern KB. Cardiopulmonary resuscitation with a novel chest compression device in a porcine model of cardiac arrest: improved hemodynamics and mechanisms. *J Am Coll Cardiol.* 2004;44:2214-2220.
294. Dickinson ET, Verdile VP, Schneider RM, Salluzzo RF. Effectiveness of mechanical versus manual chest compressions in out-of-hospital cardiac arrest resuscitation: a pilot study. *Am J Emerg Med.* 1998;16: 289-292.
295. McDonald JL. Systolic and mean arterial pressures during manual and mechanical CPR in humans. *Ann Emerg Med.* 1982;11:292-295.
296. Ward KR, Menegazzi JJ, Zelenak RR, Sullivan RJ, McSwain N Jr. A comparison of chest compressions between mechanical and manual CPR by monitoring end-tidal Pco₂ during human cardiac arrest. *Ann Emerg Med.* 1993;22:669-674.
297. Sunde K, Wik L, Steen PA. Quality of mechanical, manual standard and active compression-decompression CPR on the arrest site and during transport in a manikin model. *Resuscitation.* 1997;34:235-242.
298. Rubertsson S, Karlsten R. Increased cortical cerebral blood flow with LUCAS; a new device for mechanical chest compressions compared to standard external compressions during experimental cardiopulmonary resuscitation. *Resuscitation.* 2005;65:357-363.
299. Steen S, Liao Q, Pierre L, Paskevicius A, Sjoberg T. Evaluation of LUCAS, a new device for automatic mechanical compression and active decompression resuscitation. *Resuscitation.* 2002;55:285-299.
300. Wik L, Bircher NG, Safar P. A comparison of prolonged manual and mechanical external chest compression after cardiac arrest in dogs. *Resuscitation.* 1996;32:241-250.
301. Arntz HR, Agrawal R, Richter H, Schmidt S, Rescheleit T, Menges M, Burbach H, Schroder J, Schultheiss HP. Phased chest and abdominal compression-decompression versus conventional cardiopulmonary resuscitation in out-of-hospital cardiac arrest. *Circulation.* 2001;104: 768-772.
302. Wenzel V, Lindner KH, Prengel AW, Strohmenger HU. Effect of phased chest and abdominal compression-decompression cardiopulmonary resuscitation on myocardial and cerebral blood flow in pigs. *Crit Care Med.* 2000;28:1107-1112.
303. Tang W, Weil MH, Schock RB, Sato Y, Lucas J, Sun S, Bisera J. Phased chest and abdominal compression-decompression: a new option for cardiopulmonary resuscitation. *Circulation.* 1997;95:1335-1340.
304. Babbs CF. CPR techniques that combine chest and abdominal compression and decompression: hemodynamic insights from a spreadsheet model. *Circulation.* 1999;100:2146-2152.
305. Paiva EF, Kern KB, Hilwig RW, Scalabrini A, Ewy GA. Minimally invasive direct cardiac massage versus closed-chest cardiopulmonary resuscitation in a porcine model of prolonged ventricular fibrillation cardiac arrest. *Resuscitation.* 2000;47:287-299.
306. Buckman J, R.F., Badellino MM, Mauro LH, Aldridge SC, Milner RE, Malaspina PJ, Merchant NB, Buckman III. RF. Direct cardiac massage without major thoracotomy: feasibility and systemic blood flow. *Resuscitation.* 1995;29:237-248.
307. Buckman RF Jr, Badellino MM, Eynon CA, Mauro LH, Aldridge SC, Milner RE, Merchant NB, Buckman RF, 3rd, Mercer D, Malaspina PJ,

- Warren R. Open-chest cardiac massage without major thoracotomy: metabolic indicators of coronary and cerebral perfusion. *Resuscitation*. 1997;34:247–253.
308. Rozenberg A, Incagnoli P, Delpech P, Spaulding C, Vivien B, Kern KB, Carli P. Prehospital use of minimally invasive direct cardiac massage (MID-CM): a pilot study. *Resuscitation*. 2001;50:257–262.
309. Walcott GP, Booker RG, Ideker RE. Defibrillation with a minimally invasive direct cardiac massage device. *Resuscitation*. 2002;55:301–307.
310. Aufderheide TP, Pirralo RG, Yannopoulos D, Klein JP, von Briesen C, Sparks CW, Deja KA, Conrad CJ, Kitscha DJ, Provo TA, Lurie KG. Incomplete chest wall decompression: a clinical evaluation of CPR performance by EMS personnel and assessment of alternative manual chest compression-decompression techniques. *Resuscitation*. 2005;64:353–362.
311. Lurie KG, Voelckel WG, Zielinski T, McKnite S, Lindstrom P, Peterson C, Wenzel V, Lindner KH, Samniah N, Benditt D. Improving standard cardiopulmonary resuscitation with an inspiratory impedance threshold valve in a porcine model of cardiac arrest. *Anesth Analg*. 2001;93:649–655.
312. Lurie KG, Zielinski T, McKnite S, Aufderheide T, Voelckel W. Use of an inspiratory impedance valve improves neurologically intact survival in a porcine model of ventricular fibrillation. *Circulation*. 2002;105:124–129.
313. Lurie KG, Mulligan KA, McKnite S, Detloff B, Lindstrom P, Lindner KH. Optimizing standard cardiopulmonary resuscitation with an inspiratory impedance threshold valve. *Chest*. 1998;113:1084–1090.
314. Langhelle A, Stromme T, Sunde K, Wik L, Nicolaysen G, Steen PA. Inspiratory impedance threshold valve during CPR. *Resuscitation*. 2002;52:39–48.
315. Yannopoulos D, Sigurdsson G, McKnite S, Benditt D, Lurie KG. Reducing ventilation frequency combined with an inspiratory impedance device improves CPR efficiency in swine model of cardiac arrest. *Resuscitation*. 2004;61:75–82.
316. Pirralo R, Aufderheide T, Provo T, Lurie K. Effect of an inspiratory impedance threshold device on hemodynamics during conventional manual cardiopulmonary resuscitation. *Resuscitation*. In press.
317. Plaisance P, Lurie KG, Vicaut E, Martin D, Gueugniaud PY, Petit JL, Payen D. Evaluation of an impedance threshold device in patients receiving active compression-decompression cardiopulmonary resuscitation for out of hospital cardiac arrest. *Resuscitation*. 2004;61:265–271.
318. Lurie KG, Coffeen P, Shultz J, McKnite S, Detloff B, Mulligan K. Improving active compression-decompression cardiopulmonary resuscitation with an inspiratory impedance valve. *Circulation*. 1995;91:1629–1632.
319. Plaisance P, Lurie KG, Payen D. Inspiratory impedance during active compression-decompression cardiopulmonary resuscitation: a randomized evaluation in patients in cardiac arrest. *Circulation*. 2000;101:989–994.
320. Wolcke BB, Mauer DK, Schoefmann MF, Teichmann H, Provo TA, Lindner KH, Dick WF, Aeppli D, Lurie KG. Comparison of standard cardiopulmonary resuscitation versus the combination of active compression-decompression cardiopulmonary resuscitation and an inspiratory impedance threshold device for out-of-hospital cardiac arrest. *Circulation*. 2003;108:2201–2205.
321. Raedler C, Voelckel WG, Wenzel V, Bahlmann L, Baumeier W, Schmittinger CA, Herff H, Krismer AC, Lindner KH, Lurie KG. Vasoconstrictor response in a porcine model of hypothermic cardiac arrest is improved with active compression-decompression cardiopulmonary resuscitation using the inspiratory impedance threshold valve. *Anesth Analg*. 2002;95:1496–1502.
322. Voelckel WG, Lurie KG, Sweeney M, McKnite S, Zielinski T, Lindstrom P, Peterson C, Wenzel V, Lindner KH. Effects of active compression-decompression cardiopulmonary resuscitation with the inspiratory threshold valve in a young porcine model of cardiac arrest. *Pediatr Res*. 2002;51:523–527.
323. Chen YS, Chao A, Yu HY, Ko WJ, Wu IH, Chen RJ, Huang SC, Lin FY, Wang SS. Analysis and results of prolonged resuscitation in cardiac arrest patients rescued by extracorporeal membrane oxygenation. *J Am Coll Cardiol*. 2003;41:197–203.
324. Martin GB, Rivers EP, Paradis NA, Goetting MG, Morris DC, Nowak RM. Emergency department cardiopulmonary bypass in the treatment of human cardiac arrest. *Chest*. 1998;113:743–751.
325. Younger JG, Schreiner RJ, Swaniker F, Hirschl RB, Chapman RA, Bartlett RH. Extracorporeal resuscitation of cardiac arrest. *Acad Emerg Med*. 1999;6:700–707.
326. Anthi A, Tzelepis GE, Alivizatos P, Michalis A, Palatianos GM, Geroulanos S. Unexpected cardiac arrest after cardiac surgery: incidence, predisposing causes, and outcome of open chest cardiopulmonary resuscitation. *Chest*. 1998;113:15–19.
327. Pottle A, Bullock I, Thomas J, Scott L. Survival to discharge following Open Chest Cardiac Compression (OCCC). A 4-year retrospective audit in a cardiothoracic specialist centre—Royal Brompton and Harefield NHS Trust, United Kingdom. *Resuscitation*. 2002;52:269–272.
328. Takino M, Okada Y. The optimum timing of resuscitative thoracotomy for non-traumatic out-of-hospital cardiac arrest. *Resuscitation*. 1993;26:69–74.
329. Boczar ME, Howard MA, Rivers EP, Martin GB, Horst HM, Lewandowski C, Tomlanovich MC, Nowak RM. A technique revisited: hemodynamic comparison of closed- and open-chest cardiac massage during human cardiopulmonary resuscitation. *Crit Care Med*. 1995;23:498–503.
330. Angelos MG, DeBehnke DJ, Leasure JE. Arterial pH and carbon dioxide tension as indicators of tissue perfusion during cardiac arrest in a canine model. *Crit Care Med*. 1992;20:1302–1308.
331. DeBehnke DJ, Angelos MG, Leasure JE. Comparison of standard external CPR, open-chest CPR, and cardiopulmonary bypass in a canine myocardial infarct model. *Ann Emerg Med*. 1991;20:754–760.
332. Bircher N, Safar P. Cerebral preservation during cardiopulmonary resuscitation. *Crit Care Med*. 1985;13:185–190.
333. Kern KB, Sanders AB, Badylak SF, Janas W, Carter AB, Tacker WA, Ewy GA. Long-term survival with open-chest cardiac massage after ineffective closed-chest compression in a canine preparation. *Circulation*. 1987;75:498–503.
334. Raessler KL, Kern KB, Sanders AB, Tacker WA Jr, Ewy GA. Aortic and right atrial systolic pressures during cardiopulmonary resuscitation: a potential indicator of the mechanism of blood flow. *Am Heart J*. 1988;115:1021–1029.
335. Weiser F, Adler L, Kuhn L. Hemodynamic Effects of closed and open chest cardiac resuscitation in normal dogs and those with acute myocardial infarction. *Am J Cardiol*. 1962;10:555–561.
336. Bartlett RL, Stewart NJ Jr, Raymond J, Anstadt GL, Martin SD. Comparative study of three methods of resuscitation: closed-chest, open-chest manual, and direct mechanical ventricular assistance. *Ann Emerg Med*. 1984;13:773–777.
337. Fleisher G, Sagy M, Swedlow DB, Belani K. Open- versus closed-chest cardiac compressions in a canine model of pediatric cardiopulmonary resuscitation. *Am J Emerg Med*. 1985;3:305–310.
338. Jackson RE, Joyce K, Danosi SF, White BC, Vigor D, Hoehner TJ. Blood flow in the cerebral cortex during cardiac resuscitation in dogs. *Ann Emerg Med*. 1984;13:657–659.
339. Kern KB, Sanders AB, Janas W, Nelson JR, Badylak SF, Babbs CF, Tacker WA, Ewy GA. Limitations of open-chest cardiac massage after prolonged, untreated cardiac arrest in dogs. *Ann Emerg Med*. 1991;20:761–767.
340. Redding JS, Cozine RA. A comparison of open-chest and closed-chest cardiac massage in dogs. *Anesthesiology*. 1961;22:280–285.
341. Rubertsson S, Grenvik A, Wiklund L. Blood flow and perfusion pressure during open-chest versus closed-chest cardiopulmonary resuscitation in pigs. *Crit Care Med*. 1995;23:715–725.
342. Rubertsson S, Grenvik A, Zengulis V, Wiklund L. Systemic perfusion pressure and blood flow before and after administration of epinephrine during experimental cardiopulmonary resuscitation. *Crit Care Med*. 1995;23:1984–1996.
343. Sanders AB, Kern KB, Atlas M, Bragg S, Ewy GA. Importance of the duration of inadequate coronary perfusion pressure on resuscitation from cardiac arrest. *J Am Coll Cardiol*. 1985;6:113–118.
344. Sanders AB, Kern KB, Ewy GA, Atlas M, Bailey L. Improved resuscitation from cardiac arrest with open-chest massage. *Ann Emerg Med*. 1984;13(pt 1):672–675.
345. Chiladakis JA, Stathopoulos C, Davlourous P, Manolis AS. Intravenous magnesium sulfate versus diltiazem in paroxysmal atrial fibrillation. *Int J Cardiol*. 2001;79:287–291.
346. Wattanasuwan N, Khan IA, Mehta NJ, Arora P, Singh N, Vasavada BC, Sacchi TJ. Acute ventricular rate control in atrial fibrillation: IV combination of diltiazem and digoxin vs. IV diltiazem alone. *Chest*. 2001;119:502–506.

347. Sticherling C, Tada H, Hsu W, Bares AC, Oral H, Pelosi F, Knight BP, Strickberger SA, Morady F. Effects of diltiazem and esmolol on cycle length and spontaneous conversion of atrial fibrillation. *J Cardiovasc Pharmacol Ther.* 2002;7:81–88.
348. Shettigar UR, Toole JG, Appunn DO. Combined use of esmolol and digoxin in the acute treatment of atrial fibrillation or flutter. *Am Heart J.* 1993;126:368–374.
349. Wang HE, O'Connor R E, Megargel RE, Schnyder ME, Morrison DM, Barnes TA, Fitzkee A. The use of diltiazem for treating rapid atrial fibrillation in the out-of-hospital setting. *Ann Emerg Med.* 2001;37:38–45.
350. Martinez-Marcos FJ, Garcia-Garmendia JL, Ortega-Carpio A, Fernandez-Gomez JM, Santos JM, Camacho C. Comparison of intravenous flecainide, propafenone, and amiodarone for conversion of acute atrial fibrillation to sinus rhythm. *Am J Cardiol.* 2000;86:950–953.
351. Kalus JS, Spencer AP, Tsikouris JP, Chung JO, Kenyon KW, Ziska M, Kluger J, White CM. Impact of prophylactic i.v. magnesium on the efficacy of ibutilide for conversion of atrial fibrillation or flutter. *Am J Health Syst Pharm.* 2003;60:2308–2312.
352. Lim SH, Anantharaman V, Teo WS, Goh PP, Tan AT. Comparison of treatment of supraventricular tachycardia by Valsalva maneuver and carotid sinus massage. *Ann Emerg Med.* 1998;31:30–35.
353. Ornato JP, Hallagan LF, Reese WA, Clark RF, Tayal VS, Garnett AR, Gonzalez ER. Treatment of paroxysmal supraventricular tachycardia in the emergency department by clinical decision analysis [published correction appears in *Am J Emerg Med.* 1990;8:85]. *Am J Emerg Med.* 1988;6:555–560.
354. DiMarco JP, Miles W, Akhtar M, Milstein S, Sharma AD, Platia E, McGovern B, Scheinman MM, Govier WC. Adenosine for paroxysmal supraventricular tachycardia: dose ranging and comparison with verapamil: assessment in placebo-controlled, multicenter trials. The Adenosine for PSVT Study Group [published correction appears in *Ann Intern Med.* 1990;113:996]. *Ann Intern Med.* 1990;113:104–110.
355. Brady WJ Jr, DeBehnke DJ, Wickman LL, Lindbeck G. Treatment of out-of-hospital supraventricular tachycardia: adenosine vs verapamil. *Acad Emerg Med.* 1996;3:574–585.
356. Madsen CD, Pointer JE, Lynch TG. A comparison of adenosine and verapamil for the treatment of supraventricular tachycardia in the pre-hospital setting. *Ann Emerg Med.* 1995;25:649–655.
357. Furlong R, Gerhardt RT, Farber P, Schrank K, Willig R, Pittaluga J. Intravenous adenosine as first-line prehospital management of narrow-complex tachycardias by EMS personnel without direct physician control. *Am J Emerg Med.* 1995;13:383–388.
358. Morrison LJ, Allan R, Vermeulen M, Dong SL, McCallum AL. Conversion rates for prehospital paroxysmal supraventricular tachycardia (PSVT) with the addition of adenosine: a before-and-after trial. *Prehosp Emerg Care.* 2001;5:353–359.
359. Cheng KA. [A randomized, multicenter trial to compare the safety and efficacy of adenosine versus verapamil for termination of paroxysmal supraventricular tachycardia]. *Zhonghua Nei Ke Za Zhi.* 2003;42:773–776.
360. Lim SH, Anantharaman V, Teo WS. Slow-infusion of calcium channel blockers in the emergency management of supraventricular tachycardia. *Resuscitation.* 2002;52:167–174.
361. Gupta A, Naik A, Vora A, Lokhandwala Y. Comparison of efficacy of intravenous diltiazem and esmolol in terminating supraventricular tachycardia. *J Assoc Physicians India.* 1999;47:969–972.
362. Cybulski J, Kulakowski P, Makowska E, Czepiel A, Sikora-Frac M, Ceremuzynski L. Intravenous amiodarone is safe and seems to be effective in termination of paroxysmal supraventricular tachyarrhythmias. *Clin Cardiol.* 1996;19:563–566.
363. Schutzenberger W, Leisch F, Kerschner K, Harringer W, Herbinger W. Clinical efficacy of intravenous amiodarone in the short term treatment of recurrent sustained ventricular tachycardia and ventricular fibrillation. *Br Heart J.* 1989;62:367–371.
364. Credner SC, Klinghenben T, Mauss O, Sticherling C, Hohnloser SH. Electrical storm in patients with transvenous implantable cardioverter-defibrillators: incidence, management and prognostic implications. *J Am Coll Cardiol.* 1998;32:1909–1915.
365. Helmy I, Herre JM, Gee G, Sharkey H, Malone P, Sauve MJ, Griffin JC, Scheinman MM. Use of intravenous amiodarone for emergency treatment of life-threatening ventricular arrhythmias. *J Am Coll Cardiol.* 1988;12:1015–1022.
366. Gorgels AP, van den Dool A, Hofs A, Mulleneers R, Smeets JL, Vos MA, Wellens HJ. Comparison of procainamide and lidocaine in terminating sustained monomorphic ventricular tachycardia. *Am J Cardiol.* 1996;78:43–46.
367. Marill KA, Greenberg GM, Kay D, Nelson BK. Analysis of the treatment of spontaneous sustained stable ventricular tachycardia. *Acad Emerg Med.* 1997;4:1122–1128.
368. Armengol RE, Graff J, Baerman JM, Swiryn S. Lack of effectiveness of lidocaine for sustained, wide QRS complex tachycardia. *Ann Emerg Med.* 1989;18:254–257.
369. Domanovits H, Paulis M, Nikfardjam M, Holzer M, Stuhlinger HG, Hirschl MM, Laggner AN. Sustained ventricular tachycardia in the emergency department. *Resuscitation.* 1999;42:19–25.
370. Ho DS, Zecchin RP, Richards DA, Uther JB, Ross DL. Double-blind trial of lignocaine versus sotalol for acute termination of spontaneous sustained ventricular tachycardia. *Lancet.* 1994;344:18–23.
371. Tzivoni D, Banai S, Schuger C, Benhorin J, Keren A, Gottlieb S, Stern S. Treatment of torsade de pointes with magnesium sulfate. *Circulation.* 1988;77:392–397.
372. Nguyen PT, Scheinman MM, Seger J. Polymorphous ventricular tachycardia: clinical characterization, therapy, and the QT interval. *Circulation.* 1986;74:340–349.
373. Manz M, Pfeiffer D, Jung W, Lueritz B. Intravenous treatment with magnesium in recurrent persistent ventricular tachycardia. *New Trends in Arrhythmias.* 1991;7:437–442.
374. Keren A, Tzivoni D, Gavish D, Levi J, Gottlieb S, Benhorin J, Stern S. Etiology, warning signs and therapy of torsade de pointes: a study of 10 patients. *Circulation.* 1981;64:1167–1174.
375. Smith I, Monk TG, White PF. Comparison of transesophageal atrial pacing with anticholinergic drugs for the treatment of intraoperative bradycardia. *Anesth Analg.* 1994;78:245–252.
376. Bertolet BD, McMurtrie EB, Hill JA, Belardinelli L. Theophylline for the treatment of atrioventricular block after myocardial infarction. *Ann Intern Med.* 1995;123:509–511.
377. Atarashi H, Endoh Y, Saitoh H, Kishida H, Hayakawa H. Chronotropic effects of cilostazol, a new antithrombotic agent, in patients with bradyarrhythmias. *J Cardiovasc Pharmacol.* 1998;31:534–539.
378. Gauss A, Hubner C, Meierhenrich R, Rohm HJ, Georgieff M, Schutz W. Perioperative transcutaneous pacemaker in patients with chronic bifascicular block or left bundle branch block and additional first-degree atrioventricular block. *Acta Anaesthesiol Scand.* 1999;43:731–736.
379. Love JN, Sachdeva DK, Bessman ES, Curtis LA, Howell JM. A potential role for glucagon in the treatment of drug-induced symptomatic bradycardia. *Chest.* 1998;114:323–326.
380. Bertolet BD, Eagle DA, Conti JB, Mills RM, Belardinelli L. Bradycardia after heart transplantation: reversal with theophylline. *J Am Coll Cardiol.* 1996;28:396–399.
381. Chamberlain DA, Turner P, Sneddon JM. Effects of atropine on heart-rate in healthy man. *Lancet.* 1967;2:12–15.
382. Bernheim A, Fatio R, Kiowski W, Weilenmann D, Rickli H, Rocca HP. Atropine often results in complete atrioventricular block or sinus arrest after cardiac transplantation: an unpredictable and dose-independent phenomenon. *Transplantation.* 2004;77:1181–1185.
383. Klumbies A, Paliege R, Volkmann H. [Mechanical emergency stimulation in asystole and extreme bradycardia]. *Z Gesamte Inn Med.* 1988;43:348–352.
384. Zeh E, Rahnner E. [The manual extrathoracic stimulation of the heart. Technique and effect of the precordial thump (author's trans)]. *Z Kardiol.* 1978;67:299–304.
385. Chan L, Reid C, Taylor B. Effect of three emergency pacing modalities on cardiac output in cardiac arrest due to ventricular asystole. *Resuscitation.* 2002;52:117–119.
386. Greif R, Rajek A, Laciny S, Bastanmehr H, Sessler DI. Resistive heating is more effective than metallic-foil insulation in an experimental model of accidental hypothermia: a randomized controlled trial. *Ann Emerg Med.* 2000;35:337–345.
387. Roggla M, Frossard M, Wagner A, Holzer M, Bur A, Roggla G. Severe accidental hypothermia with or without hemodynamic instability: rewarming without the use of extracorporeal circulation. *Wien Klin Wochenschr.* 2002;114:315–320.
388. Kornberger E, Schwarz B, Lindner KH, Mair P. Forced air surface rewarming in patients with severe accidental hypothermia. *Resuscitation.* 1999;41:105–111.
389. Farstad M, Andersen KS, Koller ME, Grong K, Segadal L, Husby P. Rewarming from accidental hypothermia by extracorporeal circulation. A retrospective study. *Eur J Cardiothorac Surg.* 2001;20:58–64.

390. Silfvast T, Pettila V. Outcome from severe accidental hypothermia in Southern Finland—a 10-year review. *Resuscitation*. 2003;59:285–290.
391. Brugger H, Sumann G, Meister R, Adler-Kastner L, Mair P, Gunga HC, Schobersberger W, Falk M. Hypoxia and hypercapnia during respiration into an artificial air pocket in snow: implications for avalanche survival. *Resuscitation*. 2003;58:81–88.
392. Watson RS, Cummings P, Quan L, Bratton S, Weiss NS. Cervical spine injuries among submersion victims. *J Trauma*. 2001;51:658–662.
393. Onarheim H, Vik V. Porcine surfactant (Curosurf) for acute respiratory failure after near-drowning in 12 year old. *Acta Anaesthesiol Scand*. 2004;48:778–781.
394. Staudinger T, Bankier A, Strohmaier W, Weiss K, Locker GJ, Knapp S, Roggla M, Laczika K, Frass M. Exogenous surfactant therapy in a patient with adult respiratory distress syndrome after near drowning. *Resuscitation*. 1997;35:179–182.
395. Suzuki H, Ohta T, Iwata K, Yamaguchi K, Sato T. Surfactant therapy for respiratory failure due to near-drowning. *Eur J Pediatr*. 1996;155:383–384.
396. Dottorini M, Eslami A, Baglioni S, Fiorenzano G, Todisco T. Nasal-continuous positive airway pressure in the treatment of near-drowning in freshwater. *Chest*. 1996;110:1122–1124.
397. Foex BA, Boyd R. Towards evidence based emergency medicine: best BETs from the Manchester Royal Infirmary. Corticosteroids in the management of near-drowning. *Emerg Med J*. 2001;18:465–466.
398. Takano Y, Hirotsako S, Yamaguchi T, Saita N, Suga M, Kukita I, Okamoto K, Ando M. [Nitric oxide inhalation as an effective therapy for acute respiratory distress syndrome due to near-drowning: a case report]. *Nihon Koryuiki Gakkai Zasshi*. 1999;37:997–1002.
399. Williamson JP, Illing R, Gertler P, Braude S. Near-drowning treated with therapeutic hypothermia. *Med J Aust*. 2004;181:500–501.
400. Sumann G, Krismer AC, Wenzel V, Adelsmayr E, Schwarz B, Lindner KH, Mair P. Cardiopulmonary resuscitation after near drowning and hypothermia: restoration of spontaneous circulation after vasopressin. *Acta Anaesthesiol Scand*. 2003;47:363–365.
401. Thalmann M, Trampitsch E, Haberfellner N, Eisendle E, Kraschl R, Kobin G. Resuscitation in near drowning with extracorporeal membrane oxygenation. *Ann Thorac Surg*. 2001;72:607–608.
402. Bolte RG, Black PG, Bowers RS, Thorne JK, Corneli HM. The use of extracorporeal rewarming in a child submerged for 66 minutes. *JAMA*. 1988;260:377–379.
403. Budnick LD. Bathtub-related electrocutions in the United States, 1979 to 1982. *JAMA*. 1984;252:918–920.
404. Chambers JJ, Saha AK. Electrocution during anaesthesia. *Anaesthesia*. 1979;34:173–175.
405. Guinard JP, Chiolerio R, Buchser E, Delaloye-Bischof A, Payot M, Grbic A, Krupp S, Freeman J. Myocardial injury after electrical burns: short and long term study. *Scand J Plast Reconstr Surg Hand Surg*. 1987;21:301–302.
406. Homma S, Gillam LD, Weyman AE. Echocardiographic observations in survivors of acute electrical injury. *Chest*. 1990;97:103–105.
407. James TN, Riddick L, Embry JH. Cardiac abnormalities demonstrated postmortem in four cases of accidental electrocution and their potential significance relative to nonfatal electrical injuries of the heart. *Am Heart J*. 1990;120:143–157.
408. Martinez JA, Nguyen T. Electrical injuries. *South Med J*. 2000;93:1165–1168.
409. Romero B, Candell-Riera J, Gracia RM, Fernandez MA, Aguade S, Peracaula R, Soler-Soler J. Myocardial necrosis by electrocution: evaluation of noninvasive methods. *J Nucl Med*. 1997;38:250–251.
410. Still J, Orlet H, Law E, Wheeler M, Pickens H. Electrocution due to contact of industrial equipment with power lines. *Burns*. 1997;23:573–575.
411. Veneman TF, van Dijk GW, Boereboom E, Joore H, Savelkoul TJ. Prediction of outcome after resuscitation in a case of electrocution. *Intensive Care Med*. 1998;24:255–257.
412. Whitcomb D, Martinez JA, Daberkow D. Lightning injuries. *South Med J*. 2002;95:1331–1334.
413. Department of Health, Welsh Office, Scottish Office Department of Health, Department of Health and Social Services, Northern Ireland. Why mothers die. Report on confidential enquiries into maternal deaths in the United Kingdom, 2000–2002. In: London: The Stationery Office; 2004.
414. Goodwin H, Holmes JF, Wisner DH. Abdominal ultrasound examination in pregnant blunt trauma patients. *J Trauma*. 2001;50:689–693; discussion 694.
415. Lazebnik N, Lazebnik RS. The role of ultrasound in pregnancy-related emergencies. *Radiol Clin North Am*. 2004;42:315–327.
416. Katz VL, Dotters DJ, Droegemueller W. Perimortem cesarean delivery. *Obstet Gynecol*. 1986;68:571–576.
417. Whitten M, Irvine LM. Postmortem and perimortem caesarean section: what are the indications? *J R Soc Med*. 2000;93:6–9.
418. Bouman EA, Gutierrez y Leon JA, van der Salm PC, Christiaens GC, Bruinse HW, Broeders IA. [Complicated but successful resuscitation after amniotic fluid embolism]. *Ned Tijdschr Geneesk*. 2001;145:747–749.
419. Cardosi RJ, Porter KB. Cesarean delivery of twins during maternal cardiopulmonary arrest. *Obstet Gynecol*. 1998;92:695–697.
420. Finegold H, Darwich A, Romeo R, Vallejo M, Ramanathan S. Successful resuscitation after maternal cardiac arrest by immediate cesarean section in the labor room. *Anesthesiology*. 2002;96:1278.
421. Lanoix R, Akkapeddi V, Goldfeder B. Perimortem cesarean section: case reports and recommendations. *Acad Emerg Med*. 1995;2:1063–1067.
422. Kinsella SM. Lateral tilt for pregnant women: why 15 degrees? *Anaesthesia*. 2003;58:835–836.
423. Rees GA, Willis BA. Resuscitation in late pregnancy. *Anaesthesia*. 1988;43:347–349.
424. Nanson J, Elcock D, Williams M, Deakin CD. Do physiological changes in pregnancy change defibrillation energy requirements? *Br J Anaesth*. 2001;87:237–239.
425. Deakin CD, McLaren RM, Petley GW, Clewlow F, Dalrymple-Hay MJ. Effects of positive end-expiratory pressure on transthoracic impedance—implications for defibrillation. *Resuscitation*. 1998;37:9–12.
426. Rodrigo G, Pollack C, Rodrigo C, Rowe BH. Heliox for nonintubated acute asthma patients. *Cochrane Database Syst Rev*. 2003;CD002884.
427. Williams TJ, Tuxen DV, Scheinkestel CD, Czarny D, Bowes G. Risk factors for morbidity in mechanically ventilated patients with acute severe asthma. *Am Rev Respir Dis*. 1992;146:607–615.
428. Leatherman JW, McArthur C, Shapiro RS. Effect of prolongation of expiratory time on dynamic hyperinflation in mechanically ventilated patients with severe asthma. *Crit Care Med*. 2004;32:1542–1545.
429. Woda RP, Dzwonczyk R, Bernacki BL, Cannon M, Lynn L. The ventilatory effects of auto-positive end-expiratory pressure development during cardiopulmonary resuscitation. *Crit Care Med*. 1999;27:2212–2217.
430. Myles PS, Madder H, Morgan EB. Intraoperative cardiac arrest after unrecognized dynamic hyperinflation. *Br J Anaesth*. 1995;74:340–342.
431. Van der Touw T, Mudaliar Y, Nayyar V. Cardiorespiratory effects of manually compressing the rib cage during tidal expiration in mechanically ventilated patients recovering from acute severe asthma. *Crit Care Med*. 1998;26:1361–1367.
432. Narimatsu E, Nara S, Kita A, Kurimoto Y, Asai Y, Ishikawa A. Serious circulatory deficiency during external chest compression for asthma attack. *Am J Emerg Med*. 2001;19:169–171.
433. Diamant RH, Sloan JP. Failed resuscitation in acute severe asthma: a medical indication for emergency thoracotomy? *Arch Emerg Med*. 1987;4:233–235.
434. Smolnikoff VP. Total bronchospasm and lung massage. *Anaesthesia*. 1960;15:40–44.
435. Lee DC, Greene T, Dougherty T, Pearigen P. Fatal nifedipine ingestions in children. *J Emerg Med*. 2000;19:359–361.
436. Brown TC. Sodium bicarbonate treatment for tricyclic antidepressant arrhythmias in children. *Med J Aust*. 1976;2:380–382.
437. Hoffman JR, Votey SR, Bayer M, Silver L. Effect of hypertonic sodium bicarbonate in the treatment of moderate-to-severe cyclic antidepressant overdose. *Am J Emerg Med*. 1993;11:336–341.
438. Knudsen K, Abrahamsson J. Epinephrine and sodium bicarbonate independently and additively increase survival in experimental amitriptyline poisoning. *Crit Care Med*. 1997;25:669–674.
439. Nattel S, Mittleman M. Treatment of ventricular tachyarrhythmias resulting from amitriptyline toxicity in dogs. *J Pharmacol Exp Ther*. 1984;231:430–435.
440. Nattel S, Keable H, Sasyniuk BI. Experimental amitriptyline intoxication: electrophysiologic manifestations and management. *J Cardiovasc Pharmacol*. 1984;6:83–89.
441. Pentel P, Benowitz N. Efficacy and mechanism of action of sodium bicarbonate in the treatment of desipramine toxicity in rats. *J Pharmacol Exp Ther*. 1984;230:12–19.

442. Brown TC, Barker GA, Dunlop ME, Loughnan PM. The use of sodium bicarbonate in the treatment of tricyclic antidepressant-induced arrhythmias. *Anaesth Intensive Care*. 1973;1:203–210.
443. Brown TC. Tricyclic antidepressant overdose: experimental studies on the management of circulatory complications. *Clin Toxicol*. 1976;9:255–272.
444. Hedges JR, Baker PB, Tasset JJ, Otten EJ, Dalsey WC, Syverud SA. Bicarbonate therapy for the cardiovascular toxicity of amitriptyline in an animal model. *J Emerg Med*. 1985;3:253–260.
445. Sasyniuk BI, Jhamandas V, Valois M. Experimental amitriptyline intoxication: treatment of cardiac toxicity with sodium bicarbonate. *Ann Emerg Med*. 1986;15:1052–1059.
446. Stone CK, Kraemer CM, Carroll R, Low R. Does a sodium-free buffer affect QRS width in experimental amitriptyline overdose? *Ann Emerg Med*. 1995;26:58–64.
447. McCabe JL, Cobaugh DJ, Menegazzi JJ, Fata J. Experimental tricyclic antidepressant toxicity: a randomized, controlled comparison of hypertonic saline solution, sodium bicarbonate, and hyperventilation. *Ann Emerg Med*. 1998;32:329–333.
448. Sasyniuk BI, Jhamandas V. Mechanism of reversal of toxic effects of amitriptyline on cardiac Purkinje fibers by sodium bicarbonate. *J Pharmacol Exp Ther*. 1984;231:387–394.
449. Sasyniuk BI, Jhamandas V. Frequency-dependent effects of amitriptyline on Vmax in canine Purkinje fibers and its alteration by alkalosis. *Proc West Pharmacol Soc*. 1986;29:73–75.
450. Bou-Abboud E, Nattel S. Molecular mechanisms of the reversal of imipramine-induced sodium channel blockade by alkalization in human cardiac myocytes. *Cardiovasc Res*. 1998;38:395–404.
451. Levitt MA, Sullivan JB Jr, Owens SM, Burnham L, Finley PR. Amitriptyline plasma protein binding: effect of plasma pH and relevance to clinical overdose. *Am J Emerg Med*. 1986;4:121–125.
452. Osterwalder JJ. Naloxone—for intoxications with intravenous heroin and heroin mixtures—harmless or hazardous? A prospective clinical study. *J Toxicol Clin Toxicol*. 1996;34:409–416.
453. Sporer KA, Firestone J, Isaacs SM. Out-of-hospital treatment of opioid overdoses in an urban setting. *Acad Emerg Med*. 1996;3:660–667.
454. Wanger K, Brough L, Macmillan I, Goulding J, MacPhail I, Christenson JM. Intravenous vs subcutaneous naloxone for out-of-hospital management of presumed opioid overdose. *Acad Emerg Med*. 1998;5:293–299.
455. Hasan RA, Benko AS, Nolan BM, Campe J, Duff J, Zureikat GY. Cardiorespiratory effects of naloxone in children. *Ann Pharmacother*. 2003;37:1587–1592.
456. Sporer KA. Acute heroin overdose. *Ann Intern Med*. 1999;130:584–590.
457. Schneir AB, Vadeboncoeur TF, Offerman SR, Barry JD, Ly BT, Williams SR, Clark RF. Massive OxyContin ingestion refractory to naloxone therapy. *Ann Emerg Med*. 2002;40:425–428.
458. Buunk G, van der Hoeven JG, Meinders AE. Cerebrovascular reactivity in comatose patients resuscitated from a cardiac arrest. *Stroke*. 1997;28:1569–1573.
459. Buunk G, van der Hoeven JG, Meinders AE. A comparison of near-infrared spectroscopy and jugular bulb oximetry in comatose patients resuscitated from a cardiac arrest. *Anaesthesia*. 1998;53:13–19.
460. Roine RO, Launes J, Nikkinen P, Lindroth L, Kaste M. Regional cerebral blood flow after human cardiac arrest. A hexamethylpropyleneamine oxime single photon emission computed tomographic study. *Arch Neurol*. 1991;48:625–629.
461. Beckstead JE, Tweed WA, Lee J, MacKeen WL. Cerebral blood flow and metabolism in man following cardiac arrest. *Stroke*. 1978;9:569–573.
462. Menon DK, Coles JP, Gupta AK, Fryer TD, Smielewski P, Chatfield DA, Aigbirhio F, Skepper JN, Minhas PS, Hutchinson PJ, Carpenter TA, Clark JC, Pickard JD. Diffusion limited oxygen delivery following head injury. *Crit Care Med*. 2004;32:1384–1390.
463. Ebmeyer US, P.; Radovsky, A.; Sharma, C.; Tanigawa, K.; Wang, Y.; Capone, A.; Xiao, F.; Bircher, N.; Alexander, H. Effective combination treatments for cerebral resuscitation from cardiac arrest in dogs. Exploratory studies. *Resuscitation*. 1994;28:57.
464. Safar P, Xiao F, Radovsky A, Tanigawa K, Ebmeyer U, Bircher N, Alexander H, Stezoski SW. Improved cerebral resuscitation from cardiac arrest in dogs with mild hypothermia plus blood flow promotion. *Stroke*. 1996;27:105–113.
465. Nwaigwe CI, Roche MA, Grinberg O, Dunn JF. Effect of hyperventilation on brain tissue oxygenation and cerebrovenous PO₂ in rats. *Brain Res*. 2000;868:150–156.
466. Cohan SL, Mun SK, Petite J, Correia J, Tavelra Da Silva AT, Waldhorn RE. Cerebral blood flow in humans following resuscitation from cardiac arrest. *Stroke*. 1989;20:761–765.
467. Xu Y, Liachenko S, Tang P. Dependence of early cerebral reperfusion and long-term outcome on resuscitation efficiency after cardiac arrest in rats. *Stroke*. 2002;33:837–843.
468. Hypothermia After Cardiac Arrest Study Group. Mild therapeutic hypothermia to improve the neurologic outcome after cardiac arrest. *N Engl J Med*. 2002;346:549–556.
469. Bernard SA, Gray TW, Buist MD, Jones BM, Silvester W, Gutteridge G, Smith K. Treatment of comatose survivors of out-of-hospital cardiac arrest with induced hypothermia. *N Engl J Med*. 2002;346:557–563.
470. Hachimi-Idrissi S, Corne L, Ebinger G, Michotte Y, Huyghens L. Mild hypothermia induced by a helmet device: a clinical feasibility study. *Resuscitation*. 2001;51:275–281.
471. Bernard SA, Jones BM, Horne MK. Clinical trial of induced hypothermia in comatose survivors of out-of-hospital cardiac arrest. *Ann Emerg Med*. 1997;30:146–153.
472. Bernard S, Buist M, Monteiro O, Smith K. Induced hypothermia using large volume, ice-cold intravenous fluid in comatose survivors of out-of-hospital cardiac arrest: a preliminary report. *Resuscitation*. 2003;56:9–13.
473. Virkkunen I, Yli-Hankala A, Silfvast T. Induction of therapeutic hypothermia after cardiac arrest in prehospital patients using ice-cold Ringer's solution: a pilot study. *Resuscitation*. 2004;62:299–302.
474. Al-Senani FM, Graffagnino C, Grotta JC, Saiki R, Wood D, Chung W, Palmer G, Collins KA. A prospective, multicenter pilot study to evaluate the feasibility and safety of using the CoolGard System and Icy catheter following cardiac arrest. *Resuscitation*. 2004;62:143–150.
475. Kliegel A, Losert H, Sterz F, Kliegel M, Holzer M, Uray T, Domanovits H. Cold simple intravenous infusions preceding special endovascular cooling for faster induction of mild hypothermia after cardiac arrest—a feasibility study. *Resuscitation*. 2005;64:347–351.
476. Schmutzhard E, Engelhardt K, Beer R, Brossner G, Pfausler B, Spiss H, Unterberger I, Kampfl A. Safety and efficacy of a novel intravascular cooling device to control body temperature in neurologic intensive care patients: a prospective pilot study. *Crit Care Med*. 2002;30:2481–2488.
477. Diringer MN, Reaven NL, Funk SE, Uman GC. Elevated body temperature independently contributes to increased length of stay in neurologic intensive care unit patients. *Crit Care Med*. 2004;32:1489–1495.
478. Keller E, Imhof HG, Gasser S, Terzic A, Yonekawa Y. Endovascular cooling with heat exchange catheters: a new method to induce and maintain hypothermia. *Intensive Care Med*. 2003;29:939–943.
479. Agnew DM, Koehler RC, Guerguerian AM, Shaffner DH, Traystman RJ, Martin LJ, Ichord RN. Hypothermia for 24 hours after asphyxial cardiac arrest in piglets provides striatal neuroprotection that is sustained 10 days after rewarming. *Pediatr Res*. 2003;54:253–262.
480. Hicks SD, DeFranco DB, Callaway CW. Hypothermia during reperfusion after asphyxial cardiac arrest improves functional recovery and selectively alters stress-induced protein expression. *J Cereb Blood Flow Metab*. 2000;20:520–530.
481. Sterz F, Safar P, Tisherman S, Radovsky A, Kuboyama K, Oku K. Mild hypothermic cardiopulmonary resuscitation improves outcome after prolonged cardiac arrest in dogs. *Crit Care Med*. 1991;19:379–389.
482. Xiao F, Safar P, Radovsky A. Mild protective and resuscitative hypothermia for asphyxial cardiac arrest in rats. *Am J Emerg Med*. 1998;16:17–25.
483. Katz LM, Young A, Frank JE, Wang Y, Park K. Neurotensin-induced hypothermia improves neurologic outcome after hypoxic-ischemia. *Crit Care Med*. 2004;32:806–810.
484. Abella BS, Zhao D, Alvarado J, Hamann K, Vanden Hoek TL, Becker LB. Intra-arrest cooling improves outcomes in a murine cardiac arrest model. *Circulation*. 2004;109:2786–2791.
485. Krumholz A, Stern BJ, Weiss HD. Outcome from coma after cardiopulmonary resuscitation: relation to seizures and myoclonus. *Neurology*. 1988;38:401–405.
486. Snyder BD, Hauser WA, Loewenson RB, Leppik IE, Ramirez-Lassepas M, Gumnit RJ. Neurologic prognosis after cardiopulmonary arrest, III: seizure activity. *Neurology*. 1980;30:1292–1297.
487. Wijedicks EF, Parisi JE, Sharbrough FW. Prognostic value of myoclonus status in comatose survivors of cardiac arrest. *Ann Neurol*. 1994;35:239–243.
488. Takino M, Okada Y. Hyperthermia following cardiopulmonary resuscitation. *Intensive Care Med*. 1991;17:419–420.

489. Hickey RW, Kochanek PM, Ferimer H, Alexander HL, Garman RH, Graham SH. Induced hyperthermia exacerbates neurologic neuronal histologic damage after asphyxial cardiac arrest in rats. *Crit Care Med*. 2003;31:531–535.
490. Takasu A, Saitoh D, Kaneko N, Sakamoto T, Okada Y. Hyperthermia: is it an ominous sign after cardiac arrest? *Resuscitation*. 2001;49:273–277.
491. Zeiner A, Holzer M, Sterz F, Schorkhuber W, Eisenburger P, Havel C, Kliegel A, Laggner AN. Hyperthermia after cardiac arrest is associated with an unfavorable neurologic outcome. *Arch Intern Med*. 2001;161:2007–2012.
492. Hajat C, Hajat S, Sharma P. Effects of poststroke pyrexia on stroke outcome : a meta-analysis of studies in patients. *Stroke*. 2000;31:410–414.
493. Kasner SE, Wein T, Piriyaawat P, Villar-Cordova CE, Chalela JA, Krieger DW, Morgenstern LB, Kimmel SE, Grotta JC. Acetaminophen for altering body temperature in acute stroke: a randomized clinical trial. *Stroke*. 2002;33:130–134.
494. Dippel DW, van Breda EJ, van der Worp HB, van Gemert HM, Meijer RJ, Kappelle LJ, Koudstaal PJ. Effect of paracetamol (acetaminophen) and ibuprofen on body temperature in acute ischemic stroke PISA, a phase II double-blind, randomized, placebo-controlled trial. *BMC Cardiovasc Disord*. 2003;3:2.
495. Coimbra C, Boris-Moller F, Drake M, Wieloch T. Diminished neuronal damage in the rat brain by late treatment with the antipyretic drug dipyrone or cooling following cerebral ischemia. *Acta Neuropathol (Berl)*. 1996;92:447–453.
496. Coimbra C, Drake M, Boris-Moller F, Wieloch T. Long-lasting neuroprotective effect of postischemic hypothermia and treatment with an anti-inflammatory/antipyretic drug: evidence for chronic encephalopathic processes following ischemia. *Stroke*. 1996;27:1578–1585.
497. Rocamora R, Kurthen M, Lickfett L, Von Oertzen J, Elger CE. Cardiac asystole in epilepsy: clinical and neurophysiologic features. *Epilepsia*. 2003;44:179–185.
498. Zijlmans M, Flanagan D, Gotman J. Heart rate changes and ECG abnormalities during epileptic seizures: prevalence and definition of an objective clinical sign. *Epilepsia*. 2002;43:847–854.
499. McCall WV. Asystole in electroconvulsive therapy: report of four cases. *J Clin Psychiatry*. 1996;57:199–203.
500. Sakabe T, Tateishi A, Miyauchi Y, Maekawa T, Matsumoto M, Tsutsui T, Takeshita H. Intracranial pressure following cardiopulmonary resuscitation. *Intensive Care Med*. 1987;13:256–259.
501. Safar P. Recent advances in cardiopulmonary-cerebral resuscitation: a review. *Ann Emerg Med*. 1984;13:856–862.
502. So EL, Sam MC, Lagerlund TL. Postictal central apnea as a cause of SUDEP: evidence from near-SUDEP incident. *Epilepsia*. 2000;41:1494–1497.
503. Rello J, Diaz E, Roque M, Valles J. Risk factors for developing pneumonia within 48 hours of intubation. *Am J Respir Crit Care Med*. 1999;159:1742–1746.
504. van den Berghe G, Wouters P, Weekers F, Verwaest C, Bruyninckx F, Schetz M, Vlasselaers D, Ferdinande P, Lauwers P, Bouillon R. Intensive insulin therapy in the critically ill patients. *N Engl J Med*. 2001;345:1359–1367.
505. Krinsley JS. Effect of an intensive glucose management protocol on the mortality of critically ill adult patients. *Mayo Clin Proc*. 2004;79:992–1000.
506. Langhelle A, Tyvold SS, Lexow K, Hapnes SA, 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;56:247–263.
507. Calle PA, Buylaert WA, Vanhaute OA. Glycemia in the post-resuscitation period. The Cerebral Resuscitation Study Group. *Resuscitation*. 1989;17 Suppl:S181–S188; discussion S199–S206.
508. Longstreth WT Jr, Diehr P, Inui TS. Prediction of awakening after out-of-hospital cardiac arrest. *N Engl J Med*. 1983;308:1378–1382.
509. Longstreth WT Jr, Inui TS. High blood glucose level on hospital admission and poor neurological recovery after cardiac arrest. *Ann Neurol*. 1984;15:59–63.
510. Longstreth WT Jr, Copass MK, Dennis LK, Rauch-Matthews ME, Stark MS, Cobb LA. Intravenous glucose after out-of-hospital cardiopulmonary arrest: a community-based randomized trial. *Neurology*. 1993;43:2534–2541.
511. Mackenzie CF. A review of 100 cases of cardiac arrest and the relation of potassium, glucose, and haemoglobin levels to survival. *West Indian Med J*. 1975;24:39–45.
512. Mullner 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;17:430–436.
513. Skrifvars MB, Pettita 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;59:319–328.
514. Baird TA, Parsons MW, Phan T, Butcher KS, Desmond PM, Tress BM, Colman PG, Chambers BR, Davis SM. Persistent poststroke hyperglycemia is independently associated with infarct expansion and worse clinical outcome. *Stroke*. 2003;34:2208–2214.
515. Capes SE, Hunt D, Malmberg K, Pathak P, Gerstein HC. Stress hyperglycemia and prognosis of stroke in nondiabetic and diabetic patients: a systematic overview. *Stroke*. 2001;32:2426–2432.
516. Scott JF, Robinson GM, French JM, O'Connell JE, Alberti KG, Gray CS. Glucose potassium insulin infusions in the treatment of acute stroke patients with mild to moderate hyperglycemia: the Glucose Insulin in Stroke Trial (GIST). *Stroke*. 1999;30:793–799.
517. Yip PK, He YY, Hsu CY, Garg N, Marangos P, Hogan EL. Effect of plasma glucose on infarct size in focal cerebral ischemia-reperfusion. *Neurology*. 1991;41:899–905.
518. Katz LM, Wang Y, Ebmeyer U, Radovsky A, Safar P. Glucose plus insulin infusion improves cerebral outcome after asphyxial cardiac arrest. *Neuroreport*. 1998;9:3363–3367.
519. Zahorec R. Rescue systemic thrombolysis during cardiopulmonary resuscitation. *Bratisl Lek Listy*. 2002;103:266–269.
520. Myerburg RJ, Briesse FW, Conde C, Mallon SM, Libersohn RR, Castellanos A Jr. Long-term antiarrhythmic therapy in survivors of pre-hospital cardiac arrest. Initial 18 months' experience. *JAMA*. 1977;238:2621–2624.
521. Somberg JC, Laux B, Wynn J, Keefe D, Miura DS. Lorcaïnide therapy in a cardiac arrest population. *Am Heart J*. 1986;111:648–653.
522. Peter T, Hamer A, Weiss D, Mandel WJ. Prognosis after sudden cardiac death without associated myocardial infarction: one year follow-up of empiric therapy with amiodarone. *Am Heart J*. 1984;107:209–213.
523. Moosvi AR, Goldstein S, VanderBrug Medendorp S, Landis JR, Wolfe RA, Leighton R, Ritter G, Vasu CM, Acheson A. Effect of empiric antiarrhythmic therapy in resuscitated out-of-hospital cardiac arrest victims with coronary artery disease. *Am J Cardiol*. 1990;65:1192–1197.
524. Haverkamp W, Eckardt L, Borggrefe M, Breithardt G. Drugs versus devices in controlling ventricular tachycardia, ventricular fibrillation, and recurrent cardiac arrest. *Am J Cardiol*. 1997;80:67G–73G.
525. Hallstrom AP, Cobb LA, Yu BH, Weaver WD, Fahrenbruch CE. An antiarrhythmic drug experience in 941 patients resuscitated from an initial cardiac arrest between 1970 and 1985. *Am J Cardiol*. 1991;68:1025–1031.
526. Randomized antiarrhythmic drug therapy in survivors of cardiac arrest (the CASCADE Study). The CASCADE Investigators. *Am J Cardiol*. 1993;72:280–287.
527. Wever EFD, Hauer RNW, Van Capelle FJL, Tijssen JGP, Crijns HJGM, Algra A, Wiesfeld ACP, Bakker PFA, Robles de Medina EO. Randomized study of implantable defibrillator as first-choice therapy versus conventional strategy in postinfarct sudden death survivors. *Circulation*. 1995;91:2195–2203.
528. A comparison of antiarrhythmic-drug therapy with implantable defibrillators in patients resuscitated from near-fatal ventricular arrhythmias. The Antiarrhythmics versus Implantable Defibrillators (AVID) Investigators. *N Engl J Med*. 1997;337:1576–1583.
529. Connolly SJ, Gent M, Roberts RS, Dorian P, Roy D, Sheldon RS, Mitchell LB, Green MS, Klein GJ, O'Brien B. Canadian implantable defibrillator study (CIDS): a randomized trial of the implantable cardioverter defibrillator against amiodarone. *Circulation*. 2000;101:1297–1302.
530. Kuck KH, Cappato R, Siebels J, Ruppel R. Randomized comparison of antiarrhythmic drug therapy with implantable defibrillators in patients resuscitated from cardiac arrest : the Cardiac Arrest Study Hamburg (CASH). *Circulation*. 2000;102:748–754.
531. Hennesdorf MG, Niebch V, Vester EG, Winter J, Perings C, Strauer BE. Long-term follow-up of sudden cardiac arrest survivors and electrophysiologically guided antiarrhythmic therapy. *Cardiology*. 2003;99:190–197.

532. Jorgensen EO. Course of neurological recovery and cerebral prognostic signs during cardio-pulmonary resuscitation. *Resuscitation*. 1997; 35:9–16.
533. Jorgensen EO, Malchow-Moller AM. Cerebral prognostic signs during cardiopulmonary resuscitation. *Resuscitation*. 1978;6:217–225.
534. Fabbri LP, Nucera M, Becucci A, Grippo A, Venneri F, Merciai V, Boncinelli S. An exceptional case of complete neurologic recovery after more than 5-h cardiac arrest. *Resuscitation*. 2001;48:175–180.
535. Steen-Hansen JE, Hansen NN, Vaagenes P, Schreiner B. Pupil size and light reactivity during cardiopulmonary resuscitation: a clinical study. *Crit Care Med*. 1988;16:69–70.
536. Van Hoeyweghen R, Mullie A, Bossaert L. Decision making to cease or to continue cardiopulmonary resuscitation (CPR). The Cerebral Resuscitation Study Group. *Resuscitation*. 1989;17 Suppl:S137–S147; discussion S199–S206.
537. Geppert A, Zorn G, Karth GD, Haumer M, Gwechenberger M, Koller-Strametz J, Heinz G, Huber K, Siostrzonek P. Soluble selectins and the systemic inflammatory response syndrome after successful cardiopulmonary resuscitation. *Crit Care Med*. 2000;28:2360–2365.
538. Prengel AW, Lindner KH, Ensinger H, Grunert A. Plasma catecholamine concentrations after successful resuscitation in patients. *Crit Care Med*. 1992;20:609–614.
539. Schreiber W, Herkner H, Koreny M, Bur A, Hirschl MM, Glogar D, Huber K, Lagner AN. Predictors of survival in unselected patients with acute myocardial infarction requiring continuous catecholamine support. *Resuscitation*. 2002;55:269–276.
540. Gando S, Igarashi M, Kameue T, Nanzaki S. Ionized hypocalcemia during out-of-hospital cardiac arrest and cardiopulmonary resuscitation is not due to binding by lactate. *Intensive Care Med*. 1997;23: 1245–1250.
541. Martin GB, Nowak RM, Cisek JE, Carden DL, Tomlanovich MC. Hyperkalemia during human cardiopulmonary resuscitation: incidence and ramifications. *J Emerg Med*. 1989;7:109–113.
542. Buylaert WA, Calle PA, Houbrechts HN. Serum electrolyte disturbances in the post-resuscitation period. *Resuscitation*. 1989;17:S189–S196.
543. Weil MH, Ruiz CE, Michaels S, Rackow EC. Acid-base determinants of survival after cardiopulmonary resuscitation. *Crit Care Med*. 1985;13: 888–892.
544. Zolotov AN, Shkunova LG, Dolgikh VT, Lukach VN, Girsh AO. [Effect of early post-resuscitation endotoxemia on the myocardial function]. *Anesteziol Reanimatol*. 2003;29–32.
545. Bahlmann L, Klaus S, Baumeier W, Schmucker P, Raedler C, Schmittinger CA, Wenzel V, Voelckel W, Lindner KH. Brain metabolism during cardiopulmonary resuscitation assessed with microdialysis. *Resuscitation*. 2003;59:255–260.
546. Korth U, Krieter H, Denz C, Janke C, Ellinger K, Bertsch T, Henn C, Klein J. Intestinal ischaemia during cardiac arrest and resuscitation: comparative analysis of extracellular metabolites by microdialysis. *Resuscitation*. 2003;58:209–217.
547. Bleske BE, Song J, Chow MS, Kluger J, White CM. Hematologic and chemical changes observed during and after cardiac arrest in a canine model—a pilot study. *Pharmacotherapy*. 2001;21:1187–1191.
548. Hoxworth JM, Xu K, Zhou Y, Lust WD, LaManna JC. Cerebral metabolic profile, selective neuron loss, and survival of acute and chronic hyperglycemic rats following cardiac arrest and resuscitation. *Brain Res*. 1999;821:467–479.
549. Eleff SM, Sugimoto H, Shaffner DH, Traystman RJ, Koehler RC. Acidemia and brain pH during prolonged cardiopulmonary resuscitation in dogs. *Stroke*. 1995;26:1028–1034.
550. Taylor MJ, Bailes JE, Elrifai AM, Shih SR, Teeple E, Leavitt ML, Baust JG, Maroon JC. A new solution for life without blood. Asanguineous low-flow perfusion of a whole-body perfusate during 3 hours of cardiac arrest and profound hypothermia. *Circulation*. 1995;91:431–444.
551. Sato S, Kimura T, Okubo N, Naganuma T, Tanaka M. End-tidal CO₂ and plasma lactate level: a comparison of their use as parameters for evaluating successful CPR. *Resuscitation*. 1993;26:133–139.
552. Cairns CB, Niemann JT, Pelikan PC, Sharma J. Ionized hypocalcemia during prolonged cardiac arrest and closed-chest CPR in a canine model. *Ann Emerg Med*. 1991;20:1178–1182.
553. Bender PR, Debehne DJ, Swart GL, Hall KN. Serum potassium concentration as a predictor of resuscitation outcome in hypothermic cardiac arrest. *Wilderness Environ Med*. 1995;6:273–282.
554. Niemann JT, Cairns CB. Hyperkalemia and ionized hypocalcemia during cardiac arrest and resuscitation: possible culprits for post-countershock arrhythmias? *Ann Emerg Med*. 1999;34:1–7.
555. Salerno DM, Murakami MM, Winston MD, Elsperger KJ. Postresuscitation electrolyte changes: role of arrhythmia and resuscitation efforts in their genesis. *Crit Care Med*. 1989;17:1181–1186.
556. Martin GB, Carden DL, Nowak RM, Foreback C, Tomlanovich MC. Hyperkalemia during cardiac arrest and resuscitation in the canine model. *Crit Care Med*. 1986;14:300–302.
557. 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;34:2881–2886.
558. Fogel W, Krieger D, Veith M, Adams HP, Hund E, Storch-Hagenlocher B, Buggle F, Mathias D, Hacke W. Serum neuron-specific enolase as early predictor of outcome after cardiac arrest. *Crit Care Med*. 1997; 25:1133–1138.
559. Mussack T, Biberthaler P, Kanz KG, Wiedemann E, Gippner-Steppert C, Mutschler W, Jochum M. Serum S-100B and interleukin-8 as predictive markers for comparative neurologic outcome analysis of patients after cardiac arrest and severe traumatic brain injury. *Crit Care Med*. 2002;30:2669–2674.
560. Mussack T, Biberthaler P, Kanz KG, Wiedemann E, Gippner-Steppert C, Jochum M. S-100b, sE-selectin, and sP-selectin for evaluation of hypoxic brain damage in patients after cardiopulmonary resuscitation: pilot study. *World J Surg*. 2001;25:539–543; discussion 544.
561. Rosen H, Karlsson JE, Rosengren L. CSF levels of neurofilament is a valuable predictor of long-term outcome after cardiac arrest. *J Neurol Sci*. 2004;221:19–24.
562. 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;29:473–477.
563. Meynaar IA, Straaten HM, van der Wetering J, Verlooy P, Slaats EH, Bosman RJ, van der Spoel JJ, Zandstra DF. Serum neuron-specific enolase predicts outcome in post-anoxic coma: a prospective cohort study. *Intensive Care Med*. 2003;29:189–195.
564. 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;49:183–191.
565. Schoerhuber W, Kittler H, Sterz F, Behringer W, Holzer M, Frossard M, Spitzauer S, Lagner AN. Time course of serum neuron-specific enolase. A predictor of neurological outcome in patients resuscitated from cardiac arrest. *Stroke*. 1999;30:1598–1603.
566. Bottiger BW, Mobes S, Glatzer R, Bauer H, Gries A, Bartsch P, Motsch J, Martin E. Astroglial protein S-100 is an early and sensitive marker of hypoxic brain damage and outcome after cardiac arrest in humans. *Circulation*. 2001;103:2694–2698.
567. Martens P, Raabe A, Johnsson P. Serum S-100 and neuron-specific enolase for prediction of regaining consciousness after global cerebral ischemia. *Stroke*. 1998;29:2363–2366.
568. Zingler VC, Krumm B, Bertsch T, Fassbender K, Pohlmann-Eden B. Early prediction of neurological outcome after cardiopulmonary resuscitation: a multimodal approach combining neurobiochemical and electrophysiological investigations may provide high prognostic certainty in patients after cardiac arrest. *Eur Neurol*. 2003;49:79–84.
569. 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;27:1661–1667.
570. Madl C, Kramer L, Domanovits H, Woolard RH, Gervais H, Gendo A, Eisenhuber E, Grimm G, Sterz F. Improved outcome prediction in unconscious cardiac arrest survivors with sensory evoked potentials compared with clinical assessment. *Crit Care Med*. 2000;28:721–726.
571. Madl C, Kramer L, Yeganehfar W, Eisenhuber E, Kranz A, Ratheiser K, Zauner C, Schneider B, Grimm G. Detection of nontraumatic comatose patients with no benefit of intensive care treatment by recording of sensory evoked potentials. *Arch Neurol*. 1996;53:512–516.
572. Madl C, Grimm G, Kramer L, Yeganehfar W, Sterz F, Schneider B, Kranz A, Schneeweiss B, Lenz K. Early prediction of individual outcome after cardiopulmonary resuscitation. *Lancet*. 1993;341: 855–858.
573. Logi F, Fischer C, Murri L, Mauguere F. The prognostic value of evoked responses from primary somatosensory and auditory cortex in comatose patients. *Clin Neurophysiol*. 2003;114:1615–1627.
574. Gendo A, Kramer L, Hafner M, Funk GC, Zauner C, Sterz F, Holzer M, Bauer E, Madl C. Time-dependency of sensory evoked potentials in comatose cardiac arrest survivors. *Intensive Care Med*. 2001;27: 1305–1311.

575. Nakabayashi M, Kurokawa A, Yamamoto Y. Immediate prediction of recovery of consciousness after cardiac arrest. *Intensive Care Med.* 2001;27:1210–1214.
576. Rothstein TL. The role of evoked potentials in anoxic-ischemic coma and severe brain trauma. *J Clin Neurophysiol.* 2000;17:486–497.
577. Sherman AL, Tirschwell DL, Micklesen PJ, Longstreth WT Jr, Robinson LR. Somatosensory potentials, CSF creatine kinase BB activity, and awakening after cardiac arrest. *Neurology.* 2000;54:889–894.
578. Bassetti C, Bomio F, Mathis J, Hess CW. Early prognosis in coma after cardiac arrest: a prospective clinical, electrophysiological, and biochemical study of 60 patients. *J Neurol Neurosurg Psychiatry.* 1996;61:610–615.
579. Ahmed I. Use of somatosensory evoked responses in the prediction of outcome from coma. *Clin Electroencephalogr.* 1988;19:78–86.
580. Chen R, Bolton CF, Young B. Prediction of outcome in patients with anoxic coma: a clinical and electrophysiologic study. *Crit Care Med.* 1996;24:672–678.
581. Berek K, Lechleitner P, Luef G, Felber S, Saltuari L, Schinnerl A, Traweger C, Dienstl F, Aichner F. Early determination of neurological outcome after prehospital cardiopulmonary resuscitation. *Stroke.* 1995;26:543–549.
582. Brunko E, Zegers de Beyl D. Prognostic value of early cortical somatosensory evoked potentials after resuscitation from cardiac arrest. *Electroencephalogr Clin Neurophysiol.* 1987;66:15–24.
583. Cheliout-Heraut F, Durand MC, Clair B, Gajdos P, Raphael JC. [Importance of evoked potentials in the evolutive prognosis of coma during cerebral anoxia in adults]. *Neurophysiol Clin.* 1992;22:269–280.
584. Kano T, Shimoda O, Morioka T, Yagishita Y, Hashiguchi A. Evaluation of the central nervous function in resuscitated comatose patients by multilevel evoked potentials. *Resuscitation.* 1992;23:235–248.
585. Rothstein TL, Thomas EM, Sumi SM. Predicting outcome in hypoxic-ischemic coma. A prospective clinical and electrophysiologic study. *Electroencephalogr Clin Neurophysiol.* 1991;79:101–107.
586. Walser H, Mattle H, Keller HM, Janzer R. Early cortical median nerve somatosensory evoked potentials. Prognostic value in anoxic coma. *Arch Neurol.* 1985;42:32–38.
587. 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;352:1808–1812.
588. Synek VM. Validity of a revised EEG coma scale for predicting survival in anoxic encephalopathy. *Clin Exp Neurol.* 1989;26:119–127.
589. Moller M, Holm B, Sindrup E, Nielsen BL. Electroencephalographic prediction of anoxic brain damage after resuscitation from cardiac arrest in patients with acute myocardial infarction. *Acta Med Scand.* 1978;203:31–37.
590. Scollo-Lavizzari G, Bassetti C. Prognostic value of EEG in post-anoxic coma after cardiac arrest. *Eur Neurol.* 1987;26:161–170.
591. Bassetti C, Karbowski K. [Prognostic value of electroencephalography in non-traumatic comas]. *Schweiz Med Wochenschr.* 1990;120:1425–1434.
592. Rothstein TL. Recovery from near death following cerebral anoxia: a case report demonstrating superiority of median somatosensory evoked potentials over EEG in predicting a favorable outcome after cardiopulmonary resuscitation. *Resuscitation.* 2004;60:335–341.
593. Berkhoff M, Donati F, Bassetti C. Postanoxic alpha (theta) coma: a reappraisal of its prognostic significance. *Clin Neurophysiol.* 2000;111:297–304.
594. Kaplan PW, Genoud D, Ho TW, Jallon P. Etiology, neurologic correlations, and prognosis in alpha coma. *Clin Neurophysiol.* 1999;110:205–213.
595. Yamashita S, Morinaga T, Ohgo S, Sakamoto T, Kaku N, Sugimoto S, Matsukura S. Prognostic value of electroencephalogram (EEG) in anoxic encephalopathy after cardiopulmonary resuscitation: relationship among anoxic period, EEG grading and outcome. *Intern Med.* 1995;34:71–76.
596. Ajisaka H. Early electroencephalographic findings in patients with anoxic encephalopathy after cardiopulmonary arrest and successful resuscitation. *J Clin Neurosci.* 2004;11:616–618.
597. Edgren E, Hedstrand U, Nordin M, Rydin E, Ronquist G. Prediction of outcome after cardiac arrest. *Crit Care Med.* 1987;15:820–825.
598. Sorensen K, Thomassen A, Wernberg M. Prognostic significance of alpha frequency EEG rhythm in coma after cardiac arrest. *J Neurol Neurosurg Psychiatry.* 1978;41:840–842.
599. Geocadin RG, Sherman DL, Christian Hansen H, Kimura T, Niedermeyer E, Thakor NV, Hanley DF. Neurological recovery by EEG bursting after resuscitation from cardiac arrest in rats. *Resuscitation.* 2002;55:193–200.
600. Geocadin RG, Muthuswamy J, Sherman DL, Thakor NV, Hanley DF. Early electrophysiological and histologic changes after global cerebral ischemia in rats. *Mov Disord.* 2000;15 Suppl 1:14–21.
601. Sherman DL, Brambrink AM, Ichord RN, Dasika VK, Koehler RC, Traystman RJ, Hanley DF, Thakor NV. Quantitative EEG during early recovery from hypoxic-ischemic injury in immature piglets: burst occurrence and duration. *Clin Electroencephalogr.* 1999;30:175–183.

Worksheets Cited

- W12B. <http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC19>
- W18. <http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC26>
- W28. <http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC42>
- W29. <http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC43>
- W32. <http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC49>
- W34. <http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC54>
- W35. <http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC55>
- W42A. <http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC64>
- W42B. <http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC65>
- W43A. <http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC66>
- W43B. <http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC67>
- W44A. <http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC68>
- W44B. <http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC69>
- W45. <http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC70>
- W46A. <http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC71>
- W46B. <http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC72>
- W47. <http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC73>
- W48A. <http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC74>
- W48B. <http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC75>
- W48C. <http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC76>
- W49A. <http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC77>
- W49B. <http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC78>
- W50. <http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC79>
- W51A. <http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC80>
- W51B. <http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC81>
- W54A. <http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC84>
- W54B. <http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC85>
- W55. <http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC86>
- W57. <http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC88>
- W58. <http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC89>

Part 4: Advanced Life Support

Circulation. 2005;112:III-25-III-54

doi: 10.1161/CIRCULATIONAHA.105.166474

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

Copyright © 2005 American Heart Association, Inc. All rights reserved.

Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the
World Wide Web at:

http://circ.ahajournals.org/content/112/22_suppl/III-25

Data Supplement (unedited) at:

http://circ.ahajournals.org/content/suppl/2005/11/28/112.22_suppl.III-25.DC1

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in *Circulation* can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the [Permissions and Rights Question and Answer](#) document.

Reprints: Information about reprints can be found online at:
<http://www.lww.com/reprints>

Subscriptions: Information about subscribing to *Circulation* is online at:
<http://circ.ahajournals.org/subscriptions/>