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Writing Group Member	Employment	Research Grant	Other Research Support	Speakers Bureau/ Honoraria	Ownership Interest	Consultant/Advisory Board	Other
Abella, Benjamin	University of Chicago Hospitals	Grantee, Laerdal Medical Corporation, Stavanger, Norway	None	None	None	None	None
Abu-Laban, Riyad	Vancouver General Hospital; University of British Columbia	Grant-in Aid Recipient to carry out a study of Aminophyiline in cardiac arrest (2000), Heart and Stroke Foundation of BC and Yukon; Unrestricted grant-in-aid recipient to carry out a study of tissue plasminogen activator (TPA) in cardiac arrest (1998), Hopfmann LaRouche Pharmaceuticals (Mississauga, Ontario, 1998)	Clinical scholar award (5 years) salary support (2000—2005), Michael Smith Foundation for Health Research (Vancouver, BC)	None	None	Member, Advisory Board, Heart and Stroke Foundation of BC and Yukon (Unpaid)	None
Adam, Rosemary	The University of Iowa Hospital	None	None	None	None	Volunteer committee member, NAENT, Mississippi	None
Alcedo, Mary	AHA Staff	None	None	None	None	None	None
Allen, Emilie	Parkland Health & Hospital System	None	None	None	None	Paid Clinical Consultant, NRCPR-DAI/TAI, Bel Air, MD	None
Allison, Jeanette	AHA Staff	None	None	None	None	None	None
Alvarez, Jesús A.	Plan Nacional De RCP, Spain	None	None	None	None	None	None
Arntz, Hans-Richard	Benjamin Franklin University Hospital, Berlin	None	Travel Reimbursement: Bristol Myers Squibb	Paid Speaker: Hoffmann La Roche, Boehringer Ingelheim, Merck-Sharp Dohme, Lilly (Germany)	None	None	None
Atkins, Dianne	University of Iowa	Research Grants: Philips Medical Systems, ZOLL Medical; Completed Research Grant: Wyeth Ayerst; Grantee, Philips Medical Systems, Seattle, WA: Grantee, ZOLL Medical, Boston, MA; Grantee, 200, Wyeth Ayerst	None	None	None	Former Consultant: Medtronic; Consultant, 2002, Medtronic-Physio Control, Seattle, WA	None
Aufderheide, Tom P.	Medical College of Wisconsin	None	Advanced Circulatory Systems, Inc. (Research Collaborator); NHLBI (Funded Investigator)	None	None	Medtronic-Physio Control (Paid Consultant); Zoll Medical (Consultant); Gemarquette Medical Systems, Inc. (Consultant); Medtronic Physio-Control—$13,000 Consultant Honoraria	Gifts, Gratuities and Entertainment: Medtronic Physio-Control

Aziz, Khalid	Memorial University of Newfoundland, St. John's NL, Canada	None	None	None	None	Other Financial or Business Interests: Canadian Paediatric Society (Board Member)	None
Azzopardi, Denis	Imperial College, London, England	None	None	None	None	None	None
Babbs, Charles	Purdue University	None	None	None	None	None	None
Bahr, Jan	University of Goettingen, Germany	None	None	None	None	None	None
Bancalari, Eduardo	University of Miami School of Medicine	None	None	None	None	None	None
Bar-Joseph, Gad	Ramban Medical Center, Halfa, Israel	None	None	None	None	None	None
Barnes, Thomas	Northeastern University	None	None	None	None	Paid Consultant, AMBU, Norway; Paid Consultant, Mercury Medical, Clearwater, FL; Paid Consultant, Laerdal Medical, Wappinger Falls, NY	None
Barsan, William	University of Michigan Health System	None	None	None	None	None	None
Baskett, Peter	Retired; Editor-in-Chief, Resuscitation	None	None	None	None	None	None
Bateman, Tim	AHA Staff	None	None	None	None	None	None
Baubin, Michael	University Hospital Innsbruck	None	None	None	None	Chair of the Austrian Resuscitation Council; Chair of the Austrian Society of Emergency & Disaster Medicine	None
Becker, Lance	University of Chicago	None	Fellowship Funds, Alsius Corp, Irvine, CA	None	None	Consultant/Research Funds, Philips Medical, Seattle, WA; Consultant, Abbott Labs, Abbot Park, IL; Consultant, DSMB/PRC NIH, Bethesda, MD	None
Beerman, Steve	Self-employed family physician	None	None	None	None	Chairperson: ILS Medical Committee, Belgium; Medical Advisor: Lifesaving Society, Ottawa, Canada	None
Beiser, David	University of Chicago	None	None	None	None	None	None
Bell, Mike	AHA Staff	None	None	None	None	None	None
Berg, Marc D.	University of Arizona	None	None	None	None	None	None

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Berg, Robert	University of Arizona	Research grant studying piglet defibrillation dosage (Fall 2004—Winter 2005)	Medtronic Emergency Response Systems, Inc., Redmond, WA	None	Several [stocks] in retirement and other fully managed stock programs but have no active management role. Funds include stocks of device manufacturers, pharmaceuticals, and hospital practice entities	None	None
Berlin, Paul	Gig Harbor Fire & Medic One	None	None	None	None	Volunteer Medical Coordinator for Ironman Championship World Triathlon; Volunteer Medical Team Leader for American Stroke Association Train to End Stroke—has solicited donations and/or loans of medical equipment and supplies from Medtronic and Abbott	None
Berringer, Ross	St. Pauls Hospital, Vancouver, BC, Canada	None	None	Speaker Sabex Pharmaceuticals	None	Medical Director: Vancouver Fire Dept; ALS Medical Advisor BC Ambulane Service	None
Bhutta, Adnan	University of Arkansas for Med. Science	None	None	None	None	Member: Society of Critical Care Medicine	None
Biarent, Dominique	Hopital Universitaire des Enfants Reine Fabiola	None	None	None	None	None	None
Billi, Jack	University of Michigan Medical School	None	None	None	None	Editorial Board: Resuscitation	None
Bingham, Bob	Great Ormond St. Hospital, London	None	None	None	None	None	None
Boettiger, Bernd	Dept. of Anesthesiology, University of Heidelberg, Germany	None	Reimbursement for meetings (Steering Committee) related to the TROICA trial by Boehringer Ingelheim International	None	None	None	None
Bonifer-Tiedt, Pat	American Red Cross Staff	None	None	None	None	None	None
Borgundvaag, Bjug	Self Employed Physician	None	None	Speaker: Roche, Aventis, BMS-Sanofi, Schering Canada	None	None	None
Bork, Sue	AHA Staff	None	None	None	None	None	None

Borys, Douglas J.	Scott and White Hospital, Central Poison Center	None	None	None	None	Officer-Past President, American Association of Poison Control Centers, Washington, DC	None
Bossaert, Leo	None	None	None	None	None	Executive Director, European Resuscitation Council	None
Boyle, David	Indiana University School of Medicine	None	None	None	None	None	None
Braner, Dana	OHSU	None	None	None	None	None	None
Braslow, Alan	Self-employed	None	None	None	None	None	None
Brooks, Steven	University of British Columbia	None	None	None	None	Member: Canadian Association of Emergency Physicians, Ottawa, Canada	None
Brown, Nancy	AHA Staff	None	None	None	None	Board Member, Boomer Revolution, Inc.	None
Brunet, Fabrice	St. Michael's Hospital, Toronto, ON, Canada	None	None	None	None	None	None
Bujol, Angelle	AHA Staff	None	None	None	None	None	None
Burchfield, David	University of Florida	None	None	None	None	None	None
Butler, Janet	Self employed; Tech Law, Inc.	None	None	None	None	None	None
Byrne, Steve	South Tees Hospitals NHS Trust	None	None	None	None	None	None
Cain, Rebecca	Virginia Commonwealth University Medical Center	None	None	None	None	None	None
Caissie, Rick	Canadian Red Cross	None	None	None	None	None	None
Callaway, Clifton	University of Pittsburgh School of Medicine	Received unrestricted research grants from Medtronic; Co-investigator on studies funded by unrestricted research grants from Laerdal	None	None	None	None	Co-inventor of patent licensed by the Univ of Pittsburgh to Medtronic; intellectual interest in quantitative analysis and ECG during VF; intellectual interest in hypothermia for neurological recovery after cardiac arrest; Licensor of patent, Medtronic Emergency Response Systems, Seattle, WA

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Cantor, Warren	St. Michael's Hospital, Toronto, Canada	Honoria and/or research grants: Hoffman LaRoche, Eli Lilly, Merck Frosst, Schering Canada, Sanofi, Bristol Myers Squibb, Medtronic, Guidant Canada	None	None	None	None	None
Caracci, Barbara	National Safety Council	None	None	None	None	Director of Product Development & Training, Emergency Care Programs, National Safety Council, Itasca, IL	The National Safety Council will use the information from the consensus on science in its emergency care programs
Carli, Pierre	Paris L91V University, APHP Hospital	None	None	Chairman, CF RC, Paris, France (French Resuscitation Council, related to ERC)	None	None	None
Carlo, Waldemar	University of Alabama at Bingham	None	None	None	None	Director, Pediatrix Medical Board; Medical Board Director, Pailos, Paradigm	None
Carman, Deborah	University of Arkansas	None	None	None	None	None	None
Carrera, Renato	Santa Casa School of Medicine, Sao Paulo, Brazil	None	None	None	None	None	None
Carrington, Alan	American Heart Association	None	None	None	None	None	None
Cass, Dan	St. Michael's Hospital, Toronto, Canada	Wyeth-Ayerst (Pennsylvania) — Received grant for ''ALIVE Trial'' — clinical trial comparing Lidocaine and Amiodarone in out-of-hospital cardiac arrest. Grant covered operating expenses of study and study medications; no personal financial support/compensation received	None	Aentis Pharma Canada (Laval, Quebec)—received honourarium for lecture on DVT/Pulmonary Embolism; no input by company into content of talk. Roche Pharmaceuticals Canada (Mississauga, Ontario)—received honourarium for lecture on DVT/Pulmonary Embolism; no input by company into content of talk. Biovall—received honouraria for various lectures/presentations; no input by company into content of talk. Also appeared in video explaining to clinicians technically how to use r-PA (i.e., how to mix drug, etc.)	None	None	None

Cassan, Pascal	French Red Cross	None	None	None	None	None	None
Cave, Diana	Portland Community College-Institute for Health Professionals, Portland, OR	None	None	None	None	None	None
Celenza, Tony	University of Western Australia	None	Travel Reimbursement: American Heart Association	Paid Speaker: Divisions of General Practice, Western Australia	Director and Shareholder: Medical Emergency Solutions	None	None
Chameides, Leon	Retired; Paid Editor/Writer 2005 AHA ECC Guidelines	None	None	None	None	None	None
Cheng, Ivy	Sunnybrook and Women's College Hospital	None	None	None	None	None	None
Christenson, James	Self-employed, University of British Columbia	None	Research support, Medronic Physio; Research support, Revivant (Zoll)	Speaker, Hoffman La Roche Canada	None	None	None
Chunsheng, Li	Beijing Chaoyuan Hospital	None	None	None	None	None	None
Clendenen, William	Medic First Aid International, Inc.	None	None	None	President/Co-owner, Medic First Aid; Oxygen training programs and oxygen equipment sales account for less than 1% of Medic First Aid International's sales	None	None
Cobb, Leonard	University of Washington	None	None	None	None	None	None
Coleman, Sheri	Coleman Education Opportunities, Inc., an AHA Approved Training Center	None	None	None	100 shares Abbott Laboratories; 700 shares Amgen; 100 shares Baxter International, Inc.; 100 shares ICOS; 48 shares MedcoHealth Solutions; 400 shares MedcoHealth Solutions; 401 shares Merck & Co., Inc.; 575 shares Pfizer, Inc.; 50 shares WebMedCorp	None	None

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Cone, David	Yale University	None	None	None	None	The National Association of EMS Physicians	None
Conner, Scott	American Red Cross Staff	None	None	None	None	None	None
Cooper, Arthur	Columbia University	None	None	None	None	None	None
Coovadia, Ashraf	University of Witwatersrand, Johannesburg	None	None	None	None	Consultant: LifeWorks (an HIV disease management company)	None
Costa, Fernando	American Heart Association	None	None	None	None	None	None
Cram, Peter	University of Iowa College of Medicine	None	None	None	None	None	None
Cretikos, Michelle	Self-employed	None	PhD Candidate, Simpson Centre for Health Services Research, Liverpool, BC NSW and University of New South Wales, Sydney, Australia	None	None	None	None
Crites, Ted	American Red Cross Staff	None	None	None	None	None	None
Crocco, Todd	West Virginia University	Grantee: National Institute of Health	None	None	None	None	None
Crosby, Edward	Self-employed physi-cian/Anesthesiologist	None	None	None	None	None	None
Cummins, Richard	None	None	None	None	None	None	None
Davies, Sian	The British Heart Foundation seconded to Department of Health, UK	None	None	None	None	Member, ERC Executive & Resuscitation Council (UK)	None
Day, Craig	AHA Staff	None	None	None	None	None	None
de Caen, Allan	Self-employed	None	None	None	None	None	None
Deakin, Charles	Southhampton University Hospital, UK	None	None	None	None	None	None
Delbridge, Ted	University of Pittsburgh	None	None	None	None	None	None
Dennett, Jenny	Central Gippsland Health Service, Sale Victoria, Australia'	None	None	None	None	Chair, BLS Committee, Australian Resuscitation Council, Melbourne, Victoria, Australia; Chair — ALS Committee, Australian College of Critical Care Nurses, Melbourne, Victoria, Australia	None
DePiero, Andrew	AI du Pont Hospital for Children	None	None	None	None	None	None

Diekema, Douglas	Children's University Medical Group, Seattle WA	None	None	None	Stockholder: Microsoft	Member: American Academy of Pediatrics; Consulting Editor: American Academy of Pediatrics;	None
Dirks, Burkhard	University Clinic Ulm, Germany	None	None	None	None	Consultant, Government, State of Baden-Wurttemberg	None
Doerges, Volker	University Hospital Schleswig-Holstein, Kiel, Germany	None	None	None	None	None	None
Domanovits, Hans	Medical University of Vienna, Austria	None	None	None	None	None	None
Drajer, Saul	None	None	Travel expenses: Medtronic	None	None	None	None
Drummonds, Brenda		None	None	None	None	None	None
Eich, Christoph	Department of Anaesthesiology, Rescure and Intensive Care Medicine, Georg-August-University, Goettingen, Germany	None	None	None	None	European Resuscitation Council (ERC) Antwerp, Belgium; Clinical Fellow at Royal Hospital for Sick Children, Edinburgh, Scotland	None
Eigel, Brian	AHA Staff	None	None	None	700 shares Millennium Pharmaceuticals	None	None
Ekström, Lars	Sahlgren's University Hospital, Gothenburg, Germany	None	None	None	None	None	None
Elling, Bob	Albany Medical Center; Paid Editor/Writer for AHA ECC Guidelines	None	None	None	Senior Associate and co-owner: High Quality Endeavors, LTD	None	None
Engelbaugh, Susan L.	St. Mary's Hospital Medical Center	None	None	None	None	None	None
Epstein, Jonathan	NorthEast Emergency Medical Services, Inc.	None	None	None	None	Member: American Red Cross Advisory Council on First Aid and Safety	None
Escalante, Raffo	EMS Training, Peru	None	None	None	None	None	None
Escobedo, Marilyn	University of Oklahoma College of Medicine	None	None	None	None	None	None
Espino, Mike	American Red Cross Staff	None	None	None	None	None	None
Ewy, Gordon	University of Arizona	None	None	None	None	None	None
Fabunmi, Rosalind	AHA Staff	None	None	None	None	None	None

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Falcucci, Octavio	Virginia Commonwealth University Department of Anesthesiology	None	VCU PI for the Eclipse Study, the use of Clevidipine in Cardiac Surgery, The Medicines Company, Parsippany, NJ	None	None	None	None
Fallat, Mary	University of Louisville	None	None	None	None	Member, Board of Directors: Kentucky Organ Donor Affiliates; Member: Kentucky Board of Emergency Medical Services	None
Fendya, Diana	Emergency Medical Servics for Children National Resource Center	None	None	None	None	Board Member, Traumatec Inc, San Antonio, TX	None
Fenici, Peter	Catholic University Hospital of Sacred Heart—Rome, Italy	None	None	None	None	None	None
Fiedor, Melinda	Children's Hospital of Pittsburgh	None	None	None	None	None	None
Field, John	Penn State University College of Medicine	None	None	None	None	None	None
Figliola, Robin	AHA Staff	None	None	None	None	None	None
Finn, Judith	University of Western Australia	None	None	None	None	None	None
Fiser, Tad	University of Arkansas for Medical Sciences	None	None	None	None	None	None
Fringer, Ryan	William Bennet Hospital	None	None	None	None	None	None
Fuchs, Susan	Pediatric Faculty Foundation, Children's Memorial Hospital	None	None	None	None	American Academy of Pediatrics: PALS reviewer, Editor APLS manual, member PEPP steering committee	None
Gabrielli, Andrea	University of Florida College of Medicine	None	None	None	None	None	None
Gagnon, Cathy	Mt. San Antonio College; Pasadena Fire Dept.	None	None	None	None	None	None
Garcia Fernandez, Jose Antonio	Emergency System of Asturias, Spain (SESPA)	None	None	None	None	None	None
Gauthier, Naomi	Seacoast Children's Cardiology	None	None	None	None	None	Founder/Chair, Camp Meridian (non-profit camp for children with heart disease), Newington, NH

Gay, Marc	Corporation d'Urgences—Sautê	None	None	None	None	None	None
Gazmuri, Raul	North Chicago VA Medical Center; Rosalind Franklin University of Medicine and Science	Research Grant: Aventis Pharma; Grantee: NHLBI	None	None	None	None	None
Gerardi, Mike	Emergency Medical Associates	None	None	None	None	Director, Pediatric Emergency Medicine at Atlantic Health System and Morristown Memorial Hospital, Morristown, NJ	None
Gervais, Hendrik	Johannes Gutenburg—University Mainz, Germany	None	None	None	None	Member: European Resuscitation Council	None
Gilmore, David	Colorado Permanente Medical Group	None	None	None	None	Focused Medical Information, Denver, CO	None
Giovinazzi, Cathy	South Jersey Healthcare	None	None	Speaker/Consultant, Eli Lilly, Novo Nordisk; Speaker/Pump trainer, Medtronic Minmed	None	None	None
Goldsmith, Jay	Ochsner Clinical Foundation, New Orleans, LA	None	None	None	None	Medical Advisory Board, Discovery Labs, Doylestown, PA	None
Gonzales, Louis	Williamson County EMS, Temple College	None	None	None	None	None	None
Goodell, Heather	AHA Staff	None	None	None	None	None	None
Gope, Monica	Royal Perth Hospital, Perth Western Australia	None	None	None	None	None	None
Gordon, Donald	University of Texas Health Science Center at San Antonio	None	None	None	None	None	None
Goyal, Munish	University of Pennsylvania	None	None	None	None	None	None
Guinsburg, Ruth	Federal University of Sao Paulo	None	None	None	Translation Coordinator: Sao Paulo owns copyright of the Portugues Language Translation of the textbook of Neonatal Resuscitation—no financial gain	Director: Brazilian Neonatal Resuscitation Program; Editor: Revista, Pediatric Society of Sao Paulo	None

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Gunnerson, Kyle	Virginia Commonwealth University Medical Center	None	None	None	None	None	None
Haag, Jo	AHA Staff	None	None	None	None	None	None
Halamek, Louis	Stanford University	None	None	None	None	None	None
Hallstrom, Al	University of Washington	Grantee: Revivant Corporation; Grantee/Consultant: St. Jude Medical	None	None	None	None	None
Halperin, Henry	Johns Hopkins University	None	CPR device research	None	Shareholder, Revivant, Zoll	Consultant, Revivant, Medtori, Phillips, Abbott Labs	None
Halverson, Colleen	AHA Staff	None	None	None	None	None	None
Hammill, William	University of Virginia	None	Partner: IEC Inc.—contract with Cardioconcepts to develop web-based BLS products for healthcare providers, Cardioconcepts has partnered with the AHA to develop this product line	None	None	None	None
Handley, Anthony	Essex Rivers Healthcare Trust	None	None	None	None	Part-time consultant, Laerdal Sophus, Copenhagen; Executive member, Resuscitation Council, London	None
Harper, Richard	OHSU/Portland VANC	None	None	None	None	None	None
Hatanaka, Tetsuo	Emergency Life-Saving Technique Academy	None	None	None	None	None	None
Hatch, George	Houston Community College System	None	None	None	None	Customer Advisory Panel: Laerdal Medical Corporation	None
Hayes, Gabrielle	AHA Staff	None	None	None	None	None	None
Hazinski, Mary Fran	Vanderbilt Children's Hospital	Served as uncompensated principle investigator for Medtronic Phipio Control sponsored study of AEDS in children (1996—99, published in 2003)	None	None	None	Senior Science Editor, American Heart Association; consultation fees for AHA ECC programs	None
Hemphill, Robin	Vanderbilt University; paid editor/writer for AHA ECC Guidelines	None	None	None	None	None	None
Herlitz, Johan	Sahlgrenska University Hospital	None	None	None	None	None	None

Herrington, Rita Ann	Bloomington Hospital	None	None	None	None	None	None
Hickey, Robert	University of Pittsburgh	None	None	None	None	None	None
Holstege, Christopher	University of Virginia	None	None	None	None	None	None
Hong, Shen	General Hospital of PLA, Beijing, China	None	None	None	None	None	None
Hultman, Johan	AHA Staff	None	None	None	None	None	None
Hunt, Cheryl	AHA Staff	None	None	None	None	None	None
Hunter-Wilson, Lynn	American Heart Association	None	None	None	None	None	None
Hwang, Sung Oh	Wonju College of Medicine, Yonsei University, South Korea	None	None	None	None	Board Member: Korean Association of CPR	None
Idris, Ahamed	University of Texas Southwestern Medical Center	Research Grants: Medtronic, Laerdal, Advanced Circulatory Systems, National Institutes of Health, Department of Defense; Unpaid Consultant: Philips Science Advisory Board	None	None	None	None	None
Innes, Grant	Providence Health Care	None	None	Speaker Honorarium: Hoffman LaRoche Canada Pharmaceuticals (~$1000)	None	None	None
Jacobs, Alice	Boston University	None	None	None	None	None	Husband is Project Director Wyeth, Collegeville, PA; Consultant with MyoMend, Natick, MA
Jacobs, Ian	University of Western Australia	Research grant, National Health and Medical	Untied research support, Laerdal Australia; Research support—C.A. registry, St. John Ambulance	None	None	Chairman, Australian Resuscitation Council	None
Jacobs, Mike	San Francisco Paramedica Association	None	None	None	None	None	None
Jauch, Edward	University of Cincinnati College of Medicine	None	None	None	None	Consultant and grant recipient, Biosite, Inc.; Consultant: Johnson & Johnson, NovoNordisk, AstraZeneca, Boeringer Ingelheim	None

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Jobe, Alan	Cincinnati Children's	Grantee: Fisher & Paykel, grant to collaborator	None	None	None	Chair: NICHD Neonatal Research Network	Grant Reviewer: March of Dimes; Organize Fellows Teaching Course: Mead Johnson Nutritionals
Jude, James	Retired	None	None	None	None	None	None
Jun, Kathleen	Self-employed	None	None	None	None	Consultant: AHA, Citizen CPR Foundation	None
June, Holly	AHA Staff	None	None	None	None	None	None
Kattwinkel, John	University of Virginia	None	None	None	None	None	None
Kaye, William	Retired	None	None	None	None	Clinical Consultant, AHA NRCPR	None
Keenan, William	St. Louis University	None	None	None	None	None	None
Kerber, Richard	University of Iowa Hospital	Research Grants: Philips Medical Systems, Laerdal Foundation, NHLBI	None	None	Stock ownership: Winston Pharmaceuticals	Consultant: Guidant	None
Kern, Karl	University of Arizona	None	None	None	None	Scientific Advisory Board (SAB), Medtronic Emergency Response Systems; Scientific Advisory Board (SAB), Revivant Inc.	None
Kette, Fulvio	San Vito Al Tagliamento Hospital, Italy	None	None	None	None	None	None
Key, Timothy J.	Key Occupational Health Solutions	None	None	None	None	None	None
Kissoon, Niranjan	University of British Columbia	None	None	None	None	None	None
Kleinman, Monica	Children's Hospital Anesthesia Foundation	None	None	None	None	Progeria Research Foundation; Member, Board of Directors, Medical Research Foundation. Section on Transport Medicine, American Academy of Pediatrics, Elk Grove, IL. Member, Executive Committee; Associate Editor, Guidelines for Air and Ground Transport of the Neonatal and Pediatric Patient, 3rd edition. Boston MedFlight; Member, Board of Directors. Society of Critical Care Medicine; Member. Zoll Medical; Non-paid consultant	None

Kloeck, Walter	Academy of Advanced Life Support	None	None	None	None	Chairman: Resuscitation Council of Southern Africa	Distributor of AHA ECC Training Materials
Koster, Rudi	Academic Medical Center, Amsterdam, the Netherlands	Medtronic: recipient of research grants	Medtronic: recipient of material support, travel reimbursement, paid speaker	Medtronic: paid speaker	None	Medtronic: Paid consultant	None
Kronick, Steven	University of Michigan	None	None	None	None	None	None
Kudenchuk, Peter	University of Washington	None	None	None	None	On Speakers Bureau of Medtronic Corp, but pertaining to implantable devices	None
Kupas, Douglas F.	Geisinger Health System, Danville PA; Pennsylvania Department of Health	None	Receive and accept dinners from pharmaceutical companies in relation to journal clubs, but no payments or other gifts accepted	Speaker: Journal of Emergency Medical Services	None	Employee as Commonwealth Emergency Medical Director, Pennsylvania Dept. of Health, Harrisburg, PA	None
Laerdal, Tore	Laerdal Medical and Laerdal Foundation	None	None	None	Chairman, Laerdal Medical and Laerdal Foundation	None	None
Larkin, Gregory	University of Texas Southwestern Medical Center	None	None	None	None	None	None
Larsen, Peter	University of Otago, New Zealand	Research Grant: Wellington Medical Research, Letterea Health	Travel reimbursement: New Zealand Resuscitation Council, Medtronic, Physiological Society of New Zealand	None	None	None	None
Lee, William	AHA Staff	None	None	None	None	Consultant: Training Consulting Softek	None
Lendrum, David	University of Toronto	None	None	None	None	None	None
Lerner, Brooke	University of Rochester	None	None	None	Stockholder: General Electric, Bank of America, British Petroleum	Board of Directors and Consultant: National Association of EMS Physicians; Consultant: Erie County Health Dept.	None
Lexow, Kristian	Stavanger University Hospital, Norway	None	None	Lecturer: University of Bergen	None	Medical Director: Norwegian Air Ambulance	None
Lim, Swee Han	Singapore General Hospital	None	None	None	None	None	None

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Lindsberg, Perttu	Helsinki University Central Hospital	None	None	Paid speaker: Boehringer Ingelheim Pharmaceuticals	None	Board Member: Neurology Foundation	None
Link, Mark	Tufts-New England Medical Center	Grant Support: Guidant, Medtronic, St. Jude	None	Paid speaker: Guidant	None	None	None
Lippert, Freddy	None	None	None	None	None	None	None
Lockwood, Ron-Dee	Newton Memorial Hospital	Grants to NMF for AED Program, Medtronic Foundation; Grant to NMHF for Heartsafe Schools Program	None	None	PNC Bank Foundation; Stockholder, G.E., Pfizer; Stockholder SBC Communications; Stockholder, Selective Insurance; Stockholder, Verizon, Vodafone Airtouch; Stockholder, Eastman Kodak; Stockholder, Southern; Stockholder Citigroup; Stockholder Ford; Stockholder Exxon; Stokholder Visteon; Stockholder Avaya; Stockholder Ageresystms	Consultant, Newton Memorial Hospital Foundation, Newton, New Jersey; Local Volunteer Diseaster Preparedness, American Red Cross, Newton, NJ	None
Loftus, Bob	Paid editor/writer AHA ECC Guidelines	None	None	None	None	None	None
Long, Gayle	University of Colorado Health Sciences Center	None	None	None	None	Attending emergency physician, Colorado Permanente Medical Group, Denver, CO; Attending emergency physician, Centura Granby Medical Center, Granby, CO	None
Lu, Yiming	Rui Jin Hospital, Shanghai Second Medical University	None	None	None	None	None	None

Lukins, Jane	Box Hill Hospital, Victoria, Australia	None	None	None	None	None	Husband is employed as Staff Anaesthetist at Monash Medical Centre, Clayton Road, Clayton, Victoria Australia (not for profit, governmental, public hospital)
Lurie, Keith	CentraCare Clinic, Minnesota	None	None	None	None	Chief Medical Officer and Chairman of the Board: Advanced Circulatory Systems	None
Ma, Matthew	National Taiwan University Hospital	None	None	None	None	None	None
MacDonald, Russell	University of Toronto/Sunnybrook and Women's College Health Sciences Centre, Toronto, Ontario, Canada	None	None	None	None	None	None
Madden, Marc	American Red Cross Staff	None	None	None	None	None	None
Mallory, Julie	AHA Staff	None	None	None	None	None	None
Mancini, Beth	University of Texas, Arlington	None	None	Paid speaker: Zoll	None	Consultant: Medtronic	None
Mann, Clay	University of Utah School of Medicine	None	None	None	None	None	None
Markenson, David	Columbia University	None	None	None	None	None	None
Markus, Marcello Ricardo Paulista	Heart Institute (InCor), University of São Paulo Medical School, São Paulo, Brazil	None	None	None	None	None	None
Mason, Pip	Self-employed; New Zealand Resuscitation Council	None	None	None	None	None	None
Massey, Donna	Emergency Nurses Association	None	None	None	None	None	None
Matos, Giuliana	EMS Training, Peru	None	None	None	None	None	None
Mattes, Mark	Clarian Health Partners, Inc.					Assistant Chief, Sugar Creek TWA Fire Department, New Palestine, IN	
Mazurik, Laurie	Self-employed	None	None	None	None	None	None
McConnell, Bill	Team Health	None	None	None	None	None	None
McCune, Robin	AHA Staff	None	None	None	None	None	None
McDonald, Ed	None	None	None	None	None	None	None
McGowan, Jane	The Johns Hopkins University School of Medicine	None	None	None	None	Consultant (Grant Reviewer) NIH, Bethesda, MD	None

Writing Group Member	Employment	Research Grant	Other Research Support	Speakers Bureau/ Honoraria	Ownership Interest	Consultant/Advisory Board	Other
McMillian, Adreania	American Red Cross Staff	None	None	None	None	None	None
McNeely, Mary Ann	AHA Staff	None	None	None	None	None	None
McRae, Tagni	AHA Staff	None	None	None	None	None	None
Mears, Greg	University of North Carolina-Chapel Hill, NC	None	Funding for the development of the National EMS Information System, American Heart Association and National Highway Traffic Safety	None	None	None	None
Merchant, Raina	University of Chicago	None	None	None	None	None	None
Molsberry, Dianne	Sacred Heart Children's Hospital, Spokane, Washington	None	None	On-call Peds Educator, Empire Health Services, Spokane, WA	None	Pediatric Consultant, Hospice of Spokane; Pediatric Educator, Inland Empire EMS, Spokane	Spouse private dental practice, Family Dental Care
Monsieurs, Koen	Ghent University Hospital, Belgium	None	None	Honorary Secretary: European Resuscitation Council	None	None	None
Montgomery, William H.	Straub Clinic and Hospital	None	None	None	None	None	None
Morley, Colin	Royal Women's Hospital, Victoria, Australia	None	None	None	None	Consultant: Britannia Pharmaceuticals UK	None
Morley, Peter	Melbourne Health	None	None	None	None	Deputy Chair, Australian Resuscitation Council; Evidence worksheet expert, American Heart Association	None
Morrison, Laurie	University of Toronto, Sunnybrook & Women's University, Academic Health Science Center	Grantee, Zoll Medical, Boston, MA; Grantee, Advantis; Grantee, Hoffman La Roche, Toronto, CA	None	None	None	None	None
Mosesso, Vince	University of Pittsburgh	Research grantee, Revivant Corp (owned by Zoll)	None	None	None	Medical Director, National Center for Early Defibrillation;	None
Mount, Charles	Boakey and Associates Management Consultants	None	None	None	None	None	None
Mouw, John	Baptist Health of South Florida	None	None	None	None	None	None

Mullins, Charles	AHA Staff	None	None	None	None	None	Father is a Professor, Baylor College of Medicine, Houston, TX; At the present time he has no relationship to AHA Emergency Cardiovascular Care studies, program or grants. As a practicing physician, he takes PALS courses
Nadkarni, Vinay	University of Pennsylvania School of Medicine; Children's Anesthesiology Assoc, Division Critical Care	Research Grants: NIH/NICHD; Ross/Abbott; Sensormedics; Drager Medical	None	None	None	Unpaid education consultant: Laerdal, Medical Education Technologies, Inc.	None
Neish, Steve	Baylor College of Medicine	None	None	None	None	Member, American Academy of Pediatrics; Member American College of Cardiology	None
Nesbitt, Lisa	Ottawa Health Research Institute	None	Coordinates BIPHASIC I study funded by Medtronic	None	None	None	None
Nichol, Graham	University of Washington	None	Sponsor, Investigational Device Exemption; Wearable Cardioverter Defibrillator trial; RAFT Trial investigator (CIHR, Medtronic Inc.); Sponsor, Wenabk Defibrillator IDF	None	None	None	None
Niermeyer, Susan	University of Colorado	None	None	None	None	Volunteer, American Academy of Pediatrics, Elk Grove Village, IL; Member of Speakers'Bureau, Johnson & Johnson Pediatric Institute, New Brunswick, NJ	None
Nolan, Jerry	Royal United Hospital NHS Trust, Bath, UK	None	None	None	None	None	None
Nonogi, Hiroshi	National Cardiovascular Center, Japan	None	None	None	None	None	None
O'Connor, Robert	Christiana Care Health Systems	None	Research funding from AED manufacturers, Astra-Zeneca, McNeil, Pfizer; No salary support	None	None	President, National Association EMS Physicians; Chair, ACEP EMS Committee; Board, National Registry of EMTs	None

Writing Group Member	Employment	Research Grant	Other Research Support	Speakers Bureau/ Honoraria	Ownership Interest	Consultant/Advisory Board	Other
O'Donnell, Cathal	Sunnybrook and Women's College Health Sciences Center, Toronto, Canada	None	None	None	None	None	None
Okada, Kazuo	Japan Resuscitation Council; Teikyo University Hospital	None	None	None	None	None	None
Okamoto, Deems	Ballard Emergency Physician	None	None	None	None	Regional Faculty: Cascade Training Center, Children's Training Center; Volunteer Committee member, Center for Excellence in Education, Chico, CA	None
O'Keefe, Michael	Vermont Dept of Health	None	Advisor: AHA Research Study on ACLS ($300); Author: Brady/Prentice Hal Publishing	None	None	None	None
Okreglicki, Andrzej	Provincial Administration of the Western Cape, South Africa	None	None	None	None	None	None
Ornato, Joe	Virginia Commonwealth University Medical Center	None	None	None	None	Operational Medical Director: Richmond Ambulance Authority; Operational Medical Director: Hanover County EMS; Science Advisory Board: Meridian Medical; Science Advisory Board: Revivant (now owned by Zoll); Science Advisory Board: National Registry of Myocardial Infarction (sponsored by Genetech); Chairman Data Safety Monitor Board overseeing TROICA study (funded by Boehringer Medical)	None
Otto, Charles W.	University of Arizona College of Medicine	None	None	None	None	Vice President: American Society of Anesthesiologists; Director: Foundation for Anesthesia Education and Research	None

Paiva, Edison	University of Sao Paulo School of Medicine	None	None	None	None	None	As of 3 Jan 2005, Medical Manager for enoxaparin and teicoplanin for Sanofi-Aventis, Brazil
Paradis, Norman	University of Colorado Health Sciences Center	Grantee, Phillips Corp.	None	None	None	NIH-SBIR Rose Biomedical, Revivant/Zoll, Medivance Corp., Council of Healthcare Advisors—consultant. Med Simulation Systems, Cambridge University Press—Editor	None
Parker, Emilie	American Red Cross Staff	None	None	None	None	None	None
Peberdy, Mary Ann	VA Commonwealth University Health System	None	None	None	None	None	Joe Ornato spouse, his COIs are: Operational Medical Director: Richmond Ambulance Authority; Operational Medical Director: Hanover County EMS; Science Advisory Board: Meridian Medical; Science Advisory Board: Revivant (now owned by Zoll); Science Advisory Board: National Registry of Myocardial Infarction (sponsored by Genetech); Chairman Data Safety Monitor Board overseeing TROICA study (funded by Boehringer Medical)
Peerbaye, Yousouf	University of Toronto	None	None	None	None	None	None
Perkins, Gavin	University of Birmingham, UK	Royal College of Physicians and Resuscitation Council (UK). Grant recipient + travel expenses	None	None	None	Member BLS Committee, European Resuscitation Council, Belgium; Member ALS, ILS Committee, Resuscitation Council (UK); Intensive Care Society (UK)	None
Perlman, Jeffery	Weill-Cornell Medical Center, New York	None	None	None	None	None	None

Writing Group Member	Employment	Research Grant	Other Research Support	Speakers Bureau/ Honoraria	Ownership Interest	Consultant/Advisory Board	Other
Poirier, Pierre	Ottawa Paramedic Service, Ottawa, Ontario, Canada	None	None	None	None	Executive Director, Paramedic Association of Canada	None
Potts, Jerry	AHA Staff	None	None	None	None	None	None
Proctor, Lester	University of Wisconsin Medical School	None	None	None	None	None	None
Prudhomme, Stephen	AHA Staff	None	None	None	None	None	None
Quan, Linda	Children's University Medical Group, Seattle WA	None	Medtronics/Physiocontrol Inc—Redmond, WA—subcontracted to conduct study of CPR/AED training of high school students	None	None	None	Group Health Coop—spouse is a physician
Racht, Edward	City of Austin, TX, Medical Director, EMS System	None	None	None	None	VidaCare Corp., Advisory Board	None
Ralston, Mark	U.S. Navy, Naval Hospital, Oak Harbor, WA; Mary Bridge Childrens Hospital, Tacoma, WA	None	None	None	None	None	None
Raynovich, Bill	Creighton University	None	None	None	None	None	None
Reeves, Jay	U.S. Army	None	None	None	None	None	None
Reis, Amelia	University of Sao Paulo School of Medicine, Brazil	None	None	None	None	None	None
Richmond, Sam	City Hospitals, Sunderland NHS Trust	None	None	None	None	Chairman: Newborn Life Support, Resuscitation Council (UK); Editor: Newborn Life Support Provider Course Material (no royalties or financial gain)	None
Robinson, Kara	AHA Staff	None	None	None	None	None	None
Robles, Luis	Sunnybrook Hospital, Toronto, Canada	None	None	None	None	None	None
Rodgers, Dave	Charleston Area Medical Center Health Education and Research Institute, Charleston, WV	None	Employer is a distributor for Zoll AEDs	None	None	None	None
Rodriguez-Nunez, Antonio	Santiago de Compostela University Hospital, Spain	None	None	None	None	None	None
Roppolo, Lynn	University of Texas Southwestern Medical Center	None	None	None	None	None	None
Rubertsson, Sten	Uppsala University, Sweden	None	None	None	None	None	None

Russo, Sebastian	University Hospital, Gottinger, Germany	None	None	None	None	None	None
Samson, Ricardo	University of Arizona	None	None	None	None	None	None
Sanders, Arthur	University of Arizona	None	None	None	None	None	None
Saver, Jeffery	University of California at Los Angeles, California	Grantee: NIH				Scientific Advisory Board: Alsius Corp.; Stroke Prevention Scientific Advisory Board and Research Support: Boehringer Ingelheim; Scientific Consultant and Research Support: AGA Medical; Scientific Advisory Board and Speaker Honoraria: Bristol Myers Squibb; Scientific Advisory Board and Speaker Honoraria: Sanofi; Scientific Consultant: Biosite; Scientific Consultant: CoAxia; Research Material Support: Concentric Medical; Vice-Chair, Acute Advisory Board: National Stroke Association; Member, Professional Advisory Council: National Stroke Association	
Sayre, Michael	The Ohio State University	Research Grant: Zoll/Revivant	Travel Reimbursement: Medtronic	None	None	Ohio Board of EMS, NHTSA Dept of Transportation	None
Scherer, L.R. ''Tres''	Indiana University	None	None	None	None	None	None
Schexnayder, Stephen	University of Arkansas for Medical Sciences	Wyeth Laboratories, Madison, WI, recipient for grant support for salary (2%) for study of pantoprazole	None	None	None	None	None
Schleien, Charles L.	Columbia University	None	None	None	None	None	None
Schmidt, Terri	Oregon Health & Sciences University	None	None	None	None	EMS Medical Director for American Medical Response in Clackamas County, Oregon	None
Schwartz, Alan Jay	Children's Hospital of Philadelphia	None	None	None	Stock ownership: General Electric, GlaxoSmithKline, Colgate Palmolive, Johnson and Johnson, Medtronic, Proctor and Gamble, Teva, 3M	None	None
Semenko, Tanya	AHA Staff	None	None	None	None	None	None

Writing Group Member	Employment	Research Grant	Other Research Support	Speakers Bureau/ Honoraria	Ownership Interest	Consultant/Advisory Board	Other
Shenefelt, Ralph M.	National Instructors Resource Center, Inc.; American Safety & Health Institute	None	None	None	None	Executive Director: ASHI; Vice President: NIRC	None
Sherbino, Jonathan	University of Toronto	None	None	None	None	None	None
Shimizu, Naoki	National Center for Child Health and Development, Tokyo, Japan	None	None	None	None	Japanese Society of Pediatric Intensive and Critical Care Medcine, Tokyo, Japan; Ministry of Health, Labour and Welfare Tokyo, Japan; Japanese Association for Acute Medicine, Tokyo, Japan, Pfizer Health Research Foundation, Tokyo, Japan	None
Shore, Paul	University of Texas Southwestern	None	None	None	None	None	None
Shuster, Michael	Self-employed	None	None	None	None	Chair: ECC Policy Advisory Committee, Heart & Stroke Foundation of Canada	None
Simon, Wendy	American Academy of Pediatrics	None	None	None	None	None	None
Singer, Adam	Stony Brook University Hospital, New York	None	None	None	None	None	None
Singhal, Nalini	University of Calgary, Canada	None	None	None	None	Investigator, Canadian Institute of Health Research	None
Soar, Jasmeet	North Bristol NHS Trust, UK	None	None	None	None	None	None
Soderberg, Erik	AHA Staff	None	None	None	None	None	None
Solomon, Robert	Emergency Medicine Physicians	None	None	None	None	Councillor: American College of Physicians; Director: WV Chapter of ACEP	None
Soreide, Eldar	Stavanger University Hospital, Norway	None	None	None	None	None	None
Sorge, Kevin	AHA Staff	None	None	None	None	None	None
Speer, Michael	Baylor College of Medicine	Grant Research and Consultant: MedImmune	None	None	None	Member: American Academy of Pediatrics; Member: Texas Pediatric Society, Texas Medical Association	None
Spizzirri, Carol	Save A Life Foundation	None	None	None	None	None	None
Srinivasan, Vijay	Dr. Mark Helfaer, MD, FCCM	None	None	None	None	None	None
Stapleton, Ed	State University of New York at Stony Brook	Research Grant: Laerdal	None	None	None	President: Citizen CPR Foundation; AHA BLS Science Editor 1998—2003	None

Starkman, Sid	UCLA Emergency Medicine Center	Concentric Medical (Biomedical Device) Mountain View, CA grantee; Pharmacia & Upjohn (Pharm.) Kalamazoo MI grantee; Interneuron Pharm. Inc. (Pharm.) Lexington, MA grantee; Boehringer Ingelheim Pharm. (Pharm.) Petersburg, VA grantee; Baker Norton Pharm. (Pharm.) Miami, FL grantee; GlaxoSmithKline (Pharm.) Philadelphia, PA grantee; Warner-Lambert (now Pfizer) (Pharm.) New York, NY grantee; Bayer Pharm. Corp (Pharm) West Haven, CT grantee; Wyeth Pharm. (Pharm.) Madison, NJ grantee; AstraZeneca (Pharm) Wilmington, DE grantee; Eli Lilly Pharm. (Pharm.) Indianapolis, IN Grantee; Texas Biotechnology (Pharm. and Biotech) Houston, TX grantee; Quintiles Transnational Inc. (Pharm. & Biotech.) Research Triangle Park, North Carolina grantee; Fujisawa Research Institute of America, Inc. (Pharm.) Deerfield, IL grantee; Ono Pharma USA Inc. (Pharm.) Lawrenceville, NJ grantee; Paion (Pharm.) Aechen, Germany grantee; Ekos Corp (Biomedical Device) Seattle WA grantee	None	None	None	Genentech, Inc. (Pharm.) San Francisco, CA consultant	None
Steen, Petter	University of Oslo	None	None	None	None	Member: Laerdal Executive Board; Member: Norwegian Air Ambulance Executive Board; Member: Cardiodigital Medical Advisory Board	None

Writing Group Member	Employment	Research Grant	Other Research Support	Speakers Bureau/ Honoraria	Ownership Interest	Consultant/Advisory Board	Other
Steinhart, Brian	St. Michaels Hospital, Toronto	None	Roche Diagnostics, Laval, Quebec—investigator BNP (0 salary) study	None	None	None	None
Stenson, Ben	UK National Health Service	Research Grant and attendance at an academic meeting: Chiesi Pharmaceuticals	None	None	None	None	None
Stettler, Brian	University of Cincinnati	None	None	None	None	None	None
Stiell, Ian	Ottawa Health Research Institute	Unrestricted research grant to evaluate defibrillation energy levels, Medtronic, Seattle, WA	None	None	None	None	None
Stoy, Walt	Center for Emergency Medicine		None	None	President: Dr. Walt Stoy and Associates; Author: Various publishers	None	None
Strater, Scott	AHA Staff	None	None	None	None	None	None
Sunde, Kjetil	Ullevaal University Hospital, Department of Anaesthesiology and Institute for Experimental Medical Research, Surgical Division, Oslo	None	None	None	None	None	None
Swain, Andrew	Mid Central Health, New Zealand	None	None	None	None	None	None
Swanson, Mark	Nemours Children's Clinic	None	None	None	None	President; Critical Care Educators, Orlando, FL—not for profit PALS TC	None
Swor, Robert	William Benent Hospital	Grantee, Medtronic Physio Control; Grantee, Laerdal	None	None	None	None	None
Szpilman, David	None	None	None	None	None	None	None
Szyld, Edgardo	Hospital Diego Paroissien, Buenos Aries	None	None	None	None	None	None
Tada, Keiichi	Hiroshima City Hospital	None	None	None	None	None	None
Tally, Richard	Colorado Permanente Medical Group	None	None	None	None	Speaker's Bureau, Sanifo/Aventis; Consultant, Scios	None
Tanaka, Keiichi	Fukuoka University, Fukuoka, Japan	None	None	None	None	None	None
Tang, Wanchun	The Institute of Critical Care Medicine	AHA Grant-in-Aid, NIH ROI; Army Grant; Research grants, Philips Zoll	None	None	None	None	None

Taubert, Kathryn	AHA Staff	None	None	None	None	Adjunct Professor: UT Southwestern Medical School	None
Tenn-Lyn, Nicole	University of Toronto	None	Fellowship Recipient: Studies in Medical Education, Royal College of Physcians and Surgeons of Canada	None	None	None	None
Terndrup, Thomas	University of Alabama at Birmingham	None	None	None	None	Contract(s), Alabama Dept. of Public Health; Subcontract, Dept. of Defense	None
Thierbach, Andreas	Johannes Gutenberg University, Nainz, Germany	Speaker — Travel Reimbursement, Scientific grant for University Hospital, Karl Sherz, Germany, Ruesch, Germany	None	None	None	Member, European Resuscitation Council; Chair, Disaster and Mass Casualty Committee, International Trauma Anesthesiology and Critical Care Society;	None
Thomas, Stephen	Mass. General Hospital	None	None	None	None	None	None
Tibballs, James	Royal Children's Hospital, Melbourne Australia	None	None	None	None	None	None
Timerman, Sergio	Heart Institute, St. Paul University School of Medicine	None	None	None	None	Consultant, Baldacci; Paid Consultant, Boehringer Ingelhem	None
Timermann, Ari		None	Investigator: Cure Trial (clopidogrel); Investigator and Brazilian national coordinator and steering committee member: Tetami Trial (with enoxaparin)	Speaker: Sanofi-Aventis; Speaker: Bristol Myers Squibb	None	None	None
Tomley, Erin	AHA Staff	None	None	None	None	None	None
Trotter, Tinsla	Centennial Medical Center	None	None	None	None	None	None
Truitt, Tanya	University of Texas at Arlington	None	None	None	None	Clinical Consultant, NRCPR TAI/DAI, Bel Air, MD	None
Udaeta, Enrique	Instituto Nacional de Pediatria, Mexico	None	None	None	None	None	None
Vaillancourt, Christian	The Ottawa Hospital	None	None	None	None	None	None
Van Der Jagt, Elise	University of Rochester Medical Center	None	None	None	None	Site investigator: PECARN (feasibility of hypothermia in post-resuscitation phase of CPR in pediatric patients)	None
Vanden Hoek, Terry	University of Chicago	NIH, Grant support; DOD, Grant support; Alsius, Unrestricted grant, University of Chicago	None	None	None	None	None

Writing Group Member	Employment	Research Grant	Other Research Support	Speakers Bureau/ Honoraria	Ownership Interest	Consultant/Advisory Board	Other
Vega, Francisco Javier Garcia	Emergency Department of Pontevedra Hospital, Sergas, Spain	None	None	None	None	National Training Manager: Spanish Society of Emergency Medicine	None
Velaphi, Sithembiso	Chris Hani Baragwanath Hospital, Gauteng, South Africa	None	None	None	None	None	None
Verbeek, Rick	The Ottawa Hospital, University of Toronto, and the Ottawa Health Research Institute	None	None	None	None	None	None
Viccellio, Peter	SUNY at Stony Brook	None	None	None	None	None	None
Vidyasagar, Dharmapuri	University of Illinois	None	None	None	None	None	None
Wagoner, Robert L.	National Registry of Emergency Medical Technicians	None	None	None	None	None	None
Wassertheil, Jeff	Peninsula Health, Hastings Road, Frankston, Melbourne Victoria, Australia	None	None	None	None	Australian Resuscitation Council member; St. John Ambulance Australia Director of Training; LifeFlight Honorary Medical Advisor; Monash University Faculty of Medicine, Nursing & Health Sciences academic appointee of the University	None
Wavra, Teresa	American Association of Critical Care Nurses	None	None	None	None	None	None
Weil, Max Harry	Institute of Critical Care Medicine, Palm Springs, CA	None	None	None	Shareholder: Medtronic, Zoll	Consultant/Research Collaborator: Abbott Laboratories, Philips Medical, Tyco/Nalcor Healthcare, Vasamed Medical, Zoll Medical	None
Weiner, Gary	St. Joseph Mercy Hospital, Ann Arbor	None	None	None	None	Member: American Academy of Pediatrics	None
Weisfeldt, Myron	Johns Hopkins University	Grantee, NIH, Bethesda, MD	None	None	None	None	None
Welsford, Michelle	Self-employed; Hamilton Health Sciences	None	None	None	None	None	None
Weltge, Arlo	University of Texas, Houston Medical School, AMR Ambulance Service, Houston Community College Program in EMS	None	None	None	None	Committee Chair: American College of Emergency Physicians; Committee member: Texas Medical Association	Physician, oncologist wife—Texas Oncology PA, Houston, TX

Wenzel, Volker	University of Innsbruck, Austria	Grant recipient (US$ 50,000), Aguettant, Lyon, France — a company manufacturing Vasopressin and applying for registration to market Vasopressin	None	None	None	None	None
Werman, Howie	MedFlight of Ohio; The Ohio State University College of Medicine and Public Health	None	None	None	None	Chair: Resuscitation Committee, American Health & Safety Institute; Immediate Past Chairman, Board of Directors: National Registry of EMTs	None
White, Lynn	Ohio State University College of Medicine	None	None	None	None	Contract for research NAEMSP, Lenera, KS	None
White, Roger	Mayo Clinic	None	Travel Expense Reimbursement: Zoll, Philips, Medtronic	None	None	None	None
Whitman, Gayle	AHA Staff	None	None	None	None	None	None
Wigginton, Jane	UT Southwestern Medical Center/University, TX	None	None	None	None	None	None
Wik, Lars	Ulleval University Hospital	None	None	None	None	Medical advisor: Norwegian Air Ambulance, Medtronic, Philips, Laerdal	None
Wolcke, Benno	Johannes Gutenberg University	None	Advanced Circulatory Systems, USA study: financial support for a running study: costs for patient assurance and material	None	None	None	None
Wooley-Goekler, Susan	American School Health Association	Employer is grantee, Aventis-Pasteur (commercial)	None	None	None	Member Advisory Council on First Aid & Safety (volunteer), American Red Cross (non-profit); Adjunct faculty, Kent State University; Consultant on health videos for children, Library Media (commercial)	None
Wyckoff, Myra	University of Texas Southwestern Medical Center	Research Grant: Neonatal Resuscitation Program Research, AAP	None	None	None	None	None

Writing Group Member	Employment	Research Grant	Other Research Support	Speakers Bureau/ Honoraria	Ownership Interest	Consultant/Advisory Board	Other
Wyllie, Jonathan	South Tees Hospitals NHS Trust	None	Fellow, Royal Collee of Paediatrics and Child Health	None	None	Full Member, Resuscitation Council, London, UK; Member of the Newborn Life Support Working Group, London, UK; Member of British Association of Pernatal Medicine, London, UK; Member of the International Advanced Life Support Working Group, Advanced Life Support Group, Salford, UK Charitable proganisatio	None
Xunmei, Fan	Beijing Children's Hospital affiliated to Capital University of Medical Sciences	None	None	None	None	None	None
Yiming, Lu	Shanghai Rvi Jin Hospital, Shanghai 2nd Medical University	None	None	None	None	None	None
Young, Judy	US Air Force (Ret.); part-time Sebastian River Medical Center	None	None	None	None	None	None
Zaritsky, Arno	University of Florida College of Medicine	None	None	None	None	None	None
Zideman, David	Hammersmith Hospitals NHS Trust, UK	None	None	None	None	Chairman: European Resuscitation Council; Chairman: British Association for Immediate Care; District Medical Officer: St. John Ambulance	None
Zoch, Tom	Thedacare	None	None	None	None	None	None

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EDITORIAL

The International Liaison Committee on Resuscitation (ILCOR)—-Past and present

Compiled by the Founding Members of the International Liaison Committee on Resuscitation

This brief overview of ILCOR is dedicated to the late Peter Safar and to the scientists, researchers, trainers, and individuals worldwide who strive to develop the science and to improve the practice of resuscitation medicine.

The creation of the International Liaison Committee on Resuscitation (ILCOR) has produced a unique opportunity for worldwide collaboration in resuscitation guidelines and practice for the past 15 years. Below is a brief outline of the landmark events and progress of this organization, which has become the authoritative voice on the consensus on science behind national and international guidelines on resuscitation.

1990: In June 1990, representatives from the American Heart Association (AHA), European Resuscitation Council (ERC), Heart and Stroke Foundation of Canada (HSFC), and the Australian Resuscitation Council (ARC), attended a meeting, hosted by the Laerdal Foundation, at the Utstein Abbey on the remote Island of Mosteroy in Norway. The purpose of this meeting was to discuss the problems of resuscitation nomenclature and the lack of standardised language in reports relating to adult out-of-hospital cardiac arrest. This was the first important collaborative venture involving existing Resuscitation Councils from around the world. A follow-up meeting was held in December 1990 in Surrey, England, where the decision was made to adopt the term 'Utstein-style' for the uniform reporting of data from out-of-hospital cardiac arrests [1].

Following this first landmark meeting in the Utstein Abbey, over the following years many additional 'Utstein-style' international consensus statements were published, including the uniform reporting of neonatal [2] and paediatric advanced life support [3], laboratory CPR research [4], in-hospital resuscitation [5], and CPR registers [6].

1992: The Fifth National Conference on Cardiopulmonary Resuscitation (CPR) and Emergency Cardiac Care (ECC) was held in Dallas, Texas, USA in February 1992. Through the generosity of the AHA, over 25% of the delegates came from outside the USA, representing more than 25 countries and 53 international organisations. This offered an ideal opportunity to discuss international issues, building on the cooperation already achieved at the first Utstein meeting. The Conference addressed three international issues: (1) the desirability of international support for countries to develop effective ECC; (2) the creation of a permanent infrastructure for international cooperation; (3) the desirability of common international guidelines and an international conference on CPR and ECC. An

Abbreviations: AHA, American Heart Association; ARC, Australian Resuscitation Council; ECC, Emergency Cardiac Care; ERC, European Resuscitation Council; HSCF, Heart and Stroke Foundation of Canada; IAHF, Inter-American Heart Foundation; CoSTR, Consensus on Science and Treatment Recommendations

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International CPR and ECC Panel Discussion, co-chaired by Richard Cummins and Douglas Chamberlain included speakers from the USA, Canada, Europe, Australia and Southern Africa. The report states:

"The conference recommended that the existing major organizations with a responsibility for guidelines in CPR and ECC aim to synchronize their review of guidelines, with the objective of publishing updates in the same year. With such a schedule, the organizations could create international working groups with a worldwide membership of the principal experts in their fields. These groups could offer international reviews of the literature and, based on the shared science and experiences, could make suggestion for modifications in guidelines. The proposed modifications, supported by the science that generated them, would be offered as evidence to the major international organizations for their own meetings and deliberations: to the AHA, the Canadian Heart and Stroke Foundation, the European Resuscitation Council, the associations or societies in Latin America, Australia, Africa, and Asia, i.e. to all countries or multinational organizations that might wish to participate. The proposed modifications would be considered by these organizations. If the science was unassailable, the modifications would likely be adopted with or without change, taking into consideration local needs and realities.

Such a plan for international cooperation would have appreciable advantages over existing arrangements: (1) the world's leading experts would achieve fruitful communication and cooperation; (2) advice for guidelines would be less likely to be tainted by habit, tradition, or peer pressure; (3) guidelines generated in this way should be widely accepted within existing organizations; (4) a great similarity (or even identity) of guidelines would likely be achieved without the fear that one group was being subverted by another; (5) the potential would exist for eventual universal guidelines; (6) existing organizations would not perceive a risk to their independence or autonomy."

Resuscitation '92', held in Brighton, England in November 1992, was the first international conference held by the ERC. At the end of the conference, representatives from guidelines-producing organisations, i.e. the European Resuscitation Council, the American Heart Association, the Heart and Stroke Foundation of Canada, the Australian Resuscitation Council, and the Resuscitation Council of Southern Africa held the first meeting of the International Liaison Committee. Chaired by Douglas Chamberlain, the meeting proposed that there should be continuing international cooperation through a permanent liaison committee, comprising active, well-established organisations that were currently producing guidelines and were generally multinational or multidisciplinary in nature.

1993: Following the Update in Sudden Cardiac Death Congress in Vienna, Austria in March 1993, the newly formed "Liaison Committee on CPR" held its second meeting, at which a formal Mission Statement was adopted:

"To provide a consensus mechanism by which the international science and knowledge relevant to emergency cardiac care can be identified and reviewed. This consensus mechanism will be used to provide consistent international guidelines on emergency cardiac care for Basic Life Support (BLS), Paediatric Life Support (PLS) and Advanced Life Support (ALS). While the major focus will be upon treatment guidelines, the steering committee will also address the effectiveness of educational and training approaches and topics related to the organisation and implementation of emergency cardiac care. The Committee will also encourage coordination of dates for guidelines development and conferences by various national resuscitation councils. These international guidelines will aim for a commonality supported by science for BLS, ALS and PLS".

It was agreed that, wherever possible, meetings would be held in conjunction with international resuscitation events, being cost-effective, and allowing leaders in the field of resuscitation to meet and share information and expertise on a regular basis, with a wide multinational and multidisciplinary audience (Table 1). At the third meeting, co-chaired by Douglas Chamberlain and Richard Cummins, formal BLS, ALS and PLS Working Groups were established, tasked with reviewing scientific data in their respective area of expertise.

1994: When the ERC published its resuscitation guidelines, the Chairman, Peter Baskett, reported that "The ERC has not worked in isolation, and has enjoyed cordial and productive cooperation with the American Heart Association's Emergency Cardiac Care Committee, the Australian Resuscitation Council, the Heart and Stroke Foundation of Canada, the Resuscitation Council of Southern Africa and many Resuscitation Councils and authorities throughout Europe. Our aim in the

Table 1 Meetings of the international liaison committee on resuscitation

#	Associated International Event	Date	Host	Venue
1	Resuscitation '92 Congress	November 1992	ERC	Brighton, UK
2	Sudden Cardiac Death Congress	March 1993	ERC	Vienna, Austria
3	AHA Scientific Sessions	November 1993	AHA	Dallas, USA
4	CPR & ECC Update'94 Congress	May 1994	AHA	Richmond, USA
5	Resuscitation '94 Congress	October 1994	ERC	Mainz, Germany
6	In-Hospital Utstein Consensus	June 1995	ERC	Mosteroy, Norway
7	ASA Congress	October 1995	AHA	Atlanta, USA
8	CPR & ECC Update'96 Congress	May 1996	HSFC	Montreal, Canada
9	AHA Scientific Sessions	November 1996	AHA	Dallas, USA
10	CPR '97 Congress	April 1997	ERC	Brighton, UK
11	CPR & ECC Update 98 Congress	May 1998	AHA	Orlando, USA
12	AHA Meetings	March 1999	AHA	Dallas, USA
13	Resuscitation 2000 Congress	June 2000	ERC	Antwerp, Belgium
14	Education Utstein Consensus	June 2001	ERC	Mosteroy, Norway
15	Spark of Life 2002 Congress	April 2002	ARC	Melbourne, Australia
16	Resuscitation 2002 Congress	October 2002	ERC	Florence, Italy
17	AHA Meetings	April 2003	AHA	Dallas, USA
18	IAHF Meetings	September 2003	IAHF	Recife, Brazil
19	AHA Meetings	March 2004	AHA	Dallas, USA
20	Resuscitation 2004 Congress	September 2004	ERC	Budapest, Hungary
21	CoSTR Congress	January 2005	AHA	Dallas, USA
22	CoSTR Editorial Board	April 2005	AHA	Jersey City, USA

future is to collaborate with our colleagues to produce guidelines, which will have worldwide acceptance. There is an active International Liaison Committee currently addressing this goal, so that we can enter the 21st century with unanimity."

1995: A proposal by John Kattwinkel of the American Academy of Pediatrics to establish a Neonatal Subgroup of the Paediatric Working Group was considered. Consensus was reached on recommended guidelines for reviewing, reporting, and conducting research on in-hospital resuscitation [5]. One representative made an observation that captured the prevailing spirit of cooperation: "In the seven meetings of the International Liaison Committee, the Committee has never needed to take a vote on any question."

1996: At the suggestion of Walter Kloeck from South Africa, the name 'International Liaison Committee on Resuscitation (ILCOR)' was formally adopted in May 1996. This was a deliberate play on words relating to developing treatment guidelines for a sick heart — 'ill cor'! It was determined that there was a significant need to develop 'Advisory Statements', and each Working Groups was tasked with producing updated consensus statements.

1997: In April 1997, the 'Consejo Latino—Americano de Resucitación (CLAR)', representing the countries of Latin America, became the Seventh official member organisation of ILCOR. ILCOR Advisory Statements on Single Rescuer Basic Life Support [7], a Universal ALS Algorithm [8], Early Defibrillation [9], Paediatric Life Support [10], and Special Resuscitation Situations [11] were published worldwide.

1998: The New Zealand Resuscitation Council and the Australian Resuscitation Council joined to form a multinational resuscitation entity. Petter Steen of the ERC was appointed Co-chair of ILCOR, together with Richard Cummins, following the resignation of the Founding Co-Chairman, Douglas Chamberlain. A decision was made that guidelines development will try and move from the use of expert opinion and consensus discussions, to a much more explicit, evidence-based process, and the use of 'levels of evidence' and 'classes of recommendation.'

1999: Representatives from China, Taiwan, Thailand, Japan and Malaysia were welcomed to ILCOR meetings as observers. It was agreed that the administrative secretariat of ILCOR would be managed by the Australia and New Zealand Committee on Resuscitation (ANZCOR). ILCOR published an Advisory Statement on Resuscitation

of the Newly Born Infant [2], and an Evidence Evaluation Conference, preceding the 'Guidelines 2000 Conference' was held in Dallas in September 1999.

2000: The Guidelines 2000 Conference, held in Dallas in February 2000 was the world's first international conference assembled specifically to produce international resuscitation guidelines [12]. Bill Montgomery of the AHA was elected Co-chair of ILCOR, together with Petter Steen of the ERC.

2001: The first ILCOR symposium on Education in Resuscitation was held at Utstein Abbey in June 2001 [13], with the support of the Laerdal Foundation. A formal Constitution for ILCOR was drawn up at this meeting.

2002: ILCOR hosted a meeting in Melbourne, Australia to update and simplify the Utstein templates for reporting cardiac arrests in and out of hospital, and to develop recommendations for resuscitation registries. Jerry Nolan of the ERC was elected Co-chair of ILCOR, together with Bill Montgomery of the AHA. It was agreed that ILCOR would be an advisory group for the Cochrane Heart Group, and Ian Jacobs was appointed as the official coordinator. A Neonatal Task Force, as well as an Interdisciplinary Task Force, looking at issues such as epidemiology, education and ethics in resuscitation, was established.

2003: ILCOR published Advisory Statements on Uniform Reporting of Data from Drowning [14], Therapeutic Hypothermia after Cardiac Arrest [15], and the use of AEDs for children [16]. The Inter-American Heart Foundation (IAHF) replaced CLAR as the official member organisation representing Central and South American countries. Intense planning for the 2005 Consensus on Resuscitation Science started at an ILCOR meeting in Brazil.

2004: An update of the Utstein-style templates for resuscitation research, that were first created by representatives of international Resuscitation Councils in 1990, was published under the aegis of ILCOR [6]. An official ILCOR logo was approved and adopted, and plans are made for ILCOR to be formally incorporated as a Non-Profit Association. Preparations continue at meetings in Dallas and Budapest for the publication in 2005 of the updated consensus on the science of resuscitation, using dedicated systematic evidence evaluation tools.

2005: The 2005 International Consensus on ECC and CPR Science with Treatment Recommendations (CoSTR) Conference, hosted by the AHA, represents the most intense review of resuscitation science ever held, and involved the greatest degree of international cooperation that ILCOR has ever experienced.

As can be seen from the 22 official meetings that ILCOR has held from 1992 to 2005, the associated international events linked to each meeting (Table 1), and the accompanying landmark co-publications [1—16], a spirit of sincere cooperation and genuine desire to raise the standard of practice of emergency care will result in many additional lives being saved. The Founding Members of ILCOR salute all those that have contributed to this process.

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EDITORIAL

International collaboration in resuscitation medicine

Douglas Chamberlain, Richard O. Cummins, William H. Montgomery, Walter G.J. Kloeck, Vinay M. Nadkarni

Researchers from many countries, publishing in multiple languages, are building the scientific foundation for resuscitation practice. Universal guidelines will follow if we can find a way to gather all this information in one location and decide what it all means.

Douglas Chamberlain and Richard O. Cummins, founding cochairs of the International Liaison Committee on Resuscitation

For more than a decade, an international collaboration of clinicians and researchers has tried to identify, evaluate, and interpret the most valid resuscitation science. This issue of *Resuscitation* (simultaneously published in *Circulation*) presents these collaborators' latest attempts to reach consensus on what the science means and what resuscitation practices should follow. We have not reached our goal of universal resuscitation guidelines, but we have made a worthy attempt. Building on the International Guidelines 2000 Conference on CPR and ECC,[1] in January 2005 a total of 380 experts reviewed 276 resuscitation topics, digested countless peer-reviewed publications, and participated in six days of almost continuous discussion and debate. Particular attention was paid to disclosure of potential conflicts of interest and identification of topics that lacked good evidence to support current practice.

We can trace the pedigree of these efforts over half a century. The original reports of rescue breathing[2] and closed-chest compressions[3] and the effective combination of the two[4] created an immediate demand for CPR training and performance guidelines. In 1966, the Institute of Medicine convened the first conference to specifically review the evidence and recommend standard CPR and ECC techniques.[5] The American Heart Association sponsored subsequent conferences in 1973 and 1979.[6,7] Parallel efforts occurred internationally as other resuscitation councils faced a growing demand for training in this strange new technique of compressing the victim's chest and blowing into the victim's mouth.[8] Inevitably variations in resuscitation techniques and training methods began to emerge from one country to another.

With continued development of new drugs and medical devices, resuscitation leaders identified many questions that needed answers. At numerous small national conferences, they asked whether answers might already exist in other countries, published in both English and non-English language scientific journals. Increasing awareness of variations in resuscitation practices between countries sparked interest about gathering international experts at a single location. The AHA convened such a meeting in 1985, inviting resuscitation leaders from many countries to observe the AHA's review of standards and guidelines for CPR and ECC.[9] Passive observation by these international guests lasted only through opening introductions: these multinational experts, passionately devoted to improving resuscitation outcomes, soon demonstrated an ability to generate both heat and light.

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By 1992, when the AHA convened the next Guidelines Conference, more than 40% of the participants were from outside the United States.[9] During this 1992 conference, a panel on international cooperation on CPR and ECC endorsed the need to foster a multinational base of evidence for resuscitation practices. What was lacking, however, was a focused mechanism with which to capture and assess this growing body of evidence. That panel strongly recommended that an expanded group of international experts initiate a systematic review of the world's resuscitation literature. Under the leadership of many of these panel members, including Richard O. Cummins, Douglas Chamberlain, William Montgomery, and Walter Kloeck, the International Liaison Committee on Resuscitation (ILCOR) was formed. The founding member organisations of ILCOR were the American Heart Association, the European Resuscitation Council, the Heart and Stroke Foundation of Canada, the Resuscitation Council of Southern Africa, and the Australian Resuscitation Council. These organisations were later joined by the Consejo Latino-Americano de Resuscitatión (which now forms part of the Inter-American Heart Foundation) and the New Zealand Resuscitation Council.

With the shared vision of international cooperation, ILCOR began to assess systematically the supportive evidence for resuscitation standards and guidelines. During this project ILCOR experts identified numerous national differences in the practices of basic life support, advanced life support, and pediatric and newborn resuscitation. ILCOR eventually published 18 scientific advisory statements with the goal of explaining, eliminating, or reducing these international variations while endorsing mainly evidence-based resuscitation guidelines.[10]

Between 1992 and 2005 ILCOR has convened 22 official meetings. Guiding these ILCOR meetings was a belief that evaluation of international science by a common group of experts should lead to ''the single best set'' of evidence-based resuscitation guidelines and practices. This belief permeated the international CPR and ECC evidence evaluation conferences held in 2000 and 2005, as well as several international consensus statements.[11–13] The 2000 Guidelines Conference,[1,14] the first major assembly under the auspices of ILCOR, adopted a sophisticated process for gathering and assessing evidence; this process evolved further in 2005. With practical insight, conference participants determined how to incorporate different levels of evidence into consensus treatment recommendations, with identification of key gaps in knowledge.

The experience of developing evidence-based guidelines forced a reluctant conclusion on the ILCOR leadership: the goal of a single ''best set'' of international CPR and ECC guidelines was not yet achievable. It was recognised that universal science consensus was achievable but that localisation of the treatment recommendations using regional guidelines and training tools is necessary. Undoubtedly, international cooperation has enabled a more thorough collection and analysis of the evidence. Nevertheless, review and debate of that evidence has not always led to standard training and practice. Some obstacles were encountered in the pursuit of universal guidelines.

1. The available evidence may present an inconsistent, contradictory, or less definitive picture that fails to support universal guidelines. CPR ventilation is one example of this obstacle: fine-tuning the details of ventilation consumed considerable time and energy at the 2000 Guidelines Conference. The experts debated numerous ventilation variables, such as rate, inspiratory pressure, inspiratory duration, inspiratory/expiratory ratios, and optimal airway devices for field and hospital and lay rescuers and professionals. At the 2005 Consensus Conference many of these same resuscitation experts argued that compression-only CPR may be more effective and that perhaps ventilations should be eliminated completely from initial resuscitation actions.
2. For many questions, high-level evidence, preferably in the form of randomised controlled clinical trials, is simply not available and probably never will be, preventing the identification of definitive answers to many questions. For example, what is the best way to train lay rescuers so that they will make a vital intervention, undertake it properly and effectively, and retain the skill for years?

ILCOR and international collaboration has continued to mature. In retrospect, the goal of a single set of universal guidelines is idealistic and premature. Many problems in resuscitation require local modifications and solutions. The common goals of the resuscitation community are more important: reducing rates of morbidity and mortality from cardiovascular disease and stroke. The treatment recommendations in this publication are based on the best science known, and they have been achieved by effective international collaboration. Exponential improvements in communication technology are making international collaborative research and topic review a reality, and when indicated, will enable urgent revisions to current guidelines. We

look forward to this continual review and update of the science and the year 2010, when another international collaborative conference will be convened.

Our problems in resuscitation are similar the world over, but none of us has a monopoly of wisdom, knowledge, or experience. We must, therefore, continue to work effectively together for the good of all.

Douglas Chamberlain

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EDITORIAL

The evidence evaluation process for the 2005 International Consensus Conference on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations

Peter T. Morley, Arno Zaritsky

''In God we trust. All others must bring data.''—Robert Hayden, Plymouth State College.

Evidence-based medicine is described as ''the conscientious, explicit and judicious use of current best evidence in making decisions about individual patients''.[1] The evidence evaluation process summarised in this supplement was designed to ensure the review of all available evidence pertaining to resuscitation. Many aspects of the resuscitation process create unique challenges for the design of experimental protocols and data analysis and have not been evaluated by randomised controlled human studies. Exclusion of studies other than controlled human studies would eliminate a wealth of information that could help guide resuscitation management; for this reason, lower levels of evidence, including nonhuman studies, were included in the review.

To begin the review process, international experts (worksheet reviewers) were assigned questions to evaluate. The questions were selected from a survey of each of the International Liaison Committee on Resuscitation (ILCOR) specialty task forces (e.g. basic life support, advanced life support, paediatrics) and from the ILCOR member resuscitation councils and their training networks. The evaluation of each question was completed on a structured evidence evaluation worksheet developed for the 2005 Consensus Conference. Because many of the worksheet reviewers had never conducted a structured evidence-based review, instructional sessions were held at the twice-yearly ILCOR meetings and an instructional CD-ROM was created, demonstrating how to conduct an efficient search for evidence, complete the worksheet, and use citation management software. Two worksheet experts (Peter Morley and Arno Zaritsky) were appointed to provide further quality assurance; they reviewed all submitted worksheets. Comments, emendations, and queries were provided to the worksheet reviewers in an iterative process until the worksheets were deemed complete by the worksheet experts.

The worksheets completed for the 2005 Consensus Conference are linked from the electronic version of this document as online data supplements. Most superscript worksheet numbers are located adjacent to headings and begin with the letter W to distinguish them from other reference citations. Readers of the electronic version of this supplement can access a cited worksheet by clicking on the linked worksheet callout. Readers of the printed publication can identify the complete title and author of a cited worksheet by referring to the numbered worksheet list at the end of this issue (see Appendix 1) and then accessing that worksheet on the conference website at www.c2005.org. In

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the discussion below, a blank worksheet is cited and can be accessed for reference.

Steps for evidence evaluation

The following steps correspond with the major steps listed in the evidence evaluation worksheets.

Step 1. State the proposal (1A) and gather and select the evidence (1B)
W277

All reviewers were instructed to search their allocated questions broadly. Reviewers documented their search strategies to ensure reproducibility of the search. The minimum electronic databases to be searched included the Cochrane database for systematic reviews and the Central Register of Controlled Trials [http://www.cochrane.org/], MEDLINE [http://www.ncbi.nlm.nih.gov/PubMed/], EMBASE (www.embase.com), and the master reference library collated by the American Heart Association (AHA). To identify the largest possible number of relevant articles, reviewers were also encouraged to perform hand searches of journals, review articles, and books as appropriate.

The reviewers documented the mechanism by which studies relevant to the hypothesis were selected. Specific study inclusion and exclusion criteria and study limitations were documented. Inclusion of all relevant evidence (from animal and manikin/model studies as well as human studies) was encouraged.

Step 2. Assess the quality of evidence
W277

In this step reviewers were asked to determine the level of evidence of relevant studies (Step 2A), assess the quality of study research design and methods (Step 2B), determine the direction of results (Step 2C), and cross-tabulate assessed studies (Step 2D).

The levels of evidence used for the 2005 consensus process (see Part 1 of this issue)[2] were modified from those used in 2000.[3,4] In many situations summary conclusions were based on lower levels of evidence because human clinical trial data were not available.

The reviewers assessed the quality of research design and methods and allocated each study to one of five categories: excellent, good, fair, poor, or unsatisfactory. Studies graded as poor or unsatisfactory were excluded from further analysis.

Reviewers evaluated the direction of the study results as supportive, neutral, or opposed and then depicted the data in one of two grids. The grids were two-dimensional, showing quality and levels of evidence. The reviewers completed a Supporting Evidence grid and a Neutral or Opposing Level of Evidence grid.

Step 3. Recommendation for class of recommendation

The *2005 AHA Guidelines for CPR and ECC*[5] use a class of recommendation systems to indicate the overall strength of recommendations. These classes of recommendations were not used in the ILCOR 2005 CPR consensus document.[6]

In this step reviewers were invited to offer an opinion on the overall strength of a specific treatment recommendation for the AHA or other council-specific guidelines. Statements contained in this section reflect the reviewer's opinion and may or may not be consistent with consensus conclusions from the 2005 Consensus Conference and the *2005 AHA Guidelines for CPR and ECC* or guidelines from other resuscitation councils.

Step 4. Reviewer's perspective and potential conflict of interest
W277

All reviewers completed a conflict of interest disclosure form and also listed potential conflicts of interest on the worksheets. This ensured transparency of the review process. More details of the conflict of interest disclosure process are described in another editorial in this issue.

Step 5. Summary of the science
W277

Worksheet reviewers created a summary of the science. In the summary format reviewers were encouraged to provide a detailed discussion of the evidence, including the outcomes evaluated and the strengths and limitations of the data.

The final step in the science summary process was the creation of draft consensus on science statements and treatment recommendations. Statement templates were provided to standardise the comprehensive summary of information. Elements of the consensus on science statement template included the specific intervention or assessment tool, number of studies, levels of evidence, clinical outcome, population studied, and

the study setting. Elements of the treatment recommendation template included specific intervention or assessment tool, population and setting, and strength of recommendation.

The statements drafted by the reviewers in the worksheets reflect the recommendations of the reviewers and may or may not be consistent with the conclusions of the 2005 Consensus Conference.

Step 6. References

Worksheet reviewers were asked to provide a database file containing the references that were used. The submitted references were added to the master reference library collated by the AHA.

Step 7. Posting on the internet

Completed worksheets were posted on the internet for further review. The initial process involved posting the worksheet to a password-protected area of the AHA intranet (accessible to worksheet reviewers). In December 2004 the completed worksheets were posted on an internet site that could be accessed by the public for further review and feedback before the 2005 Consensus Conference in Dallas (http://www.c2005.org/).

Controversies encountered

Studies on related topics (LOE 7)

Many reviewers identified studies that answered related questions but did not specifically address the reviewer's initial hypothesis. Examples include the extrapolation of adult data for pediatric worksheets and extrapolation of the results of glucose control in critically ill patients to the postresuscitation setting. Worksheet reviewers were instructed to clearly designate evidence that represented extrapolations. Reviewers could designate such studies as LOE 7, or they could assign a level of evidence-based on the study design but include terms such as ''extrapolated from'' with specific relevant details in the draft consensus on science statements to indicate clearly that these were extrapolations from data collected for other purposes.

Animal studies and mechanical models

Animal studies can be performed under highly controlled experimental conditions using extremely sophisticated methodology. Irrespective of methodology, all animal studies and all studies involving mechanical models (e.g. manikin studies) were classified as LOE 6. Specific details about these studies (including methodology) are included in the summary of science wherever appropriate.

Studies evaluating diagnosis or prognosis

The default levels of evidence used for the 2005 consensus process were not designed for the review of studies that evaluate diagnosis or prognosis. For these studies other methods of assigning levels of evidence were considered (such as those proposed by the Oxford Centre for Evidence-Based Medicine [CEBM http://www.cebm.net/]). Worksheet reviewers planning to include alternative levels of evidence were asked to define such levels clearly and to retain the default levels of evidence.

Summary

The 2005 consensus process provided a large number of detailed literature reviews published on the internet and summarised in this supplement. This review suggests that the evidence evaluation process for resuscitation literature will continue to evolve, providing a comprehensive process for collating data, summarising the science, and facilitating its translation into treatment recommendations.

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EDITORIAL

Conflict of interest management before, during, and after the 2005 International Consensus Conference on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations

John E. Billi, David A. Zideman, Brian Eigel, Jerry P. Nolan, William H. Montgomery, Vinay M. Nadkarni,

To preserve the public trust and integrity of the International Liaison Committee on Resuscitation (ILCOR) evidence evaluation process, in 2004 ILCOR established a conflict of interest (COI) policy[1] to manage any real or potential conflicts of interest in an open and effective manner. This editorial explains the ILCOR and American Heart Association (AHA) COI policies and their application throughout the 2005 evidence evaluation process. ILCOR and the AHA also invite readers' questions and feedback on this process.

The value of the ILCOR evidence evaluation process depends on rigorous expert review of published science. Therefore, it is essential that any potential professional conflict of interest be fully disclosed and managed effectively during the planning and conduct of the evidence evaluation process, especially when issues arise. Because many of the world's most qualified scientific experts may have professional relationships that could pose a real or perceived conflict of interest, it is not always possible to avoid all involvement by such persons. It is necessary, however, to limit and manage their involvement in areas of potential conflict, especially to minimise their influence over consensus statements or recommendations in such areas. ILCOR COI procedures applied to all ILCOR delegates, 2005 Consensus Conference participants, observers, worksheet experts, worksheet authors, editors of the ILCOR 2005 CPR Consensus document (published in this supplement), and all others working on ILCOR projects.

As host of the 2005 Consensus Conference, the AHA also required every participant to complete an AHA COI disclosure questionnaire and to comply with all AHA COI policies. The purpose of the AHA COI policies and procedures[2] is to protect the integrity of the AHA's decision-making processes and the *2005 AHA Guidelines for CPR and ECC*, as well as to protect the public's trust in the AHA and AHA volunteers and staff.

Summary of COI procedures

Each participant in the 2005 evidence evaluation process completed and submitted both an ILCOR and an AHA COI disclosure form before attending

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the 2005 Consensus Conference.[3] Late registrants were required to complete the COI disclosure forms when they registered on-site. AHA staff reviewed the forms and ensured that completed versions of both forms were submitted by each conference participant and worksheet author. ILCOR task force cochairs (e.g. cochairs of the Basic Life Support, Advanced Life Support, and Pediatric Resuscitation Task Forces) reviewed the forms for potential conflicts of interest. COI-related questions or concerns were submitted to the ILCOR COI cochairs (John Billi and David Zideman) for resolution. Corrective actions included reassigning topics or moderator roles to persons without a significant conflict of interest or limiting persons with a significant conflict of interest to the role of reviewer of the evidence. In the latter instance, panellists with no conflict of interest made any final judgments based on the evidence and drafted any consensus statements or summaries. The AHA and ILCOR have retained all disclosure forms together with written records of actions taken.

Each evidence evaluation worksheet (see the editorial on evidence evaluation in this supplement) included a section for the author to disclose potential conflicts of interest. Worksheets without a completed COI section were not accepted. The COI information submitted for each worksheet was cross-referenced for accuracy and consistency with the COI information on file with the AHA and ILCOR.

At the start of the 2005 Consensus Conference each participant was given a printed COI disclosure booklet listing each attendee's name and institution and the basic details of any declared professional relationship that could pose a potential conflict of interest (see COI listing at doi: 10.1016/j.resuscitation.2005.11.001 or www.elsevier.com/locate/resuscitation). Each participant was assigned a participant number. COI information for each participant was listed numerically in the COI booklet, which was updated daily with additional COI disclosure information from late registrants.

Throughout the 2005 Consensus Conference, continuous COI disclosure for all speakers (scheduled or unscheduled) was provided without interruption or delay in the proceedings. Every speaker, whether moderator, presenter, panelist, or someone making comments from the floor, was required to state his or her name and participant number. A slide listing the speaker's institution and COI disclosure information was projected on a designated screen for the duration of the speaker's comments. This provided conference participants with immediate and continuous information on any relationships the speaker had that could pose a COI issue. Participant numbers enabled participants to immediately crosscheck disclosures in the conference COI disclosure booklet. Late registrants were required to make verbal disclosures until their information could be posted on a slide.

All moderated sessions, questions from the audience, comments, and statements were audiorecorded for future reference. All speakers stated their participant numbers each time they spoke, making the task of identifying recorded speakers easier and assessment of the impact of potential conflicts of interest possible.

A COI monitor was assigned to each session to ensure that policies were followed and to record any irregularities. The monitors' reports were reviewed and retained as part of the AHA COI documentation file. Conference participants were repeatedly reminded to raise COI issues with COI monitors, moderators, or cochairs. Participants were also given the number of a confidential COI phone ''hotline'' to enable them to report issues anonymously if they did not wish to make their comments in person. The methods through which participants could raise potential COI issues were displayed on the screens in the plenary sessions several times each day.

During the conference any new COI problems or questions that could not be resolved by the session moderators were referred to the ILCOR COI cochairs for rapid resolution. If an issue was deemed sufficiently challenging, it was referred to the Ad Hoc COI Committee (see Results). The Ad Hoc COI Committee was composed of the 2005 Consensus Conference coordinator (William Montgomery), conference cochairs (Vinay Nadkarni and Jerry Nolan), and COI cochairs (John Billi and David Zideman). Moderators were instructed to stop discussion immediately if they believed that the session should not continue until a specific COI issue was resolved and to go on to the next presentation to enable the COI cochairs time to resolve the issue. After resolution the panel was permitted to resume the earlier presentation and discussion.

Results of COI Policy implementation

All 380 participants in the 2005 Consensus Conference completed COI disclosure forms, most before the conference. Staff added information from late registrants to daily updates of the COI disclosure booklet and slides. Although a few reminders were needed on the first day of the conference, all conference participants quickly adopted the habit of giving their name and participant number whenever they spoke.

COI cochairs investigated and recommended resolution for 8 concerns before the conference and 12 concerns during the conference. One COI issue required that the Ad Hoc COI Committee convene. On another occasion a discussion was stopped when a floor debate appeared centered on a detail of interest to device manufacturers and the debaters had potential or perceived links with the manufacturers as disclosed on the COI slides. In this instance the COI monitor and session moderators conferred, then asked all participants to send any further written comments to the Task Force for consideration. The comments included the authors' participant numbers so that their COI disclosures could be considered when their input was weighed. Throughout the poster sessions a COI policy/rationale poster was displayed and attended by one of the COI cochairs. This stimulated much discussion, raising awareness of the importance of good COI management.

No anonymous calls were received on the COI hotline. Twelve participants voluntarily revised their COI disclosure forms once they observed the comprehensive level of disclosure of their peers or were reminded of relationships that might pose a potential conflict. In two instances one participant was aware of a potentially conflicting, undisclosed relationship of another participant. In both instances a COI cochair investigated the issue, and the disclosure forms, booklet, and slides were updated.

A participant survey conducted after the 2005 Consensus Conference indicated almost uniform support for the COI disclosure method. The common responses were ''very effective'' and ''nonintrusive''. A few participants indicated that the disclosure was too continuous, but several others thought it did not go far enough. Ninety percent of the 120 respondents ''strongly agreed'' or ''agreed'' that speakers' relationships with commercial entities were clearly disclosed during the 2005 Consensus Conference. One unintended benefit of the simultaneous projection of the COI slide was that the audience always knew who was speaking, something that can be difficult to discern in a large meeting with floor microphones.

Readers are welcome to provide feedback on any aspect of the ILCOR or AHA COI policies and implementation. Please contact any of the authors at www.C2005.org.

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EDITORIAL

Controversial Topics from the 2005 International Consensus Conference on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations

Jerry P. Nolan, Mary Fran Hazinski, Petter A. Steen, Lance B. Becker

Cardiopulmonary resuscitation (CPR) and emergency cardiovascular care (ECC) constitute a relatively young field of medicine concerned literally with issues of life and death. The scientific evidence is scant and opinions are strong. It is difficult to perform clinical intervention studies with sufficient power, and this has been compounded by the severe restrictions on research created by consent legislation in North America[1] and Europe.[2] There is very little high-level evidence for resuscitation therapies, and many traditional treatment recommendations such as the use of adrenaline/epinephrine, are based on animal studies and reluctance to change an existing treatment recommendation until it is proven ineffective or less effective than a novel therapy.

A rigorous evidence evaluation worksheet process,[3] full disclosure and management of potential conflicts of interest,[4] and focus on science rather than treatment guidelines enabled the 380 international participants at the 2005 Consensus Conference ultimately to achieve consensus constructively and transparently. Participants agreed to focus on the few factors known to have the greatest impact on outcome, specifically recommendations most likely to improve survival rates without adding to the complexity of rescuer training. It was feared that complexity of training could have a negative impact by reducing attention to the most important factors.

There was unanimity about the need for increased emphasis on the quality of CPR, particularly the quality and number of chest compressions provided and the need to minimise interruptions in chest compressions. Participants also considered the need for altering the sequence of actions (i.e. compression first or shock delivery first) based on the interval from collapse of the victim to the arrival of rescuers (i.e. on the phase of resuscitation).

Selection and debate of controversial topics during the 2005 Consensus Conference

Plenary sessions were scheduled daily for presentation and additional debate on the most controversial issues from the previous day. Controversial topics were identified by panel moderators, conference participants, and the International Liaison Committee on Resuscitation (ILCOR) task force cochairs. During the final day of the conference the entire group of experts focused on the most controversial issue of the conference: selection and sequence of the critical actions needed

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to treat sudden cardiac arrest (SCA). This session crystallised discussion of controversial topics that had been debated daily and enabled the group to reach consensus on these topics. The topics included the relative merits of a compression-first sequence versus a shock-first sequence for treatment of ventricular fibrillation (VF) SCA, the compression—ventilation ratio, and the concept of a one-shock strategy (followed by immediate CPR) versus the three-shock strategy for treatment of VF/pulseless ventricular tachycardia (VT), and other topics (see below).

Summary of debate and decision about the most controversial topics

Compression first versus shock first for VF SCA

Recent data challenge the standard practice of providing defibrillation first to every victim with VF, particularly when 4—5 min or longer has elapsed from collapse to rescuer intervention. Only three human studies plus a somewhat larger body of animal data were available for experts to consider.

If the emergency medical services (EMS) response interval (interval between call to EMS and EMS arrival) for out-of-hospital VF arrest is more than 4—5 min, a period of CPR before attempted defibrillation may improve outcome.[5,6] If all of the human evidence had been positive, there would have been no debate. But one randomised study (LOE 2)[7] failed to show any effect of CPR before defibrillation at any collapse-to-response or collapse-to-defibrillation interval. An added factor is the realisation that rescuers may not know the interval since collapse of the victim.

Some conference participants proposed a treatment recommendation for rescuers to ''perform CPR for 3 min (or some specified interval or number of CPR cycles) before the first shock if more than 4—5 min had elapsed since arrest.'' Animal evidence[8—10] and one large case series (LOE 5)[11] suggests that ventilation is unnecessary for the first few minutes after primary VF cardiac arrest. But ventilation is important in asphyxial arrest (e.g. most arrests in children and many noncardiac arrests, such as drowning and drug overdose). Some conference participants suggested that recommendations provide the option of omitting ventilation for the first few minutes unless the victim is a child or the possibility of asphyxial cardiac arrest exists (e.g. drowning). To simplify lay rescuer education, the consensus among conference participants was to strive for a universal sequence of resuscitation by lay rescuers that would be identical for all victims.

Because the improvement in survival rates associated with provision of CPR before defibrillation was observed only in the subset of victims for whom EMS response intervals were 4—5 min or longer, the consensus was that there were insufficient data to justify recommending CPR before defibrillation for all victims of VF SCA. The experts wanted the treatment recommendations to allow rescuers the option of providing CPR first, particularly for out-of-hospital cardiac arrest in settings where the EMS response interval is >4—5 min. Therefore, the final decision was that 1.5—3 min of CPR before attempting defibrillation may be considered for treatment of out-of-hospital VF or pulseless VT when the EMS response interval is typically greater than 4—5 min.

There were insufficient data to determine (1) whether this recommendation should be applied to in-hospital cardiac arrest; (2) the ideal duration of CPR before attempted defibrillation; or (3) the duration of VF at which rescuers should switch from defibrillation first to CPR first.

Compression—ventilation ratio

The compression—ventilation ratio was one of the most controversial topics of the conference. The experts began the conference acknowledging that rates of survival to hospital discharge from witnessed out-of-hospital VF SCA are low, averaging $\leq$6% internationally (LOE 5),[12—14] and that survival rates have not increased substantially in recent years.[6] The North American Public Access Defibrillation trial showed that lay rescuer AED programs produced higher survival than lay rescuer CPR programs without AEDs, and that organised lay rescuer AED and CPR programs improved survival for witnessed VF SCA over the international average of 6%.[15] High (49—74%) survival rates for out-of-hospital witnessed VF SCA have been reported in some lay rescuer programs using CPR plus automated external defibrillation (AED) in casinos (LOE 5),[16] airports (LOE 5),[17] and commercial passenger planes (LOE 5),[18,19] and in some first responder AED programs (LOE 2,[20] LOE 3,[21,22] LOE 4,[23] and LOE 5[24]). Typically the higher rates were associated with provision of both early CPR and early (within 3—5 min of collapse) defibrillation.

No human data have identified an optimal compression—ventilation ratio for CPR in victims of any age. Compelling animal data indicate that frequent and prolonged interruption of chest compressions is deleterious. Recent clinical data showed frequent hands-off periods without chest

compressions even for advanced CPR providers in both out-of-hospital[25] and in-hospital[26] settings, and laypeople require hands-off intervals of 14—16 s (during which chest compressions are interrupted) to give two rescue breaths.[27,28]

In animal models better results were achieved with a compression—ventilation ratio higher than 15:2.[29] In animals with sudden VF cardiac arrest and open airways, good results were achieved with continuous compressions without any ventilatory support.[30] One study of dispatcher-assisted CPR with apparent cardiac arrest and short (4 min) EMS call-to-ambulance response intervals had good results with chest compressions only.[31] However, it is difficult to determine the relevance of these studies to victims of out-of-hospital arrest with no patent airway, victims of asphyxial arrest, and victims in areas where EMS response intervals are longer than 4 min.

There was substantial evidence that the current practice of CPR provides too much ventilation to victims of cardiac arrest. Participants agreed that fewer ventilations are needed during CPR than previously recommended. One observational study showed that experienced paramedics provided ventilations at excessive rates to intubated patients during treatment for out-of-hospital cardiac arrest and that these excessive rates of ventilation persisted despite intensive retraining (LOE 5).[32] An in-hospital study also showed delivery of ventilation at excessive rates during CPR to patients with and without an advanced airway in place.[26] Although no human outcome studies were identified, one animal study showed that hyperventilation is associated with excessive intrathoracic pressure, decreased coronary and cerebral perfusion pressures, and decreased rates of survival (LOE 6).[32]

The obvious challenge was how to translate the need to increase chest compressions into recommendations that would be simple and appropriate for both asphyxial and VF cardiac arrest. There was agreement that continuous chest compressions could be appropriate in the first minutes of VF arrest, but ventilations would be more important for asphyxial arrest and all forms of prolonged arrest. There was also agreement that it would be too complicated to teach lay rescuers different sequences of CPR for different circumstances. For simplicity, a universal compression—ventilation ratio of 30:2 for lone rescuers of victims from infancy (excluding neonates) through adulthood was agreed on by consensus based on integration of the best human, animal, manikin, and theoretical models available. For two-rescuer CPR in children, a compression—ventilation ratio of 15:2 was recommended.

Oxygenation and ventilation are crucial for the newborn infant and few newborn infants require chest compressions. No new data were discussed to support a higher compression—ventilation ratio in newborns. For this reason, the 3:1 compression—ventilation ratio was retained for newborns.

One-shock versus three-shock sequence for attempted defibrillation

The *ECC Guidelines* 2000[33] recommended the use of a stacked sequence of up to three shocks without interposed chest compressions if VF/VT persists after the first or second shock. The 2005 Consensus Conference participants challenged this strategy, partly because the three shocks require prolonged interruption of compressions that is likely to be needless in the face of relatively high first-shock efficacy (defined as termination of VF for at least five seconds following the shock) of modern biphasic defibrillators.[34]

Researchers found no studies of three-shock defibrillation compared with one-shock defibrillation strategies in humans or animals. But there was consensus that interruptions in effective CPR should be minimised. Several relevant studies reported on the magnitude of success of initial or subsequent shocks, and these studies were compared to determine success rates for shocks. The experts reached consensus that the best overall strategy would be to recommend delivery of one shock with immediate resumption of CPR, beginning with chest compressions, with no check of rhythm or pulse until after a period of CPR.

Resumption of chest compressions immediately after each shock is novel and not based on outcome data. This recommendation follows concern about the excessive interruptions in chest compressions during resuscitation and the dramatic fall in predicted return of spontaneous circulation (ROSC) with even short periods of no compressions before defibrillation attempts.[35]

Shock dose

The recommendation to use a one-shock strategy creates a new challenge: to define the optimal energy for the initial shock. The consensus is that for the initial shock it is reasonable to use selected energies of 150—200 J for a biphasic truncated exponential waveform or 120 J for a rectilinear biphasic waveform.

In a study of out-of-hospital cardiac arrest, first-shock efficacy was no higher using a 360-J shock than a 200-J shock, and repeated shocks at the

higher dose were associated with more atrioventricular block but no evidence of long-term harm.[36] The consensus recommendation was that when using a monophasic waveform defibrillator, it is reasonable to use 360 J for the initial and subsequent shocks.

Role of vasopressors in treatment of cardiac arrest

One of the most contentious topics debated during the conference was the role of vasopressin in advanced life support. It was conceded that despite the widespread use of epinephrine and several studies involving vasopressin, no placebo-controlled study shows that routine administration of any vasopressor at any stage during human cardiac arrest increases rates of survival to hospital discharge. Despite animal data indicating the advantages of vasopressin over epinephrine, a meta-analysis of five randomised trials showed no statistically significant differences between vasopressin and epinephrine for ROSC, death within 24 h, or death before hospital discharge.[37] Individual resuscitation councils will need to determine the role of vasopressin in their resuscitation guidelines.

Post-resuscitation care

Optimal treatment in the post-resuscitation period has not been well researched and is not standardised across healthcare communities.[38] In two studies therapeutic hypothermia improved neurological outcome among initially comatose survivors from out-of-hospital VF cardiac arrest, but the role of this therapy after in-hospital cardiac arrest or arrest from other rhythms remains inconclusive.[39,40] It is hoped that additional studies will add precision to our use of hypothermia in the future.

Summary

We acknowledge the limited data that we have to support many resuscitation interventions; further research is needed in virtually all facets of CPR and ECC. Ethics committees must empower investigators to challenge the unproven dogma that we have tolerated for far too long.

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Part 1: Introduction

International Liaison Committee on Resuscitation

Toward international consensus on science

The International Liaison Committee on Resuscitation (ILCOR) was formed in 1993. Its mission is to identify and review international science and knowledge relevant to cardiopulmonary resuscitation (CPR) and emergency cardiovascular care (ECC) and to offer consensus on treatment recommendations.[1] Emergency cardiovascular care includes all responses necessary to treat sudden life-threatening events affecting the cardiovascular and respiratory systems but with a particular focus on sudden cardiac arrest.

In 1999, the American Heart Association (AHA) hosted the first ILCOR conference to evaluate resuscitation science and develop common resuscitation guidelines. The conference recommendations were published in the international *Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care*.[2,3] Since that time researchers from the ILCOR member councils have continued to evaluate resuscitation science in a process that culminated in the *2005 International Consensus Conference on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations* (2005 Consensus Conference). This publication summarises the conclusions and recommendations of that evidence evaluation process.

The goal of every resuscitation organisation and resuscitation expert is to prevent premature cardiovascular death. When cardiac arrest or life-threatening emergencies occur, prompt and skilful response can make the difference between life and death and between intact survival and debilitation. This document summarises current evidence for the recognition and response to sudden life-threatening events, particularly sudden cardiac arrest in victims of all ages. The broad range and number of topics reviewed and the inevitable limitations of journal space require succinctness in science statements and, where recommendations were appropriate, brevity in treatment recommendations. This is not a comprehensive review of every aspect of resuscitation medicine; some topics were omitted if there was no evidence or no new information.

Evidence evaluation process

To begin the current evidence evaluation process, ILCOR representatives established six task forces: basic life support, advanced life support, acute coronary syndromes, paediatric life support, neonatal life support, and an interdisciplinary task force to consider overlapping topics such as educational issues. Each task force identified topics requiring evidence evaluation and appointed international experts to review them. To ensure a consistent and thorough approach, a worksheet template was created with step-by-step directions to help the experts document their literature review (Table 1.1), evaluate studies, determine levels of evidence (Table 1.2), and develop treatment recommendations. When possible, two expert reviewers were recruited to undertake independent evaluations for each topic. In addition, two evidence evaluation experts reviewed all worksheets and assisted the worksheet reviewers to ensure that the worksheets met a consistently high standard. This process is described in detail in an accompanying editorial.[4] Two additional task forces were established by the AHA to review evidence about stroke

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Table 1.1 Steps in evidence integration

Integrate all evidence following these steps:
1. Perform literature review and record search terms and databases searched
2. Select studies relevant to hypothesis
3. Determine level of evidence based on methodology (see Table 1.2)
4. Perform critical appraisal (*poor to excellent*)
5. Integrate evidence into a science summary and possible treatment recommendation

Experts must develop consensus based on scientific evidence. Steps used include:
Evidence evaluation and worksheet preparation by experts, plus
2005 Consensus Conference presentations and discussions
ILCOR task force discussions
Approval by ILCOR member organisations
Final editorial review and approval by international editorial board
Blinded peer review
Publication

and first aid. These topics were included in the 2005 Consensus Conference, but they were not part of the ILCOR process.

A total of 281 experts completed 403 worksheets on 276 topics. Two hundred and forty-nine worksheet authors (141 from the United States and 108 from 17 other countries) attended the 2005 Consensus Conference. In December 2004,

Table 1.2 Levels of evidence

Evidence	Definition
Level 1	Randomised clinical trials or meta-analyses of multiple clinical trials with substantial treatment effects
Level 2	Randomised clinical trials with smaller or less significant treatment effects
Level 3	Prospective, controlled, non-randomised cohort studies
Level 4	Historic, non-randomised cohort or case-control studies
Level 5	Case series; patients compiled in serial fashion, control group lacking
Level 6	Animal studies or mechanical model studies
Level 7	Extrapolations from existing data collected for other purposes, theoretical analyses
Level 8	Rational conjecture (common sense); common practices accepted before evidence-based guidelines

the evidence review and summary portions of the evidence evaluation worksheets, with worksheet author conflict of interest statements, were posted on the Internet at http://www.c2005.org. Journal advertisements and e-mails invited public comment. Persons who submitted comments were required to indicate their potential conflicts of interest. Such comments were sent to the appropriate ILCOR task force chair and worksheet author for consideration.

To provide the widest possible dissemination of the science reviews performed for the 2005 Consensus Conference, the worksheets prepared for the conference are linked from the electronic version of this document. Worksheet numbers begin with W to distinguish them from other reference citations. Most worksheet numbers are located adjacent to headings rather than in the body of the text. Readers of the electronic version of this supplement can access a cited worksheet by clicking on the linked callout. Readers of the printed publication can identify the complete title and author of a cited worksheet by referring to the numbered worksheet list at the end of this issue (Appendix 1) and then accessing that worksheet on the website: http://www.c2005.org.

All 380 participants at the 2005 Consensus Conference received a copy of the worksheets on CD-ROM. Internet access was available to all conference participants during the conference to facilitate real-time verification of the literature. Expert reviewers presented topics in plenary, concurrent, and poster conference sessions, expert reviewers presented each topic. Presenters and participants then debated the evidence, conclusions, and draft summary statements. Each day the most controversial topics from the previous day, as identified by the task force chairs, were presented and debated in one or more additional sessions. The ILCOR task forces met daily during the conference to discuss and debate the experts' recommendations and develop interim consensus science statements. Each science statement summarised the experts' interpretation of all the relevant data on a specific topic. Draft treatment recommendations were added if a consensus was reached. Wording of science statements and treatment recommendations were refined after further review by ILCOR member organisations and the international editorial board. This format ensured that this final document represents a truly international consensus process.

At the time of submission this document represented a summary of the state-of-the-art science of many topics in resuscitation medicine. Several papers that were accepted for publication in

a peer-reviewed journal before the 2005 Consensus Conference but had not yet been published were circulated, with the permission of the relevant journal editors, to the ILCOR task forces and contributed to the consensus statements.

This manuscript was ultimately approved by all ILCOR member organisations and by an international editorial board (listed on the title page of this issue). The AHA Science Advisory and Coordinating Committee and the editor of *Circulation* obtained peer reviews of this document before it was accepted for publication. The document is being published simultaneously in *Circulation* and *Resuscitation*, although the version in *Resuscitation* does not include the sections on stroke and first aid.

Management of conflict of interest

The world's leading experts in resuscitation science establish their expertise by undertaking and publishing research and related scholarly work (e.g. presentation of research abstracts and participation in scientific conferences). This work potentially creates financial and intellectual conflicts of interest (COI) for the expert.[5,6] Grants and other support for scientific research, speaker fees, and honoraria can also create financial conflicts of interest. Non-financial conflicts of interest include in-kind support, intellectual collaboration or intellectual investment in personal ideas, and long-term research agendas in which investigators have invested a substantial amount of time. A robust COI policy was developed to ensure full disclosure of potential conflicts and to protect the objectivity and credibility of the evidence evaluation and consensus development process. This policy is described in detail in an accompanying editorial.[7] Representatives of manufacturers and industry did not participate in this conference.

Potential conflicts of interest of the editorial board are listed in Appendix 3 at the end of this issue. Potential conflicts of interest of the worksheet authors are included in the worksheets and can be accessed through the links to the worksheets contained in this document and also from the worksheet home page at http://www.c2005.org. All 380 attendees were required to complete forms in order to document their potential conflicts of interest. Most attendees were also worksheet authors. The information from the conflict of interest forms completed by all conference attendees, including those who are not worksheet authors, can also be accessed at doi: 10.1016/j.resuscitation.2005.11.001 or www.elsevier.com/locate/resuscitation.

Applying science to improve survival

From consensus on science to guidelines

This document presents international consensus statements on the science of resuscitation and, wherever possible, treatment recommendations. ILCOR member organisations will publish resuscitation guidelines subsequently that are consistent with the science in this consensus document, but they will also take into account geographic, economic, and system differences in practice and the availability of medical devices and drugs. All ILCOR member organisations strive to minimise international differences in resuscitation practice and to optimise the effectiveness of instructional methods, teaching aids, and training networks.

The recommendations of the 2005 Consensus Conference confirm the safety and effectiveness of some current approaches, acknowledge that other approaches may not be optimal, and introduce new treatments resulting from evidence-based evaluation. *New and revised treatment recommendations do not imply that clinical care that involves the use of previously published guidelines is unsafe*. ILCOR scientists and member organisations consider these new recommendations to be the most effective and easily learned interventions that can be supported by current knowledge, research, and experience. Implications for education and retention were also considered when developing the final treatment recommendations.

Ischaemic heart disease is the leading cause of death in the world.[8] Sudden cardiac arrest is responsible for >60% of the estimated 340,000 annual deaths from coronary heart disease in emergency departments or out-of-hospital in the United States.[8,9] Most victims die out of hospital without receiving the interventions described in this publication. The actions linking the victim of sudden cardiac arrest with survival are called the adult Chain of Survival. The links in the Chain of Survival are early recognition of the emergency and activation of the emergency medical services (EMS) system, early CPR, early defibrillation, and early advanced life support, including post-resuscitation care. The links in the infant and child Chain of Survival are prevention of conditions leading to cardiopulmonary arrest, early CPR, early activation of the EMS system, and early advanced life support.

The most important determinant of survival from sudden cardiac arrest is the presence of a trained lay rescuer who is ready, willing, able, and equipped to act. Although some advanced life support techniques may improve survival,[10] these improvements are usually less significant than the

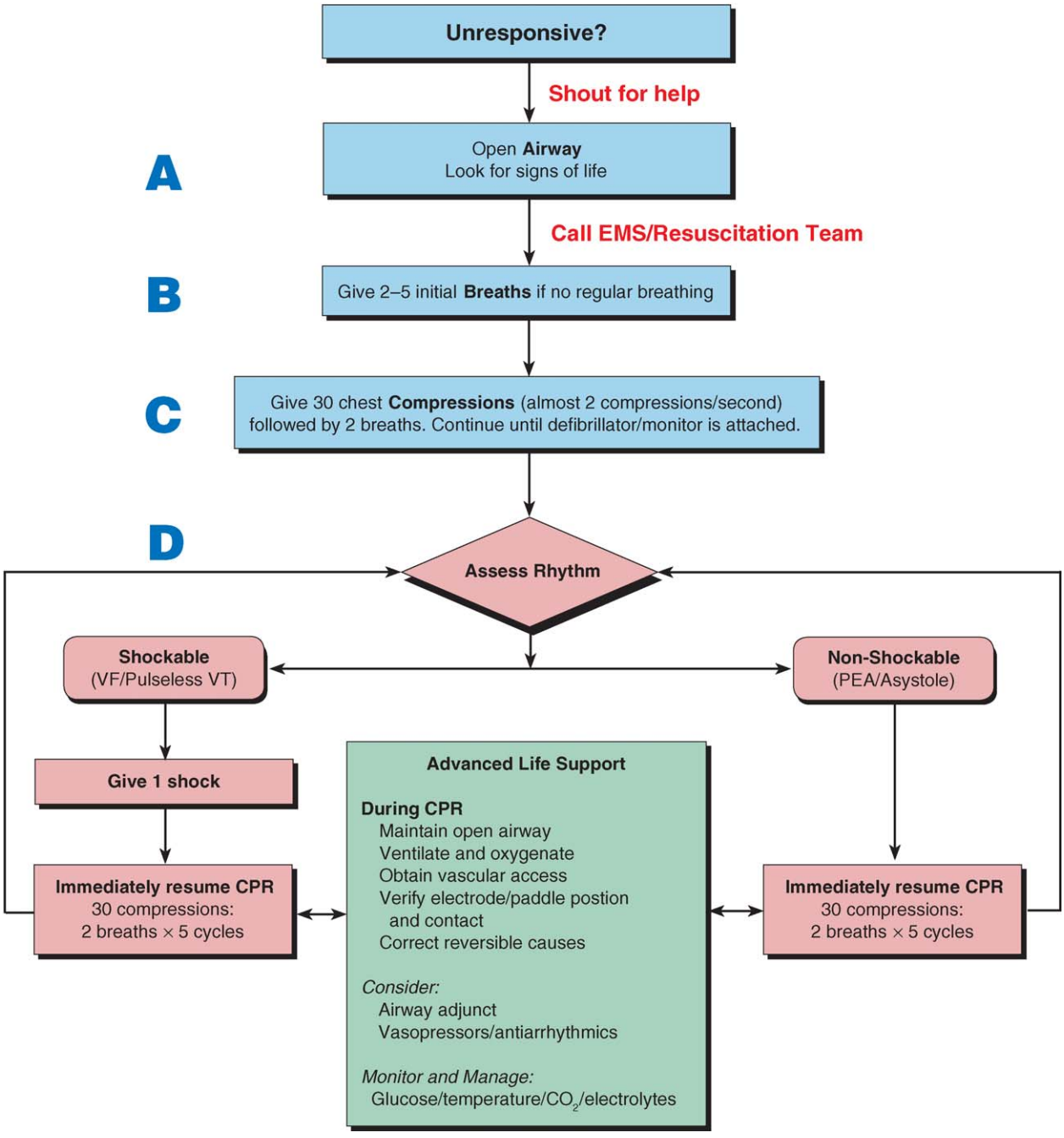

Figure 1.1 ILCOR Universal Cardiac Arrest Algorithm.

increased survival rates reported by lay rescuer CPR and automated external defibrillation programs in the community.[11–15] Thus, our greatest challenge remains the education of the lay rescuer. We must increase the effectiveness and efficiency of instruction, improve skills retention, and reduce barriers to action for both basic and advanced life support providers.[16,17] The science of resuscitation education is addressed in this publication.

The Universal Algorithm

Several of the new treatment recommendations to emerge from this document are included in the updated ILCOR Universal Cardiac Arrest Algorithm (Figure 1.1). This algorithm is intended to apply to attempted resuscitation of infant, child, and adult victims of cardiac arrest (excluding newborns). Every effort has been made to keep this

algorithm simple yet make it applicable to cardiac arrest victims of all ages and in most circumstances. Inevitably modification will be required in some situations, and these exceptions are highlighted elsewhere in this document. Each resuscitation organisation will base its guidelines on this ILCOR algorithm, although there will be subtle regional modifications.

Rescuers begin CPR if the victim is unconscious or unresponsive, not moving, and not breathing (ignoring occasional gasps). A single compression—ventilation ratio of 30:2 is used for the single rescuer of an infant, child, or adult victim (excluding newborns); this applies for the lay rescuer and for all adult CPR. This single ratio is designed to simplify teaching, promote skills retention, increase the number of compressions given, and decrease interruption to compressions.

Once a defibrillator is attached, if a ''shockable'' rhythm (i.e. ventricular fibrillation or rapid ventricular tachycardia) is confirmed, a single shock is delivered. Irrespective of the resultant rhythm, chest compressions and ventilations (five cycles of 30:2—approximately 2 min) are resumed immediately after the shock to minimise the ''no flow'' time (i.e. time during which compressions are not delivered for actions such as rhythm analysis). Advanced life support interventions are outlined in a box at the centre of the algorithm. Once an advanced airway (e.g. tracheal tube, laryngeal mask airway [LMA] or Combitube) has been inserted, during two-rescuer CPR, one rescuer should provide 8—10 ventilations min^{-1} while the other delivers 100 compressions min^{-1}. The rescuer performing the chest compressions should not pause chest compressions for delivery of ventilations.

The theme of minimal interruption of chest compressions is emphasised throughout this document; recent evidence indicates that such interruptions occur frequently both in and out of hospital.[18—20] Interruptions in chest compressions during CPR must be minimised.

Future directions

The science of resuscitation is evolving rapidly. It would not be in the best interests of patients if we waited five or more years to inform healthcare professionals of therapeutic advances in this field. ILCOR members will continue to review new science and, when necessary, publish interim advisory statements to update treatment guidelines so that resuscitation practitioners may provide state-of-the-art treatment. Existing gaps in our knowledge will be closed only by continuing high-quality research into all facets of CPR.

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Part 2: Adult basic life support

International Liaison Committee on Resuscitation

The consensus conference addressed many questions related to the performance of basic life support. These have been grouped into (1) epidemiology and recognition of cardiac arrest, (2) airway and ventilation, (3) chest compression, (4) compression—ventilation sequence, (5) postresuscitation positioning, (6) special circumstances, (7) emergency medical services (EMS) system, and (8) risks to the victim and rescuer. Defibrillation is discussed separately in Part 3 because it is both a basic and an advanced life support skill.

There have been several important advances in the science of resuscitation since the last ILCOR review in 2000. The following is a summary of the evidence-based recommendations for the performance of basic life support:

- Rescuers begin CPR if the victim is unconscious, not moving, and not breathing (ignoring occasional gasps).
- For mouth-to-mouth ventilation or for bag-valve-mask ventilation with room air or oxygen, the rescuer should deliver each breath in 1 s and should see visible chest rise.
- Increased emphasis on the process of CPR: push hard at a rate of 100 compressions per min, allow full chest recoil, and minimise interruptions in chest compressions.
- For the single rescuer of an infant (except newborns), child, or adult victim, use a single compression—ventilation ratio of 30:2 to simplify teaching, promote skills retention, increase the number of compressions given, and decrease interruptions in compressions. During two-rescuer CPR of the infant or child, healthcare providers should use a 15:2 compression—ventilation ratio.
- During CPR for a patient with an advanced airway (i.e. tracheal tube, Combitube, laryngeal mask airway [LMA]) in place, deliver ventilations at a rate of 8—10 per min for infants (excepting neonates), children and adults, without pausing during chest compressions to deliver the ventilations.

Epidemiology and recognition of cardiac arrest

Many people die prematurely from sudden cardiac arrest (SCA), often associated with coronary heart disease. The following section summarises the burden, risk factors, and potential interventions to reduce the risk.

Epidemiology

Incidence
W137, W138A

Consensus on science. Approximately 400,000—460,000 people in the United States (LOE 5)[1] and 700,000 people in Europe (LOE 7)[2] experience SCA each year; resuscitation is attempted in approximately two thirds of these victims.[3] Case series and cohort studies showed wide variation in the incidence of cardiac arrest, depending on the method

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of assessment:

1.5 per 1000 person-years based on death certificates (LOE 5),[4]
0.5 per 1000 person-years based on activation of emergency medical services (EMS) systems (LOE 5).[5,6]

In recent years the incidence of ventricular fibrillation (VF) at first rhythm analysis has declined significantly.[7–9]

Prognosis
W138B

Consensus on science. Since the previous international evidence evaluation process (the International Guidelines 2000 Conference on CPR and ECC),[10] there have been three systematic reviews of survival-to-hospital discharge from out-of-hospital cardiac arrest (LOE 5).[5,11,12] Of all victims of cardiac arrest treated by EMS providers, 5–10% survive; of those with VF, 15% survive to hospital discharge. In data from a national registry, survival to discharge from in-hospital cardiac arrest was 17% (LOE 5).[13] The aetiology and presentation of in-hospital arrest differ from that of out-of-hospital arrests.

Risk of cardiac arrest is influenced by several factors, including demographic, genetic, behavioural, dietary, clinical, anatomical, and treatment characteristics (LOE 4–7).[4,14–19]

Recognition

Early recognition is a key step in the early treatment of cardiac arrest. It is important to determine the most accurate method of diagnosing cardiac arrest.

Signs of cardiac arrest
W142A,W142B

Consensus on science. Checking the carotid pulse is an inaccurate method of confirming the presence or absence of circulation (LOE 3)[20]; however, there is no evidence that checking for movement, breathing, or coughing (i.e. ''signs of circulation'') is diagnostically superior (LOE 3).[21,22] Agonal gasps are common in the early stages of cardiac arrest (LOE 5).[23] Bystanders often report to dispatchers that victims of cardiac arrest are ''breathing'' when they demonstrate agonal gasps; this can result in the withholding of CPR from victims who might benefit from it (LOE 5).[24]

Treatment recommendation. Rescuers should start CPR if the victim is unconscious (unresponsive), not moving, and not breathing. Even if the victim takes occasional gasps, rescuers should suspect that cardiac arrest has occurred and should start CPR.

Airway and ventilation

The best method of obtaining an open airway and the optimum frequency and volume of artificial ventilation were reviewed.

Airway

Opening the airway
W149

Consensus on science. Five prospective clinical studies evaluating clinical (LOE 3)[25,26] or radiological (LOE 3)[27–29] measures of airway patency and one case series (LOE 5)[30] showed that the head tilt–chin lift manoeuvre is feasible, safe, and effective. No studies have evaluated the routine use of the finger sweep manoeuvre to clear an airway in the absence of obvious airway obstruction.

Treatment recommendation. Rescuers should open the airway using the head tilt–chin lift manoeuvre. Rescuers should use the finger sweep in the unconscious patient with a suspected airway obstruction only if solid material is visible in the oropharynx.

Devices for airway positioning
W1,W49A,W49B

Consensus on science. There is no published evidence on the effectiveness of devices for airway positioning. Collars that are used to stabilise the cervical spine can make airway management difficult and increase intracranial pressure (LOE 4[31–33]; LOE 5[34]).

Foreign-body airway obstruction
W151A,W151B

Like CPR, relief of foreign-body airway obstruction (FBAO) is an urgent procedure that should be taught to laypersons. Evidence for the safest, most effective, and simplest methods was sought.

Consensus on science. It is unclear which method of removal of FBAO should be used first. For conscious victims, case reports showed success in relieving FBAO with back blows (LOE 5),[35–37]

abdominal thrusts (LOE 5),[36–44] and chest thrusts (LOE 5).[36] Frequently, more than one technique was needed to achieve relief of the obstruction.[36,45–50] Life-threatening complications have been associated with the use of abdominal thrusts (LOE 5).[48,51–72]

For unconscious victims, case reports showed success in relieving FBAO with chest thrusts (LOE 5)[49] and abdominal thrusts (LOE 5).[73] One randomised trial of manoeuvres to clear the airway in cadavers (LOE 7)[74] and two prospective studies in anaesthetised volunteers (LOE 7)[75,76] showed that higher airway pressures can be generated by using the chest thrust rather than the abdominal thrust.

Case series (LOE 5)[36,37,45] reported the finger sweep as effective for relieving FBAO in unconscious adults and children aged >1 year. Four case reports documented harm to the victim's mouth (LOE 7)[77,78] or biting of the rescuer's finger (LOE 7).[29,30]

Treatment recommendation. Chest thrusts, back blows, or abdominal thrusts are effective for relieving FBAO in conscious adults and children >1 year of age, although injuries have been reported with the abdominal thrust. There is insufficient evidence to determine which should be used first. These techniques should be applied in rapid sequence until the obstruction is relieved; more than one technique may be needed. Unconscious victims should receive CPR. The finger sweep can be used in the unconscious patient with an obstructed airway if solid material is visible in the airway. There is insufficient evidence for a treatment recommendation for an obese or pregnant patient with FBAO.

Ventilation

Mouth-to-nose ventilation
W157A, W157B

Consensus on science. A case series suggested that mouth-to-nose ventilation of adults is feasible, safe, and effective (LOE 5).[79]

Treatment recommendation. Mouth-to-nose ventilation is an acceptable alternative to mouth-to-mouth ventilation.

Mouth-to-tracheal stoma ventilation
W158A, 158B

Consensus on science. There was no published evidence of the safety or effectiveness of mouth-to-stoma ventilation. A single crossover study of patients with laryngectomies showed that a paediatric face mask provided a better seal around the stoma than a standard ventilation mask (LOE 4).[80]

Treatment recommendation. It is reasonable to perform mouth-to-stoma breathing or to use a well-sealing, round pediatric face mask.

Tidal volumes and ventilation rates
W53, W156A

Consensus on science. There was insufficient evidence to determine how many initial breaths should be given. Manikin studies (LOE 6)[81–83] and one human study (LOE 7)[84] showed that when there is no advanced airway (such as a tracheal tube, Combitube, or LMA) in place, a tidal volume of 1 L produced significantly more gastric inflation than a tidal volume of 500 mL. Studies of anaesthetised patients with no advanced airway in place showed that ventilation with 455 mL of room air was associated with an acceptable but significantly reduced oxygen saturation when compared with 719 mL (LOE 7).[85] There was no difference in oxygen saturation with volumes of 624 and 719 mL (LOE 7).[85] A study of cardiac arrest patients compared tidal volumes of 500 mL versus 1000 mL delivered to patients with advanced airways during mechanical ventilation with 100% oxygen at a rate of 12 min^{-1} (LOE 2).[86] Smaller tidal volumes were associated with higher arterial PCO_2 and worse acidosis but no differences in PaO_2.

Reports containing both a small case series (LOE 5) and an animal study (LOE 6)[87,88] showed that hyperventilation is associated with increased intrathoracic pressure, decreased coronary and cerebral perfusion, and, in animals, decreased return of spontaneous circulation (ROSC). In a secondary analysis of the case series that included patients with advanced airways in place after out-of-hospital cardiac arrest, ventilation rates of >10 min^{-1} and inspiration times >1 s were associated with no survival (LOE 5).[87,88] Extrapolation from an animal model of severe shock suggests that a ventilation rate of six ventilations per minute is associated with adequate oxygenation and better haemodynamics than $\geq$12 ventilations min^{-1} (LOE 6).[89] In summary, larger tidal volumes and ventilation rates can be associated with complications, whereas the detrimental effects observed with smaller tidal volumes appear to be acceptable.

Treatment recommendation. For mouth-to-mouth ventilation with exhaled air or bag-valve-mask ventilation with room air or oxygen, it

is reasonable to give each breath within a 1-s inspiratory time to achieve chest rise. After an advanced airway (e.g. tracheal tube, Combitube, LMA) is placed, ventilate the patient's lungs with supplementary oxygen to make the chest rise. During CPR for a patient with an advanced airway in place, it is reasonable to ventilate the lungs at a rate of 8–10 ventilations min^{-1} without pausing during chest compressions to deliver ventilations. Use the same initial tidal volume and rate in patients regardless of the cause of the cardiac arrest.

Mechanical ventilators and automatic transport ventilators
W55, W152A

Consensus on science. Three manikin studies of simulated cardiac arrest showed a significant decrease in gastric inflation with manually triggered, flow-restricted, oxygen-powered resuscitators when compared with ventilation by bag-valve-mask (LOE 6).[90–92] One study showed that firefighters who ventilated anaesthetised patients with no advanced airway in place produced less gastric inflation and lower peak airway pressure with manually triggered, flow-limited, oxygen-powered resuscitators than with a bag-valve-mask (LOE 5).[93] A prospective cohort study of intubated patients, most in cardiac arrest, in an out-of-hospital setting showed no significant difference in arterial blood gas values between those ventilated with an automatic transport ventilator and those ventilated manually (LOE 4).[94] Two laboratory studies showed that automatic transport ventilators can provide safe and effective management of mask ventilation during CPR of adult patients (LOE 6).[95,96]

Treatment recommendation. There are insufficient data to recommend for or against the use of a manually triggered, flow-restricted resuscitator or an automatic transport ventilator during bag-valve-mask ventilation and resuscitation of adults in cardiac arrest.

Chest compressions

Several components of chest compressions can alter effectiveness: hand position, position of the rescuer, position of the victim, depth and rate of compression, decompression, and duty cycle (see definition below). Evidence for these techniques was reviewed in an attempt to define the optimal method.

Chest compression technique

Hand position
W167A, W167C

Consensus on science. There was insufficient evidence for or against a specific hand position for chest compressions during CPR in adults. In children who require CPR, compression of the lower one third of the sternum may generate a higher blood pressure than compressions in the middle of the chest (LOE 4).[97]

Manikin studies in healthcare professionals showed improved quality of chest compressions when the dominant hand was in contact with the sternum (LOE 6).[98] There were shorter pauses between ventilations and compressions if the hands were simply positioned ''in the center of the chest'' (LOE 6).[99]

Treatment recommendation. It is reasonable for laypeople and healthcare professionals to be taught to position the heel of their dominant hand in the centre of the chest of an adult victim, with the non-dominant hand on top.

Chest compression rate, depth, decompression, and duty cycle
W167A, W167B, W167C

Consensus on science

Rate. The number of compressions delivered per minute is determined by the compression rate, the compression–ventilation ratio, the time required to provide mouth-to-mouth or bag-valve-mask ventilation, and the strength (or fatigue) of the rescuer. Observational studies showed that responders give fewer compressions than currently recommended (LOE 5).[100–103] Some studies in animal models of cardiac arrest showed that high-frequency CPR (120–150 compressions min^{-1}) improved haemodynamics without increasing trauma when compared with standard CPR (LOE 6),[104–107] whereas others showed no effect (LOE 6).[108] Some studies in animals showed more effect from other variables, such as duty cycle (see below).[109] In humans, high-frequency CPR (120 compressions min^{-1}) improved haemodynamics over standard CPR (LOE 4).[110] In mechanical CPR in humans, however, high-frequency CPR (up to 140 compressions min^{-1}) showed no improvement in haemodynamics when compared with 60 compressions min^{-1} (LOE 5).[111,112]

Depth. In both out-of-hospital[102] and in-hospital[100] studies, insufficient depth of compression was observed during CPR when compared

with currently recommended depths (LOE 5).[100,102] Studies in animal models of adult cardiac arrest showed that deeper compressions (i.e. 3—4 in.) are correlated with improved ROSC and 24-h neurological outcome when compared with standard-depth compressions (LOE 6).[107,113,114] A manikin study of rescuer CPR showed that compressions became shallow within one minute, but providers became aware of fatigue only after 5 min (LOE 6).[115]

Decompression. One observational study in humans (LOE 5)[88] and one manikin study (LOE 6) [116] showed that incomplete chest recoil was common during CPR. In one animal study incomplete chest recoil was associated with significantly increased intrathoracic pressure, decreased venous return, and decreased coronary and cerebral perfusion during CPR (LOE 6).[117] In a manikin study, lifting the hand slightly but completely off the chest during decompression allowed full chest recoil (LOE 6).[116]

Duty cycle. The term *duty cycle* refers to the time spent compressing the chest as a proportion of the time between the start of one cycle of compression and the start of the next. Coronary blood flow is determined partly by the duty cycle (reduced coronary perfusion with a duty cycle >50%) and partly by how fully the chest is relaxed at the end of each compression (LOE 6).[118] One animal study that compared duty cycles of 20% with 50% during cardiac arrest chest compressions showed no statistical difference in neurological outcome at 24 h (LOE 6).[107]

A mathematical model of Thumper CPR showed significant improvements in pulmonary, coronary, and carotid flow with a 50% duty cycle when compared with compression—relaxation cycles in which compressions constitute a greater percentage of the cycle (LOE 6).[119] At duty cycles ranging between 20 and 50%, coronary and cerebral perfusion in animal models increased with chest compression rates of up to 130—150 compressions min^{-1} (LOE 6).[104,105,109] In a manikin study, duty cycle was independent of the compression rate when rescuers increased progressively from 40 to 100 compressions min^{-1} (LOE 6).[120] A duty cycle of 50% is mechanically easier to achieve with practice than cycles in which compressions constitute a smaller percentage of cycle time (LOE 7).[121]

Treatment recommendation. It is reasonable for lay rescuers and healthcare providers to perform chest compressions for adults at a rate of at least 100 compressions min^{-1} and to compress the sternum by at least 4—5 cm. Rescuers should allow complete recoil of the chest after each compression. When feasible, rescuers should frequently alternate ''compressor'' duties, regardless of whether they feel fatigued, to ensure that fatigue does not interfere with delivery of adequate chest compressions. It is reasonable to use a duty cycle (i.e. ratio between compression and release) of 50%.

Firm surface for chest compressions
W167A

Consensus on science. When manikins were placed on a bed supported by a pressure-relieving mattress, chest compressions were less effective than those performed when the manikins were placed on the floor. Emergency deflation of the mattress did not improve the efficacy of chest compressions (LOE 6).[122,123] These studies did not involve standard mattresses or backboards and did not consider the logistics of moving a victim from a bed to the floor.

Treatment recommendation. Cardiac arrest victims should be placed supine on a firm surface (i.e. backboard or floor) during chest compressions to optimise the effectiveness of compressions.

CPR process versus outcome
W182A, W182B, W194

Consensus on science. CPR compression rate and depth provided by lay responders (LOE 5),[124] physician trainees (LOE 5),[100] and EMS personnel (LOE 5)[102] were insufficient when compared with currently recommended methods. Ventilation rates and durations higher or longer than recommended when CPR is performed impaired haemodynamics and reduced survival rates (LOE 6).[88] It is likely that poor performance of CPR impairs haemodynamics and possibly survival rates.

Treatment recommendation. It is reasonable for instructors, trainees, providers, and EMS agencies to monitor and improve the process of CPR to ensure adherence to recommended compression and ventilation rates and depths.

Alternative compression techniques

CPR in the prone position
W166D

Consensus on science. Six case series that included 22 intubated hospitalised patients documented survival to discharge in 10 patients who received CPR in a prone position (LOE 5).[125—130]

Treatment recommendation. CPR with the patient in a prone position is a reasonable alternative for intubated hospitalised patients who cannot be placed in the supine position.

Leg-foot chest compressions
W166C

Consensus on science. Three studies in manikins showed no difference in chest compressions, depth, or rate when leg-foot compressions were used instead of standard chest compressions (LOE 6).[131–133] Two studies[132,133] reported that rescuers felt fatigue and leg soreness when using leg-foot chest compressions. One study[132] reported incomplete chest recoil when leg-foot chest compressions were used.

'Cough' CPR
W166A

Consensus on science. Case series (LOE 5)[134–136] show that repeated coughing every one to three seconds during episodes of rapid VF in supine, monitored, trained patients in the cardiac catheterisation laboratory can maintain a mean arterial pressure > 100 mmHg and maintain consciousness for up to 90 s. No data support the usefulness of cough CPR in any other setting, and there is no specific evidence for or against use of cough CPR by laypersons in unsupervised settings.

Compression—ventilation sequence

Any recommendation for a specific CPR compression—ventilation ratio represents a compromise between the need to generate blood flow and the need to supply oxygen to the lungs. At the same time any such ratio must be taught to would-be rescuers, so that skills acquisition and retention are also important factors.

Effect of ventilations on compressions

Interruption of compressions
W147A,W147B

Consensus on science. In animal studies interruption of chest compressions is associated with reduced ROSC and survival as well as increased postresuscitation myocardial dysfunction (LOE 6).[137–139]

Observational studies (LOE 5)[100,102] and secondary analyses of two randomised trials (LOE 5)[140,141] have shown that interruption of chest compressions is common. In a retrospective analysis of the VF waveform, interruption of CPR was associated with a decreased probability of conversion of VF to another rhythm (LOE 5).[141]

Treatment recommendation. Rescuers should minimise interruptions of chest compressions.

Compression—ventilation ratio during CPR
W154

Consensus on science. An observational study showed that experienced paramedics performed ventilation at excessive rates on intubated patients during treatment for out-of-hospital cardiac arrest (LOE 5).[88] An in-hospital study also showed delivery of excessive-rate ventilation to patients with and without advanced airways in place.[100] Two animal studies showed that hyperventilation is associated with excessive intrathoracic pressure and decreased coronary and cerebral perfusion pressures and survival rates (LOE 6).[87,88]

Observational studies in humans showed that responders gave fewer compressions than currently recommended (LOE 5).[100–102]

Multiple animal studies of VF arrests showed that continuous chest compressions with minimal or no interruptions is associated with better haemodynamics and survival than standard CPR (LOE 6).[137,139,142–144]

Results of varying compression—ventilation ratios in intubated animal models and even theoretical calculations have yielded mixed results. In one animal model of cardiac arrest, use of a compression—ventilation ratio of 100:2 was associated with significantly improved neurological function at 24 h when compared with a ratio of 15:2 or continuous-compression CPR, but there was no significant difference in perfusion pressures or survival rates (LOE 6).[145] In an animal model of cardiac arrest, use of a compression—ventilation ratio of 50:2 achieved a significantly greater number of chest compressions than using either 15:2 or 50:5 (LOE 6).[146] Carotid blood flow was significantly greater at a ratio of 50:2 compared with 50:5 and not significantly different from that achieved with a ratio of 15:2. Arterial oxygenation and oxygen delivery to the brain were significantly higher with a ratio of 15:2 when compared with a ratio of either 50:5 or 50:2. In an animal model of cardiac arrest, a compression—ventilation ratio of 30:2 was associated with significantly shorter time to ROSC and greater systemic and cerebral oxygenation than with continuous chest compressions (LOE 6).[147] A theoretical analysis suggests that a compression—ventilation ratio of 30:2 would provide the best blood flow and oxygen delivery (LOE 7).[148]

An animal model of asphyxial arrest showed that compression-only CPR is associated with significantly greater pulmonary oedema than both compression and ventilation, with or without oxygenation (LOE 6).[149]

Treatment recommendation. There is insufficient evidence that any specific compression—ventilation ratio is associated with improved outcome in patients with cardiac arrest. To increase the number of compressions given, minimise interruptions of chest compressions, and simplify instruction for teaching and skills retention, a single compression—ventilation ratio of 30:2 for the lone rescuer of an infant, child, or adult victim is recommended. Initial steps of resuscitation may include (1) opening the airway while verifying the need for resuscitation, (2) giving 2—5 breaths when initiating resuscitation, and (3) then providing compressions and ventilations using a compression—ventilation ratio of 30:2.

Chest compression-only CPR
W52, W164A, W164B

Consensus on science. No prospective studies have assessed the strategy of implementing chest compression—only CPR. A randomised trial of telephone instruction in CPR given to untrained lay responders in an EMS system with a short (mean: four minutes) response interval suggests that a strategy of teaching chest compressions alone is associated with similar survival rates when compared with a strategy of teaching chest compressions and ventilations (LOE 7).[150]

Animal studies of nonasphyxial arrest demonstrate that chest compression—only CPR may be as efficacious as compression—ventilation CPR in the initial few minutes of resuscitation (LOE 6).[142,150] In another model of nonasphyxial arrest, however, a compression—ventilation ratio of 30:2 maintained arterial oxygen content at two thirds of normal, but compression-only CPR was associated with desaturation within two minutes (LOE 6).[147] In observational studies of adults with cardiac arrest treated by lay responders trained in standard CPR, survival was better with compression-only CPR than with no CPR but not as good as with both compressions and ventilations (LOE 3;[151] LOE 4[124]).

Treatment recommendation. Rescuers should be encouraged to do compression-only CPR if they are unwilling to do airway and breathing manoeuvres or if they are not trained in CPR or are uncertain how to do CPR. Researchers are encouraged to evaluate the efficacy of compression-only CPR.

Postresuscitation positioning

Recovery position
W155, W146A, W146B

Consensus on science. No studies were identified that evaluated any recovery position in an unconscious victim with normal breathing. A small cohort study (LOE 5)[152] and a randomised trial (LOE 7)[153] in normal volunteers showed that compression of vessels and nerves occurs infrequently in the dependent limb when the victim's lower arm is placed in front of the body; however, the ease of turning the victim into this position may outweigh the risk (LOE 5).[154,155]

Treatment recommendation. It is reasonable to position an unconscious adult with normal breathing on the side with the lower arm in front of the body.

Special circumstances

Cervical spine injury

For victims of suspected spinal injury, additional time may be needed for careful assessment of breathing and circulation, and it may be necessary to move the victim if he or she is found face-down. In-line spinal stabilisation is an effective method of reducing risk of further spinal damage.

Airway opening
W150A, W150B

Consensus on science. The incidence of cervical spine injury after blunt trauma was 2.4% (LOE 5)[156] but increased in patients with craniofacial injuries (LOE 4),[157] a Glasgow Coma Scale score of <8 (LOE 4),[158] or both (LOE 4).[159] A large cohort study (LOE 4)[160] showed that the following features are highly sensitive (94% to 97%) predictors of spinal injury when applied by professional rescuers: mechanism of injury, altered mental status, neurological deficit, evidence of intoxication, spinal pain or tenderness, and distracting injuries (i.e. injuries that distract the victim from awareness of cervical pain). Failure to stabilise an injured spine was associated with an increased risk of secondary neurological injury (LOE 4).[161,162] A case—control study of injured patients with and without stabilisation showed that the risk of secondary injury may be lower than previously thought (LOE 4).[163]

All airway manoeuvres cause spinal movement (LOE 5).[164] Studies in human cadavers showed that both chin lift (with or without head tilt) and jaw thrust were associated with similar, substantial movement of the cervical vertebrae (LOE 6;[164—166] LOE 7[167,168]). Use of manual in-line stabilisation (MILS)[168] or spinal collars (LOE 6)[164] did not prevent spinal movement. Other studies have shown that application of MILS during airway manoeuvres reduces spinal movement to physiological levels

(LOE 5,6).[169,170] Airway manoeuvres can be undertaken more safely with MILS than with collars (LOE 3, 5).[171–173] But a small study of anaesthetised paralysed volunteers showed that use of the jaw thrust with the head maintained in neutral alignment did not improve radiological airway patency (LOE 3).[28] No studies evaluated CPR on a victim with suspected spinal injuries.

Treatment recommendation. Maintaining an airway and adequate ventilation is the overriding priority in managing a patient with a suspected spinal injury. In a victim with a suspected spinal injury and an obstructed airway, the head tilt—chin lift or jaw thrust (with head tilt) techniques are feasible and may be effective for clearing the airway. Both techniques are associated with cervical spinal movement. Use of MILS to minimise head movement is reasonable if a sufficient number of rescuers with adequate training are available.

Face-down victim
W143A,W143B

Consensus on science. Head position was an important factor in airway patency (LOE 5),[174] and it was more difficult to check for breathing with the victim in a face-down position. Checking for breathing by lay and professional rescuers was not always accurate when done within the recommended 10 s (LOE 7).[21,22] A longer time to check for breathing will delay CPR and may impair outcome.

Treatment recommendation. It is reasonable to roll a face-down, unresponsive victim carefully into the supine position to check for breathing.

Drowning

Drowning is a common cause of death worldwide. The special needs of the drowning victim were reviewed.

CPR for drowning victim in water
W160A,W160B

Consensus on science. Expired-air resuscitation in the water may be effective when undertaken by a trained rescuer (LOE 5;[175,176] LOE 6[177]). Chest compressions are difficult to perform in water and could potentially cause harm to both the rescuer and victim.

Treatment recommendation. In-water expired-air resuscitation may be considered by trained rescuers, preferably with a flotation device, but chest compressions should not be attempted in the water.

Removing drowning victim from water
W161

Consensus on science. Human studies showed that drowning victims without clinical signs of injury or obvious neurological deficit, a history of diving, use of a waterslide, trauma, or alcohol intoxication are unlikely to have a cervical spine injury (LOE 4;[178,179] LOE 5[180–184]).

Treatment recommendation. Drowning victims should be removed from the water and resuscitated by the fastest means available. Only victims with risk factors or clinical signs of injury or focal neurological signs should be treated as a victim with a potential spinal cord injury, with immobilisation of the cervical and thoracic spine.

EMS system

Dispatcher instruction in CPR
W165

Consensus on science. Observational studies (LOE 4)[185,186] and a randomised trial (LOE 2)[187] of telephone instruction in CPR by dispatchers to untrained lay responders in an EMS system with a short (mean 4 minutes) response interval showed that dispatcher instruction in CPR increases the likelihood of performance of bystander CPR but may or may not increase the rate of survival from cardiac arrest.

Treatment recommendation. Providing telephone instruction in CPR is reasonable.

Improving EMS response interval
W148A

Consensus on science. Cohort studies (LOE 3)[188–191] and a systematic review (LOE 1)[12] of cohort studies of patients with out-of-hospital cardiac arrest show that reducing the interval from EMS call to arrival increases survival to hospital discharge. Response time may be reduced by using professional first responders such as fire or police personnel or other methods.

Treatment recommendation. Administrators responsible for EMS and other systems that respond to patients with cardiac arrest should evaluate their process of delivering care and make resources available to shorten response time intervals when improvements are feasible.

Risks to victim and rescuer

Risks to trainees
W141B, W141C, W196

Consensus on science. Few adverse events from training in CPR have been reported by instructors and trainees even though millions of people are trained annually throughout the world. Case series reported the following infrequent adverse occurrences in trainees (LOE 5): infections, including herpes simplex virus (HSV);[192] *Neisseria meningitides*;[193] hepatitis B virus (HBV);[194] stomatitis;[195] tracheitis;[196] and others, including chest pain or near-syncope attributed to hyperventilation[197] and fatal myocardial infarction.[198] There was no evidence that a prior medical assessment of ''at-risk'' trainees reduces any perceived risk (LOE 7).[199]

Commonly used chemical disinfectants effectively removed bacteriologic and viral contamination of the training manikin (LOE 6).[200,201] Another study showed that 70% ethanol with or without 0.5% chlorhexidine did not completely eradicate herpes simplex contamination after several hours (LOE 6).[192]

Treatment recommendation. Training manikins should be cleaned between trainee ventilation sessions. It is acceptable to clean them with commercially available antiseptic, 30% isopropyl alcohol, 70% alcohol solution, or 0.5% sodium hypochlorite, allowing at least 1 minute of drying time between trainee ventilation sessions.

Risks to responders
W141A, W159A, W159B, W184A, W184B

Consensus on science. Few adverse events resulting from providing CPR have been reported, even though CPR is performed frequently throughout the world. There were only isolated reports of persons acquiring infections after providing CPR, e.g. tuberculosis[202] and severe acute respiratory distress syndrome (SARS).[203] Transmission of HIV during provision of CPR has never been reported. Responders exposed to infections while performing CPR might reduce their risk of becoming infected by taking appropriate prophylactic steps (LOE 7).[193] Responders occasionally experienced psychological distress.[204–208]

No human studies have addressed the safety, effectiveness, or feasibility of using barrier devices during CPR. Laboratory studies showed that nonwoven fibre filters or barrier devices with one-way valves prevented oral bacterial flora transmission from victim to rescuer during mouth-to-mouth ventilation (LOE 6).[209,210] Giving mouth-to-mouth ventilation to victims of organophosphate or cyanide intoxication was associated with adverse effects for responders (LOE 5).[211,212] One study showed that a high volume of air transmitting a highly virulent agent (i.e. SARS coronavirus) can overwhelm the protection offered by gowns, 2 sets of gloves, goggles, a full face shield, and a non–fit-tested N95 disposable respirator (LOE 5).[203]

Treatment recommendation. Providers should take appropriate safety precautions when feasible and when resources are available to do so, especially if a victim is known to have a serious infection (e.g. HIV, tuberculosis, HBV, or SARS).

Risks for the victim
W140A

Consensus on science. The incidence of rib fractures among survivors of cardiac arrest who received standard CPR is unknown. Rib fractures and other injuries are commonly observed among those who die following cardiac arrest and provision of standard CPR (LOE 4).[213] One study (LOE 4)[214] showed an increased incidence of sternal fractures in an active compression–decompression (ACD)-CPR group when compared with standard CPR alone. The incidence of rib fractures after mechanically performed CPR appeared to be similar to that occurring after performance of standard CPR (LOE 6).[215] There is no published evidence of the incidence of adverse effects when chest compressions are performed on someone who does not require resuscitation.

Treatment recommendation. Rib fractures and other injuries are common but acceptable consequences of CPR given the alternative of death from cardiac arrest. After resuscitation all patients should be reassessed and re-evaluated for resuscitation-related injuries.

If available, the use of a barrier device during mouth-to-mouth ventilation is reasonable. Adequate protective equipment and administrative, environmental, and quality control measures are necessary during resuscitation attempts in the event of an outbreak of a highly transmittable microbe such as the SARS coronavirus.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.resuscitation.2005.09.016.

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Part 3: Defibrillation

International Liaison Committee on Resuscitation

The 2005 Consensus Conference considered questions related to the sequence of shock delivery and the use and effectiveness of various waveforms and energies. These questions have been grouped into the following categories: (1) strategies before defibrillation; (2) use of automated external defibrillators (AEDs); (3) electrode-patient interface; (4) use of the electrocardiographic (ECG) waveform to alter management; (5) waveform and energy levels for the initial shock; (6) sequence after failure of the initial shock (i.e. second and subsequent shocks; and (7) other related topics.

The International Guidelines 2000[1] state that defibrillation should be attempted as soon as ventricular fibrillation (VF) is detected, regardless of the response interval (i.e. time between collapse and arrival of the AED). If the response interval is >4—5 min, however, there is evidence that 1.5—3 min of CPR before attempted defibrillation may improve the victim's chance of survival. The data in support of out-of-hospital AED programmes continue to accumulate, and there is some evidence supporting the use of AEDs in the hospital. Analysis of the VF waveform enables prediction of the likelihood of defibrillation success; with this information the rescuer can be instructed to give CPR or attempt defibrillation. This technology was developed by analysis of downloads from AEDs; it has yet to be applied prospectively to improve defibrillation success and is not available outside research programmes.

All new defibrillators deliver a shock with a biphasic waveform. There are several varieties of biphasic waveform, but the best variant and the optimal energy level and shock strategy (fixed versus escalating) have yet to be determined. Biphasic devices achieve higher first-shock success rates than monophasic defibrillators. This fact, combined with the knowledge that interruptions to chest compressions are harmful, suggests that a one-shock strategy (one shock followed immediately by CPR) may be preferable to the traditional three-shock sequence for VF and pulseless ventricular tachycardia (VT).

Strategies before defibrillation

Precordial thump
W59,W166B

Consensus on science. No prospective studies have evaluated the use of the precordial (chest) thump. In three case series (LOE 5)[2—4] VF or pulseless VT was converted to a perfusing rhythm by a precordial thump. The likelihood of conversion of VF decreased rapidly with time (LOE 5).[4] The conversion rate was higher for unstable or pulseless VT than for VF (LOE 5).[2—6]

Several observational studies indicated that an effective thump was delivered by a closed fist from a height of 5—40 cm (LOE 5).[3,4,6—8] Other observational studies indicated that additional tachyarrhythmias, such as unstable supraventricular tachycardia (SVT), were terminated by precordial thump (LOE 5).[9,10] Potential complications of the precordial thump include rhythm deteriorations, such as rate acceleration of VT, conversion of VT into VF, complete heart block, and asystole (LOE 5;[3,5,6,8,11,12] LOE 6[13]). Existing data do not enable an accurate estimate of the likelihood of these complications.

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Treatment recommendation. One immediate precordial thump may be considered after a monitored cardiac arrest if an electrical defibrillator is not immediately available.

CPR before defibrillation
W68,W177

Consensus on science. In a before—after study (LOE 4)[14] and a randomised trial (LOE 2),[15] 1.5—3 min of CPR by paramedics or EMS physicians before attempted defibrillation improved return of spontaneous circulation (ROSC) and survival rates for adults with out-of-hospital VF or VT when the response interval (ambulance dispatch to arrival) and time to defibrillation was ≥4—5 min. This contrasts with the results of another trial in adults with out-of-hospital VF or VT, in which 1.5 min of paramedic CPR before defibrillation did not improve ROSC or survival to hospital discharge (LOE 2).[16] In animal studies of VF lasting ≥5 min, CPR (often with administration of adrenaline (epinephrine)) before defibrillation improved haemodynamics and survival rates (LOE 6).[17—21]

Treatment recommendation. A 1.5- to 3-min period of CPR before attempting defibrillation may be considered in adults with out-of-hospital VF or pulseless VT and EMS response (call to arrival) intervals >4—5 min. There is no evidence to support or refute the use of CPR before defibrillation for in-hospital cardiac arrest.

Use of AEDS

AED programmes
W174,W175

Consensus on science. A randomised trial of trained lay responders in public settings (LOE 2)[22] and observational studies of CPR and defibrillation performed by trained professional responders in casinos (LOE 5)[23] and lay responders in airports (LOE 5)[24] and on commercial passenger aircraft (LOE 5)[25,26] showed that AED programmes are safe and feasible and significantly increase survival from out-of-hospital VF cardiac arrest if the emergency response plan is effectively implemented and sustained. In some studies defibrillation by trained first responders (e.g. firefighters or police officers) has improved survival rates from witnessed out-of-hospital VF sudden cardiac arrest (LOE 2;[27] LOE 3;[28,29] LOE 4;[30,31] LOE 5[32]). In other studies AED defibrillation by trained first-responders has not improved survival.[14,33]

Approximately 80% of out-of-hospital cardiac arrests occur in a private or residential setting (LOE 4).[34] However, there are insufficient data to support or refute the effectiveness of home AED programmes.

Treatment recommendation. Use of AEDs by trained lay and professional responders is recommended to increase survival rates in patients with cardiac arrest. Use of AEDs in public settings (airports, casinos, sports facilities, etc.) where witnessed cardiac arrest is likely to occur can be useful if an effective response plan is in place. The response plan should include equipment maintenance, training of likely responders, coordination with local EMS systems, and programme monitoring. No recommendation can be made for or against personal or home AED deployment.

AED Programme quality assurance and maintenance
W178

Consensus on science. No published trials evaluated specifically the effectiveness of AED programme quality improvement efforts to further improve survival rates. Case series and reports suggest that potential improvements can be made by reviewing AED function (rhythm analysis and shock), battery and pad readiness, operator performance, and system performance (e.g. mock codes, time to shock, outcomes) (LOE 5).[35—42]

Treatment recommendation. AED programmes should optimise AED function (rhythm analysis and shock), battery and pad readiness, operator performance, and system performance (e.g. mock codes, time to shock, outcomes).

AED use in hospitals
W62A

Consensus on science. No published randomised trials have compared AEDs with manual defibrillators in hospitals. One study of adults with in-hospital cardiac arrest with shockable rhythms showed higher survival-to-hospital discharge rates when defibrillation was provided through an AED than by manual defibrillation alone (LOE 4).[43] In an animal model, use of an AED substantially interrupted and delayed chest compressions compared with manual defibrillation (LOE 6).[44] A manikin study showed that use of an AED significantly increased the likelihood of delivering three shocks but increased the time to deliver the shocks when compared with manual defibrillators (LOE 6).[45] In contrast, a study of mock arrests in simulated

patients showed that use of monitoring leads and fully automated defibrillators reduced time to defibrillation when compared with manual defibrillators (LOE 7).[46]

Treatment recommendation. Use of AEDs is reasonable to facilitate early defibrillation in hospitals.

Electrode-patient interface

Electrode pad/paddle position and size
W63A,W63B,W173A

Consensus on science.

Position. No studies of cardiac arrest in humans have evaluated the effect of pad/paddle position on defibrillation success or survival rates. Most studies evaluated cardioversion (e.g. atrial fibrillation [AF]) or secondary end points (e.g. transthoracic impedance [TTI]).

Placement of paddles or electrode pads on the superior-anterior right chest and the inferior-lateral left chest were effective (paddles studied in AF, LOE 2;[47] pads studied in AF, LOE 3;[48] effect of pad position on TTI, LOE 3[49]). Alternative paddle or pad positions that were reported to be effective were apex-posterior (pads studied in VF and AF, LOE 4;[50] effect of pad position on TTI, LOE 3[49]), and anteroposterior (paddles studied in AF, LOE 2;[51] pads studied in AF, LOE 2;[52] LOE 3;[53] effect of pad position on TTI, LOE 3[49]). One study showed lower TTI with longitudinal placement of the apical paddle (LOE 3).[54] Placement of the pad on the female breast increased impedance and may decrease efficacy of defibrillation (LOE 5).[55] High-voltage alternating current (e.g. from high power lines) interfered with AED analysis (LOE 6).[56]

Size. One human study (LOE 3)[57] and one animal study (LOE 6)[58] documented higher defibrillation success rates with larger paddles: 12.8-cm paddles were superior to 8-cm paddles. Eight studies (LOE 3;[53,57,59,60] LOE 5[61] LOE 6[55,62,63]) demonstrated that increased pad size decreased TTI. In one canine study, significantly increased myocardial damage was reported after defibrillation with small (4.3 cm) electrodes compared with larger (8 and 12 cm) electrodes (LOE 6).[64]

Treatment recommendation. Paddles and electrode pads should be placed on the exposed chest in an anterolateral position. Acceptable alternative positions are anteroposterior (paddles and pads) and apex-posterior (pads). In large-breasted patients it is reasonable to place the left electrode pad (or paddle) lateral to or underneath the left breast. Defibrillation success may be higher with 12-cm electrodes than with 8-cm electrodes. Small electrodes (4.3 cm) may be harmful (myocardial injury can occur).

Self-adhesive defibrillation pads versus paddles
W71

Consensus on science. One randomised trial (LOE 2)[65] and two retrospective comparisons (LOE 4)[50,66] showed that TTI is similar when either pads or paddles are used. One prospective comparison of pads and paddles (LOE 3)[67] showed lower TTI when paddles were applied at an optimal force of 8 kg compared with pads. One randomised study of chronic AF showed similar effectiveness for self-adhesive pads and manual paddles when monophasic damped sinusoidal or BTE waveforms were evaluated separately (LOE 7).[68] Several studies (LOE 5;[69–71] LOE 6[72]) showed the practical benefits of pads over paddles for routine monitoring and defibrillation, prehospital defibrillation, and perioperative defibrillation.

Treatment recommendation. Self-adhesive defibrillation pads are safe and effective and are an acceptable alternative to standard defibrillation paddles.

Waveform analysis

VF waveform analysis has the potential to improve the timing and effectiveness of defibrillation attempts; this should minimise interruptions in precordial compressions and reduce the number of unsuccessful high-energy shocks, which cause postresuscitation myocardial injury. The technology is advancing rapidly but is not yet available to assist rescuers.

Prediction of shock success from VF waveform
W64A,W64B,W64C,W65A

Consensus on science. Retrospective analyses of the VF waveform in clinical and animal studies and theoretical models (LOE 4;[73–82] LOE 6[83–93]) suggest that it is possible to predict with varying reliability the success of defibrillation from the fibrillation waveform. No studies evaluated specifically whether treatment can be altered by the prediction of defibrillation success to improve survival from cardiac arrest.

Initial shock waveform and energy levels

Several related questions were reviewed. Outcome after defibrillation has been studied by many

investigators. When evaluating these studies the reviewer must consider the setting (e.g. out-of-hospital versus in-hospital), the initial rhythm (e.g. VF/pulseless VT), the duration of arrests (e.g. out-of-hospital with typical EMS response interval versus electrophysiology study with 15-s arrest interval), and the specific outcome measured (e.g. termination of VF at 5 s).

Biphasic versus monophasic waveforms for ventricular defibrillation
W61A,W61B,W172

Consensus on science. In three randomised cardiac arrest studies (LOE 2),[94–96] a re-analysis of one of these studies (LOE 2),[97] two observational cardiac arrest studies (LOE 4),[98,99] a meta-analysis of seven randomised trials in the electrophysiology laboratory (LOE 1),[100] and multiple animal studies, defibrillation with a biphasic waveform, using equal or lower energy levels, was at least as effective for termination of VF as monophasic waveforms. No specific waveform (either monophasic or biphasic) was consistently associated with a greater incidence of ROSC or higher hospital discharge rates from cardiac arrest than any other specific waveform. One retrospective study (LOE 4)[99] showed a lower survival-to-hospital-discharge rate after defibrillation with a biphasic truncated exponential (BTE) waveform when compared with a monophasic truncated exponential (MTE) device (20% versus 39.7%, $P = .01$), but survival was a secondary end point. This study had multiple potential confounders, including the fact that CPR was provided to more subjects in the MTE group.

No direct comparison of the different biphasic waveforms has been reported as of 2005.

Treatment recommendation. Biphasic waveform shocks are safe and effective for termination of VF when compared with monophasic waveform shocks.

Energy level for defibrillation
W60A,W60B

Consensus on science. Eight human clinical studies (LOE 2;[94] LOE 3;[101] LOE 5;[95,96,98,99,102,103]) described initial biphasic selected shock energy levels ranging from 100 to 200 J with different devices but without demonstrating an optimal energy level clearly. These human clinical studies also described use of subsequent selected shock energy levels with different devices for shock-refractory VF/VT ranging from 150 to 360 J but without demonstrating an optimal energy level clearly.

Seven more laboratory studies (LOE 7)[104–110] in stable patients evaluated termination of induced VF with energy levels of 115–200 J.

Neither human clinical nor laboratory studies demonstrated evidence of significantly greater benefit or harm from any energy level used currently. One human study showed an increased incidence of transient heart block following two or more 320-J monophasic damped sine wave (MDS) shocks when compared with an equal number of 175-J MDS shocks, but there was no difference in long-term clinical outcome (LOE 2).[111]

Only one of the reviewed animal studies showed harm caused by attempted defibrillation with doses in the range of 120–360 J in adult animals; this study indicated that myocardial damage was caused by higher-energy shocks (LOE 6).[112]

One in-hospital study of 100 patients in VF compared MDS shocks of low (200–240 J), intermediate (300–320 J), and high (400–440 J) energy (LOE 2).[113] First-shock efficacy (termination of VF for $\geq$5 s) was 39% for the low-energy group, 58% for the intermediate-energy group, and 56% for the high-energy dose group. These differences did not achieve statistical significance. A study of electrical cardioversion for AF indicated that 360-J MDS shocks were more effective than 100- or 200-J MDS shocks (LOE 7).[114] Cardioversion of a well-perfused myocardium, however, is not the same as defibrillation attempted during VF cardiac arrest, and any extrapolation should be interpreted cautiously.

Treatment recommendation. There is insufficient evidence for or against specific selected energy levels for the first or subsequent biphasic shocks. With a biphasic defibrillator it is reasonable to use 150–200 J with BTE waveforms or 120 J with the rectilinear biphasic waveform for the initial shock. With a monophasic waveform defibrillator, an initial shock of 360 J is reasonable.

Second and subsequent shocks

Fixed versus escalating energy
W171

Consensus on science. Only one small human clinical study (LOE 3)[101] compared fixed energy with escalating energies using biphasic defibrillators. The study did not identify a clear benefit for either strategy.

Treatment recommendation. Nonescalating- and escalating-energy biphasic waveform defibrillation can be used safely and effectively to terminate VF of both short and long duration.

One-shock protocol versus three-shock sequence
W69A,W69B,W69C

Consensus on science. No published human or animal studies compared a one-shock protocol with a three-stacked shock sequence for any outcome. The magnitude of success of initial or subsequent shocks depended on the specific group of patients, the initial rhythm, and the outcome considered. Shock success was defined as termination of VF for ≥5 s after the shock. Resuscitation success can include ROSC and survival to hospital discharge. Only shock success is cited below.

First-shock success. Six studies of defibrillation in out-of-hospital cardiac arrest reported first-shock success in patients whose initial rhythm was shockable (VF/pulseless VT):

- In studies that used a 200-J MDS waveform, the first-shock success rate was 77—91% (LOE 2;[94,97] LOE 5[95,99]). In studies that used a 200-J MTE waveform, the first-shock success rate was 54—63% (LOE 4).[97,99]
- In studies that used a 150-J BTE waveform[97,99,115,116] and one study that used a 200-J BTE waveform,[95] the first-shock success rate was 86—98%.[95,97,99,115,116]
- The first-shock success rate with a 120-J rectilinear biphasic waveform was 85% (according to L.J. Morrison, MD, in oral discussion at the 2005 Consensus Conference).[94]

Although the first-shock success rate was relatively high in patients with out-of-hospital cardiac arrest with an initial rhythm of VF, the average rate of ROSC with the first shock (for MDS, MTE, and BTE waveforms) was 21% (range 13—23%) (LOE 5).[99]

Second- and third-shock success rates. Six studies of defibrillation in out-of-hospital cardiac arrest reported the shock success (defined above) rate of the first shock and subsequent two shocks (if the initial shock was unsuccessful) for patients whose initial rhythm was VF/pulseless VT. The figures below refer to only those patients who remained in VF after the first shock, and they represent the proportion of these cases successfully defibrillated by either the second or third shock.

In two studies that used the MDS waveform with increasing energy levels (200 J to 200—300 J to 360 J), the combined shock success of the second and/or third shocks when the first shock failed was 68—72% (LOE 5).[94,99] In two studies that used the MTE waveform with increasing energy levels (200 J to 200—360 J), the combined shock success of the second and third shocks when the first shock failed was 27—60% (LOE 5).[97,99]

In four studies that used the fixed-energy 150-J BTE waveform, the combined shock success of the second and third shocks when the first shock failed was 50—90% (LOE 5).[97,99,115,116]

In the one study that used a rectilinear waveform with increasing energy levels (120 J to 150—200 J), the combined success rate of the second and third shocks when the first shock failed was 85% (LOE 5).[94]

One study of defibrillation for out-of-hospital cardiac arrest in which the initial rhythm was VF reported a 26% rate of ROSC with the initial series of up to three shocks (for BTE waveforms) combined with pre-shock or post-shock CPR or both (LOE 5).[116]

Treatment recommendation. Priorities in resuscitation should include early assessment of the need for defibrillation (Part 2. Adult Basic Life Support), provision of CPR until a defibrillator is available, and minimisation of interruptions in chest compressions. Rescuers can optimise the likelihood of defibrillation success by optimising the performance of CPR, timing of shock delivery with respect to CPR, and the combination of waveform and energy levels. A one-shock strategy may improve outcome by reducing interruption of chest compressions. A three-stacked shock sequence can be optimised by immediate resumption of effective chest compressions after each shock (irrespective of the rhythm) and by minimising the hands-off time for rhythm analysis.

Related defibrillation topics

Defibrillator data collection
W66

Consensus on science. Collection of data from defibrillators enables a comparison of actual performance during cardiac arrests and training events. The results of three observational studies (LOE 5)[117—119] suggest that the rate and depth of external cardiac compressions and ventilation rate were at variance with current guidelines.

Treatment recommendation. Monitor/defibrillators modified to enable collection of data on compression rate and depth and ventilation rate may be useful for monitoring and improving process and outcomes after cardiac arrest.

Oxygen and fire risk during defibrillation
W70A,W70B

Consensus on science. Several case reports (LOE 5)[120–125] described instances of fires ignited by sparks from poorly attached defibrillator paddles in the presence of an oxygen-enriched atmosphere. The oxygen-enriched atmosphere rarely extends >0.5 m in any direction from the oxygen outflow point, and the oxygen concentration returns quickly to ambient when the source of enrichment is removed (LOE 5;[122] LOE 6[126]). The most severe fires were caused when ventilator tubing was disconnected from the tracheal tube and then left adjacent to the patient's head during attempted defibrillation (LOE 5).[121,123,125] In at least one case a spark generated during defibrillation ignited oxygen delivered by a simple transparent face mask that was left in place (LOE 5).[120]

In a manikin study (LOE 6)[126] there was no increase in oxygen concentration anywhere around the manikin when the ventilation device was left attached to the tracheal tube, even with an oxygen flow of 15 L min^{-1}.

Treatment recommendation. Rescuers should take precautions to minimise sparking (by paying attention to pad/paddle placement, contact, etc) during attempted defibrillation. Rescuers should try to ensure that defibrillation is not attempted in an oxygen-enriched atmosphere.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.resuscitation.2005.09.017.

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Part 4: Advanced life support

International Liaison Committee on Resuscitation

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 neurological 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
- Randomised controlled trials on several new devices to assist circulation during CPR
- Randomised 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 aetiology of cardiac arrest

W119A,W120,W121

Consensus on science. Very few data address the aetiology of cardiac arrest directly. One prospective study (LOE 3)[1] and one retrospective study (LOE 4)[2]

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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 aetiology.

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 (e.g. survival and length of stay in the intensive care unit [ICU]) after introduction of a MET. One cluster randomised 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 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 (e.g. 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 categorised 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,W46B

Consensus on science. Despite frequent successful use of nasopharyngeal airways by anaesthetists, there are no published data on the use of these airway adjuncts during CPR. One study in anaesthetised patients showed that nurses inserting nasopharyngeal airways were no more likely than anaesthesiologists to cause nasopharyngeal trauma (LOE 7).[12] One LOE 5 study[13] 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 (i.e. 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 unrecognised oesophageal 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 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 randomised 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 randomised 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 paediatric study is to adult resuscitation. The study had some important limitations, including the provision of only 6 h 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 3[18]; LOE 4[19]). 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 unrecognised 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 provide 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 unrecognised oesophageal intubation misplacement and unrecognised 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 randomised controlled trials conducted on adult patients undergoing resuscitation (LOE 2)[24–28] and three additional randomised controlled trials involving patients undergoing anaesthesia (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 3[32]; LOE 4[33]; LOE 5[34–37]). Successful ventilation was achieved with the Combitube during CPR in 78.9—98% of patients (LOE 2[26,27,38]; LOE 3[32]; LOE 4[33]; LOE 5[34,35]).

LMA. Seven randomised controlled trials involving anaesthetised patients (LOE 7)[39–45] that compared the LMA with tracheal intubation and another seven randomised 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 randomised 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.

Nonrandomised studies (LOE 3[53–55]; LOE 4[33]; LOE 5[56–61]) have also shown high insertion success rates by inexperienced providers both in and out of the hospital. Complication rates in nonrandomised studies (LOE 3[58]; LOE 4[53]; LOE 5[56]) have been extremely low.

Successful ventilation was achieved with the LMA during CPR in 71.5–98% of cases (LOE 2[38]; LOE 3[54]; LOE 4[33]; LOE 5[56,58–60]).

Additional airway devices. Use of the laryngeal tube during CPR was described in just a few cases included in two LOE 5 studies[62,63] and one LOE 8 paper.[64] There were no studies comparing the laryngeal tube with the tracheal tube in any patient population, although four randomised controlled trials compared the laryngeal tube favourably with the LMA in anaesthetised 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

Unrecognised oesophageal 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 are no data quantifying the capability of these devices to monitor tube position after initial placement.

Exhaled CO_2
W47,W48,W50

Consensus on science. Evidence from one meta-analysis in adults (LOE 1),[69] one prospective controlled cohort study (LOE 3),[70] case series (LOE 5),[71–79] and animal models (LOE 6)[80,81] indicate that exhaled CO_2 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] four to digital,[69–71,77] and four 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: 33–100%
- Percentage of oesophageal placements detected: 97–100%
- Probability of tracheal placement if test result is positive (exhaled CO_2 is detected): 100%
- Probability of oesophageal placement if test result is negative (exhaled CO_2 is not detected): 20–100%

One adult case series (LOE 5)[82] shows that in the presence of a perfusing rhythm, exhaled CO_2 detection can be used to monitor tracheal tube position during transport.

No studies directly evaluated exhaled CO_2 to confirm placement of the Combitube or LMA during cardiac arrest in humans.

Treatment recommendation. Healthcare providers should recognise that evaluation of exhaled CO_2 is not infallible for confirming correct placement of a tracheal tube, particularly in patients in cardiac arrest. Exhaled CO_2 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.

Oesophageal detector device
W48A,W48B,W51A,W51B

Consensus on science. Eight studies of at least fair quality evaluated the accuracy of the syringe or self-inflating bulb type of oesophageal detector device (EDD) (LOE 3[21,77,83]; LOE 5[84]; 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 oesophagus (LOE 5[84]; LOE 7[85–88]). In two 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 of

the EDD suggested oesophageal 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]: two 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 six cases in which resuscitation had ceased and ventilation was shown on repeated occasions (or was highly likely) to cause gas trapping and consequent haemodynamic compromise (LOE 5).[100,108–110] The authors of all these studies suggested a period of disconnection from ventilation during resuscitation from PEA may be useful to exclude gas trapping.

Automatic transport ventilators
W55,W152A,W152B

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] Anaesthetised 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 categorised as (1) vasopressors, (2) antiarrhythmics, (3) other drugs and fluids, and (4) alternative routes of delivery.

Vasopressors

Despite the widespread use of adrenaline/epinebreak phrine 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.

Adrenaline and vasopressin
W83B, W83E, W83F, W83G, W83H, W84A, W84B, W84D, W85A, W85B, W85C, W112

Consensus on science. Despite promising lower-level data (LOE 2[118]; LOE 5[119–121]) and multiple well-performed animal studies [LOE 6]), two large randomised 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 one study) when compared with adrenaline (1 mg, repeated) as the initial vasopressor. In one 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 adrenaline (1 mg, repeated if necessary). A meta-analysis of five randomised trials (LOE 1)[124] showed no statistically significant differences between vasopressin and adrenaline for ROSC, death within 24 h, 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, adrenaline 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, adrenaline in any cardiac arrest rhythm.

Alpha-methyl noradrenaline
W83B, W48C

Consensus on science. Preliminary animal studies (LOE 6)[125–127] have suggested some potential short-term benefits with the use of alpha-methyl noradrenaline in animal models of VF. At this stage no published human studies have been identified.

Endothelin
W83D, W83I

Consensus on science. Evidence from five 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 two blinded randomised controlled clinical trials in adults (LOE 1),[133,134] administration of amiodarone (300 mg[133]; 5 mg kg^{-1}[134]) 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 placebo[133] or lidocaine 1.5 mg kg^{-1}.[134] Additional studies (LOE 7)[135–139] document consistent improvement in defibrillation response when amiodarone is given to humans or animals with VF or haemodynamically 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 giving other drugs routinely (e.g. 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 three small randomised trials (LOE 2)[141–143]

indicate that aminophylline does not increase ROSC when given for bradyasystolic 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 bradyasystolic cardiac arrest (LOE 2[141–143]; LOE 5[140]).

Atropine
W97A,W97B

Consensus on science. Five prospective controlled nonrandomised cohort studies in adults (LOE 3)[19,144–147] and one LOE 4 study[148] 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 study[149] showed no advantage of Tribonate over placebo (neutral), and five retrospective analyses of uncontrolled clinical use of sodium bicarbonate were inconclusive (LOE 4).[150–154] One LOE 4 study[155] 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 neurological 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 one LOE 5 publication[156] 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 3[161]; LOE 7[162]) and animal studies (LOE 6)[163–166] indicated no increase in the rate of ROSC when magnesium was given during CPR. Results from one small case series of five patients (LOE 5)[167] indicated benefit from giving magnesium in shock-resistant and adrenaline/lidocaine-resistant VF.

Treatment recommendation. Magnesium should be given for hypomagnesemia and torsades de pointes, but there are 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 resuscitated successfully 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 3[168]; 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 3[168]; LOE 4[169–171]) and five 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 are 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 normovolaemic 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 routes.

Intraosseous route
W29

Consensus on science. Two prospective trials in adults and children (LOE 3)[184,185] and six other studies (LOE 4[186]; 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 adrenaline. In one historic nonrandomised cohort study (LOE 4)[192] in adults, the rate of ROSC (27% versus 15%, $P = .01$) and rate of survival to hospital admission (20% versus 9%, $P = .01$) 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.

Adrenaline. During CPR the equipotent adrenaline dose given endobronchially was approximately 3–10 times higher than the IV dose (LOE 5[193]; LOE 6[194]). Endobronchial adrenaline (2–3 mg) diluted in 5–10 mL 0.9% NaCl achieved therapeutic plasma concentrations (LOE 5).[193] Endobronchial adrenaline achieved higher plasma concentrations when diluted with water rather than 0.9% saline (LOE 6).[195]

During CPR lung perfusion is only 10–30% of the normal value, resulting in a pulmonary adrenaline depot. When cardiac output is restored after a high dose of endobronchial adrenaline, prolonged reabsorption of adrenaline 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 haemodynamically 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 anaesthetised 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 2[199]; LOE 5[196]), 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 PaO_2 when diluted with water instead of 0.9% saline (LOE 5).[201]

Vasopressin. Endobronchial vasopressin was more effective in increasing diastolic blood pressure than equivalent doses of endobronchial adrenaline (LOE 6).[202] In a small animal study, endobronchial vasopressin was more effective than placebo in increasing coronary perfusion pressure during CPR and improved 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 CO_2 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 CO_2 monitoring to guide therapy during cardiac arrest
W92A,W92B

Consensus on science. No studies have addressed this question directly. The studies published over the past 5 years were consistent with the older literature, which showed that higher end-tidal CO_2 values during CPR correlate with ROSC (LOE 5).[204–207]

In experimental models, end-tidal CO_2 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 CO_2 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 6[220]).

In case series totaling 744 patients, intubated adults in cardiac arrest receiving CPR who had a *maximum* end-tidal CO_2 of <10 mmHg 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 two studies (LOE 5)[217,223] show no difference in ROSC and survival in those with an *initial* end-tidal CO_2 of <10 mmHg. Two additional studies (LOE 5)[221,222] reported that five patients achieved ROSC despite an *initial* end-tidal CO_2 of <10 mmHg (one patient survived).

Treatment recommendation. End-tidal CO_2 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 one LOE 5 study[224] 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 hypoxaemia (LOE 5[235]; LOE 6[236,237]; LOE 7[225,227,231,238–240]). Arterial blood gas analysis can also highlight the extent of metabolic acidosis (LOE 5[241]; LOE 6[236]; LOE 7[225,227,230,231,238,239]).

Arterial CO_2 is an indicator of adequacy of ventilation during CPR (LOE 2[242]; LOE 5[235]; LOE 6[236]; LOE 7[92,227,239,243]). If ventilation is constant, an increase in $PaCO_2$ is a potential marker of improved perfusion during CPR (LOE 5[244]; LOE 6[209,245]; LOE 7[246]).

Treatment recommendation. Arterial blood gas monitoring during cardiac arrest enables estimation of the degree of hypoxaemia 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,W95B,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 $\geq$15 mmHg is predictive of ROSC. Increased CPP correlated with improved 24-h survival in animal studies (LOE 6)[249] and is associated with improved myocardial blood flow and ROSC in studies of adrenaline, 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 were 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 randomised controlled trials (LOE 2)[252–254] and additional studies (LOE 3[255]; LOE 5[256–259]; LOE 6[260]; LOE 7[261]) 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 CO_2 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 randomised controlled trials (LOE 1[273]; LOE 2[274]) 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. These data and those from a crossover study (LOE 3)[275] were combined in two meta-analyses (LOE 1).[276,277] One randomised 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 one child (LOE 5).[279] Although only a small proportion of patients had postmortem examinations, there was no evidence of significant harm.

High-frequency CPR
W74,163H

Consensus on science. One clinical trial of nine patients (LOE 4)[280] showed that high-frequency CPR (120 compressions min^{-1}) improved haemodynamics over standard CPR. Three laboratory studies (LOE 6)[281–283] showed that high-frequency CPR (120–150 compressions min^{-1}) improved haemodynamics without increasing trauma. In one additional laboratory study (LOE 6),[284] high-frequency CPR did not improve haemodynamics 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 neurological 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 two 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-analysis[288] 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 haemodynamics in one in-hospital study of end-stage patients (LOE 3)[291] and two laboratory studies (LOE 6).[292,293]

Mechanical (piston) CPR
W77A,W77B,W163B,W163E

Consensus on science. One prospective randomised study and two prospective randomised crossover studies in adults (LOE 2)[294–296] indicated improvement in end-tidal CO_2 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 CO_2, cardiac output, cerebral blood flow, mean arterial pressure, and short-term neurological 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 randomised human studies comparing LUCAS-CPR with standard CPR. A single study of pigs with VF showed that LUCAS-CPR improved haemodynamic 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
W78A,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 haemodynamics. One clinical, randomised study in adults (LOE 2)[301] and additional experimental studies (LOE 6[302,303]; LOE 7[304]) 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 delay starting CPR substantially 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 two 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 one 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 randomised study of 230 adults documented increased admissions to the ICU and 24-h 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 haemodynamics during standard CPR in five laboratory studies (LOE 6)[311–315] and one clinical study (LOE 2).[316]

A randomised study of 400 adults showed increased ROSC and 24-h 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 haemodynamics during ACD-CPR in one laboratory study (LOE 6)[318] and one clinical study (LOE 2).[319] One laboratory study failed to show an improvement in haemodynamics with the use of the ITD during ACD-CPR (LOE 6).[314] Compared with standard CPR, ROSC and 24-h survival were increased when the ITD was used with ACD in a randomised study of 210 prehospital patients (LOE 1),[320] and haemodynamics were improved in two laboratory studies (LOE 6).[321,322]

Extracorporeal techniques and invasive perfusion devices
W28,W82

Consensus on science. The only adult data come from three case series (LOE 5).[323–325] One of these[323] indicated that extracorporeal CPR (ECPR) was more successful in postcardiotomy patients than those in cardiac arrest from other causes. The other two 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
W81A,W81B

Consensus on science. No prospective randomised studies of open-chest CPR for resuscitation have been published. Four relevant human studies were reviewed, two after cardiac surgery (LOE 4[326]; LOE 5[327]) and two after out-of-hospital cardiac arrest (LOE 4[328]; LOE 5[329]). The observed benefits of open-chest cardiac massage included improved coronary perfusion pressure[329] 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 four options for the treatment of narrow-complex tachycardia in the periarrest setting: electrical conversion, physical manoeuvers, pharmacological conversion, or rate control. The choice depends on the stability of the patient and the rhythm. In a haemodynamically unstable patient, narrow-complex tachycardia is best treated with electrical cardioversion.

Drug therapy for atrial fibrillation
W86

Consensus on science. One randomised controlled trial in adults and three 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 randomised 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 randomised 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 manoeuver (LOE 2).[352] One study (LOE 4)[353] showed that stable paroxysmal supraventricular tachycardia (PSVT) in younger patients may be treated first with vagal manoeuvers but will be unsuccessful 80% of the time.

Five prospective controlled nonrandomised cohort studies (LOE 2[354]; LOE 3[355–358]) indicated that adenosine is safe and effective in converting PSVT in the hospital and out-of-hospital settings. Two randomised 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 more severe with verapamil. One randomised 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 randomised 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 manoeuvers (avoiding carotid sinus massage in the elderly); these will terminate about 20% of PSVTs. If vagal manoeuvers 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 10–15% 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 randomised parallel study (LOE 2)[138] indicated that aqueous amiodarone is more effective than lidocaine in the treatment of shock-resistant VT. One randomised 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 randomised 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 haemodynamically 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 terminate torsades de pointes effectively 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, adrenaline, 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 one randomised clinical trial in adults (LOE 2)[375] and one 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 two prospective controlled nonrandomised 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^{-1} 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 0.5–1 mg i.v., repeated every 3–5 min, 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, adrenaline, 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 two electrical modes in the same patient and found all three 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—70 beats min^{-1} (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 haemodynamically 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 categorised as environmental (hypothermia, submersion, electrocution), pregnancy, asthma, and drug overdose/poisoning.

Environmental

Hypothermia
W131,W162A,W162B

Consensus on science. *Hypothermic patients with pulse*. One randomised 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,W160,W161

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; two victims survived. A case report described the use of noninvasive positive-pressure ventilation in two 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 neurological signs should be treated as having a potential spinal cord injury, with stabilisation 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

neurological 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 neurological and cardiac sequelae.

Pregnancy

Aetiology 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 aetiology 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 haemorrhage 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 7[417]; 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 min of cardiac arrest in pregnancy if initial resuscitative efforts by skilled rescuers in the hospital setting failed.

Extrapolation from anaesthesia (LOE 7)[422] and a manikin study (LOE 6[423]) 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 min 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 three 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 apnoea 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 two noncardiac arrest case reports (LOE 7[433]; LOE 8[434]) 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 apnoea 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,197D,197E

Consensus on science. Evidence from the use of bicarbonate in calcium channel blocker overdose in two 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—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 8[457] 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 categorised 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 neurological 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 h 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 3)[460]; LOE 5[461]; LOE 7[462]) and numerous animal studies (LOE 6)[463–465] documented harmful effects of hypocapnia (cerebral ischemia) after cardiac arrest. Two studies provide neutral evidence (LOE 5[466]; LOE 6[467]).

Treatment recommendation. There are no data to support the targeting of a specific $PaCO_2$ 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 randomised clinical trials (LOE 1[468]; LOE 2[469]) showed improved outcome in adults who remained comatose after ini-

tial 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 °C[468] or to the range of 32–34 °C[469] for 12–24 h. The Hypothermia After Cardiac Arrest (HACA) study[468] included a small subset of patients with in-hospital cardiac arrest.

One study (LOE 2)[470] documented improved metabolic end points (lactate and O_2 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 1[468]; LOE 2[469,470]; LOE 5[472–475]).The only studies documenting improved outcome with therapeutic hypothermia after cardiac arrest used external cooling (LOE 1[468]; LOE 2[469,470]). An infusion of 30 ml kg^{-1} 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 three 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 1[468]; 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 (e.g. 12–24 h). Optimal variables, 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 32–34 °C for 12–24 h when the initial rhythm was VF. Cooling to 32–34 °C for 12–24 h 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 h 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 unfavourable neurological 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 paracetamol or ibuprofen (LOE 7)[493,494]; however, antipyretics or physical cooling methods have been associated with decreased infarct volumes in animal models of global ischaemia (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 8[501]) 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 (e.g. intracranial haemorrhage, electrolyte imbalance, etc) are excluded.

Sedation and pharmacological 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 h after prehospital or in-hospital cardiac arrest.

Other supportive therapies

Blood glucose control
W115A,W115B

Consensus on science. Tight control of blood glucose (range 80–110 mg dl^{-1} or 4.4–6.1 mmol l^{-1})

with insulin reduces hospital mortality rates in critically ill adults (LOE 1[504]; LOE 4[505]), 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 neurological outcome (LOE 4[506]; LOE 5[507–513]). There was good evidence that persistent hyperglycaemia after stroke is associated with a worse neurological 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 unrecognised hypoglycaemia, 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 hyperglycaemia (LOE 2).[469]

Treatment recommendation. Providers should monitor blood glucose frequently after cardiac arrest and should treat hyperglycaemia with insulin but avoid hypoglycaemia.

Coagulation control
W116

Consensus on science. There are no studies evaluating the role of anticoagulation alone to improve outcome after ROSC. In three nonexperimental reports (LOE 4[168]; LOE 5[519]; LOE 6[179]) using fibrinolytics combined with heparin (anticoagulation) after prolonged cardiac arrest in humans, ROSC, but not 24-h 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 3[531]) 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 aetiology, can neither be recommended nor rejected. It may be reasonable, however, to continue an infusion of an antiarrhythmic drug that restored a stable rhythm successfully during resuscitation.

Prognostication

Prognostication during cardiac arrest

Predictive value of neurological examination
W122A,W122B,W122C

Consensus on science. Five studies (LOE 4[532,533]; LOE 5[534–536]) documented some ability to predict outcome in adults when neurological 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 neurological exam during cardiac arrest to predict outcome is not recommended and should not be used.

Prognostication after resuscitation

Predictive value of standard laboratory analyses
W12B

Consensus on science. In eight 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 unfavourable outcomes. None of these studies could formulate a predictive model conclusively to identify 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 randomised 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 one meta-analysis (LOE 1)[587] indicated that median nerve somatosensory-evoked potentials in normothermic patients comatose for at least 72 h 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 h 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 24–48 h 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 24–48 h after a cardiac arrest can help define the prognosis in patients with grade I, IV, and V EEGs.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.resuscitation.2005.09.018.

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Part 5: Acute coronary syndromes

International Liaison Committee on Resuscitation

The American Heart Association and the American College of Cardiology,[1,2] the European Society of Cardiology[3,4] and others[5] have developed comprehensive guidelines for the in-hospital management of patients with ST-elevation myocardial infarction (STEMI)[2] and for unstable angina (UA) and non-ST-elevation MI (NSTEMI).[1] The International Liaison Committee on Resuscitation (ILCOR) Acute Coronary Syndromes (ACS)/Acute Myocardial Infarction (AMI) Task Force reviewed the evidence specifically related to diagnosis and treatment of ACS/AMI in the out-of-hospital setting and the first hours of care in the in-hospital setting, typically in the emergency department (ED).

Much of the research concerning the care of the patient with ACS has been conducted on in-hospital populations rather than in the ED or out-of-hospital settings. By definition, extending the conclusions from such research to the early ED management strategy or the out-of-hospital setting requires extrapolation classified as level of evidence 7.

Diagnostic tests in ACS and AMI

The sensitivity, specificity, and clinical impact of various diagnostic strategies in ACS/AMI have been evaluated. These include signs and symptoms, cardiac markers, and 12-lead electrocardiogram (ECG). The standard ILCOR/AHA levels of evidence (described in Part 1: ''Introduction'') pertain largely to therapeutic interventions. For this reason, in the evaluation of evidence for diagnostic accuracy the reviewers used the Centre for Evidence-Based Medicine (CEBM) levels of evidence for diagnostic tests (http://www.cebm.net/levels_of_evidence.asp). The CEBM levels are cited as ''levels'' and the ILCOR/AHA levels of evidence are designated with ''LOE,'' for ''level of evidence.''

Neither signs and symptoms nor cardiac markers alone are sufficiently sensitive to diagnose AMI or ischaemia in the prehospital setting or the first 4—6 h in the ED. The 12-lead ECG in the ED and out-of-hospital settings is central to the initial triage of patients with possible ACS.

Diagnostic and prognostic test characteristics of signs and symptoms of ACS/AMI

W221A, W221B

Consensus on science

Diagnosis. Four CEBM level 1B validating cohort studies[6—9] and nine CEBM level 2A-4 studies[10—18] do not support the use of any clinical signs and symptoms independent of ECG, cardiac biomarkers, or other diagnostic tests to rule in or rule out ACS/AMI in prehospital or ED settings. Although some signs are more sensitive and specific than others, no sign or symptom evaluated exceeded 92% sensitivity in the higher LOE studies (most reported sensitivity of 35—38%) or 91% specificity (range 28—91% in highest CEBM levels).[7]

Prognosis and clinical impact. In three CEBM level 1a systematic reviews,[10,19,20] 10 CEBM level 1b validating cohort studies[6—9,21—26] and 21 CEBM level 2a-4 studies,[11—13,15—18,27—40] a variety of signs and symptoms assisted in the diagnosis of ACS/AMI and had clinical impact (defined as triage and some treatment and investigational decisions) on the out-of-hospital emergency management and

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risk assessment for coronary atherosclerosis and unstable syndromes.

Treatment recommendation Signs and symptoms of ACS/AMI may be useful in combination with other important information (biomarkers, risk factors, ECG, and other diagnostic tests) in making triage and some treatment and investigational decisions in the out-of-hospital setting and the ED. Signs and symptoms are not independently diagnostic of ACS/AMI.

Diagnostic and prognostic test characteristics of cardiac biomarkers for ACS/AMI
W222A,W222B

Consensus on science

Diagnosis. All literature reviewed showed that biomarkers (creatine kinase [CK], creatine kinase myocardial band [CK-MB], myoglobin, troponin I [TnI], troponin T [TnT]) were helpful in the diagnosis of ACS/AMI. But only six studies[41–44] (CEBM level 4[45,46]; ILCOR LOE 7) showed a sensitivity of >95% within the first 4–6 h of the patient's arrival in the ED. Multimarker strategies[20,41–43,45–61] (CEBM level 1b; ILCOR/AHA LOE 7 [extrapolated from in-hospital setting]), and serial marker testing over time[41–43,45–49,51,56,58,60–69] (CEBM level 1b; ILCOR/AHA LOE 7 [extrapolated from in-hospital setting]) improved test performance.

Six out-of-hospital studies[70–75] (CEBM level 1b) showed consistent lack of support for the use of cardiac biomarkers in diagnosing AMI in the out-of-hospital phase (sensitivity 10–25%; specificity 92–100%).

Prognosis. Two systematic reviews (CEBM level 1a)[76,77] and 21 additional studies[78–98] (18 CEBM level 1b and 3 ILCOR/AHA LOE 7) documented consistent ability of cardiac biomarker testing to identify patients at increased risk of adverse outcome. One systematic review (CEBM level 1a)[76] suggested that risk assessment cannot be based exclusively on cardiac biomarker results (30-day mortality range for patients with suspected ACS and negative troponin results: 0.7–4.4%).

Treatment recommendation. Emergency physicians should obtain cardiac biomarkers for all patients with suspected ACS/AMI. Serial time points (increasing interval from onset of symptoms to testing), and multimarker strategies greatly improve sensitivity for detection of myocardial ischaemia or infarction but are insensitive for ruling out these diagnoses in the out-of-hospital setting or within the first 4–6 h of evaluation in the ED.

ED interpretation of 12-lead ECG for STEMI

Consensus on science

Diagnostic characteristics—out-of-hospital. One meta-analysis plus five prospective nonrandomised consecutive case series of patients with chest pain (CEBM level 1b-1c)[99–104] and five review articles ILCOR/AHA LOE 7[11,20,105–107] showed that trained out-of-hospital care providers (paramedics and nurses) could identify ST elevation accurately in the resting out-of-hospital 12-lead ECG of patients with chest pain suspected of having STEMI. The out-of-hospital care providers achieved a specificity of 91–100% and sensitivity of 71–97% compared with emergency physicians or cardiologists. Of note, left bundle branch block paced rhythm and idioventricular rhythm may affect the diagnostic test accuracy because they were excluded in some studies and not mentioned in others.

Prognostic characteristics—ED. ST elevation (>0.1 mV elevation in two or more adjacent limb leads or in two or more adjacent precordial leads with reciprocal depression) was the most discriminating single ECG feature for diagnosis of STEMI (likelihood ratio [LR] of 13.1; 95% confidence interval [CI], 8.28–20.6).[11] Emergency physicians blinded to biomarker results established the diagnosis of STEMI using admission ECGs with a very high specificity of 99.7% (95% CI, 98–99.9%; LR+ 145; 95% CI, 20.2–1044), although sensitivity was low at 42% (95% CI, 32–52%)[103,108,109] (CEBM 1b-1c; ILCOR/AHA LOE 7).[11]

Treatment recommendation

Out-of-hospital. Trained out-of-hospital personnel can accurately identify acute STEMI in prehospital 12-lead ECGs obtained in patients with ACS. The ECG is used in combination with chest pain symptoms, assessment of risk factors, and other diagnostic tests to rule out alternative diagnoses. Out-of-hospital interpretation of a single 12-lead ECG with stringent inclusion criteria (i.e. ST elevation >0.1 mV in two or more adjacent precordial leads or two or more adjacent limb leads and with reciprocal depression) has a high specificity for the diagnosis of STEMI.

Emergency department. In the ED the interpretation of a single 12-lead ECG with rigid inclusion criteria (see above) is discriminating for the diagnosis of STEMI with a relatively low sensitivity but a high specificity for this diagnosis.

Acute therapeutic interventions

Few studies have been published to guide out-of-hospital interventions for ACS and AMI. Extrapolat-

ing from the evidence for many of the adjunctive therapies used in-hospital within 24—48 h may provide some guidance for out-of-hospital and early ED management.

Adjunctive therapies

Oxygen therapy
W224

Consensus on science. One animal study (LOE 6)[110] showed a reduction in infarct size when supplementary oxygen was provided during left anterior descending coronary artery occlusion. One human study (LOE 5)[111] showed improvement in ECG findings, but one double-blind, randomised human trial (LOE 2)[112] of supplementary oxygen versus room air failed to show a long-term benefit of oxygen therapy for patients with MI.

Treatment recommendation. Supplementary oxygen should be given to patients with arterial oxygen desaturation (arterial oxygen saturation [SaO_2] <90%). Given the safety profile of oxygen in this population and the potential benefit in the patient with unrecognized hypoxia, it is reasonable to give supplementary oxygen to all patients with uncomplicated STEMI during the first 6 h of emergency management.

Aspirin (acetylsalicylic acid)
W225A,W225B

Consensus on science. Eight randomised controlled trials (RCTs) (LOE 1)[113—120] showed decreased mortality rates when acetylsalicylic acid (ASA) (75—325 mg) was given to hospitalized patients with ACS. The International Study of Infarct Survival (ISIS)-2 trial used 160 mg day^{-1} orally (odds reduction = 0.23; 95% CI, 0.15—0.30).[115]

Four RCTs (LOE 1)[115,116,120,121] and three additional studies (LOE 7)[122—124] indicated decreased mortality rates when ASA was given as early as possible.

Two studies (LOE 1)[125,126] addressed specific ASA dose, but the standard of 160 mg enteric-coated ASA has still been maintained from ISIS-2. Two studies showed that chewed (LOE 3)[127] or soluble ASA (LOE 6)[128] provides more rapid bioavailability than swallowed tablets. Two nonblinded studies (LOE 7)[124,129] showed that 50 mg of intravenous (IV) ASA was >90% effective in inhibiting thromboxane A_2 and inhibits platelets effectively.

One post hoc study suggested decreased mortality rates with out-of-hospital administration of ASA (LOE 7).[123]

Seven hospital-based RCTs indicated that giving ASA to patients with suspected ACS is safe (LOE 1).[113—115,117,118,120,121]

Treatment recommendation. It is reasonable for dispatchers to advise the patient with suspected ACS and without a true aspirin allergy to chew a single dose (160—325 mg) of ASA. It is also reasonable for EMS providers to administer ASA because there is good evidence that it is safe and that the earlier ASA is given, the greater the reduction in risk of mortality.

Limited evidence from several very small studies suggests that the bioavailability and pharmacologic action of other formulations of ASA (soluble, IV) may be as effective as chewed tablets.

Heparins
W226A

Consensus on science

UA/NSTEMI Six in-hospital RCTs (LOE 1[130,131] and LOE 2[121,132,133] <24 h; LOE 1[134] <36 h) and additional studies (including seven meta-analyses, [135—141]) documented similar or improved composite outcomes (death, MI or recurrent angina, or recurrent ischaemia or revascularisation) after giving low-molecular-weight heparin (LMWH) instead of unfractionated heparin (UFH) to patients with UA/NSTEMI within the first 24—36 h after onset of symptoms. No study evaluated the early use of LMWH versus UFH in the first 6 h of management.

Extrapolation (LOE 7) from one RCT[133] and one meta-analysis (LOE 1)[135] suggests that changing from one form of heparin to another (crossover of antithrombin therapy) during initial treatment of an acute event may not be safe or effective in patients with UA/NSTEMI.

There is no evidence that LMWH is superior to UFH in the group of patients who will receive early percutaneous coronary intervention (PCI).

STEMI. In two RCTs (LOE 1[142]; LOE 2[143]) and additional studies, including one meta-analysis (LOE 1),[144] LMWH (specifically enoxaparin) improved overall TIMI flow[145] (coronary reperfusion) and ischemic outcomes better than UFH when given to patients with STEMI within 6 h of onset of symptoms. TIMI flow grade was defined by investigators from the TIMI study[145] as the degree of reperfusion, ranging from 0 for no flow through 3 for complete, brisk flow.

Two studies (LOE 1[146]; LOE 2[147]) in the out-of-hospital setting documented improved composite outcomes with LMWH (specifically enoxaparin) in comparison with UFH, when given to patients with STEMI as adjunctive therapy to fibrinolysis. This must be balanced against the increase in intracranial haemorrhage in patients >75 years receiving LMWH (enoxaparin) that was observed in one of these RCTs (LOE 2).[147]

In patients with STEMI proceeding to PCI, there is no evidence in favour of LMWH.

In one RCT (LOE 1)[148] there was no difference in the incidence of death, reinfarction, or recurrent angina with LMWH (enoxaparin) in comparison with UFH when given to patients who were ineligible for reperfusion therapy.

Treatment recommendation

UA/NSTEMI. In the ED giving LMWH instead of UFH in addition to aspirin to patients with UA/NSTEMI may be helpful. There is insufficient evidence to identify the optimal time for administration after onset of symptoms. In-hospital administration of UFH is recommended if reperfusion is planned within the first 24–36 h after onset of symptoms. There is insufficient evidence to recommend for or against treatment with LMWH in UA/NSTEMI in the out-of-hospital setting. Changing from one form of heparin to another (crossover of antithrombin therapy) during an acute event is not recommended.

STEMI. LMWH is an acceptable alternative to UFH as ancillary therapy for patients with STEMI who are <75 years and receiving fibrinolytic therapy. LMWH should not be given if significant renal dysfunction (serum creatinine >2.5 mg dl^{-1} in men or 2 mg dl^{-1} in women) is present. UFH is recommended for patients ≥75 years as ancillary therapy to fibrinolysis.

Heparin may be given to STEMI patients who do not receive reperfusion therapy. These include patients at high risk for cardioembolic events and those on prolonged bedrest. UFH or LMWH may be used. Patients receiving LMWH should have no significant renal dysfunction.

Clopidogrel
W228A

Consensus on science. In two in-hospital, randomised, double-blind, controlled trials (LOE 1)[149,150] and four post hoc analyses (LOE 7),[151–154] clopidogrel was effective in reducing the combined event rate (stroke, nonfatal infarction, deaths from cardiovascular causes, refractory ischaemia, heart failure, and need for revascularisation) in patients with suspected ACS with evidence of ischaemia but no infarction. In these studies clopidogrel was given within the first 4 h of presentation to the hospital in addition to standard care (ASA, heparin) to patients with ACS who had a rise in serum level of cardiac biomarkers or new ECG changes consistent with ischaemia but no ST-segment elevation.

One large randomised, double-blind, controlled trial (LOE 7)[155] documented no significant increase in risk of bleeding with clopidogrel in comparison with ASA. One large multicenter RCT (LOE 1)[156] documented a significant reduction in adverse ischemic events at 28 days after elective PCI when clopidogrel was given at least 6 h before elective PCI.

One multicenter, randomised, double-blind, controlled trial (LOE 1)[157] documented a significant reduction in the composite end point of an occluded infarct-related artery (defined by a TIMI flow grade of 0 or 1) on angiography or death or recurrent MI before angiography when clopidogrel (300 mg oral loading dose) was given at the time of initial management (followed by a 75-mg daily dose for up to 8 days in hospital) to patients up to 75 years with STEMI who were treated with fibrinolysis, ASA, and heparin (LMWH or UFH).

In one large prospective STEMI trial (the CURE [Clopidogrel in Unstable angina to prevent Recurrent Events] trial),[152] preoperative clopidogrel was associated with a trend toward increased postoperative reoperation for bleeding in the 2072 patients who underwent coronary artery bypass graft (CABG) surgery. A second prospective trial (LOE 1)[157] failed to show any increase in bleeding in the 136 patients who underwent CABG within 5–7 days of receiving clopidogrel. A subsequent risk to benefit ratio analysis concluded that the bleeding risk with clopidogrel in patients undergoing CABG was overestimated.[154]

Treatment recommendation. Give a 300-mg oral loading dose of clopidogrel in addition to standard care (ASA, heparin) to patients with ACS within 4–6 h of contact if they have:

- A rise in serum cardiac biomarkers or new ECG changes consistent with ischaemia when a medical approach or PCI is planned in the absence of ST-segment elevation.
- STEMI in patients up to 75 years of age receiving fibrinolysis, ASA, and heparin.

Although in one large trial[152] preoperative clopidogrel was associated with increased reoperation for postoperative bleeding, the recent CLARITY TIMI 28 trial[157] did not document increased bleed-

ing in 136 patients undergoing CABG within 5–7 days of receiving clopidogrel. Current ACC/AHA recommendations[2] advise withholding clopidogrel for 5–7 days before planned CABG.

It is reasonable to give clopidogrel 300 mg orally to patients with suspected ACS (without ECG or cardiac marker changes) who have hypersensitivity to or gastrointestinal intolerance of ASA.

Glycoprotein IIb/IIIa inhibitors

Consensus on science

UA/NSTEMI. Two studies (LOE 1[158]; LOE 2[159]) and two meta-analyses (LOE 1)[158,160] showed a reduction in the combined end point of death or recurrent ischaemia when glycoprotein (GP) IIb/IIIa inhibitors were added to standard therapy (including ASA and heparin) for patients with high-risk UA/NSTEMI treated with PCI. High-risk features include persistent ongoing pain due to ischaemia, haemodynamic or rhythm instability due to ongoing ischaemia, acute or dynamic ECG changes, and any elevation in cardiac troponins attributed to ACS.

Two studies (LOE 1)[158,161] and three meta-analyses (LOE 1)[160,162,163] failed to show a reduction in the combined end point of death or recurrent ischaemia in patients with UA/NSTEMI treated with tirofiban or eptifibatide without PCI. Two studies (LOE 1)[164,165] showed that abciximab given in addition to standard therapy but without PCI in patients with UA/NSTEMI did not reduce the combined end point of death or recurrent ischaemia.

No published studies evaluated the out-of-hospital use of GP IIb/IIIa inhibitors. Three studies (LOE 1)[158,160,163] showed the safety (as defined by incidence of major haemorrhagic complications) of GP IIb/IIIa inhibitors when given to ACS patients within 24–48 h of onset of symptoms.

STEMI. In multiple studies (LOE 1[166–168]; LOE 2 [130,169–174]; LOE 4[175]; LOE 7[176]) there was no reduction in the combined end point of death or recurrent ischaemia when tirofiban or eptifibatide were given in combination with reduced-dose fibrinolytics to patients with STEMI in the absence of PCI.

Two RCTs (LOE 1)[165,177] in patients with STEMI treated with abciximab and fibrinolytics showed no reduction in the combined end point of death or recurrent ischaemia. One meta-analysis (LOE 1)[178] showed reduction in short-term reinfarction rate when abciximab was given with fibrinolytics or PCI, whereas the benefits in mortality-rate reduction were seen only in patients treated with PCI.

One RCT failed to show a benefit with tirofiban in addition to standard therapy when given out-of-hospital (LOE 2).[171] Another study demonstrated the feasibility of using abciximab in the out-of-hospital setting (LOE 7).[175] A third study showed a trend toward improved patency of infarct-related artery with PCI (LOE 3).[179]

Treatment recommendation

High-risk UA/NSTEMI. If revascularisation therapy (PCI or surgery) is planned, it is safe to give GP IIb/IIIa inhibitors in addition to standard therapy (including ASA and heparin) to patients with high-risk UA/NSTEMI in the ED. This therapy may reduce the risk of death or recurrent ischaemia. High-risk features of UA/NSTEMI are defined in the consensus on science statement above.

If revascularisation therapy is not planned, the recommendation for use of GP IIb/IIIa varies by drug. Tirofiban and eptifibatide may be used in patients with high-risk UA/NSTEMI in conjunction with ASA and LMWH if PCI is not planned. But abciximab can be harmful in patients with high-risk UA/NSTEMI if early (e.g. 24 h) PCI is not planned.

STEMI Abciximab is not currently recommended in patients receiving fibrinolytics for STEMI. In patients treated with PCI without fibrinolysis, abciximab may be helpful in reducing mortality rates and short-term reinfarction. There is no evidence documenting a better outcome by giving GP IIb/IIIa inhibitors out of hospital or early in the ED.

Reperfusion strategies

Out-of-hospital fibrinolytics for STEMI
W227A

Consensus on science. One meta-analysis (LOE 1)[180] and multiple studies (LOE 1[181,182]; LOE 2[183–185]; LOE 3[147,186–188]; LOE 4[189–192]; LOE 5[193]; LOE 7[102,194–196]) documented reduced time to injection of fibrinolytics when given by out-of-hospital providers (physicians, nurses, or paramedics) to patients with STEMI and no contraindications to fibrinolysis. In most studies the duration of symptoms was from 30 min to 6 h from onset of symptoms. Using the same criteria, one meta analysis (LOE 1)[180] and eight additional studies (LOE 1[181,197]; LOE 2[184,198]; LOE 3[187]; LOE 4[191,192]; LOE 5[199]) documented reduced risk of mortality with out-of-hospital fibrinolysis.

Treatment recommendation. Out-of-hospital administration of fibrinolytics by paramedics, nurses, or physicians using an established protocol is safe and feasible for patients with STEMI and no

contraindications. This requires adequate provisions for the diagnosis and treatment of STEMI and its complications, including strict treatment directives, fibrinolytic checklist, ECG acquisition and interpretation, defibrillators, experience in ACLS protocols, and the ability to communicate with medical control. Physicians may give out-of-hospital fibrinolytics to patients with symptoms compatible with ACS and signs of true posterior infarctions (no ST elevation).

Fibrinolytics in the ED management of STEMI
W227B

Consensus on science. A prospective cohort study (LOE 3)[200] and 11 additional studies (LOE 3[201–208]; LOE 4[209]; LOE 5[210,211]) documented reduced delay to injection of fibrinolytics and some decrease in mortality (LOE 3)[200,212] and improved left ventricular function (LOE 3)[206] when fibrinolytics were given in the ED to selected patients with STEMI (defined in studies with variable ST-elevation criteria with or without new onset left bundle branch block (LBBB) ± posterior infarct) and no contraindications.

Treatment recommendation. In the ED patients with symptoms of ACS and ECG evidence of either STEMI (presumably) new LBBB, or true posterior infarction should be given fibrinolytics if fibrinolysis is the treatment of choice and there are no contraindications. The emergency physician should give fibrinolytics as early as possible according to a predetermined protocol.

Primary PCI compared with ED or out-of-hospital fibrinolysis
W234A,W234B

Consensus on science. Six randomised studies (LOE 1),[213–218] three meta-analyses (LOE 1),[219–221] and 24 additional studies (LOE 2–4)[222–245] compared primary PCI with fibrinolysis in patients with STEMI. These studies documented consistent improvement in the combined end point of death, stroke, and reinfarction when PCI was undertaken by skilled personnel in a high-volume center (i.e. >75 procedures per operator annually) with minimal delay. Minimal delay was defined as balloon inflation ≤90 min after first medical contact (i.e. contact with a healthcare provider who can make a decision to treat or transfer). In these studies the typical additional delay from decision to treat to either PCI or ED fibrinolysis was ≤60 min.

One study (LOE 1)[217] and a post hoc subgroup analysis (LOE 7)[246] of fibrinolysis compared with PCI showed no difference in survival rates when fibrinolysis was initiated within 2 h[246] or 3 h[217] after onset of symptoms.

One RCT and a 1-year follow-up of the same study (LOE 1)[216,247] comparing early revascularisation (e.g. surgery, facilitated PCI, and primary PCI) with medical therapy in patients with cardiogenic shock showed decreased six-month and 1-year mortality rates, especially for patients <75 years. Direct comparison of the outcome of primary PCI patients to patients who received only fibrinolytic therapy was not reported.

Treatment recommendation. All patients presenting with STEMI within 12 h of the onset of symptoms should be evaluated for reperfusion therapy (i.e. fibrinolysis or PCI).

Primary PCI is the preferred reperfusion strategy in STEMI with symptom duration >3 h if a skilled team can perform primary PCI in ≤90 min after first medical contact with the patient or if there are contraindications to fibrinolysis.

If the duration of symptoms is ≤3 h, treatment is more time-sensitive, and the superiority of out-of-hospital fibrinolysis, immediate in-hospital fibrinolysis, or transfer for primary PCI is not established (see below for further discussion of transfer).

Early revascularisation (i.e. surgery, primary or early PCI, defined as PCI ≤24 h after fibrinolysis) is reasonable in patients with cardiogenic shock, especially for patients <75 years.

Primary and secondary prevention interventions

Traditional preventive interventions usually start with the first admission with a confirmed diagnosis of ACS. Therapeutic options include antiarrhythmics, β-blockers, angiotensin-converting enzyme (ACE) inhibitors, and HMG-CoA reductase inhibitors (statins). The current evidence indicates that with the exception of β-blockers, none plays a significant role in the out-of-hospital and ED management of ACS.

Antiarrhythmics
W230

Lidocaine

Consensus on science. When lidocaine was given by physicians or paramedics for primary prophylaxis within the first 4 h of a suspected STEMI in the out-of-hospital setting, four meta-analyses (LOE 1)[248–251] and two RCTs (LOE 2)[250,252] showed a trend toward increased mortality rates. In addi-

tion, two meta-analyses[253,254] and 15 RCTs (LOE 1[255]; LOE 2[256–269]), one case series (LOE 5),[270] and one retrospective trial (LOE 5)[271] showed no effect of lidocaine on mortality in this setting. Only one small study (LOE 2)[272] showed a decrease in mortality with prophylactic lidocaine. Several trials (LOE 2[258,259,262,264,265]; LOE 5[270]) reported more side effects (including paraesthesia, tinnitus, confusion, bradycardia requiring treatment, seizures, coma, and respiratory arrest) in patients receiving prophylactic lidocaine.

Magnesium

Consensus on science. Giving magnesium prophylactically to patients with STEMI has produced mixed results. One study (LOE 2)[273] showed a decrease in mortality and symptomatic arrhythmias. One meta-analysis (LOE 1)[274] and two RCTs (LOE 1[275]; LOE 2[276]) showed a decrease in mortality but no reduction in ventricular arrhythmias. One small RCT (LOE 2)[277] showed that magnesium reduced the incidence of ventricular tachycardia, but it was underpowered to assess mortality. The definitive study on the subject is the ISIS-4 study (LOE 1).[278] ISIS-4 enrolled >58,000 patients and showed a trend toward increased mortality when magnesium was given in-hospital for primary prophylaxis to patients within the first 4 h of known or suspected AMI.

Disopyramide, mexiletine, and verapamil

Consensus on science. One multi-antiarrhythmic meta-analysis (LOE 1)[279] and four RCTs (LOE 2[280–282]; LOE 7[283]) showed no effect on mortality when a variety of antiarrhythmic drugs (disopyramide, mexiletine, and verapamil) were given for primary prophylaxis by paramedics or physicians to patients within the first 4 h of known or suspected AMI.

Treatment recommendation for antiarrhythmics. There is insufficient evidence to support the routine use of any antiarrhythmic drug as primary prophylaxis within the first 4 h of proven or suspected AMI.

This conclusion does not take into account the potential effect of β-blockers discussed below.

β-Blockers
W232

Consensus on science. Two in-hospital RCTs (LOE 1)[284,285] and two supporting studies (LOE 2)[286,287] completed before the advent of fibrinolytics documented decreased mortality, reinfarction, ventricular fibrillation, supraventricular arrhythmias, and cardiac rupture in patients treated with β-blockers. In patients with AMI who received fibrinolytics, treatment with IV β-blockade within 24 h of onset of symptoms reduced rates of reinfarction and cardiac rupture. IV β-blockade may reduce mortality in patients undergoing primary PCI who are not on oral β-blockers (LOE 7).[288] β-Blocker therapy was initiated in the ED for most of these trials; only one included out-of-hospital administration.[289]

One small trial (LOE 2)[290] showed a trend toward decreased mortality when IV β-blockers were given for unstable angina.

Treatment recommendation. In the ED treat ACS patients promptly with IV β-blockers followed by oral β-blockers. β-Blockers are given irrespective of the need for revascularisation therapies. Contraindications to β-blockers include hypotension, bradycardia, heart block, moderate to severe congestive heart failure, and reactive airway disease.

ACE inhibitors
W231

Consensus on science. Seven large clinical trials (LOE 1),[278,291–296] two meta-analyses (LOE 1),[297,298] and 11 minor trials (LOE 1)[296,299–308] documented consistent improvement in mortality when oral ACE inhibitors were given to patients with AMI with or without early reperfusion therapy. ACE inhibitors should not be given if hypotension (systolic blood pressure <100 mmHg or more than 30 mmHg below baseline) is present or a contraindication to these drugs exists.

One large, randomised, double-blind, placebo-controlled trial (LOE 1)[309] and two small randomised trials (LOE 2)[310,311] in adults documented a trend toward a higher mortality rate if an IV ACE inhibitor was started within the first 24 h after onset of symptoms in the hospital setting. There is no literature evaluating the therapeutic role of ACE inhibitors in the out-of-hospital setting.

Treatment recommendation. Start an oral ACE inhibitor within 24 h after onset of symptoms in patients with MI whether or not early reperfusion therapy is planned. Do not give an ACE inhibitor if the patient has hypotension (systolic blood pressure <100 mmHg or more than 30 mmHg below baseline) or if the patient has a known contraindication to these drugs. ACE inhibitors are most effective in patients with anterior infarction, pulmonary congestion, or left ventricular ejection fraction <40%.

There is no evidence to recommend for or against starting ACE inhibitors in the out-of-hospital setting. Avoid giving IV ACE inhibitors within the first 24 h after onset of symptoms because they can cause significant hypotension during this phase.

HMG CoA reductase inhibitors (statins)
W233

Consensus on science. Nine RCTs (LOE 7)[312–320] and additional small studies (LOE 3–7)[321–323] documented a consistent decrease in the incidence of major adverse cardiovascular events (reinfarction, stroke, necessary intervention for recurrent angina, and rehospitalisation) when statins were given within a few days after onset of ACS. There are few data on patients treated within 24 h of the onset of symptoms.

One retrospective analysis (LOE 4)[324] and data from one registry (LOE 4)[325] showed that patients presenting with ACS who are already taking statins should continue to take them.

There are no data on the initiation of statin therapy out-of-hospital or in the ED for patients with ACS.

Treatment recommendation. It is safe and feasible to start statin therapy early (within 24 h) in patients with ACS or AMI; once started, continue statin therapy uninterrupted.

Healthcare system interventions for ACS/AMI

Novel strategies have been developed and evaluated to improve the speed and quality of care delivered to patients with ACS. Many strategies have been shown to be safe, effective, and feasible in the prehospital setting and ED. Such strategies include out-of-hospital 12-lead ECG and advance ED notification, interfacility transfer of the patient for PCI, and a combined strategy of interfacility transfer after fibrinolysis.

12-lead out-of-hospital ECG and advance ED notification
W235A,W235B

Consensus on science. Two RCTs (LOE 2),[326,327] six nonrandomised controlled trials (LOE 3),[101,328–332] one retrospective cross-sectional study (LOE),[106] and extrapolations from two feasibility studies (LOE 4[333]; LOE 3[103]) showed a reduction of 10–60 min in the door-to-reperfusion interval for patients with STEMI when a 12-lead out-of-hospital ECG was obtained and interpreted by a physician, nurse, or paramedic and sent to the receiving hospital in advance (cellular ECG transmission or verbal communication).

One RCT (LOE 2)[326] and five other studies (LOE 5[103,334]; LOE 4[333]; LOE 3[101]; LOE 5[335]) showed that 12-lead out-of-hospital ECGs with advance notification undertaken by out-of-hospital personnel does not increase on-scene time interval significantly (0.2–5.6 min) in patients with suspected AMI.

Four studies (LOE 3[103,334,336]; LOE 5[335]) showed that out-of-hospital personnel can acquire and transmit diagnostic-quality 12-lead out-of-hospital ECGs.

Treatment recommendation. Routine use of the 12-lead out-of-hospital ECG with advance ED notification may benefit STEMI patients by reducing the time interval to fibrinolysis.

Advance ED notification may be achieved with direct transmission of the ECG itself or verbal report (via telephone) of the ECG interpretation by out-of-hospital personnel.

Interfacility transfer for primary PCI
W237A,W237B

Consensus on science. Three RCTs (LOE 2)[213,217,240] and one meta-analysis (LOE 1)[219] documented safety and improved combined event rate (30-day combined rate of death, reinfarction, or stroke) when patients with STEMI from hospitals without the capability for primary PCI were transferred promptly for primary PCI at a skilled facility. A skilled facility provides access to PCI undertaken by a skilled operator in a high-volume center (i.e. >75 procedures per operator annually) with minimal delay.[214,225,226]

When combined in a meta-analysis (LOE 1),[219] five RCTs (LOE 2)[213,217,233,240,241] showed reduced mortality rates when patients with STEMI from hospitals without the capability for primary PCI were transferred promptly to a facility with such capability.

In one RCT (LOE 2)[217] and one post hoc subgroup analysis of an RCT (LOE 7),[246] it is unclear whether immediate out-of-hospital fibrinolysis, in-hospital fibrinolysis, or transfer for primary PCI is most efficacious for patients presenting with STEMI within 2–3 h of the onset of symptoms.

Treatment recommendation. For patients with STEMI presenting >3 h but <12 h from the onset of symptoms, interfacility transfer from hospitals that lack primary PCI capability to centres capable of providing primary PCI is indicated if such a transfer

can be accomplished as soon as possible. Optimally PCI should occur ≤90 min from first medical contact (i.e. contact with a healthcare provider who can make the decision to treat or transfer).

In patients with STEMI presenting ≤3 h from onset of symptoms, treatment is more time-sensitive, and there are inadequate data to indicate the superiority of out-of-hospital fibrinolysis, immediate hospital fibrinolysis, or transfer for primary PCI.

The time recommendations do not apply to patients in cardiogenic shock. In such patients the evidence supports early revascularisation therapy (primary PCI, early PCI, or surgery) compared with medical therapy.[216]

Out-of-hospital triage for PCI
W236A,W236B

Consensus on science. A single study (LOE 2)[337] with insufficient power and some methodological concerns and a second post hoc subgroup analysis (LOE 7)[246] failed to show that out-of-hospital triage for primary PCI was any better than out-of-hospital fibrinolysis in patients with STEMI in systems involving the presence of physicians in mobile intensive care units (MICUs).

No randomised studies directly compared out-of-hospital triage for primary PCI with fibrinolytics given at a community hospital.

Extrapolations from four RCTs on interfacility transfer (LOE 7)[213,217,240] suggest that out-of-hospital STEMI patients may do better with direct triage to a primary PCI facility because of the potential for earlier treatment. A cost-effectiveness substudy of the Comparison of Angioplasty and Prehospital Thrombolysis in Acute Myocardial Infarction (CAPTIM) trial[337] using critical-care physicians during transport and for administration of fibrinolytics suggests that direct transport to a primary PCI facility may be more cost-effective than out-of hospital fibrinolysis when transport can be completed in ≤60 min. But this study excluded patients considered to be at high risk for complications during transfer (e.g. cardiogenic shock).

Treatment recommendation. There is some limited evidence to recommend out-of-hospital triage for primary PCI for patients with uncomplicated STEMI who are ≤60 min away from a PCI site in systems that use MICUs with physicians on board with the proviso that the delay from decision to treat to balloon inflation is ≤90 min. Further studies are required to define appropriate triage and transport criteria.

Interfacility transfer for early PCI
W237A,W237B

Consensus on science. A strategy of fibrinolysis combined with transfer for early PCI (defined as PCI performed ≤24 h after fibrinolysis) is supported by six randomised trials (LOE 1[223,338,339] and LOE 2[241,340,341]). The efficacy of this strategy is also supported by a post hoc nonrandomised comparison (LOE 3).[342] But this strategy is not supported by other RCTs (LOE 1[343–345]; LOE 2[223,240]) and other nonrandomised studies or secondary analyses of the above trials (LOE 7).[346] Several meta-analyses showed no benefit of early PCI (LOE 1).[347–349] All but one of these trials were carried out in the 1990s before the era of coronary stenting. These studies did not use modern drugs or contemporary PCI techniques.

The feasibility of fibrinolysis combined with transfer for early PCI is supported by three low-level studies. One study is a small trial in which PCI was performed routinely (LOE 7),[350] one is a randomised trial of low-dose fibrinolytics compared with placebo before immediate cardiac catheterization and PCI as necessary (LOE 7),[351] and one is a retrospective analysis (LOE 7).[352]

The efficacy of early PCI for patients with cardiogenic shock was shown in an RCT that showed improved mortality at six months and 1 year with early revascularisation (LOE 1),[216] especially in patients <75 years. This was supported by a retrospective analysis (LOE 7).[353]

One RCT (LOE 2) showed improvement in secondary nonfatal outcomes when early PCI was used for patients who did not achieve reperfusion after fibrinolysis.[354]

All of the above studies involved in-hospital fibrinolysis. The use of prehospital fibrinolysis followed by early PCI has not been studied.

Treatment recommendation. There is inadequate evidence to recommend the routine transfer of patients for early PCI after successful fibrinolysis in community hospital EDs or out of hospital.

Transfer for early PCI is recommended as one strategy for early revascularisation for patients with cardiogenic shock, especially patients <75 years; or with haemodynamic instability or persistent symptoms of ischaemia after fibrinolysis.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.resuscitation.2005.09.019.

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Part 6: Paediatric basic and advanced life support

International Liaison Committee on Resuscitation

The ILCOR Paediatric Task Force included expert reviewers from Africa, Asia, Australia, Asia, Europe, North America, and South America. These experts reviewed 45 topics related to paediatric resuscitation. Topics were selected from previous recommendations (the *ECC Guidelines 2000*),[1,2] emerging science, and newly identified issues. Some well-established topics without controversies or new evidence (e.g. adenosine for the treatment of supraventricular tachycardia (SVT)) are not included in this document.

Evidence-based worksheets on some topics were prepared and discussed but are not included here because there was insufficient evidence (e.g. fibrinolytics in cardiac arrest,[W13] securing the tracheal tube in children[W1], use of impedance threshold device in children,[W2] sodium bicarbonate for prolonged resuscitation attempts[W34]) or because no new evidence was found (e.g. evaluation of capillary refill,[W10] ventilation before naloxone,[W18] delayed volume resuscitation in trauma,[W17] use of hypertonic saline in shock[W16]).

The following is a summary of the most important changes in recommendations for paediatric resuscitation since the last ILCOR review in 2000.[1,2] The scientific evidence supporting these recommendations is summarised in this document:

- Emphasis on the quality of CPR is increased: ''Push hard, push fast, minimise interruptions; allow full chest recoil, and don't hyperventilate''.
  - Recommended chest compression—ventilation ratio:
    - for one lay rescuer: 30:2;
    - for healthcare providers performing two-rescuer CPR: 15:2.
  - Either the two- or one-hand technique is acceptable for chest compressions in children.
  - One initial shock followed by immediate CPR for attempted defibrillation instead of three stacked shocks.
- Biphasic attenuated shocks with an automated external defibrillator (AED) are acceptable for children >1 year.
- Routine use of high-dose adrenaline is no longer recommended.
- Either cuffed or uncuffed tracheal tubes are acceptable in infants and children.
- Exhaled CO_2 monitoring is recommended for confirmation of tracheal tube placement and during transport.
- Consider induced hypothermia for patients who remain comatose following resuscitation.
- Emphasis is increased on intravascular (intravenous (IV) and intraosseous (IO)) rather than tracheal administration of drugs.

The ILCOR Paediatric Task Force re-evaluated the definitions of newborn, infant, child, and adult. These definitions are somewhat arbitrary but are important because some recommendations for treatment differ according to patient size and the most likely aetiology of arrest. The distinction between child and adult victims has been de-emphasised by the recommendation of a universal compression—ventilation ratio for lay rescuers and the same chest compression technique for lay rescuers of children and adults. Some differences in treatment recommendations remain between the

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newborn and infant and between an infant and child, but those differences are chiefly linked to resuscitation training and practice. They are noted below.

Identified knowledge gaps in paediatric resuscitation include:

- Sensitive and specific indicators of cardiac arrest that lay rescuers and healthcare providers can recognise reliably.
- Effectiveness of aetiology-based versus age-based resuscitation sequences.
- The ideal ratio of chest compressions to ventilations during CPR.
- Mechanisms to monitor and optimise quality of CPR during attempted resuscitation.
- Best methods for securing a tracheal tube.
- Clinical data on the safety and efficacy of automated external defibrillators (AEDs).
- Clinical data on the safety and efficacy of the laryngeal mask airway (LMA) during cardiac arrest.
- The benefits and risks of supplementary oxygen during and after CPR.
- Clinical data on antiarrhythmic and pressor medications during cardiac arrest.
- Data on induced hypothermia in paediatric cardiac arrest.
- The identification and treatment of post-arrest myocardial dysfunction.
- The use of fibrinolytics and anticoagulants in cardiac arrest.
- Use of emerging technologies for assessment of tissue perfusion.
- Predictors of outcome from cardiac arrest.

Initial steps of CPR

The *ECC Guidelines 2000*[1] recommended that lone rescuers of *adult* victims of cardiac arrest phone the emergency medical services (EMS) system and get an AED (''call first'') before starting CPR. The lone rescuer of an unresponsive *infant or child* victim was instructed to provide a brief period of CPR before leaving the victim to phone for professional help and an AED (''call fast''). These sequence differences were based on the supposition that cardiac arrest in adults is due primarily to ventricular fibrillation (VF) and that a hypoxic-ischaemic mechanism is more common in children. But this simplistic approach may be inaccurate and may not provide the ideal rescue sequence for many victims of cardiac arrest. Hypoxic-ischaemic arrest may occur in adults, and VF may be the cause of cardiac arrest in up to 7% to 15% of infants and children. Resuscitation results might be improved if the sequence of lay rescuer CPR actions, (i.e. the priority of phoning for professional help, getting an AED, and providing CPR) is based on the aetiology of cardiac arrest rather than age.

The pulse check was previously eliminated as an assessment for the lay rescuer. There is now evidence that healthcare professionals may take too long to check for a pulse and may not determine the presence or absence of the pulse accurately. This may lead to interruptions in chest compressions and affect the quality of CPR.

Experts reviewed the data on the technique of rescue breathing for infants and the two-thumb-encircling hands versus two-finger chest compression techniques for infants.

One of the most challenging topics debated during the 2005 Consensus Conference was the compression—ventilation ratio. The scientific evidence on which to base recommendations was sparse, and it was difficult to arrive at consensus. Evidence was presented that the ratio should be higher than 5:1, but the optimal ratio was not identified. The only data addressing a compression—ventilation ratio greater than 15:2 came from mathematical models. The experts acknowledged the educational benefit of simplifying training for lay rescuers (specifically one-rescuer CPR) by adopting a single ratio for infants, children, and adults with the hope that simplification might increase the number of bystanders who will learn, remember, and perform CPR. On this basis experts agreed that this single compression—ventilation ratio should be 30:2. Healthcare providers typically will be experienced in CPR and practice it frequently. This group of experienced providers will learn two-person CPR, and for them the recommended compression—ventilation ratio for two rescuers is 15:2.

Some laypeople are reluctant to perform mouth-to-mouth ventilation. For treatment of cardiac arrest in infants and children, chest compressions alone are better than no CPR but not as good as a combination of ventilations and compressions.

In the past one-handed chest compressions were recommended for CPR in children. A review of the evidential basis for this recommendation was conducted. From an educational standpoint, we agree that it will simplify training to recommend a single technique for chest compressions for children and adults.

Activating emergency medical services and getting the AED
W4

Consensus on science. Most cardiac arrests in children are caused by asphyxia (LOE 4).[3–6]

Observational studies of non-VF arrests in children show an association between bystander CPR and intact neurological outcome (LOE 4).[6–8] Animal studies show that in asphyxial arrest, chest compressions plus ventilation CPR is superior to either chest compression-only CPR or ventilation-only CPR (LOE 6).[9]

Observational studies of children with VF report good (17% to 20%) rates of survival after early defibrillation (LOE 4).[5,6,10] The merits of ''call first'' versus ''call fast'' CPR sequences have not been studied adequately in adults or children with cardiac arrest of asphyxial or VF etiologies. Three animal studies (LOE 6)[9,11,12] show that even in prolonged VF, CPR increases the likelihood of successful defibrillation, and seven adult human studies (LOE 7)[13–19] document improved survival with the combination of CPR with minimal interruptions in chest compression and early defibrillation.

Treatment recommendation. A period of immediate CPR before phoning emergency medical services (EMS) and getting the AED (''call fast'') is indicated for most paediatric arrests because they are presumed to be asphyxial or prolonged. In a witnessed sudden collapse (e.g. during an athletic event), the cause is more likely to be VF, and the lone rescuer should phone for professional help and get the AED (when available) before starting CPR. Rescuers should perform CPR with minimal interruptions in chest compressions until attempted defibrillation.

In summary, the priorities for *unwitnessed* or non-sudden collapse in children are as follows:

- Start CPR immediately.
- Activate EMS/get the AED.

The priorities for *witnessed* sudden collapse in children are as follows:

- Activate EMS/get the AED.
- Start CPR.
- Attempt defibrillation.

Pulse check
W5A,W5B

Consensus on science. Ten studies (LOE 2[20,21]; LOE 4[22–26]; LOE 5[27]; LOE 6[28,29]) show that lay rescuers[23,25,30] and healthcare providers[20,21,24,26–29] show that rescuers are often unable to accurately determine the presence of a pulse within 10 s. Two studies in infants (LOE 5)[31,32] reported that rescuers detected cardiac activity rapidly by direct chest auscultation but were biased because they knew that the infants were healthy.

Treatment recommendation. Lay rescuers should start chest compressions for an unresponsive infant or child who is not moving or breathing. Healthcare professionals may also check for a pulse but should proceed with CPR if they cannot feel a pulse within 10 s or are uncertain if a pulse is present.

Ventilations in infants
W7A,W7B

Consensus on science. One LOE 5[33] study and 10 LOE 7[34–43] reports assessed a mouth-to-nose ventilation technique for infants. The LOE 5 study[33] is an anecdotal report of three infants ventilated with mouth-to-nose technique. The LOE 7 reports describe postmortem anatomy,[34] physiology of nasal breathing,[35–37] related breathing issues,[38,39] and measurements of infants' faces compared with the measurement of adult mouths.[40–43] There is great variation in these measurements, probably because of imprecise or inconsistent definitions.

Treatment recommendation. There are no data to justify a change from the recommendation that the rescuer attempt mouth-to—mouth-and-nose ventilation for infants. Rescuers who have difficulty achieving a tight seal over the mouth and nose of an infant, however, may attempt either mouth-to-mouth or mouth-to-nose ventilation (LOE 5).[33]

Circumferential versus two-finger chest compressions
W9A,W9B

Consensus on science. Two manikin (LOE 6)[44,45] and two animal (LOE 6)[46,47] studies showed that the two thumb-encircling hands technique of chest compressions with circumferential thoracic squeeze produces higher coronary perfusion pressures and more consistently correct depth and force of compression than the two-finger technique.

Case reports (LOE 5)[48,49] of haemodynamic monitoring in infants receiving chest compressions showed higher systolic and diastolic arterial pressures in the two-thumb encircling hands technique compared with the two-finger technique.

Treatment recommendation. The two thumb-encircling hands chest compression technique with thoracic squeeze is the preferred technique for two-rescuer infant CPR. The two-finger technique is recommended for one-rescuer infant CPR to facilitate rapid transition between compression and ventilation to minimise interruptions in chest compressions. It remains an acceptable alternative method of chest compressions for two rescuers.

One- versus two-hand chest compression technique
W276

Consensus on science. There are no outcome studies that compare one- versus two-hand compressions of the chest in children. One (LOE 6)[50] study reported higher pressures generated in child manikins using the two-hand technique to compress over the lower part of the sternum to a depth of approximately one-third the anterior-posterior diameter of the chest. Rescuers reported that this technique was easy to perform.

Treatment recommendation. Both the one- and two-hand techniques for chest compressions in children are acceptable provided that rescuers compress over the lower part of the sternum to a depth of approximately one-third the anterior-posterior diameter of the chest. To simplify education, rescuers can be taught the same technique (i.e. two-hand) for adult and child compressions.

Compression—ventilation ratio
W3A,W3B,W3C

Consensus on science. There are insufficient data to identify an optimal compression—ventilation ratio for CPR in children. Manikin studies (LOE 6)[51–54] have examined the feasibility of compression—ventilation ratios of 15:2 and 5:1. Lone rescuers cannot deliver the desired number of chest compressions per minute at a ratio of 5:1. A mathematical model (LOE 7)[55] supports compression—ventilation ratios higher than 5:1 for infants and children.

Two animal (LOE 6)[56,57] studies show that coronary perfusion pressure, a major determinant of success in resuscitation, declines with interruptions in chest compressions. In addition, once compressions are interrupted, several chest compressions are needed to restore coronary perfusion pressure. Frequent interruptions of chest compressions (e.g. with a 5:1 compression—ventilation ratio) prolongs the duration of low coronary perfusion pressure. Long interruptions in chest compressions have been documented in manikin studies (LOE 6)[58,59] and both in- and out-of-hospital adult CPR studies (LOE 7).[60,61] These interruptions reduce the likelihood of a return of spontaneous circulation (LOE 7).[62–64]

Five animal (LOE 6)[9,56,57,65,66] studies and one review (LOE 7)[67] review suggest that ventilations are relatively less important in victims with VF or pulseless ventricular tachycardia (VT) cardiac arrest than in victims with asphyxia-induced arrest. But even in asphyxial arrest, few ventilations are needed to maintain an adequate ventilation-perfusion ratio in the presence of the low cardiac output (and, consequently low pulmonary blood flow) produced by chest compressions.

Treatment recommendation. For ease of teaching and retention, a universal compression—ventilation ratio of 30:2 is recommended for the lone rescuer responding to infants (for neonates see Part 7: ''Neonatal Resuscitation''), children, and adults. For healthcare providers performing two-rescuer CPR, a compression—ventilation ratio of 15:2 is recommended. When an advanced airway is established (e.g. a tracheal tube, Combitube, or laryngeal mask airway (LMA)), ventilations are given without interrupting chest compressions.

Some CPR versus no CPR
W8

Consensus on science. Numerous reports (LOE 5)[4,5,8,68–70] document survival of children after cardiac arrest when bystander CPR was provided. Bystander CPR in these reports included rescue breathing alone, chest compressions alone, or a combination of compressions and ventilations.

One prospective and three retrospective studies of adult VF (LOE 7)[71–74] and numerous animal studies of VF cardiac arrest (LOE 6)[56,57,66,75–79] document comparable long-term survival after chest compressions alone or chest compressions plus ventilations, and both techniques result in better outcomes compared with no CPR. In animals with asphyxial arrest (LOE 6),[9] the more common mechanism of cardiac arrest in infants and children, best results are achieved with a combination of chest compressions and ventilations. But resuscitation with either ventilations only or chest compressions only is better than no CPR.

Treatment recommendation. Bystander CPR is important for survival from cardiac arrest. Trained rescuers should be encouraged to provide both ventilations and chest compressions. If rescuers are reluctant to provide rescue breaths, however, they should be encouraged to perform chest compressions alone without interruption.

Disturbances in cardiac rhythm

Evidence evaluation for the treatment of haemodynamically stable arrhythmias focused on vagal manoeuvres, amiodarone, and procainamide. There were no new data to suggest a change in the indications for vagal manoeuvres or procainamide. Several case series described the safe and effective

use of amiodarone in children, but these studies involved selected patient populations (often with postoperative arrhythmias) treated by experienced providers in controlled settings. Although there is no change in the recommendation for amiodarone as a treatment option in children with stable arrhythmias, providers are encouraged to consult with an expert knowledgeable in paediatric arrhythmias before initiating drug therapy.

There is insufficient evidence to identify an optimal shock waveform, energy dose, and shock strategy (e.g. fixed versus escalating shocks, one versus three stacked shocks) for defibrillation. The new recommendation for the sequence of defibrillation in children is based on extrapolated data from adult and animal studies with biphasic devices, data documenting the high rates of success for first shock conversion of VF with biphasic waveforms, and knowledge that interruption of chest compressions reduces coronary perfusion pressure. Thus, a one-shock strategy may be preferable to the three-shock sequence recommended in the *ECC Guidelines 2000*.[2] For further details, see Part 3: Defibrillation.

Many, but not all, AED algorithms have been shown to be sensitive and specific for recognising shockable arrhythmias in children. A standard AED (adult AED with adult pad-cable system) can be used for children older than about 8 years and weighing more than about 25 kg. Many manufacturers now provide a method for attenuating the energy delivered to make the AED suitable for smaller children (e.g. use of a pad-cable system or an AED with a key or switch to select a smaller dose).

Management of supraventricular tachycardias

If the child with SVT is haemodynamically stable, we recommend early consultation with a paediatric cardiologist or other physician with appropriate expertise. This recommendation is common for all of the SVT topics below.

Vagal manoeuvres for SVT
W36

Consensus on science. One prospective (LOE 3)[80] and nine observational studies (LOE 4[81]; LOE 5[82,83]; LOE 7[84–89]) show that vagal manoeuvres are effective in terminating SVT in children. There are reports of complications from carotid sinus massage and application of ice to the face to stimulate the diving reflex (LOE 5),[90,91] but virtually none from the Valsalva manoeuvre.

Treatment recommendation. The Valsalva manoeuvre and ice application to the face may be used to treat haemodynamically stable SVT in infants and children. When performed correctly, these manoeuvres can be initiated quickly and safely and without altering subsequent therapies if they fail.

Amiodarone for haemodynamically stable SVT
W38

Consensus on science. One prospective (LOE 3)[92] and 10 observational (LOE 5)[93–102] studies show that amiodarone is effective for treating SVT in children. A limitation of this evidence is that most of the studies in children describe treatment for postoperative junctional ectopic tachycardia.

Treatment recommendation. Amiodarone may be considered in the treatment of haemodynamically stable SVT refractory to vagal manoeuvres and adenosine. Rare but significant acute side effects include bradycardia, hypotension, and polymorphic VT (LOE 5).[103–105]

Procainamide for haemodynamically stable SVT
W37

Consensus on science. Experience with procainamide in children is limited. Twelve LOE 5[106–117] and four LOE 6[118–121] observational studies show that procainamide can terminate SVT that is resistant to other drugs. Most of these reports include mixed populations of adults and children. Hypotension following procainamide infusion results from its vasodilator action rather than a negative inotropic effect (LOE 5[122,123]; LOE 6[124]).

Treatment recommendation. Procainamide may be considered in the treatment of haemodynamically stable SVT refractory to vagal manoeuvres and adenosine.

Management of stable wide-QRS tachycardia

If a child with wide-QRS tachycardia is haemodynamically stable, early consultation with a paediatric cardiologist or other physician with appropriate expertise is recommended. In general, amiodarone and procainamide should not be administered together because their combination may increase risk of hypotension and ventricular arrhythmias.

Amiodarone
W39A,W39B,W40

Consensus on science. One case series (LOE 5)[125] suggests that wide-QRS tachycardia in children is

more likely to be supraventricular than ventricular in origin. Two prospective studies (LOE 3)[92,126] and 13 case series (LOE 5)[93–102,127–129] show that amiodarone is effective for a wide variety of tachyarrhythmias in children. None of these reports specifically evaluates the role of amiodarone in the setting of a stable, unknown wide-complex tachycardia.

Treatment recommendation. Wide-QRS tachycardia in children who are stable may be treated as SVT. If the diagnosis of VT is confirmed, amiodarone should be considered.

Procainamide for stable VT
W35

Consensus on science. Twenty (LOE 5)[106,115,123,130–146] and two LOE 6[118,124] observational studies, primarily in adults, but including some children show that procainamide is effective in the treatment of stable VT.

Treatment recommendation. Procainamide may be considered in the treatment of haemodynamically stable VT.

Management of unstable VT

Amiodarone
W39A,W40

Consensus on science. In small paediatric case series (LOE 3[100]; LOE 5[93,95,97,99,147–149]) and extrapolation from animal (LOE 6)[150,151] and adult (LOE 7)[152–165] studies, amiodarone is safe and effective for haemodynamically unstable VT in children.

Treatment recommendation. Synchronised cardioversion remains the treatment of choice for unstable VT. Amiodarone may be considered for treatment of haemodynamically unstable VT.

Paediatric defibrillation

For additional information about consensus on science and treatment recommendations for defibrillation (e.g. one versus three stacked shock sequences and sequence of CPR first versus defibrillation first), see Part 3: ''Defibrillation.''

Manual and automated external defibrillation
W41A,W41B

Consensus on science. The ideal energy dose for safe and effective defibrillation in children is unknown. Extrapolation from adult data (LOE 1[166,167]; LOE 2[168–170]) and paediatric animal studies (LOE 6)[171–173] shows that biphasic shocks are at least as effective as monophasic shocks and produce less postshock myocardial dysfunction. One LOE 5[174] and one LOE 6[171] study show that an initial monophasic or biphasic shock dose of 2 J kg^{-1} generally terminates paediatric VF. Two paediatric case series (LOE 5)[175,176] report that doses >4 J kg^{-1} (up to 9 J kg^{-1}) have effectively defibrillated children <12 years, with negligible adverse effects.

In five animal studies (LOE 6)[172,173,177–179] large (per kilogram) energy doses caused less myocardial damage in young hearts than in adult hearts. In three animal studies (LOE 6)[173,179,180] and one small paediatric case series (LOE 5),[176] a 50-J biphasic dose delivered through a paediatric pad/cable system terminated VF and resulted in survival. One piglet (13–26 kg) study (LOE 6)[179] showed that paediatric biphasic AED shocks (50/75/86 J) terminated VF and caused less myocardial injury and better outcome than adult AED biphasic shocks (200/300/360 J).

Treatment recommendation. The treatment of choice for paediatric VF/pulseless VT is prompt defibrillation, although the optimum dose is unknown. For manual defibrillation, we recommend an initial dose of 2 J kg^{-1} (biphasic or monophasic waveform). If this dose does not terminate VF, subsequent doses should be 4 J kg^{-1}.

For automated defibrillation, we recommend an initial paediatric attenuated dose for children 1–8 years and up to about 25 kg and 127 cm in length. There is insufficient information to recommend for or against the use of an AED in infants <1 year. A variable dose manual defibrillator or an AED able to recognise paediatric shockable rhythms and equipped with dose attenuation are preferred; if such a defibrillator is not available, a standard AED with standard electrode pads may be used. A standard AED (without a dose attenuator) should be used for children ≥25 kg (about 8 years) and adolescent and adult victims.

Management of shock-resistant VF/pulseless VT

Amiodarone
W20,W21A,W21B

Consensus on science. Evidence extrapolated from three (LOE 1) studies in adults (LOE 7 when applied to children)[154,159,181] shows increased survival to hospital admission but not discharge when amiodarone is compared with placebo or lidocaine for shock-resistant VF. One study in children (LOE

3)[100] showed effectiveness of amiodarone for life-threatening ventricular arrhythmias.

Treatment recommendation. Intravenous amiodarone can be considered as part of the treatment of shock-refractory or recurrent VT/VF.

Airway and ventilation

Maintaining a patent airway and ventilation are fundamental to resuscitation. Adult and animal studies during CPR suggest detrimental effects of hyperventilation and interruption of chest compressions. For children requiring airway control or ventilation for short periods in the out-of-hospital setting, bag-valve-mask (BVM) ventilation produces equivalent survival rates compared with ventilation with tracheal intubation.

The risks of tracheal tube misplacement, displacement, and obstruction are well recognized, and an evidence-based review led to a recommendation that proper tube placement and patency be monitored by exhaled CO_2 throughout transport. A review also found that cuffed tracheal tubes could be used safely even in infants.

Following the return of spontaneous circulation from cardiac arrest, toxic oxygen by-products (reactive oxygen species, free radicals) are produced that may damage cell membranes, proteins, and DNA (reperfusion injury). There are no clinical studies in children outside the newborn period comparing different concentrations of inspired oxygen during and immediately after resuscitation, and it is difficult to differentiate sufficient from excessive oxygen therapy.

Bag-valve-mask ventilation
W6

Consensus on science. One out-of-hospital paediatric randomised controlled study (LOE 1)[182] in an EMS system with short transport times showed that BVM ventilation compared with tracheal intubation resulted in equivalent survival to hospital discharge rates and neurological outcome in children requiring airway control, including children with cardiac arrest and trauma.

One study in paediatric cardiac arrest (LOE 4)[183] and four studies in children with trauma (LOE 3[184,185]; LOE 4[186,187]) found no advantage of tracheal intubation over BVM ventilation.

Treatment recommendation. In the out-of-hospital setting with short transport times, BVM ventilation is the method of choice for children who require ventilatory support. When transport times are long, the relative benefit versus potential harm of tracheal intubation compared with BVM ventilation is uncertain. It is affected by the level of training and experience of the healthcare professional and the availability of exhaled CO_2 monitoring during intubation and transport.

Advanced airways

Advanced airways include the tracheal tube, the Combitube, and the LMA. Experts at the 2005 Consensus Conference reviewed the available evidence on use of the tracheal tube and LMA in infants and children. There were no data on use of the Combitube in this age group.

Cuffed versus uncuffed tracheal tubes
W11A,W11B

Consensus on science. One randomised controlled trial (LOE 2),[188] three prospective cohort studies (LOE 3),[189–191] and one cohort study (LOE 4)[192] document no greater risk of complications for children <8 years when using cuffed tracheal tubes compared with uncuffed tubes in the operating room and intensive care unit.

Evidence from one randomised controlled trial (LOE 2)[188] and one small, prospective controlled study (LOE 3)[193] showed some advantage in cuffed over uncuffed tracheal tubes in children in the paediatric anaesthesia and intensive care settings, respectively.

Treatment recommendation. Cuffed tracheal tubes are as safe as uncuffed tubes for infants (except newborns) and children if rescuers use the correct tube size and cuff inflation pressure and verify tube position. Under certain circumstances (e.g. poor lung compliance, high airway resistance, and large glottic air leak), cuffed tracheal tubes may be preferable.

Laryngeal mask airway
W26A,W26B

Consensus on science. There are no studies examining the use of the LMA in children during cardiac arrest. Evidence extrapolated from paediatric anaesthesia shows a higher rate of complications with LMAs in smaller children compared with LMA experience in adults. The complication rate decreases with increasing operator experience (LOE 7).[194,195] Case reports document that the LMA can be helpful for management of the difficult airway.

Treatment recommendation. There are insufficient data to support or refute a recommendation for the routine use of an LMA for children in cardiac arrest. The LMA may be an acceptable initial alternative airway adjunct for experienced providers during paediatric cardiac arrest when tracheal intubation is difficult to achieve.

Confirmation of tube placement

Exhaled CO_2
W25

Consensus on science. Misplaced, displaced, or obstructed tracheal tubes are associated with a high risk of death. No single method of tracheal tube confirmation is always accurate and reliable. One study (LOE 3)[196] showed that clinical assessment of tracheal tube position (observation of chest wall rise, mist in the tube, and auscultation of the chest) can be unreliable for distinguishing oesophageal from tracheal intubation.

In three studies (LOE 5),[197–199] when a perfusing cardiac rhythm was present in infants >2 kg and children, detection of exhaled CO_2 using a colourmetric detector or capnometer had a high sensitivity and specificity for tracheal tube placement. In one study (LOE 5)[198] during cardiac arrest, the sensitivity of exhaled CO_2 detection for tracheal tube placement was 85% and specificity 100%. Both with a perfusing rhythm and during cardiac arrest, the presence of exhaled CO_2 reliably indicates tracheal tube placement, but the absence of exhaled CO_2 during cardiac arrest does not prove tracheal tube misplacement.

Treatment recommendation. In all settings (i.e. prehospital, emergency departments, intensive care units, operating rooms), confirmation of tracheal tube placement should be achieved using detection of exhaled CO_2 in intubated infants and children with a perfusing cardiac rhythm. This may be accomplished using a colourmetric detector or capnometry. During cardiac arrest, if exhaled CO_2 is not detected, tube position should be confirmed using direct laryngoscopy.

Oesophageal detector device
W23

Consensus on science. A study in the operating room (LOE 2)[200] showed that the oesophageal detector device (EDD) was highly sensitive and specific for correct tracheal tube placement in children weighing >20 kg with a perfusing cardiac rhythm. There have been no studies of the EDD in children during cardiac arrest. A paediatric animal study (LOE 6)[201] showed only fair results with the EDD, but accuracy improved with use of a larger syringe. The same animal study showed no difference when the tracheal tube cuff was either inflated or deflated.

Treatment recommendation. The EDD may be considered for confirmation of tracheal tube placement in children weighing >20 kg.

Confirmation of tracheal tube placement during transport
W24

Consensus on science. Studies (LOE 1[202]; LOE 7[203]) have documented the high rate of inadvertent displacement of tracheal tubes during prehospital transport. There are no studies to evaluate the frequency of these events during intra- or inter-hospital transport.

Two studies (LOE 5)[204,205] show that in the presence of a perfusing rhythm, exhaled CO_2 detection or measurement can confirm tracheal tube position accurately during transport. In two animal studies (LOE 6),[206,207] loss of exhaled CO_2 detection indicated tracheal tube displacement more rapidly than pulse oximetry. On the basis of one case series (LOE 5),[204] continuous use of colourmetric exhaled CO_2 detectors may not be reliable for long (>30 min) transport duration.

Treatment recommendation. We recommend monitoring tracheal tube placement and patency in infants and children with a perfusing rhythm by continuous measurement or frequent intermittent detection of exhaled CO_2 during prehospital and intra- and inter-hospital transport.

Oxygen

Oxygen during resuscitation
W14A,W14B

Consensus on science. Meta-analyses of four human studies (LOE 1)[208–211] showed a reduction in mortality rates and no evidence of harm in newborns resuscitated with air compared with 100% oxygen (see Part 7: Neonatal Resuscitation). The two largest studies,[210,211] however, were not blinded, so results should be interpreted with caution. Two animal studies (LOE 6)[212,213] suggest that ventilation with room air may be superior to 100% oxygen during resuscitation from cardiac arrest, whereas one animal study (LOE 6)[214] showed no difference.

Treatment recommendation. There is insufficient information to recommend for or against the use of any specific inspired oxygen concentration during and immediately after resuscitation from cardiac arrest. Until additional evidence is published, we support healthcare providers' use of 100% oxygen during resuscitation (when available). Once circulation is restored, providers should monitor oxygen saturation and reduce the inspired oxygen concentration while ensuring adequate oxygen delivery.

Vascular access and drugs for cardiac arrest

Vascular access can be difficult to establish during resuscitation of children. Review of the evidence showed increasing experience with IO access and resulted in a de-emphasis of the tracheal route for drug delivery. Evidence evaluation of resuscitation drugs was limited by a lack of reported experience in children. There was little experience with vasopressin in children in cardiac arrest and inconsistent results in adult patients. In contrast, there was a good study in children showing no benefit and possibly some harm in using high-dose adrenaline for cardiac arrest.

Routes of drug delivery

Intraosseous access
W29

Consensus on science. Two prospective randomised trials in adults and children (LOE 3)[215,216] and six other studies (LOE 4[217]; LOE 5[218–220]; LOE 7[221,222]) document that IO access is safe and effective for fluid resuscitation, drug delivery, and blood sampling for laboratory evaluation.

Treatment recommendation. We recommend establishing IO access if vascular access is not achieved rapidly in any infant or child for whom IV drugs or fluids are urgently required.

Drugs given via tracheal tube
W32

Consensus on science. One study in children (LOE 2),[223] five studies in adults (LOE 2[224–226]; LOE 3[227,228]), and multiple animal studies (LOE 6)[229–231] indicate that atropine, adrenaline, naloxone, lidocaine, and vasopressin are absorbed via the trachea. Administration of resuscitation drugs into the trachea results in lower blood concentrations than the same dose given intravascularly. Furthermore, animal studies (LOE 6)[232–235] suggest that the lower adrenaline concentrations achieved when the drug is delivered by tracheal route may produce transient β-adrenergic effects. These effects can be detrimental, causing hypotension, lower coronary artery perfusion pressure and flow, and reduced potential for return of spontaneous circulation.

Treatment recommendation. Intravascular, including IO, injection of drugs is preferable to administration by the tracheal route. The recommended tracheal dose of atropine, adrenaline, or lidocaine is higher than the vascular dose and is as follows:

- Adrenaline 0.1 mg kg^{-1} (multiple LOE 6 studies).
- Lidocaine 2–3 mg kg^{-1} (LOE 3)[228] and multiple LOE 6 studies.
- Atropine 0.03 mg kg^{-1} (LOE 2)[224].

The optimal tracheal doses of naloxone or vasopressin have not been determined.

Drugs in cardiac arrest

Dose of adrenaline for cardiac arrest
W31A,W31B

Consensus on science. In four paediatric studies (LOE 2[236,237]; LOE 4[238,239]) there was no improvement in survival rates and a trend toward worse neurological outcome after administration of high-dose adrenaline for cardiac arrest. A randomised, controlled trial (LOE 2)[236] comparing high-dose with standard-dose adrenaline for the second and subsequent (''rescue'') doses in paediatric in-hospital cardiac arrest showed reduced 24-h survival rates in the high-dose adrenaline group. In subgroup analysis, survival rates in asphyxia and sepsis were significantly worse with high-dose rescue adrenaline.

Treatment recommendation. Children in cardiac arrest should be given 10 μg kg^{-1} of adrenaline as the first and subsequent intravascular doses. Routine use of high-dose (100 μg kg^{-1}) intravascular adrenaline is not recommended and may be harmful, particularly in asphyxia. High-dose adrenaline may be considered in exceptional circumstances (e.g. β-blocker overdose).

Vasopressin in cardiac arrest
W19A,W19B

Consensus on science. Based on a small series of children (LOE 5),[240] vasopressin given after adrenaline may be associated with return of spontaneous circulation after prolonged cardiac arrest. Animal data (LOE 6)[241,242] indicate that a

combination of adrenaline and vasopressin may be beneficial. Adult data are inconsistent. Giving vasopressin after adult cardiac arrest (LOE 7)[243–247] has produced improved short-term outcomes (e.g. return of spontaneous circulation or survival to hospital admission) but no improvement in neurologically intact survival to hospital discharge when compared with adrenaline.

Treatment recommendation. There is insufficient evidence to recommend for or against the routine use of vasopressin during cardiac arrest in children.

Magnesium in cardiac arrest
W15

Consensus on science. The relationship between serum magnesium concentrations and outcome of CPR was analyzed in three studies in adults (LOE 3[248]; LOE 4[249]) and one animal study (LOE 6).[250] The first two studies indicated that a normal serum concentration of magnesium was associated with a higher rate of successful resuscitation, but it is unclear whether the association is causative. Six adult clinical studies (LOE 1[251]; LOE 2[252–255]; LOE 3[256]) and one study in an adult animal model (LOE 6)[257] indicated no significant difference in any survival end point in patients who received magnesium before, during, or after CPR.

Treatment recommendation. Magnesium should be given for hypomagnesaemia and torsades de pointes VT, but there is insufficient evidence to recommend for or against its routine use in cardiac arrest.

Postresuscitation care

Postresuscitation care is critical to a favourable outcome. An evidence-based literature review was performed on the topics of brain preservation and myocardial function after resuscitation from cardiac arrest. It showed the potential benefits of induced hypothermia on brain preservation, the importance of preventing or aggressively treating hyperthermia, the importance of glucose control, and the role of vasoactive drugs in supporting haemodynamic function.

Ventilation

Hyperventilation
W27

Consensus on science. One study in cardiac arrest patients (LOE 3)[258] and extrapolation from 12 other studies (LOE 6[259]; LOE 2[260]; LOE 3[261–267]; LOE 4[268]; LOE 5[269,270]) suggest that hyperventilation may cause decreased venous return to the heart and cerebral ischaemia and may be harmful in the comatose patient after cardiac arrest.

Treatment recommendation. Hyperventilation after cardiac arrest may be harmful and should be avoided. The target of postresuscitation ventilation is normocapnoea. Short periods of hyperventilation may be performed as a temporising measure for the child with signs of impending cerebral herniation.

Temperature control

Therapeutic hypothermia
W22B,W22C

Consensus on science. Immediately after resuscitation from cardiac arrest, children often develop hypothermia followed by delayed hyperthermia (LOE 5).[271] Hypothermia (32 °C–34 °C) may be beneficial to the injured brain. Although there are no paediatric studies of induced hypothermia after cardiac arrest, support for this treatment is extrapolated from:

- Two prospective randomised studies of adults with VF arrest (LOE 1[272]; LOE 2[273]).
- One study of newborns with birth asphyxia (LOE 2)[274].
- Numerous animal studies (LOE 6) of both asphyxial and VF arrest.
- Acceptable safety profiles in adults (LOE 7)[272,273] and neonates (LOE 7)[275–278] treated with hypothermia (32 °C–34 °C) for up to 72 h.

Treatment recommendation. Induction of hypothermia (32 °C–34 °C) for 12–24 h should be considered in children who remain comatose after resuscitation from cardiac arrest.

Treatment of hyperthermia
W22A,W22D

Consensus on science. Two studies (LOE 5)[271,279] show that fever is common after resuscitation from cardiac arrest, and three studies (LOE 7)[280–282] show that it is associated with worse outcome. Animal studies suggest that fever causes a worse outcome. One study (LOE 6)[283] shows that rats resuscitated from asphyxial cardiac arrest have a worse outcome if hyperthermia is induced within the first 24 h of recovery. In rats with global ischaemic brain injury (which produces endogenous fever), prevention of fever with a nonsteroidal anti-inflammatory drug (NSAID) class of antipyretic attenuated neuronal damage (LOE 6).[284,285]

Treatment recommendation. Healthcare providers should prevent hyperthermia and treat it aggressively in infants and children resuscitated from cardiac arrest.

Haemodynamic support

Vasoactive drugs
W33A,W33B,W33C,W33D

Consensus on science. Two studies in children (LOE 5),[286,287] multiple studies in adults (LOE 7[288–290]), and animal studies (LOE 6)[291–293] indicate that myocardial dysfunction is common after resuscitation from cardiac arrest. Multiple animal studies (LOE 6)[294–296] document consistent improvement in haemodynamics when selected vasoactive drugs are given in the post-cardiac arrest period. Evidence extrapolated from multiple adult and paediatric studies (LOE 7)[297–302] of cardiovascular surgical patients with low cardiac output documents consistent improvement in haemodynamics when vasoactive drugs are titrated in the period after cardiopulmonary bypass.

Treatment recommendation. Vasoactive drugs should be considered to improve haemodynamic status in the post-cardiac arrest phase. The choice, timing, and dose of specific vasoactive drugs must be individualised and guided by available monitoring data.

Blood glucose control

Treatment of hypoglycaemia and hyperglycaemia
W30A,W30B,W30C

Consensus on science. Adults with out-of-hospital cardiac arrest and elevated blood glucose on admission have poor neurological and survival outcomes (LOE 7).[303–308] In critically ill children, hypoglycaemia (LOE 5)[309] and hyperglycaemia (LOE 5)[310] are associated with poor outcome. It is unknown if the association of hyperglycaemia with poor outcome after cardiac arrest is causative or an epiphenomenon related to the stress response.

In critically ill adult surgical patients, (LOE 7)[311] strict glucose control improves outcome, but there are currently insufficient data in children showing that the benefit of tight glucose control outweighs the risk of inadvertent hypoglycaemia.

Several adult and animal studies (LOE 6)[312–316] and an adult clinical study (LOE 4)[317] show poor outcome when glucose is given immediately before or during cardiac arrest. It is unknown if there is harm in giving glucose-containing maintenance fluids to children after cardiac arrest.

Hypoglycaemia is an important consideration in paediatric resuscitation because:

- Critically ill children are hypermetabolic compared with baseline and have increased glucose requirement ($6–8\,mg\,kg^{-1}\,min^{-1}$) to prevent catabolism.
- The combined effects of hypoglycaemia and hypoxia/ischaemia on the immature brain (neonatal animals) appears more deleterious than the effect of either insult alone.[318]
- Four retrospective studies of human neonatal asphyxia show an association between hypoglycaemia and subsequent brain injury (LOE 4[319,320]; LOE 5[321,322]).

Treatment recommendation. Healthcare providers should check glucose concentration during cardiac arrest and monitor it closely afterward with the goal of maintaining normoglycaemia. Glucose-containing fluids are not indicated during CPR unless hypoglycaemia is present (LOE 7).[323]

Prognosis

One of the most difficult challenges in CPR is to decide the point at which further resuscitative efforts are futile. Unfortunately, there are no simple guidelines. Certain characteristics suggest that resuscitation should be continued (e.g. ice water drowning, witnessed VF arrest), and others suggest that further resuscitative efforts will be futile (e.g. most cardiac arrests associated with blunt trauma or septic shock).

Predictors of outcome in children
W12B,W28

Consensus on science. Multiple studies in adults have linked characteristics of the patient or of the cardiac arrest with prognosis following in-hospital or out-of-hospital cardiac arrest. Experience in children is more limited. Six paediatric studies (LOE 5)[3,324–328] show that prolonged resuscitation is associated with a poor outcome. Although the likelihood of a good outcome is greater with a short duration of CPR, two paediatric studies (LOE 3)[328,329] reported good outcomes in some patients following 30–60 min of CPR in the in-patient setting when the arrests were witnessed and prompt and presumably excellent CPR was provided. Children with cardiac arrest associated with environmental hypothermia or immersion in icy water can have excellent outcomes despite >30 min of cardiac arrest (LOE 5).[7,330]

One large paediatric study (LOE 4)[331] and several smaller studies (LOE 5)[332–336] show that good outcome can be achieved when extracorporeal CPR is started after 30—90 min of refractory standard CPR for in-hospital cardiac arrests. The good outcomes were reported primarily in patients with isolated heart disease. These data show that 15 or 30 min of CPR does not define the limits of cardiac and cerebral viability.

Witnessed events, bystander CPR, and a short interval from collapse to arrival of EMS system personnel are important prognostic factors associated with improved outcome in adult resuscitation, and it seems reasonable to extrapolate these factors to children. At least one paediatric study (LOE 5)[328] showed that the interval from collapse to initiation of CPR is a significant prognostic factor.

Children with prehospital cardiac arrest caused by blunt trauma[337] and in-hospital cardiac arrest caused by septic shock[329] rarely survive.

Treatment recommendation. The rescuer should consider whether to discontinue resuscitative efforts after 15—20 min of CPR. Relevant considerations include the cause of the arrest, preexisting conditions, whether the arrest was witnessed, duration of untreated cardiac arrest (no flow), effectiveness and duration of CPR (low flow), prompt availability of extracorporeal life support for a reversible disease process, and associated special circumstances (e.g. icy water drowning, toxic drug exposure).

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.resuscitation.2005.09.020.

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RESUSCITATION

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Part 7: Neonatal resuscitation

International Liaison Committee on Resuscitation

Approximately 10% of newborns require some assistance to begin breathing at birth, and about 1% require extensive resuscitation. Although the vast majority of newborn infants do not require intervention to make the transition from intrauterine to extrauterine life, the large number of births worldwide means that many infants require some resuscitation. Newborn infants who are born at term, have had clear amniotic fluid, and are breathing or crying and have good tone must be dried and kept warm but do not require resuscitation.

All others need to be assessed for the need to receive one or more of the following actions in sequence:

A. initial steps in stabilisation (clearing the airway, positioning, stimulating);
B. ventilation;
C. chest compressions;
D. medications or volume expansion.

Progression to the next step is based on simultaneous assessment of three vital signs: respirations, heart rate, and colour. Progression occurs only after successful completion of the preceding step. Approximately 30 s is allotted to complete one step successfully, re-evaluate, and decide whether to progress to the next (Figure 7.1).

Since publication of the last International Liaison Committee on Resuscitation (ILCOR) document,[1] several controversial neonatal resuscitation issues have been identified. The literature was researched and a consensus was reached on the role of supplementary oxygen, peripartum management of meconium, ventilation strategies, devices to confirm placement of an advanced airway (e.g. tracheal tube or laryngeal mask airway [LMA]), medications, maintenance of body temperature, postresuscitation management, and considerations for withholding and discontinuing resuscitation.

Initial resuscitation

Supplementary oxygen

Supplementary oxygen versus room air
W202A,W202B

There is growing evidence from both animal and human studies that air is as effective as 100% oxygen for the resuscitation of most infants at birth. There are concerns about potential adverse effects of 100% oxygen on breathing physiology, cerebral circulation, and potential tissue damage from oxygen free radicals.

Consensus on science. Studies examining blood pressure, cerebral perfusion, and biochemical indicators of cell damage in asphyxiated animals resuscitated with 100% versus 21% oxygen show conflicting results (LOE 6).[2—6] One study of preterm infants (<33 weeks of gestation) exposed to 80% oxygen found lower cerebral blood flow when compared with those stabilised with 21% oxygen (LOE 2).[7] Some animal data indicate the opposite effect, i.e. reduced blood pressure and cerebral perfusion with air versus 100% oxygen (LOE 6).[2]

Meta-analysis of four human studies showed a reduction in mortality and no evidence of harm in infants resuscitated with air compared with those

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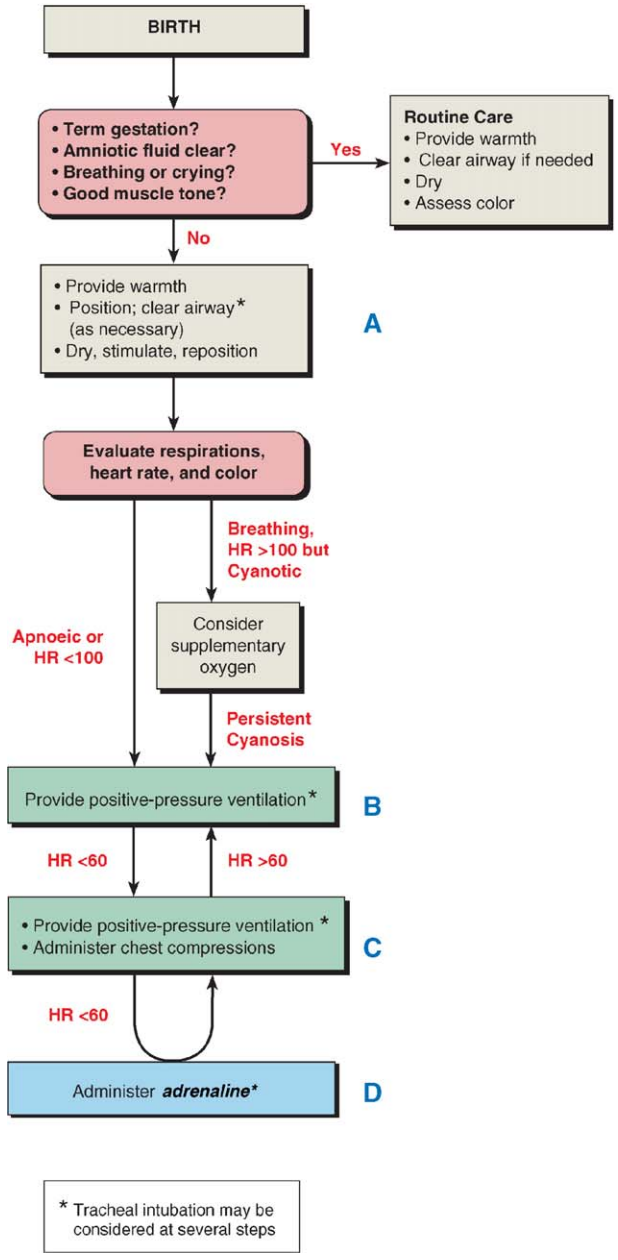

Figure 7.1 ILCOR neonatal flow algorithm.

resuscitated with 100% oxygen (LOE 1).[8,9] The two largest newborn human studies of room air versus oxygen resuscitation were not blinded. In those studies, if there was no response after 90 s, those resuscitated with air were switched to supplementary oxygen; a similar proportion who failed to respond while receiving oxygen were not crossed over to room air.[10,11] These results require careful interpretation because of significant methodological concerns (regarding patient selection, lack of blinding, randomisation methods, and follow-up).

Trials have not examined in sufficient detail infants with a birth weight of <1000 g, those with known congenital pulmonary or cyanotic heart disease, and those without discernible signs of life at birth.[10–13] Continuous oximetry studies show that term healthy newborns may take >10 min to achieve a preductal oxygen saturation >95% and nearly 1 h to achieve this postductally (LOE 5).[14–16]

Treatment recommendation. There is currently insufficient evidence to specify the concentration of oxygen to be used at initiation of resuscitation. After initial steps at birth, if respiratory efforts are absent or inadequate, lung inflation/ventilation should be the priority. Once adequate ventilation is established, if the heart rate remains low, there is no evidence to support or refute a change in the oxygen concentration that was initiated. Rather the priority should be to support cardiac output with chest compressions and coordinated ventilations. Supplementary oxygen should be considered for babies with persistent central cyanosis. Some have advocated adjusting the oxygen supply according to pulse oximetry measurements to avoid hyperoxia, but there is insufficient evidence to determine the appropriate oximetry goal because observations are confounded by the gradual increase in oxyhaemoglobin saturation that normally occurs following birth. Excessive tissue oxygen may cause oxidant injury and should be avoided, especially in the premature infant.

Peripartum management of meconium

Management of meconium was examined from two perspectives: (1) suctioning of the meconium from the infant's airway after delivery of the head but before delivery of the shoulders (intrapartum suctioning) and (2) suctioning of the infant's trachea immediately after birth (tracheal suctioning).

Intrapartum suctioning
W206

Consensus on science. Previous studies have yielded conflicting results about the value of intrapartum oropharyngeal and nasopharyngeal suctioning of babies born with meconium-stained fluid (LOE 3[17]; LOE 4[18,19]). A recent large multicenter randomised trial found that intrapartum suctioning of meconium does not reduce the incidence of meconium aspiration syndrome (LOE 1).[20]

Treatment recommendation. Routine intrapartum oropharyngeal and nasopharyngeal suctioning

for infants born with meconium-stained amniotic fluid is no longer recommended.

Tracheal suctioning
W206

Consensus on science. A randomised controlled trial showed that tracheal intubation and suctioning of meconium-stained but vigorous infants at birth offers no benefit (LOE 1).[17] The benefit of tracheal suctioning in meconium-stained, depressed infants has not been systematically studied (LOE 5).[21–23]

Treatment recommendation. Meconium-stained, depressed infants should receive tracheal suctioning immediately after birth and before stimulation, presuming the equipment and expertise is available. Tracheal suctioning is not necessary for babies with meconium-stained fluid who are vigorous.

Ventilation strategies

Ventilation strategy was examined from four perspectives: (1) the characteristics of the initial assisted breaths, (2) devices to assist ventilation, (3) special considerations for babies born preterm, and the role of positive end-expiratory pressure (PEEP) or continuous positive air pressure (CPAP) during or following resuscitation.

Initial breaths
W203A,W203C

Consensus on science. When performed properly, positive-pressure ventilation alone is effective for resuscitating almost all apnoeic or bradycardic newborn infants (LOE 5).[24] The primary measure of adequate initial ventilation is prompt improvement in heart rate (LOE 6).[25–27] The presence or absence of chest wall movement has been described but not assessed adequately (LOE 5).[28]

In term infants, initial inflations, either spontaneous or assisted, create a functional residual capacity (FRC) (LOE 5).[28–33] The optimum pressure, inflation time, and flow required to establish an effective FRC has not been determined. In case series reporting the physiological changes associated with initial ventilation of term human neonates, peak pressures used to initiate ventilation varied widely (18–60 cm H_2O). Average initial peak inflating pressures of 30–40 cm H_2O were used to successfully ventilate unresponsive term infants (LOE 5).[31–35] In a single small series a sustained inflation pressure of 30 cm H_2O for 5 s for the first breath was effective in establishing lung volume in term infants requiring resuscitation (LOE 5)[31]; the risk and benefits of this practice have not been evaluated. Ventilation rates of 30–60 breaths min^{-1} are commonly used, but the relative efficacy of various rates has not been investigated (LOE 8).

Treatment recommendation. Establishing effective ventilation is the primary objective in the management of the apnoeic or bradycardic newborn infant in the delivery room. In the bradycardic infant, prompt improvement in heart rate is the primary measure of adequate initial ventilation; chest wall movement should be assessed if heart rate does not improve. Initial peak inflating pressures necessary to achieve an increase in heart rate or movement of the chest are variable and unpredictable and should be individualised with each breath. If pressure is being monitored, an initial inflation pressure of 20 cm H_2O may be effective, but a pressure $\geq$30–40 cm H_2O may be necessary in some term babies. If pressure is not being monitored, the minimal inflation required to achieve an increase in heart rate should be used. There is insufficient evidence to recommend optimal initial or subsequent inflation times.

Assisted ventilation devices
W203B

Consensus on science. Studies on humans and manikins suggest that effective ventilation can be achieved with either a flow-inflating or self-inflating bag or with a T-piece mechanical device designed to regulate pressure (LOE 4[36,37]; LOE 5[38]). The pop-off valves of self-inflating bags are flow-dependent, and pressures generated during resuscitation may exceed the target values (LOE 6).[39] Target inflation pressures and long inspiratory times are achieved more consistently in mechanical models when using T-piece devices than when using bags (LOE 6),[40] although the clinical implications are not clear. To provide the desired pressure, healthcare providers need more training to use flow-inflating bags than they need to use self-inflating bags (LOE 6).[41]

Treatment recommendation. A self-inflating bag, a flow-inflating bag, or a T-piece mechanical device designed to regulate pressure as needed can be used to provide bag-valve-mask ventilation to a newborn.

Laryngeal mask airway
W215A,W215B

Consensus on science. Masks that fit over the laryngeal inlet are effective for ventilating new-

born full-term infants (LOE 2[42]; LOE 5[43]). There are limited data on the use of these devices in small preterm infants (LOE 5).[44,45] There is currently no evidence directly comparing the laryngeal mask airway (LMA) with bag-valve-mask ventilation during neonatal resuscitation. Data from two case series show that use of the LMA can provide effective ventilation in a time frame consistent with current resuscitation guidelines (LOE 5).[43,46] A single randomised controlled trial found no significant difference between the LMA and tracheal intubation during resuscitation of babies by experienced providers after Caesarean section (LOE 2).[42] Case reports suggest that when ventilation via a face mask has been unsuccessful and tracheal intubation is unsuccessful or not feasible, the LMA may provide effective ventilation (LOE 5).[47,48]

Treatment recommendation. The LMA may enable effective ventilation during neonatal resuscitation if bag-mask ventilation is unsuccessful and tracheal intubation is unsuccessful or not feasible. There is insufficient evidence to recommend use of the LMA as the primary airway device during neonatal resuscitation or in the settings of meconium-stained amniotic fluid, when chest compressions are required, or for the delivery of drugs into the trachea.

Ventilation strategies for preterm infants
W203A,W203C

Consensus on science. There has been little research evaluating initial ventilation strategies in the resuscitation of preterm infants. Animal studies indicate that preterm lungs are more easily injured by large-volume inflations immediately after birth (LOE 6).[49] Additional studies in animals indicate that when positive-pressure ventilation is applied immediately after birth, the application of end-expiratory pressure protects against lung injury and improves lung compliance and gas exchange (LOE 6).[50,51] Case series in infants indicate that most apnoeic preterm infants can be ventilated with an initial inflation pressure of 20–25 cm H_2O, although some infants who do not respond require a higher pressure (LOE 5).[52,53]

Treatment recommendation. Providers should avoid creation of excessive chest wall movement during ventilation of preterm infants immediately after birth. Although measured peak inflation pressure does not correlate well with volume delivered in the context of changing respiratory mechanics, monitoring of inflation pressure may help provide consistent inflations and avoid unnecessarily high pressures. If positive-pressure ventilation is required, an initial inflation pressure of 20–25 cm H_2O is adequate for most preterm infants. If prompt improvement in heart rate or chest movement is not obtained, then higher pressures may be needed.

Use of CPAP or PEEP
W204A,W204B

Consensus on science. Spontaneously breathing newborns establish functional residual capacity more quickly and with lower transpulmonary pressures than sick neonates (LOE 5).[32] In the sick neonate CPAP helps stabilise and improve lung function (LOE 4).[54] Excessive CPAP, however, can overdistend the lung, increase the work of breathing, and reduce cardiac output and regional blood flow (LOE 6).[55,56] There are no prospective, randomised, controlled clinical trials of sufficient power to compare CPAP and positive-pressure ventilation (via bag-mask or bag-tracheal tube) during resuscitation of either the preterm or term neonate. When compared with historical controls, use of CPAP for extremely premature babies in the delivery room was associated with a decrease in: requirement for intubation, days on mechanical ventilation, and use of postnatal steroids (LOE 4).[53] A small underpowered feasibility trial of delivery room CPAP/PEEP versus no CPAP/PEEP did not show a significant difference in immediate outcomes (LOE 2).[57]

Treatment recommendation. There are insufficient data to support or refute the routine use of CPAP during or immediately after resuscitation in the delivery room.

Exhaled CO_2 detectors to confirm tracheal tube placement
W212A,W212B

Consensus on science. After tracheal intubation, adequate ventilation is associated with a prompt increase in heart rate (LOE 5).[35] Exhaled CO_2 detection is a reliable indicator of tracheal tube placement in infants (LOE 5).[58–61] A positive test (detection of exhaled CO_2) confirms tracheal placement of the tube, whereas a negative test strongly suggests oesophageal intubation (LOE 5).[58,60,61] Poor or absent pulmonary blood flow may give false-negative results, but tracheal tube placement is identified correctly in nearly all patients who are not in cardiac arrest (LOE 7).[62] In critically ill infants with poor cardiac output, a false-negative result may lead to unnecessary extubation.

Exhaled CO_2 detectors identify oesophageal intubations faster than clinical assessments (LOE 5).[58,61] Clinical techniques for confirmation of correct tracheal tube placement (e.g. evaluation of condensed humidified gas during exhalation, chest movement) have not been evaluated systematically in neonates.

Treatment recommendation. Tracheal tube placement must be confirmed after intubation, especially in infants with a low heart rate that is not rising. Exhaled CO_2 detection is useful to confirm tracheal tube placement. During cardiac arrest, if exhaled CO_2 is not detected, tube placement should be confirmed with direct laryngoscopy.

Medications

The primary considerations about medications focused on which drugs should be used and the route by which they should be given. Medications are rarely needed in neonatal resuscitation. Those that may be used include adrenaline and fluids. Very rarely, a narcotic antagonist, sodium bicarbonate, [W200] or vasopressors may be useful after resuscitation.

Adrenaline

Route and dose of adrenaline
W213A,W213B,W217,W220

Consensus on science. Despite the widespread use of adrenaline/epinephrine during resuscitation, no placebo-controlled studies have evaluated either the tracheal or intravenous (IV) administration of epinephrine at any stage during cardiac arrest in human neonates. A paediatric study (LOE 7)[63] and studies in newborn animals (LOE 6)[64,65] showed no benefit and a trend toward reduced survival rates and worse neurological status after administration of high-dose IV adrenaline ($100\,\mu g\,kg^{-1}$) during resuscitation. Animal and adult human studies show that when given tracheally, considerably higher doses of adrenaline than currently recommended are required to show a positive effect (LOE 6).[66–68]

One neonatal animal study using the currently recommended dose of tracheal adrenaline ($10\,\mu g\,kg^{-1}$) showed *no* benefit (LOE 6).[69] One neonatal cohort study of nine preterm babies requiring resuscitation showed that tracheal adrenaline was absorbed, but the study used 7–25 times the dose recommended currently (LOE 5).[70]

Treatment recommendation. Despite the lack of human data, it is reasonable to continue to use adrenaline when adequate ventilation and chest compressions have failed to increase the heart rate to >60 beats/min. Use the IV route for adrenaline as soon as venous access is established. The recommended IV dose is $0.01–0.03\,mg\,kg^{-1}$. If the tracheal route is used, give a higher dose (up to $0.1\,mg\,kg^{-1}$). The safety of these higher tracheal doses has not been studied. Do not give high doses of intravenous adrenaline.

Volume expansion

Crystalloids and colloids
W208

Consensus on science. Three randomised controlled trials in neonates showed that isotonic crystalloid is as effective as albumin for the treatment of hypotension (LOE 7).[71–73] No studies have compared the relative effectiveness of crystalloid during resuscitation.

Treatment recommendation. In consideration of cost and theoretical risks, an isotonic crystalloid solution rather than albumin should be the fluid of choice for volume expansion in neonatal resuscitation.

Other drugs

Naloxone
W214A,W214B

Consensus on science. There are no studies examining the use of naloxone in infants with severe respiratory depression from maternal opioids. Vigorous newborns whose mothers received opioids had brief improvement in alveolar ventilation with naloxone without affecting Apgar score, pH, $PaCO_2$, or respiratory rate (LOE 7).[74] Compared with intramuscular naloxone, IV naloxone produces higher plasma concentrations but has a shorter half-life (LOE 5).[75] Tracheal or subcutaneous administration has not been examined in neonates, nor has the current recommended dose of $0.1\,mg\,kg^{-1}$ been studied.

Naloxone may interfere with critical functions of endogenous opioids and exacerbate long-term neurohistological injury of cerebral white matter in asphyxiated animals (LOE 6).[76,77] Cardiac arrhythmias, hypertension, and noncardiogenic pulmonary oedema have been reported in adolescents and adults, especially when high doses have been used (LOE 7).[78] Naloxone given to a baby born to an opioid-addicted mother was associated with seizures.[79]

Treatment recommendation. Naloxone is not recommended as part of the initial resuscitation of newborns with respiratory depression in the delivery room. Before naloxone is given, providers should restore heart rate and colour by supporting ventilation. The preferred route should be IV or intramuscular. Tracheal administration is not recommended. There is no evidence to support or refute the current dose of 0.1 mg kg^{-1}.

Supportive therapy

Temperature control

Maintenance of body temperature
W210A,W210B

Consensus on science. Numerous observational studies showed an association between hypothermia and increased mortality in premature newborns. Premature infants continue to be at risk for hypothermia when treated according to current recommendations (dry the infant, remove wet linens, place the infant on a radiant warmer) (LOE 5).[80] Two randomised controlled trials (LOE 2)[81,82] and three observational studies (LOE 4[83,84]; LOE 5[85]) confirm the efficacy of plastic bags or plastic wrapping (food-grade, heat-resistant plastic) in addition to the customary radiant heat in significantly improving the admission temperature of premature babies of <28 weeks gestation when compared with standard care (LOE 2[81,82]; LOE 4[83,84]; LOE 5[85]). There is no direct evidence that this improves mortality or long-term outcomes. Temperature must be monitored closely because there is a small risk that this technique may produce hyperthermia (LOE 2).[82]

Other techniques have been used to maintain temperature in the delivery room during stabilisation (drying and swaddling, warming pads, placing the newborn skin-to-skin with the mother and covering both, etc.) but have not been compared with the plastic wrap technique for premature babies (LOE 8).[86,87]

Treatment recommendation. Very low birth weight preterm babies remain at risk for hypothermia. Consider the use of plastic bags or plastic wrapping under radiant heat as well as standard techniques to maintain temperature. All initial resuscitation steps, including intubation, chest compression, and insertion of lines, can be performed with these temperature-controlling interventions in place.

Postresuscitation management

Temperature

Hyperthermia
W201

Consensus on science. Babies born to febrile mothers (temperature >38 °C) have an increased risk of death, perinatal respiratory depression, neonatal seizures, and cerebral palsy (LOE 4).[88,89] During the first 24 h after adult stroke, fever is associated with a marked increase in neurological morbidity and mortality (LOE 7).[90,91] Adult animal studies indicate that hyperthermia during or after ischaemia is associated with a progression of cerebral injury (LOE 6).[92,93]

Treatment recommendation. The goal is to achieve normothermia and to avoid iatrogenic hyperthermia in babies who require resuscitation.

Therapeutic hypothermia
W211A,W211B

Consensus on science. A reduction of body temperature by 2—3 °C (modest hypothermia) following cerebral hypoxia-ischaemia reduces cerebral metabolic and biochemical abnormalities and cerebral injury and improves function in experimental neonatal models (LOE 6).[94—96] In adults, induced hypothermia (temperature of 32—34 °C) for 12—24 h improves neurological outcome after cardiac arrest due to ventricular arrhythmias but not after trauma or stroke (LOE 7).[97] In a multicentre trial involving newborns with suspected asphyxia (indicated by need for resuscitation at birth, metabolic acidosis, and early encephalopathy), selective head cooling to achieve a rectal temperature of 34—35 °C was associated with a nonsignificant reduction in the overall number of survivors with severe disability at 18 months but a significant benefit in the subgroup with moderate encephalopathy (LOE 2).[98]

Infants with severe electroencephalographic (EEG) suppression and seizures did not benefit from treatment with modest hypothermia (LOE 2).[98] A second small controlled pilot study in asphyxiated infants with early induced systemic hypothermia that achieved a rectal temperature of 33 °C resulted in fewer deaths and disability at 12 months (LOE 2).[99]

Modest hypothermia is associated with bradycardia and elevated blood pressure that do not usually require treatment, but a rapid increase in body temperature may cause hypotension (LOE 5).[100] Profound hypothermia (core temperature <33 °C) may cause arrhythmia, bleeding, thrombosis, and

sepsis, but these complications have not been reported in infants treated with modest hypothermia (LOE 2).[98,99,101,102]

Treatment recommendation. There are insufficient data to recommend the routine use of systemic or selective cerebral hypothermia after resuscitation of infants with suspected asphyxia. Further clinical trials are needed to confirm that treatment with cooling is beneficial, to identify infants who will benefit most, and to determine the most effective method and timing of cooling.

General supportive care

Glucose
W218A,W218B,W219A,W219B

Consensus on science. Low blood glucose is associated with adverse neurological outcomes in a neonatal animal model of asphyxia and resuscitation (LOE 6).[103] Hypoglycaemia in animals at the time of an anoxic or hypoxic-ischaemic insult resulted in larger areas of cerebral infarction and/or decreased survival rates when compared with controls (LOE 6).[104,105] One clinical study showed an association between hypoglycaemia (blood glucose <40 mg dl^{-1}) measured shortly after resuscitation and poor neurological outcome following perinatal asphyxia (LOE 4).[106]

Hyperglycaemia induced in neonatal animal models of hypoxia-ischaemia had conflicting effects on the extent of brain injury (LOE 6).[107,108] No clinical neonatal studies have investigated this topic.

Treatment recommendation. Based on available evidence, the optimal range of blood glucose concentration to minimise brain injury following asphyxia and resuscitation cannot be defined. Infants requiring resuscitation should be monitored and treated to maintain glucose in the normal range.

Timing of cord clamping
W216A,W216B

Consensus on science. Although delayed cord clamping (30—120 s after birth) in premature infants was associated with higher mean blood pressure and haematocrit and less intraventricular haemorrhage, most study subjects did not require resuscitation (LOE 1[109] and LOE 2[110]). Delayed cord clamping in term infants not requiring resuscitation resulted in no clinically significant improvement in stability over the first 4—6 h after birth (LOE 3).[111,112]

Treatment recommendation. No recommendation can be made about the timing of cord clamping when resuscitation is required.

Withholding or discontinuing resuscitative efforts
W209A,W209B

Consensus on science. Mortality and morbidity for newborns varies according to region and availability of resources (LOE 5).[113] Social science studies indicate that parents would like a greater role in decisions to start resuscitation and continue life support of severely compromised newborns. Opinions among neonatal providers vary widely regarding the benefits and disadvantages of aggressive therapies in such newborns (LOE 5).[114,115]

Some data are available to help identify conditions associated with high mortality and poor outcome (LOE 5).[80,116] In some settings with adequate resources, such conditions may include extreme prematurity and infants with anomalies that predict extreme morbidity or early death. Data from infants without signs of life lasting at least 10 min or longer from birth despite continuous and adequate resuscitation efforts document either high mortality or severe neurodevelopmental disability (LOE 5).[117,118]

Treatment recommendation. A consistent and coordinated approach to individual cases by obstetric and neonatal teams and parents is an important goal. Not starting resuscitation and discontinuation of life-sustaining treatment during or after resuscitation are ethically equivalent, and clinicians should not be hesitant to withdraw support when functional survival is highly unlikely. The following guidelines must be interpreted according to current regional outcomes and societal principles:

- When gestation, birth weight, or congenital anomalies are associated with almost certain early death and an unacceptably high morbidity is likely among the rare survivors, resuscitation is not indicated. Examples from the published literature from developed countries include
  - extreme prematurity (gestational age <23 weeks or birth weight <400 g);
  - anomalies such as anencephaly and confirmed trisomy 13 or 18.
- In conditions associated with a high rate of survival and acceptable morbidity, resuscitation is nearly always indicated.
- In conditions associated with uncertain prognosis, when there is borderline survival and a rel-

atively high rate of morbidity, and where the burden to the child is high, the parents' views on starting resuscitation should be supported.

If there are no signs of life after 10 min of continuous and adequate resuscitative efforts, it may be justifiable to stop resuscitation.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.resuscitation.2005.09.014.

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Part 8: Interdisciplinary topics

International Liaison Committee on Resuscitation

The Interdisciplinary Task Force discussed topics that applied to several task forces and in particular focused on questions about educational methods, ethics, and outcomes. Some of these topics are discussed in other sections of this document (e.g. the topic of medical emergency teams is discussed in Part 4: 'Advanced Life Support').

To maintain consistency with the science statements in other sections, studies using manikins were recorded as LOE 6, irrespective of the study design.

Educational methods

Acquisition and retention of skills are poor after conventional CPR training.[1] Evidence for and against several resuscitation training methods was reviewed, highlighting the need for further research.

Devices

CPR prompt devices
W190A,W190B

Consensus on science. Twenty-seven randomised studies using models from the motor skills literature (LOE 6)[2—28] and one randomised study using manikins (LOE 6)[29] showed that the use of audio or visual prompts during motor skills acquisition training improved student skills performance during or immediately after training. These studies and supporting theory from two studies (LOE 7)[30,31] indicate that the overuse of guiding prompts during training reduced skills retention in the long term.

Treatment recommendation. Audio and visual prompts and other forms of directive or corrective feedback that guide action sequences and timing of chest compressions and ventilations may help early learning of CPR skills. Training must include ample practice time without prompting devices to optimize skills retention for situations in which prompting devices are not available.

Instructional methods

Effective AED instructional methods
W191A,W191B

Consensus on science. Seven studies (LOE 4[32—35]; LOE 5[36,37]; LOE 7[38]) showed improved rates of survival from out-of-hospital cardiac arrest when CPR plus automated external defibrillation training (traditional 4-h course) was made widely available to lay first responders. The prospective randomised trial of lay rescuer automated external defibrillation programs did not specifically evaluate the training provided, but sites where rescuers were trained and equipped to provide CPR or CPR plus automated external defibrillator (AED) use showed higher survival rates compared with national reports (LOE 7).[38]

Twenty studies (LOE 5[39]; LOE 6[40—58]) document consistent improvement in simulated AED use and skills retention using diverse training methods and durations. Three studies (LOE 6)[59—61] show that within a simulated arrest scenario the correct and

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appropriate use of an AED depends on the AED user interface.

Treatment recommendation. Community lay responder AED training is recommended. There is insufficient evidence to recommend a specific instructional method for AED training. AED manufacturers should increase the ease of AED user interface to improve efficacy.

Effective BLS instructional methods
W185A,W185B,W192

Consensus on science. Nineteen randomised manikin studies (LOE 6)[48,62–79] and one extrapolated study (LOE 7)[80] showed considerable variability in BLS skills acquisition and retention with the use of different instructional formats (video instruction, computer-assisted instruction, and traditional instruction). Four randomised studies using manikins (LOE 6)[66–69] indicated that one video instruction program (a self-instructional synchronous ''watch-while-you-practice'' program) achieved better skills acquisition and retention than other educational formats. One randomised study of adult learners using manikins showed that a brief video self-instruction program produced CPR skills performance equivalent to or better than traditional training (LOE 6).[81]

Treatment recommendation. Instruction methods should not be limited to traditional techniques; newer training methods (e.g. ''watch-while-you-practice'' video programs) may be more effective. Training programs should be evaluated to verify that they enable effective skills acquisition and retention.

Instructional methods for hand position in chest compressions
W189

Consensus on science. Six randomised controlled trials (RCTs) using manikins (LOE 6)[67,69,82–85] evaluated hand positioning in detail. One trial[82] compared a simplified message (''place hands in the centre of the chest'') versus the standard method (anatomical landmarks) for teaching correct hand placement. Three of the six trials[83–85] compared a staged teaching approach with standard teaching. Two of the trials[67,69] compared the results of video self-instruction with standard teaching on CPR performance. The likelihood of achieving an acceptable hand position was no different between those who had received detailed instruction on anatomical landmarks and those who were instructed to simply compress the centre of the chest.

In four manikin RCTs (LOE 6)[82–85] the use of anatomical landmarks to determine hand placement delayed delivery of the first chest compression after a ventilation; thus, fewer compressions were delivered per minute. Incorrect rescuer hand placement can injure the victim (LOE 6).[86,87]

Treatment recommendation. Teaching hand placement for chest compression should be simplified with less attention to anatomical landmarks and emphasis on the importance of minimising interruption to chest compressions and performing an adequate number of chest compressions per minute.

Retraining intervals

Retraining intervals in advanced and basic life support
W186A,W86

Consensus on science. One prospective cohort study (LOE 3),[88] one survey (LOE 5),[89] and 10 manikin studies (LOE 6),[90–99] documented decay in healthcare provider ALS skills and knowledge after ALS training and retraining from as little as 6 weeks to 2 years. Refresher courses based only on knowledge did not prevent the decay in psychomotor skills.

A single randomised manikin study (LOE 6)[100] concluded that retraining at either 3- or 6-month intervals resulted in similar BLS performance at 12 months and providers who were retrained performed significantly better than controls with no retraining.

Treatment recommendation. Frequent retraining (theory and practice) is required to maintain both BLS and ALS skills. The optimal interval for retraining has not been established.

Media campaigns

Media campaigns targeting chest pain
W193A,W193B

Consensus on science. One large RCT (LOE 1),[101] a Cochrane systematic review (LOE 1),[102] and four additional studies (LOE 3[103,104]; LOE 4[105,106]) evaluating the impact of mass media campaigns indicate that they do not reduce the delay to presentation at the hospital following onset of chest pain. Conversely seven studies (LOE 3)[107–113] did report reduced delay in the patient's response to chest pain.

The evidence that mass media campaigns reduce patient delay from the onset of symptoms to pre-

sentation at hospital is equivocal and suggests that the impact of such campaigns, particularly on prehospital delay times, may be greater for populations in which the baseline delay time is long.

There is evidence that mass media campaigns can increase the use of ambulance transport (LOE 1)[101] in patients with symptoms that suggest myocardial ischemia. In several studies (LOE 1[102]; LOE 3[107,110,114]; LOE 4[105]) the number of patients presenting to the emergency department increased in the early stages of the media campaign but soon returned to baseline.

The impact of mass media campaigns on rates of mortality from ischemic heart disease remains inconclusive (LOE 3)[109]; however, the inference is that by reducing prehospital delay time, the mortality rate should decrease.

Treatment recommendation. Given that the data are inconsistent, mass media campaigns should not be considered the only option for reducing patient delay but rather part of an overall system approach to reduce the interval from onset of symptoms of chest pain to hospital presentation.

Educational evaluation

Although there is considerable literature on the evaluation of educational processes in general, there are few studies of resuscitation education.

Attitude toward performing CPR

Barriers to performing CPR
W184A,W184B

Consensus on science. One RCT (LOE 2),[115] one prospective controlled cohort study (LOE 3),[116] two cohort and case studies (LOE 4),[117,118] supported by 27 cohort and case studies (LOE 5[119–138]; LOE 7[139–145]) indicate hesitancy or unwillingness to perform CPR, particularly mouth-to-mouth ventilation, on adult patients in and out of hospital, even after CPR training.

Reasons for the hesitancy or unwillingness to perform CPR include, but are not limited to, fear of contracting a disease while performing mouth-to-mouth ventilations, fear of performing the skills incorrectly, and fear of hurting the patient.

Treatment recommendation. CPR training programs should include discussion of the minimal risk of contracting infectious diseases while performing mouth-to-mouth ventilation. ''Chest compression only'' resuscitation may be considered when there is a reluctance to perform mouth-to-mouth ventilation (see Part 2: ''Adult Basic Life Support'').

Written test scores and skills competence
W188A,W188B

Consensus on science. Do written test scores correlate with competence in CPR skills? None of the studies reviewed was designed specifically to answer this question. In 14 of 17 studies test scores correlated with CPR proficiency. Of the seven studies with good written test scores (LOE 6 manikin studies), four studies were associated with good CPR skills[146–149] and three studies with poor CPR skills.[150–152] In two manikin studies (LOE 6)[68,153] mediocre written test scores correlated with mediocre or borderline CPR performance. In six manikin studies (LOE 6),[72,147,153–156] poor written test performance was associated with poor CPR capability. In five manikin studies (LOE 6),[150–152,157,158] written test scores did not correlate with CPR proficiency.

Treatment recommendation. A written test score does not always reflect BLS skills competence. Therefore, a written test or questionnaire should not be used as the sole determinant of a person's acquisition of the skills needed to perform CPR.

Ethics

The ethical issues surrounding resuscitation are dependent on local culture and law. Consideration of the patient's wishes, the family's desires, cultural issues, and local laws makes specific recommendations about ethical decisions generally inappropriate.

Impact of DNAR on resuscitation
W179A,W179B,W179C

Consensus on science. The emergency medical services (EMS) system is activated for many patients in cardiac arrest who are chronically ill, have a terminal illness, or have do-not-attempt-resuscitation (DNAR) orders (LOE 4).[159–161] Studies from the United States and Australia indicate that Caucasians and better-educated persons are more likely to have advance directives (LOE 4[162–165]; LOE 7[166–168]). There is evidence that out-of-hospital healthcare providers can interpret and use DNAR orders and other documents to limit treatment (LOE 3[169,170]; LOE 4[171]; LOE 7[172]).

The most studied DNAR form is the Physician Orders for Life-Sustaining Treatment (POLST) form.[170,171,173–175]

Treatment recommendation. We recommend the use of standardised out-of-hospital physician orders for patients who are chronically ill or have a terminal illness. These must be easily understood by EMS personnel. Additional instructions should indicate whether EMS personnel are to initiate or continue life-sustaining interventions for patients in cardiac arrest and those in near-arrest. Because laws governing the use of DNAR forms and advance directives vary by jurisdiction, providers should be aware of local laws and regulations.[176]

Family member presence during CPR
W180A,W180B

Consensus on science. No studies evaluated the effect of the presence of parents during resuscitation of children. Studies on parents' opinions indicate their preference to be at the side of the child who is dying (LOE 5),[177] during CPR (LOE 5),[177] or during procedures (LOE 7).[177–184] However, five studies (LOE 3)[185–189] found that staff members were reluctant to allow parents to be present during resuscitation.

Most relatives of adult patients requiring CPR state that they would like to be offered the option of being present in the resuscitation room (LOE 5).[190–194] A survey of adult patients indicated that many, but not all, would prefer to have certain family members present (LOE 5).[195] Family presence during resuscitation did not impact on self-reported stress among staff (LOE 3),[196] nor was it disruptive for staff (LOE 5).[191,194] Family members considered their presence to be beneficial (LOE 5)[191,193,194,197] and their adjustment to the death of the patient made easier by their presence during the resuscitation attempt (LOE 2[198]; LOE 5[191,197]).

There are no data to support or refute the importance of having a dedicated staff member available to support family members during resuscitation for either adults or children, but this practice is well described (LOE 2[198]; LOE 5[191]).

Treatment recommendation. There are no data indicating that the presence of relatives in the resuscitation room is harmful. Therefore, it is reasonable to give select family members the opportunity to be present during resuscitation unless the adult patient has raised a prior objection.

Outcomes and cost-effectiveness

Research about the ''quality of life'' for survivors of cardiac arrest is plagued by the lack of a consistent definition of quality of life and how best to measure it. Nonetheless, the increasing demand for limited healthcare resources makes it important to measure the effectiveness of CPR in terms of quality of survival and not just the number of survivors.

Outcomes

Quality of life outcomes after CPR
W182A,W182B

Consensus on science. In six nonrandomised prospective cohort studies (LOE 3)[144,199–203] and 20 additional studies (LOE 4[204–210]; LOE 5[211–223]) of long-term survivors of in- and out-of-hospital cardiac arrest, the quality of life among the majority of adult survivors is similar to that of the general population. Cognitive deficits in survivors, such as memory loss and depression, are common. In two studies (LOE 4)[224,225] neurologic outcomes were poor after cardiac arrest in children. Two studies indicate that the quality of life may not be as good in some cohorts, such as long-term care patients (LOE 5).[226,227]

Treatment recommendation. The quality of life for most adult survivors of cardiac arrest and CPR is good. There are few reports about longer-term quality of life in children. For more information about prognosis in adults, children, and neonates, see Part 2: ''Adult Basic Life Support,'' Part 6: ''Paediatric Basic and Advanced Life Support,'' and Part 7: ''Neonatal Resuscitation.''

Cost-effectiveness

Cost-effectiveness in CPR training programs
W183

Consensus on science. In the single study (LOE 3)[148] that considers the cost-effectiveness of CPR training programs, traditional CPR training in an unselected population of laypeople is expensive compared with accepted cost-effectiveness thresholds. Conversely, selective training of laypeople at high risk of witnessing a cardiac arrest (i.e. persons living in households with a recent survivor of myocardial infarction) is much more cost-effective.

Treatment recommendation. It is reasonable for CPR programs to emphasise the enrolment of laypeople with the highest probability of encoun-

tering cardiac arrest. Other potentially more cost-effective methods of training should be considered (see previous sections).

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at 10.1016/j.resuscitation.2005.09.021.

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Appendix 1: Worksheet topics and authors

International Liaison Committee on Resuscitation

Topic no.	Topic	Worksheet author and WS abbreviation	
		AHA author	ILCOR author
1	Securement of endotracheal tube following intubation	George W. Hatch, Jr, EdD, LP, EMT-P **W1_Hatch.doc**	
2	Impedance threshold valve in pediatric CPR	Lester T. Proctor, MD **W2_Proctor.doc**	
3	15:2 vs. 5:1 compression:ventilation ratio	Robert W. Hickey, MD **W3A_Hickey.doc** Robert A. Berg, MD **W3B_Berg.doc**	James Tibballs, MD **W3C_Tibballs.doc**
4	. . . the age-based sequence (''phone fast'' for infants and children, ''phone first'' for children >8 years old and adults) was retained (Class Indeterminate)	Linda Quan, MD **W4_Quan.doc**	
5	Lay rescuers are instructed to assess for signs of circulation rather than attempt to check a pulse (Class IIa)	Arno Zaritsky, MD **W5A_Zaritsky.doc** Melinda L. Fiedor, MD **W5B_Fiedor.doc**	
6	BMV ventilation vs. endotracheal intubation		Dominique Biarent **W6_Biarent.doc**
7	Mouth-to-nose rescue breathing	Vinay M. Nadkarni, MD **W7A_Nadkarni.doc**	David Zideman, MD **W7B_Zideman.doc**
8	Some CPR vs. no CPR	Robert A. Berg, MD **W8_Berg.doc**	
9	Two thumb circumferential CPR vs. two finger CPR	Monica E. Kleinman, MD **W9A_Kleinman.doc**	James Tibballs, MD **W9B_Tibballs.doc**
10	Capillary fill time		James Tibballs, MD **W10_Tibballs.doc**
11	Cuffed vs. uncuffed ET tubes	Lester T. Proctor, MD Ashraf Coovadia, MD **W11A_Coovadia.doc** **W11B_Proctor_Coovadia.doc**	

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Topic no.	Topic	Worksheet author and WS abbreviation	
		AHA author	ILCOR author
12	Predictors of return of spontaneous circulation (ROSC) and/or short term survival		David Zideman, MD **W12B_Zideman.doc**
13	Thrombolytics for pediatric cardiac arrest	Vinay M. Nadkarni, MD **W13_Nadkarni.doc**	
14	Potential for oxygen toxicity	Robert W. Hickey, MD **W14A_Hickey.doc**	David Zideman, MD **W14B_Zideman.doc**
15	Magnesium in pediatric cardiac arrest		Amelia Gorete Reis, PhD **W15_Reis.doc**
16	Hypertonic saline for resuscitation (traumatic and non-traumatic)		Renato Carrera, MD **W16_Carrera.doc**
17	Graded volume resuscitation for traumatic shock		Jesús López-Herce, MD **W17_Lopez-Hence.doc**
18	Ventilation prior to naloxone (discrepancy with peds and adult guidelines)	Anthony J. Scalzo, MD **W18_Scalzo.doc**	
19	Vasopressin for pediatric shock-refractory VF	Stephen M. Schexnayder, MD **W19A_Schexnayder.doc**	Dominique Biarent **W19B_Biarent.doc**
20	Lidocaine for pediatric shock-resistant VF or pulseless VT	Dianne L. Atkins, MD **W20_Atkins.doc**	
21	Amiodarone for pediatric shock resistant VF/VT	Dianne L. Atkins, MD **W21A_Atkins.doc**	Antonio Rodriguez-Nuñez **W21B_Rodrigues-Nunez.doc**
22	Post-resuscitation temperature management: a. induced hypothermia b. hyperthermia	Robert W. Hickey, MD **W22A_Hickey.doc** **W22B_Hickey.doc** Elise W. van der Jagt, MD, MPH **W22C_van der Jagt.doc**	Antonio Rodriguez-Nuñez **W22D_Rodrigues-Nuez.doc**
23	Secondary confirmation of tracheal tube placement using esophageal detector device	Monica E. Kleinman, MD **W23_Kleinman.doc**	
24	End-tidal CO_2 monitoring during transport.	Monica E. Kleinman, MD **W24_Kleinman.doc**	
25	Secondary confirmation of tracheal tube placement using exhaled CO_2	Monica E. Kleinman, MD **W25_Kleinman.doc**	
26	LMAs for pediatric arrest	Richard T. Fiser, MD **W26A_Fiser.doc**	Robert Bingham, MD **W26B_Bingham.doc**
27	Hyperventilation is no longer routinely recommended		Naoki Shimizu, MD, PhD **W27_Shimizu.doc**
28	ECMO and emergency cardiopulmonary bypass for children	Marilyn Morris **W28_Morris.doc**	
29	Intraosseous cannula		Allan de Caen, MD **W29_de Caen.doc**

Topic no.	Topic	Worksheet author and WS abbreviation	
		AHA author	ILCOR author
30	Use of glucose during and after a resuscitation	Paul M. Shore, MD **W30A_Shore.doc** Vijay Srinivasan, MD **W30B_Srinivasan.doc**	Amelia Gorete Reis, PhD **W30C_Reis.doc**
31	Dose of epinephrine for cardiac arrest in children	Adnan T. Bhutta **W31A_Bhutta.doc**	Robert Bingham, MD **W31B_Bingham.doc**
32	Administration and dosage of drugs given via the endotracheal tube		Allan de Caen, MD **W32_de Caen.doc**
33	Can vasoactive agents improve hemodynamics in the setting of post-arrest myocardial dysfunction?	Robert A. Berg, MD **W33A_Berg.doc** Allan de Caen, MD **W33B_de Caen.doc** Elise W. van der Jagt, MD, MPH **W33C_van der Jagt.doc** Berg, de Caen, van der Jagt Summary: **W33D_Berg_de Caen_van der Jagt.doc**	
34	Sodium bicarbonate for pediatric prolonged cardiac arrest	Douglas S. Diekema, MD, MPH **W34_Diekema.doc**	
35	Procainamide for a perfusing rhythm associated with VT	Ricardo Samson, MD **W35_Samson.doc**	
36	Vagal maneuvers	Ricardo Samson, MD **W36_Samson.doc**	
37	Procainamide is an alternative agent for hemodynamically stable SVT.	Ricardo Samson, MD **W37_Samson.doc**	
38	Amiodarone is an alternative agent for hemodynamically stable SVT.	Ricardo Samson, MD **W38_Samson.doc**	
39	Amiodarone for hemodynamically stable wide QRS tachycardia	William Hammill, MD **W39A_Hammil.doc** Ricardo Samson, MD **W39B_Samson.doc**	
40	Amiodarone for hemodynamically unstable VT	William Hammill, MD **W40_Hammil.doc**	
41	What is the appropriate dose for biphasic defibrillation in children	James Tibballs, MD **W41A_Tibballs.doc**	Linda Quan, MD **W41B_Quan.doc**
276	Method of chest (cardiac) compression for children	James Tibballs, MD Edward Stapleton **W276_Tibballs_Stapleton.doc**	
42	Is the **laryngeal mask airway (LMA—including variations)** as safe and effective as tracheal intubation for the management of the airway during cardiac arrest?	Michael Shuster, MD **W42A_Shuster.doc**	Jerry Nolan, MD **W42B_Nolan.doc**
43	Is the **Combitube** as safe and effective as tracheal intubation for the management of the airway during cardiac arrest?	Michael Shuster, MD **W43A_Shuster.doc**	Andreas R. Thierbach, MD **W43B_Thierbach.doc**

Topic no.	Topic	Worksheet author and WS abbreviation	
		AHA author	ILCOR author
44	Is the **laryngeal tube** as safe and effective as tracheal intubation for the management of the airway during cardiac arrest?	Michael Shuster, MD **W44A_Shuster.doc**	Andreas R. Thierbach, MD **W44B_Thierbach.doc**
45	Does the **oropharyngeal airway** provide a patent airway during CPR	Keiichi Tanaka, MD **W45_Tanaka.doc**	
46	Does the **nasopharyngeal airway** provide a patent airway during CPR	Keiichi Tanaka, MD **W46A_Tanaka.doc**	
47	Is use of end-tidal CO_2 safe and effective to confirm placement of tracheal tubes during cardiac arrest?	Henry E. Wang, MD MPH Frances X. Guyette **W47_Wang_Guyette.doc**	
48	Is use of an oesophageal detector device safe and effective for confirming placement of tracheal tubes during cardiac arrest?	Douglas F. Kupas, MD **W48A_Kupas.doc**	
49	Do commercial devices for securing the tracheal tube, backboards, cervical collars, other strategies provide a more effective method for preventing accidental tube displacement during resuscitation?	Robert O'Connor, MD **W49A_O'Connor.doc**	Burkhard Dirks, MD, PhD **W49B_Dirks.doc**
50	Is use of end-tidal CO_2 safe and effective to confirm placement of alternative airway devices during cardiac arrest?	Henry E. Wang, MD MPH Frances X. Guyette **W50_Wang_Guyette.doc**	
51	Is use of an oesophageal detector device safe and effective for confirming placement of alternative airway devices during cardiac arrest?	Douglas F. Kupas, MD **W51A_Kupas.doc**	Volker Dörges, MD **W51B_Dörges.doc**
52	When should we commence ventilation during cardiac arrest?	Octavio A. Falcucci, MD Rebecca L. Cain **W52_Falcucci_Cain.doc**	
53	What are the optimal respiratory rates and tidal volumes during cardiac arrest?		Volker Dörges, MD **W53_Dörges.doc**
54	Is the use of intermittent disconnection safe and effective for the management of ventilation during cardiac arrest?	Imo P. Aisiku Chris Hogan, MD **W54A_Aisiku_Hogan.doc**	Peter Morley, MD **W54B_Morley.doc**
55	Are automatic transport ventilators safe and effective for the management of ventilation during cardiac arrest?	Douglas F. Kupas, MD **W55_Kupas.doc**	
56	Are oxygen-powered, manually triggered devices safe and effective for the management of ventilation during cardiac arrest?		
57	Is the **bag-valve mask** as safe and effective as tracheal intubation for ventilation during cardiac arrest?		Jerry Nolan, MD **W57_Nolan.doc**
58	Does the use of fist pacing in cardiac arrest achieve adequate circulation?		Christoph Eich, MD **W58_Eich.doc**
59	Does the use of a pre-cordial thump in cardiac arrest successfully achieve cardioversion of VF or pulseless VT		Christoph Eich, MD **W59_Eich.doc**
60	What is the optimal energy level for defibrillation?	Richard E. Kerber, MD Colin Robertson, MD Karl B. Kern, MD **W60A_Kern.doc**	Ian G. Stiell, MD **W60B_Stiell.doc**

Topic no.	Topic	Worksheet author and WS abbreviation	
		AHA author	ILCOR author
61	What is the optimal waveform for defibrillation?	Richard E. Kerber, MD Colin Robertson, MD Karl B. Kern, MD **W61A_Kern.doc**	Peter Morley, MD **W61B_Morley.doc**
62	Does the use of AEDs in hospital improve outcome when compared with manual defibrillation?	Richard E. Kerber, MD	Peter Morley, MD **W62A_Morley.doc**
63	Does paddle size/orientation and position effect outcome during cardiac arrest?	Dianne L. Atkins, MD **W63A_Atkins.doc**	Benno Wolcke, MD **W63B_Wolcke.doc**
64	Is it possible to reliably predict success of defibrillation from the fibrillation waveform?	Max Harry Weil, MD, PhD, DSc (HON) Mary Ann Peberdy, MD **W64A_Weil_Peberdy.doc**	Petter Andreas Steen, MD, PhD **W64B_Steen.doc** Trygve Eftestøl, Dr.Ing **W64C_Eftestol.doc**
65	Does the prediction of the likelihood of success of defibrillation enable treatment to be altered to improve outcome?	Max Harry Weil, MD, PhD, DSc (HON) Mary Ann Peberdy, MD **W65A_Weil_Peberdy.doc**	
66	Does the collection of the data acquired from a defibrillator provide valuable information for quality control and education?		Dr. Michael Baubin, MSc **W66_Baubin.doc**
67	Does the delay for rhythm analysis, either manually or automatically, adversely effect outcome?		
68	Does chest compression before defibrillation improve outcome?	Vincent N Mosesso, Jr, MD Edison Ferreira de Paiva, MD Leo Bossaert, MD, PhD **W68_Gazmuri_Mosesso_de Paiva_Bossaert.doc**	
69	Does the use of up to 3 shocks for subsequent shocks improve outcome compared with single shock?	Wanchun Tang, MD Max Harry Weil, MD, PhD, DSc (HON) **W69A_Tang_Weil.doc**	Max Harry Weil, MD, PhD, DSc (HON) **W69B_Weil.doc** Rudolph W. Koster, MD, PhD **W69C_Koster.doc**
70	Does the presence of supplementary oxygen in the immediate vicinity increase the risks of fire during defibrillation	Joseph P. Ornato, MD **W70A_Ornato.doc**	Jerry Nolan, MD **W70B_Nolan.doc**
71	Do self-adhesive defibrillation pads have benefit over standard paddles		Charles D. Deakin, MA, MD **W71_Deakin.doc**
72	Does the composition of conductive material affect transthoracic impedance		Dr. Michael Baubin, MSc **W72_Baubin.doc**
73	Does IAC-CPR improve outcome from cardiac arrest when compared with standard CPR?	Charles F. Babbs, MD, PhD **W73A_Babbs.doc**	Peter Morley, MD **W73B_Morley.doc**

Topic no.	Topic	Worksheet author and WS abbreviation	
		AHA author	ILCOR author
74	Does High-Frequency CPR improve outcome from cardiac arrest when compared with standard CPR?	Henry Halperin, MD **W74_Halperin.doc**	
75	Does ACD-CPR improve outcome from cardiac arrest when compared with standard CPR?	Henry Halperin, MD **W75A_Halperin.doc**	Peter Morley, MD **W75B_Morley.doc**
76	Does Vest CPR improve outcome from cardiac arrest when compared with standard CPR?	Henry Halperin, MD **W76A_Halperin.doc**	Pierre Carli, MD **W76B_Carli.doc**
77	Does Mechanical (Piston) CPR improve outcome from cardiac arrest when compared with standard CPR?	Henry Halperin, MD **W77A_Halperin.doc** Sten Rubertsson, MD, PhD **W77B_Rubertsson.doc**	
78	Does Phased Thoracic-Abdominal Compression-Decompression CPR improve outcome from cardiac arrest when compared with standard CPR?		Kjetil Sunde, MD, PhD **W78B_Sunde.doc**
79	Does MID-CM improve outcome from cardiac arrest when compared with standard CPR?	Henry Halperin, MD **W79A_Halperin.doc**	Pierre Carli, MD **W79B_Carli.doc**
80	Does Impedance Threshold Valve improve outcome from cardiac arrest when compared with standard CPR?	Henry Halperin, MD **W80_Halperin.doc**	
81	Does Open Chest CPR improve outcome from cardiac arrest when compared with standard CPR?		Lars Widklund, MD Sten Rubertsson, MD, PhD **W81B_Wiklund_Rubertsson.doc**
82	Do extracorporeal techniques or invasive perfusion devices improve outcome from cardiac arrest when compared with standard CPR?	Octavio A. Falcucci, MD Seshendra Chirumamilla **W82_Falcucci_Chirumamilla.doc**	
83	What is the optimal drug therapy for VF?	Jason S. Haukoos, MD, MS Norman A. Paradis, MD **W83A_Haukoos_Paradis.doc** Jason Bartsch Charles M. Little, DO Norman A. Paradis, MD **W83B_Bartsch_Little_Paradis.doc** Charles M. Little, DO Norman A. Paradis, MD **W83C_Little_Paradis.doc** **W83D_Little_Paradis.doc** Gayle Long, MD Norman A. Paradis, MD **W83E_Long_Paradis.doc** Gayle Long, MD Charles M. Little, DO Norman A. Paradis, MD **W83F_Long_Little_Paradis.doc** **W83G_Long_Little_Paradis.doc**	Hendrik W. Gervais, MD, PhD **W83H_Gervais.doc** **W83I_Gervais.doc** **W83J_Gervais.doc** **W83K_Gervais.doc**

Topic no.	Topic	Worksheet author and WS abbreviation	
		AHA author	ILCOR author
84	What is the optimal drug therapy for asystole?	Gayle Long, MD Charles M. Little, DO Norman A. Paradis, MD **W84A_Long_Little_Paradis.doc** **W84B_Long_Little_Paradis.doc** Jason Bartsch Charles M. Little, DO Norman A. Paradis, MD **W84C_Bartsch_Little_Paradis.doc**	Volker Wenzel, MD **W84D_Wenzel.doc**
85	What is the optimal drug therapy for PEA?	Gayle Long, MD Charles M. Little, DO Norman A. Paradis, MD **W85A_Long_Little_Paradis.doc** **W85B_Long_Little_Paradis.doc**	Hendrik W. Gervais, MD, PhD **W85C_Gervais.doc**
86	What is the optimal drug therapy for atrial fibrillation?	Robert O'Connor, MD **W86_O'Connor.doc**	
87	What is the optimal drug therapy for narrow complex tachycardia?		Swee Han Lim, MD **W87_Lim Swee Han.doc**
88	What is the optimal drug therapy for monomorphic (wide complex) tachycardia?		Hans Domanovits, MD **W88_Domanovits.doc**
89	What is the optimal drug therapy for polymorphic (wide complex) tachycardia?		Hans Domanovits, MD **W89_Domanovits.doc**
90	What is the optimal drug therapy for Torsades do Pointes?		Hans Domanovits, MD **W90_Domanovits.doc**
91	What is the optimal drug therapy for significant bradycardia?		Swee Han Lim, MD **W91_Lim Swee Han.doc**
92	Does the use of end-tidal CO_2 monitoring during cardiac arrest guide more appropriate management?	Arthur B. Sanders, MD **W92A_Sanders.doc**	Benno Wolcke, MD **W92B_Wolcke.doc**
93	Does the use of arterial blood gas monitoring during cardiac arrest guide more appropriate management?	Max Harry Weil, MD, PhD, DSc (HON) **W93A_Weil.doc**	Fulvio Kette, MD **W93B_Kette.doc**
94	Does alteration of management based on the use of ultrasound during cardiac arrest improve outcome?		
95	Does the use of coronary perfusion pressure guide more appropriate management?	Charles M. Little, DO Norman A. Paradis, MD **W95A_Little_Paradis.doc**	Wolfgang G. Voelckel, MD **W95C_Voelckel.doc**
96	Does the use of thrombolytics improve outcome when used during the management of cardiac arrest?	Joseph P. Ornato, MD **W96A_Ornato.doc** Riyad B. Abu-Laban, MD, MHSc **W96B_Abu-Laban.doc**	Bernd W. Böttiger, MD **W96C_Boettiger.doc**
97	Does the use of atropine improve outcome when used during the management of cardiac arrest?	Munish Goyal, MD **W97A_Goyal.doc**	Swee Han Lim, MD **W97B_Swee Han Lim.doc**

Topic no.	Topic	Worksheet author and WS abbreviation	
		AHA author	ILCOR author
98	Does the use of aminophylline improve outcome when used during the management of cardiac arrest?	Riyad B. Abu-Laban, MD, MHSc **W98A_Abu-Laban.doc**	Burkhard Dirks, MD, PhD **W98B_Dirks.doc**
99	Does the use of calcium improve outcome when used during the management of cardiac arrest?		
100	Does the use of buffers improve outcome when used during the management of cardiac arrest?	Kyle Gunnerson, MD **W100A_Gunnerson.doc**	Fulvio Kette, MD Gad Bar-Joseph **W100B_Kette_Bar-Joseph.doc**
101	Does the use of magnesium improve outcome when used during the management of cardiac arrest?	Ross Berringer **W101A_Berringer.doc**	Miguel Ruano-Marco, MD **W101B_Ruano.doc**
102	Does the use of potassium improve outcome when used during the management of cardiac arrest?		
103	Does the use of drug X improve outcome when used during the management of cardiac arrest due to drug toxicity with drug Y? (digoxin, cyanide, opiates, organophosphates, cocaine, beta-blockers, calcium channel blockers, tricyclics)		
104	Does the use of pacing for asystolic cardiac arrest improve outcome?		Miguel Ruano-Marco, MD **W104_Ruano.doc**
105	Does the routine use of fluids during resuscitation improve outcome from cardiac arrest?	Terry L. Vanden Hoek, MD Raina Merchant Jasmeet Soar, MD **W105_vanden Hoek_Merchant_Soar.doc**	
106	Does ventilation before giving naloxone improve outcome when used during the management of cardiac arrest due to opioid toxicity?		Jasmeet Soar, MD **W106_Soar.doc**
107	Does the use intraosseus fluid and drugs improve outcome during cardiac arrest		
108	What is the role and optimal dose of drugs given via the tracheal route during cardiac arrest		Volker Wenzel, MD **W108_Wenzel.doc**
109	Does the use of **therapeutic hypothermia** in the management of the patient after a cardiac arrest improve outcome?	Terry L. Vanden Hoek, MD **W109A_vanden Hoek.doc**	Peter Morley, MD Jerry Nolan, MD **W109B_Morley_Nolan.doc**
110	Does the **prevention of hyperthermia/use of antipyretics** in the management of the patient after a cardiac arrest improve outcome?	David G. Beiser, MD/MS Terry L. Vanden Hoek, MD **W110_Beiser_Vanden Hoek.doc**	
111	Does the **prevention of seizures** in the management of the patient after a cardiac arrest improve outcome?	Kyle Gunnerson, MD **W111A_Gunnerson.doc**	Nabil El Sanadi, MD, MBA **W111B_El Sanadi.doc**

Topic no.	Topic	Worksheet author and WS abbreviation	
		AHA author	ILCOR author
112	Does the use of **cardiovascular support**, including vasopressor and inotropic drugs, in the management of the patient after a cardiac arrest improve cerebral and/or cardiovascular outcome?		Matthias Fischer, MD **W112_Fischer.doc**
113	Does the use of **sedation/paralysis** for a specified duration in the management of the patient after a cardiac arrest improve outcome?	Imo P. Aisiku Chris Hogan, MD **W113_Aisiku_Hogan.doc**	
114	Does the **control of arterial CO_2** in the management of the patient after a cardiac arrest improve outcome?		Hendrik W. Gervais, MD, PhD **W114B_Gervais.doc**
115	Does the use of **tight blood glucose control** in the management of the patient after a cardiac arrest improve outcome?	Mary Ann Peberdy, MD Terry L. Vanden Hoek, MD **W115A_Peberdy_vanden Hoek.doc**	Jerry Nolan, MD **W115B_Nolan.doc**
116	Does the use of **thrombolytics** in the management of the patient following cardiac arrest improve outcome?		Bernd W. Böttiger, MD **W116_Boettiger.doc**
117	Does the use of **anticoagulation** in the management of the patient after a cardiac arrest improve outcome?		
118	Does the use of **prophylactic anti-arrhythmics** in the management of the patient after a cardiac arrest improve outcome?	Steven Kronick, MD, MS **W118A_Kronick.doc**	Nabil El Sanadi, MD, MBA **W118B_El Sanadi.doc**
119	Can the rescuer identify the aetiology of the cardiac arrest during the cardiac arrest? (e.g. asphyxia induced cardiac arrest, drug/toxin induced VT/VF [cocaine], drug induced PEA, Hypothermia, Drowning, Trauma, Electrolytes, Anaphylaxis, Asthma, Pulmonary	Arlo Weltge, MD, MPH **W119A_Weltge.doc**	Sebastian Russo, MD - Asthma **W119B_Russo.doc** Jasmeet Soar, MD—Pregnancy **W119C_Soar.doc**
120	Does the identification of the aetiology during the cardiac arrest allow tailored cardiac arrest management (BLS/ALS)?	Arlo Weltge, MD, MPH **W120_Weltge.doc**	
121	Does the identification of the aetiology, and tailored cardiac arrest management during the cardiac arrest improve outcome?	Arlo Weltge, MD, MPH **W121_Weltge.doc**	
122	Can neurological examination, e.g., pupil dilation, allow the rescuer to predict the likely outcome of the cardiac arrest during the cardiac arrest?	Steven Kronick, MD, MS **W122A_Kronick.doc**	Petter Andreas Steen, MD, PhD **W122B_Steen.doc**
123	Can any stat laboratory analyses or other investigations allow the rescuer to predict the likely outcome of the cardiac arrest during the cardiac arrest?		
124	Can the use of somatosensory evoked potentials allow the rescuer to predict the likely outcome of the cardiac arrest after the cardiac arrest?	Octavio A. Falcucci, MD **W124A_Falcucci.doc**	Rien de Vos, MD Albert Hijdra, MD **W124B_de Vos_Hijdra.doc**

Topic no.	Topic	Worksheet author and WS abbreviation	
		AHA author	ILCOR author
125	Can the use EEG allow the rescuer to predict the likely outcome of the cardiac arrest after the cardiac arrest?		
126	Can the use of serum analyses allow the rescuer to predict the likely outcome of the cardiac arrest after the cardiac arrest?	Douglas Franzen Gregory Christiansen, DO **W126_Franzen_ Christiansen.doc**	
127	Can the use of CSF analyses allow the rescuer to predict the likely outcome of the cardiac arrest after the cardiac arrest?		
128	Can the use of early warning scoring systems reduce the number of in-hospital cardiac arrests?	Mary Ann Peberdy, MD **W128A_Peberdy.doc**	Michael Parr, MBBS, MRCP Michelle Cretikos, MBBS, MPH **W128B_Parr_ Cretikos.doc**
129	Does the use of a Medical Emergency Team reduce the number of in-hospital cardiac arrests	Mary Ann Peberdy, MD **W129A_Peberdy.doc**	Michael Parr, MBBS, MRCP Michelle Cretikos, MBBS, MPH **W129B_Parr_ Cretikos.doc**
130	Does the use of a Medical Emergency Team improve outcome from in-hospital cardiac arrest	Mary Ann Peberdy, MD **W130A_Peberdy.doc**	Michael Parr, MBBS, MRCP Michelle Cretikos, MBBS, MPH **W130B_Parr_ Cretikos.doc**
131	What modifications are applicable to resuscitation technique for: Hypothermia	Arlo Weltge, MD, MPH **W131_ Weltge.doc**	
132	What modifications are applicable to resuscitation technique for: Drowning	Arlo Weltge, MD, MPH **W132_Weltge.doc**	
133	What modifications are applicable to resuscitation technique for: Asthma		Jasmeet Soar, MD **W133_Soar.doc**
134	What modifications are applicable to resuscitation technique for: Pregnancy		Jasmeet Soar, MD **W134_Soar.doc**
135	What modifications are applicable to resuscitation technique for: Electrocution		Nabil El Sanadi, MD, MBA **W135_El Sanadi.doc**
136	What modifications are applicable to resuscitation technique for: Anaphylaxis		
137	What is the incidence, prevalence, etiology of cardiopulmonary arrest?	Tom Rea **W137_Rea.doc**	
138	What are the independent predictors of cardiopulmonary arrest? What are the independent predictors of outcomes after CPA?	Tom Rea **W138A_Rea.doc** **W138B_Rea.doc**	
139	What interventions are feasible, safe and effective in individuals at risk of impending CPA (i.e. within 24 h)?	N. Clay Mann, PhD, MS **W139A_Mann.doc** **W139B_Mann.doc**	Jennifer Dennett **W139C_Dennett.doc**

Topic no.	Topic	Worksheet author and WS abbreviation	
		AHA author	ILCOR author
140	What are adverse effects for the patient who receives cardiopulmonary resuscitation?	Graham Nichol, MD **W140A_Nichol.doc**	
141	What are the adverse effects for the responder who performs cardiopulmonary resuscitation (Incl infection control)?	Graham Nichol, MD **W141A_Nichol.doc** **W141B_Nichol.doc**	Franklin HG Bridgewater, MD **W141C_Bridgewater.doc**
142	What is the sensitivity, specificity and clinical impact of signs of need for resuscitation, including agonal respirations, shaking and signs of circulation?	Wanchun Tang, MD **W142A_Tang.doc**	Ian Jacobs, RN, PhD **W142B_Jacobs.doc**
143	What is sensitivity and specificity, clinical of signs of need for resuscitation in facedown victim? . . . in suspected neck injury	Mike Jacobs, EMT-P **W143A_Jacobs.doc**	Jeff Wassertheil, MD **W143B_Wassertheil.doc**
144	(For above, consider any differences in S, E and F according age of victim and availability of responders?)		
145	(For above consider aetiology e.g. trauma, drowning, intoxication,arrythmia, respiratory arrest		
146	What is the feasibility, safety and effectiveness of repositioning a victim?	Lei Huang Wanchun Tang, MD **W146A_Huang_Tang.doc**	Tony Walker **W146B_Walker.doc**
147	What is the sensitivity, specificity and clinical impact of interruption of CPR to check circulation?	Ting Yu, MD Wanchun Tang, MD **W147A_Yu_Tang.doc**	Jeff Wassertheil, MD **W147B_Wassertheil.doc**
148	What is the safety, effectiveness and feasibility of improving response time?	Lynn J. White, MS **W148A_White.doc**	
149	Which methods for opening the airway, are feasible, safe and effective?		Gavin Perkins, MD **W149_Perkins.doc**
150	What interventions are safe effective and feasible when performing CPR in victims with suspected cervical spine injury? For above, consider over the head position for CPR? . . . body position of victim? Body positions of responder?	Edward Crosby, MD **W150A_Crosbie.doc**	Gavin Perkins, MD **W150B_Perkins.doc**
151	Are methods for removal of FBAO feasible, safe and effective? For above, consider chest compression/finger sweep or alternatives For above, consider Heimlich, chest thrust? Consider responsive and unresponsive victim? Consider obese, pregnant?	Thomas A. Barnes, EdD, RRT **W151A_Barnes.doc**	Gavin Perkins, MD **W151B_Perkins.doc**
152	Are mechanical ventilators used by basic-trained rescuers (first responders) and professional health care providers safe and effective for ventilating unintubated adult patients during cardiac arrest	Thomas A. Barnes, EdD, RRT Richard Branson **W152A_Barnes_Branson.doc**	
153	Are devices/adjuncts for airway positioning, ventilation feasible, safe and effective?		
154	Which compression-ventilation ratio is feasible, safe and effective for which etiology, condition and age-group?	Andrea Gabrielli, MD Peter Fenici, MD **W154_Gabrielli_Fenici.doc**	

Topic no.	Topic	Worksheet author and WS abbreviation	
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155	What recovery positions are feasible, safe and effective?		Anthony J. Handley, MD E. Brooke Lerner, PhD **W155_Handley_Lerner.doc**
156	Which tidal volume and ventilation rate are feasible, safe and effective using MMV/BVM/with or without O_2 for which aetiology, condition, and age-group?	Andrea Gabrielli, MD E. Brooke Lerner, PhD **W156A_Gabrielli_Lerner.doc**	
157	Is MNV safe, effective and feasible compared to MMV?	E. Brooke Lerner, PhD **W157A_Lerner.doc**	Benno Wolcke, MD **W157B_Wolcke.doc**
158	Which methods of ventilation are feasible, safe and effective in MSV?	E. Brooke Lerner, PhD **W158A_Lerner.doc**	Benno Wolcke, MD **W158B_Wolcke.doc**
159	What is the safety, effectiveness and feasibility of protective devices to protect a rescuer while performing CPR? Incl barrier devices	Andrea Gabrielli, MD E. Brooke Lerner, PhD **W159A_Gabrielli_Lerner.doc**	Benno Wolcke, MD **W159B_Wolcke.doc**
160	What is the safety, effectiveness, and feasibility of performing CPR on a near drowning victim in the water? (consider C-spine injury, VF, call first/call fast)	Jane G. Wigginton, MD Ahamed H. Idris, MD David Szpilman, MD **W160A_Wigginton_Idris_Szpilman.doc** **W160B_Wigginton_Idris_Szpilman.doc**	
161	What is the safest most feasible and effective intervention for removing a near drowning victim from the water?	Jane G. Wigginton, MD Ahamed H. Idris, MD David Szpilman, MD **W161_Wigginton_Idris_Szpilman.doc**	
162	What interventions are safe, effective and feasible for immersion, exposure, or accidental hypothermia? Consider active rewarming	Benjamin S. Abella, MD, MPhil **W162A_Abella.doc**	
163	What CPR devices are safe, effective and feasible? (limited to circulation: chest compressors, boards to be placed under neck? are there simple first line devices for diagnosing circulatory arrest?)	Jane G. Wigginton, MD **W163A_Wigginton.doc** **W163B_Wigginton.doc** **W163C_Wigginton.doc** **W163D_Wigginton.doc** **W163E_Wigginton.doc** **W163F_Wigginton.doc** **W163G_Wigginton.doc** **W163H_Wigginton.doc** **W163I_Wigginton.doc** **W163J_Wigginton.doc**	
164	Is compression only CPR safe, effective and feasible? When should ventilation begin?	Vincent N Mosesso, Jr, MD E. Brooke Lerner, PhD **W164A_Mosesso_Lerner.doc**	Rudolph W. Koster, MD, PhD **W164B_Koster.doc**
165	Is dispatcher-assisted CPR safe, effective and feasible?	Lynn Roppolo, MD Ahamed H. Idris, MD **W165_Roppolo_Idris.doc**	

Topic no.	Topic	Worksheet author and WS abbreviation	
		AHA author	ILCOR author
166	Alternative methods of CPR including cough CPR and precordial thump	Ahamed H. Idris, MD **W166A_Idris.doc** **W166B_Idris.doc** **W166C_Idris.doc** **W166D_Idris.doc**	
167	What hand position/depth of chest compression is safe, effective and feasible?	Andrea Gabrielli, MD Peter Fenici, MD **W167A_Gabrielli_Fenici.doc** **W167B_Gabrielli_Fenici.doc**	Rudolph W. Koster, MD, PhD **W167C_Koster.doc**
168	What compression-decompression method is safe, effective and feasible?	Jane G. Wigginton, MD **W168_Wigginton.doc**	
169	Are rectilinear first-phase biphasic waveform shocks with escalation to 200J or non-escalating 150-200J biphasic shocks safer, more effective, and feasible?		
170	Are rectilinear first-phase biphasic waveform shocks with escalation to 200J or 200-360J escalating energy biphasic shocks safer, more effective and feasible?		
171	Escalating-energy biphasic waveform shocks delivering energy in the range of 200-360J are safer, more effective, and more feasible than are non-escalating-energy biphasic waveforms delivering 200J or less	Karl B. Kern, MD Ian G. Stiell, MD **W171_Kern_Stiell.doc**	
172	Biphasic waveforms for use in transthoracic defibrillation of VF cardiac arrest are more efficacious (higher rates of VF termination, ROSC, and survival) as well as safer (fewer adverse effects) than monophasic waveforms	Karl B. Kern, MD Colin Robertson, MD **W172_Kern_Robertson.doc**	
173	What pad position is safe, effective and feasible for AED use?	Vincent N Mosesso, Jr, MD **W173A_Mosesso.doc**	
174	What is the safety, effectiveness and feasibility of AED programmes?	Keith Lurie, MD Rudolph W. Koster, MD, PhD **W174_Lurie_Koster.doc**	
175	(For above, consider defibrillation by EMS, first responder, public access, home use, wearable cardioverter defibrillators)	Keith Lurie, MD Rudolph W. Koster, MD, PhD **W175_Lurie_Koster.doc**	
176	What algorithms should be recommended for AED users? One shock or three?		
177	Is CPR before defibrillation safe, effective and feasible?	Vincent N Mosesso, Jr, MD Edison Ferreira de Paiva, MD Raúl J. Gazmuri, MD, PhD Leo Bossaert, MD, PhD **W177_Gazmuri_Mosesso_de Paiva_Bossaert.doc**	
178	What quality assurance is appropriate for AED users? Does the collection of data from the AED affect quality control and education?	Vincent N Mosesso, Jr, MD **W178_Mosesso.doc**	

Topic no.	Topic	Worksheet author and WS abbreviation	
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179	What is the impact of 'advanced directives', 'living wills' and 'do not resuscitate orders' in directing resuscitative efforts?	Deems Okamoto, MD **W179A_Okamoto.doc** **W179B_Okamoto.doc** Terri Schmidt, MD, MS Kenneth V. Iserson, MD, MBA **W179C_Schmidt_Iserson.doc**	
180	Should family members be present during resuscitation?	Douglas S. Diekema, MD, MPH **W180A_Diekema.doc**	Dominique Biarent **W180B_Biarent.doc**
181	Ethical issues in paediatric resuscitation		
182	What are the outcomes associated with resuscitation after CPA (incl health-related quality of life)	David Rodgers, EdS, NREMT-P **W182A_Rodgers.doc**	Dr Judith Finn, PhD, RN **W182B_Finn.doc**
183	What is the cost-effectiveness of lay-responder training in CPR?	Peter Cram, MD, MBA **W183_Cram.doc**	
184	Are people who are trained in CPR willing to perform it? (Chest compression only)	Judy Young, RN, MSN, Lt Col, USAF (Ret) **W184A_Young.doc**	Jennifer Dennett **W184B_Dennet.doc**
185	What instructional methods are most effective in BLS skill acquisition and retention at 6 months? - traditional lecture/practice session - interactive computer programmes - video self-instruction	David Rodgers, EdS, NREMT-P **W185A_Rodgers.doc**	Ana Paula Quilici Marcello Ricardo Paulista Markus, PhD **W185B_Quilici_Markus.doc**
186	How frequently are ACLS/BLS re-training/update sessions required in order to maintain skills in a) laypersons and b) health professionals?		Jennifer Dennett **W186A_Dennet.doc** Anthony J. Handley, MD **W186B_Handley.doc**
187	Does the use of audio/visual CPR performance aids during training improve the acquisition of CPR psychomotor skills?		
188	Does a written test score reflect BLS skill competence?	Cheryl Hamel, PhD **W188A_Hamel.doc**	Jeff Wassertheil, MD **W188B_Wassertheil.doc**
189	What instructional methods are most effective for teaching hand position in external cardiac compression		Dr Judith Finn, PhD, RN **W189_Finn.doc**
190	What CPR prompt devices are safe, effective and feasible?	David Rodgers, EdS, NREMT-P **W190A_Rodgers.doc** Jane G. Wigginton, MD **W190B_Wiggington.doc**	
191	What instructional methods are most effective in training and skill-retention in AED use?	Judy Young, RN, MSN, Lt Col, USAF (Ret) **W191A_Young.doc**	Antonio Celenza **W191B_Celenza.doc**
192	What is the Effectiveness of CPR Self-Instruction to Train Lay Rescuers in the Community		Antonio Celenza **W192_Celenza.doc**
193	Do community-wide media campaigns decrease patient delay in response to chest pain?	Charles Mount, MEd, Capt, USN (Ret) Sharon Coleman, RN, MSN, CNS **W193A_Mount_Coleman.doc**	Dr Judith Finn, PhD, RN **W193B_Finn.doc**

Topic no.	Topic	Worksheet author and WS abbreviation	
		AHA author	ILCOR author
194	Does the chain of survival result in improved outcomes from cardiac arrest, in and out of hospital?		Ian Jacobs, RN, PhD **W194_Jacobs.doc**
195	Does the use of Medical Emergency Team reduce the number (and outcome) of in-hospital cardiac arrest?	Mary Ann Peberdy, MD **W195A_Peberdy.doc** Elise W. van der Jagt, MD, MPH **W195B_van der Jagt.doc**	Michelle Cretikos, MBBS, MPH Michael Parr, MBBS, MRCP **W195C_Cretikos_Parr.doc** **W195D_Cretikos_Parr.doc** **W195E_Cretikos_Parr.doc**
196	What is the risk of infection or other adverse event during CPR training		Franklin HG Bridgewater, MD **W196_Bridgewater.doc**
197	Sodium bicarbonate for hyperkalemia, hypermagnesemia, tricyclic antidepressant overdose, or overdose from other sodium channel blocking agents (from PEDs)	Douglas S. Diekema, MD, MPH **W197A_Diekema.doc** **W197B_Diekema.doc** **W197C_Diekema.doc**	James Tibballs, MD **W197D_Tibballs.doc** **W197E_Tibballs.doc**
198	Calcium for hypocalcemia, hyperkalemia, hypermagnesemia and calcium channel blocker overdose Resolve discrepancy between adult and pediatric dose (from PEDs)	Anthony J. Scalzo, MD **W198_Scalzo.doc**	
199	What interventions are safe, effective and feasible for treatment of anaphylaxis and severe allergic reactions by BLS providers?	Ron Roth, MD David C. Cone, MD **W199_Roth_Cone.doc**	
200	Sodium bicarbonate infusion during DR resuscitation	Jeffrey Perlman, MB, Ch B **W200_Perlman.doc**	
201	Hyperthermia in the DR	Jeffrey Perlman, MB, Ch B **W201_Perlman.doc**	
202	Room Air/O_2	Jay P. Goldsmith, MD **W202A_Goldsmith.doc**	Sam Richmond, MD **W202B_Richmond.doc**
203	Initial Ventilation strategies during DR resuscitation	David Boyle, MD **W203A_Boyle.doc**	Edgardo Szyld, MD **W203B_Szyld.doc** Ben J. Stenson, MD **W203C_Stenson.doc**
204	The use of CPAP during DR resuscitation	Louis P. Halamek, MD **W204A_Halamek.doc**	Colin Morley, MD **W204B_Morley.doc**
205	Meconium — Oro-pharyngeal suctioning at theperineum with meconium staining		
206	ET suctioning of meconium post-delivery		Sithembiso Velaphi, MB **W206_Velaphi.doc**
207	Amnioinfusion during labor to reduce Meconium Aspiration Syndrome	Dharmapuri Vidyasagar **W207A_Vidyasagar.doc** Sithembiso Velaphi, MB **W207B_Velaphi.doc**	
208	Crystalloid/Albumin Infusions during DR resuscitation	Susan Niermeyer, MD **W208_Niermeyer.doc**	
209	Delivery room ethics—emphasis on the initiation and discontinuation of resuscitation	Jay P. Goldsmith, MD **W209A_Goldsmith.doc**	Steve Byrne **W209B_Byrne.doc**

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		AHA author	ILCOR author
210	Maintaining temperature in the delivery room with specific emphasis on the preterm infant	Marilyn B Escobedo, MD **W210A_Escobedo.doc**	Mike Watkinson, MD **W210B_Watkinson.doc**
211	Hypothermia as a neuroprotective therapy	Michael Speer, MD **W211A_Speer.doc** Jeffrey Perlman, MB, Ch B **W211B_Perlman.doc**	
212	CO_2 detectors to verify ET placement	Wally Carlo, MD **W212A_Carlo.doc**	Jonathan Wyllie, MD **W212B_Wyllie.doc**
213	Administration of endotracheal medications	Myra H. Wyckoff, MD **W213A_Wyckoff.doc**	Jonathan Wyllie, MD **W213B_Wyllie.doc**
214	Naloxone administration in the DR	Myra H. Wyckoff, MD **W214A_Wyckoff.doc**	Ruth Guinsburg **W214B_Guinsburg.doc**
215	Laryngeal Mask Airway to establish airway patency during neonatal resuscitation	Gary Weiner, MD **W215A_Weiner.doc**	Enrique Udaeta, MD **W215B_Udaeta.doc**
216	Placental transfusion	Susan Niermeyer, MD **W216A_Niermeyer.doc**	Nalini Singhal **W216B_Singhal.doc**
217	Interosseous Infusion of medications		William A. Engle, MD **W217_Engle.doc**
218	Glucose post Resuscitation	Jeffrey Perlman, MB, Ch B **W218A_Perlman.doc** Jane E. McGowan, MD **W218B_McGowan.doc**	
219	Glucose homeostasis during DR resuscitation	Jeffrey Perlman, MB, Ch B **W219A_Perlman.doc** Jane E. McGowan, MD **W219B_McGowan.doc**	
220	Intravenous High Dose Epinephrine during DR resuscitation	Jeffrey Perlman, MB, Ch B **W220_Perlman.doc**	
221	What is the sensitivity, specificity and clinical impact on signs and symptoms in prehospital and emergency department management of ACS and AMI?	David Lendrum, MD **W221A_Lendrum.doc**	Andrzej Okreglicki, MD **W221B_Okreglicki.doc**
222	What is the sensitivity, specificity and clinical impact on protein markers in the prehospital and emergency department management of ACS and AMI?	Bjug Borgundvaag MD, PhD **W222A_Borgundvaag.doc**	Brian Steinhart **W222B_Steinhart.doc**
223	What is the sensitivity, specificity and clinical impact of prehospital and emergency department 12 lead ECG interpretation on the prehospital and emergency department management of ACS AMI?		
224	What is the safety, efficacy and feasibility of Oxygen vs. Room Air in prehospital and emergency department management of ACS and AMI?	Dave Hostler, PhD, NREMT-P **W224_Hostler.doc**	
225	What is the safety, efficacy and feasibility of ASA in prehospital and emergency department ACS and AMI?	Ivy Cheng **W225A_Cheng.doc**	Bjug Borgundvaag MD, PhD **W225B_Borgundvaag.doc**

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226	What is the safety, efficacy and feasibility of Heparin UF vs. LMW in prehospital and emergency department management of ACS and AMI?	Jane Lukins, MD **W226A_Lukins.doc**	
227	What is the safety, efficacy and feasibility of Fibrinolytics in prehospital and emergency department management of ACS and AMI?	Monica Gope **W227A_Gope.doc** **W227B_Gope.doc**	
228	What is the safety, efficacy and feasibility of Clopidigrel in prehospital and emergency department management of ACS and AMI?	Nicole Tenn-Lyn, MD **W228A_Tenn-Lyn.doc**	
229	What is the safety, efficacy and feasibility of IIB IIIA Inhibitors in prehospital and emergency department management of ACS and AMI?		
230	What is the safety, efficacy and feasibility of Prophylactic Antiarrhythmics in prehospital and emergency department management of ACS and AMI?	Dan Cass **W230_Cass.doc**	
231	What is the safety, efficacy and feasibility of ACE Inhibitors in prehospital and emergency department management of ACS and AMI?	Uwe Zeymer, Priv. Doz. Dr. med. **W231_Zeymer.doc**	
232	What is the safety, efficacy and feasibility of Beta Blockers in prehospital and emergency department management of ACS and AMI?	Jonathan Sherbino, MD **W232_Sherbino.doc**	
233	What is the safety, efficacy and feasibility of Statins in prehospital and emergency department management of ACS and AMI?		Hans-Richard Arntz, MD, PhD **W233_Arntz.doc**
234	What is the safety, efficacy and feasibility of PTCA vs. fibrinolytics in prehospital and emergency department management of ACS and AMI?	Russell D. MacDonald, MD **W234A_MacDonald.doc**	Hans-Richard Arntz, MD, PhD **W234B_Arntz.doc**
235	What is the safety, efficacy and feasibility of PH ECG and ED advance notification vs. standard EMS care or vs. PH fibrinolytics in prehospital and emergency department management of ACS and AMI?	Dave Hostler, PhD, NREMT-P **W235A_Hostler.doc**	Steven Brooks, MD **W235B_Brooks.doc**
236	What is the safety, efficacy and feasibility of PH bypass for PTCA in prehospital and emergency department management of ACS and AMI?	Michelle Welsford, MD **W236A_Welsford.doc**	Cathal O'Donnell **W236B_O'Donnell.doc**
237	What is the safety, efficacy and feasibility of Community lytics combined with immediate transfer for PTCA vs. delayed transfer for PTCA (standard care) in prehospital and emergency department management of ACS and AMI?	Warren J. Cantor **W237A_Cantor.doc**	Fabrice Brunet, MD **W237B_Brunet.doc**
238	What is the sensitivity, specificity and clinical impact of prehospital stroke scales?	E. Brooke Lerner, PhD **W238_Lerner.doc**	
239	What is the safety, effectiveness and feasibility of ''stroke centers''? (Are there items shown to be effective in stroke care in the first hours of stroke)	Michael R. Sayre, MD **W239_Sayre.doc**	

Topic no.	Topic	Worksheet author and WS abbreviation	
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240	What is the safety, effectiveness and feasibility of prehospital personnel triage of potential stroke patients to specific stroke hospitals?	Todd Crocco, MD **W240A_Crocco.doc**	
241	What is the safety, effectiveness and feasibility of supplemental oxygen in acute stroke?	Jeffrey L. Saver, MD Werner Hacke, MD, PhD Simone Wagner, MD **W241_Saver_Hacke_Wagner.doc**	
242	What is the safety, effectiveness and feasibility of blood pressure management in acute ischemic and hemorrhagic stroke?		
243	What is the safety, effectiveness and feasibility of hypothermia for acute stroke?	Edward C. Jauch, MD, MS **W243A_Jauch.doc** Andy Jagoda **W243B_Jagoda.doc**	
244	What is the safety, effectiveness and feasibility of glucose management in acute ischemic stroke?	Michael R. Sayre, MD **W244A_Sayre.doc**	
245	What is the safety, effectiveness and feasibility of intravenous rt-PA in acute ischemic stroke?	Todd Crocco, MD **W245_Crocco.doc**	
246	What is the safety, effectiveness and feasibility of intra-arterial thrombolysis in acute ischemic stroke?	Brian A. Stettler, MD **W246_Stettler.doc**	
247	What is the safety, efficacy, and feasibility of cooling in the first aid management of a thermal cutaneous burn?	Adam J. Singer, MD **W247_Singer.doc**	Andrew DePiero, MD Debra G. Perina, MD
248	What is the most appropriate first aid of the burn blister?	Debra G. Perina, MD **W248_Perina.doc**	Adam J. Singer, MD
249	What is the safety, efficacy, and feasibility of charcoal in an oral poisoning?	Christopher P. Holstege, MD **W249_Holstege.doc**	Ryan C. Fringer, MD Edward Sargeant
250	What is the safety, efficacy, and feasibility of syrup of ipecac in the first aid management of a toxic ingestion (oral poisoning)?	Ryan C. Fringer, MD **W250_Fringer.doc**	
251	What is the safety, efficacy, and feasibility of dilution with water or milk or taking nothing by mouth?	David Markenson, MD **W251_Markenson.doc**	Ryan C. Fringer, MD
252	What is the safety and feasibility of *assisting* the victim in the administration of the victim's own self-administered epinephrine (adrenaline) in first aid management of a *severe* allergic reaction?	Jonathan L. Epstein, MEMS, NREMT-P **W252_Epstein.doc**	Jeff Wassertheil, MD William Brady, MD
253	What is the safety and feasibility of *assisting* the victim in the administration of the victim's own self-administered albuterol in first aid management of a *breathing difficulty* in the asthmatic patient?	David Markenson, MD **W253_Markenson.doc**	Susan F. Wooley, PhD
254	What is the safety, efficacy, and feasibility of direct pressure, pressure points and elevation in the first aid management of a hemorrhage?	Leon Chameides, MD **W254_Chameides.doc**	Richard Bissell, PhD

Topic no.	Topic	Worksheet author and WS abbreviation	
		AHA author	ILCOR author
255	What is the safety, efficacy, and feasibility of the tourniquet in the first aid management of a hemorrhage?	Leon Chameides, MD **W255_Chameides.doc**	Sherri-Lynne Almeida, DrPH Ralph M. Shenefelt
256	What is the safety, efficacy, and feasibility of spinal immobilization in the first aid management of a suspected spinal injury?	Bill Raynovich, EMSA **W256_Raynovich.doc**	William Brady, MD James A. Judge II, CEM, BPA, EMT-P
257	Under what conditions should the lay rescuer suspect spinal injury?	William Brady, MD **W257_Brady.doc**	Bill Raynovich, EMSA Jonathan L. Epstein, MEMS, NREMT-P
258	What is the safety, efficacy, and feasibility of irrigation in the first aid management of a toxic exposure to the skin and/or eye?	James A. Judge II, CEM, BPA, EMT-P **W258_Judge.doc**	Christopher P. Holstege, MD
259	What is the safety, efficacy, and feasibility of treatment of an eye injury by a first aider?	Cartland Burns, MD **W259_Burns.doc**	Donald J. Gordon, PhD, MD
260	What is the safety, efficacy, and feasibility of stabilization in the first aid management of an injured (suspected fracture) extremity?	William Hammill, MD **W260_Hammill.doc**	Richard Bissell, PhD
261	What is the safety, efficacy, and feasibility of compression in the first aid management of an injured extremity joint?	Rita Ann Herrington **W261_Herrington.doc**	Ryan C. Fringer, MD Rick Caissie
262	What is the safety, efficacy, and feasibility of cooling in the first aid management of an injured extremity joint?	Thomas W. Zoch, MD **W262_Zoch.doc**	
263	What is the safety, efficacy, and feasibility of psychological first aid (may also need to define psychological first aid)?		
264	What is the safety, efficacy, and feasibility of oxygen administration in the first aid management of the dyspneic patient?	James A. Judge II, CEM, BPA, EMT-P **W264_Judge.doc**	Bill Clendenen, MBA James A. Judge II, CEM, BPA, EMT-P
265	What is the most appropriate first aid management of the cutaneous abrasion including safety, efficacy, and feasibility of antibiotic ointment?	Mary Fry Davis, RN **W265_Davis.doc**	Ricky Davidson, MD Carol Spizzirri
266	What is the most appropriate first aid management of the cutaneous abrasion including safety, efficacy, and feasibility of tap water?	Mary Fry Davis, RN Carol Spizzirri **W266_Davis_Spizzirri.doc**	Ricky Davidson, MD
267	What is the safety, efficacy, and feasiblity of body-part rewarming in the first aid management of a localized cold injury?	David Markenson, MD **W267_Markenson.doc**	Arthur Cooper, MD, MS Rick Caissie Rick Murray, EMT-P
268	What are the risk factors for possible spinal injury that can be used by the lay rescuer?	Arthur Cooper, MD, MS **W268_Cooper.doc**	
269	What is the incidence of spinal injury?	Arthur Cooper, MD, MS **W269_Cooper.doc**	
270	What is the safety, efficacy and feasibility of compressive wrapping for coral snake (elapid) envenomation?	Naomi Gauthier, MD Stephen H. Thomas, MD, MPH **W270_Gauthier_Thomas.doc**	Donald J. Gordon, PhD, MD
271	What is the safety, efficacy and feasibility of incision-mediated wound suctioning for pit viper envenomation?	Christopher P. Holstege, MD **W271_Holstege.doc**	

Topic no.	Topic	Worksheet author and WS abbreviation	
		AHA author	ILCOR author
272	What is the best first aid treatment for burns wet or dry dressings?		
273	What is the safety, efficacy, and feasibility of straightening angulated long bone fractures?	Michael Bosse, MD **W273_Bosse.doc**	Rick Murray, EMT-P
274	What is the safety, efficacy, and feasibility of the left lateral recumbenant position or the recovery position?	David Markenson, MD **W274_Markenson.doc**	
275	Is it safe, feasible and effective to place the avulsed tooth in milk until definitive therapy can be provided	David Markenson, MD **W275_Markenson.doc**	
277	**Blank worksheet**	**W277**	

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Appendix 2: Previous ''giants'' honorees

International Liaison Committee on Resuscitation

The American Heart Association began the tradition of honoring ''giants'' in resuscitation science at the 1985 conference establishing new resuscitation guidelines. ILCOR joined this tradition in 2000. ''Giants'' honorees are selected based on ongoing landmark contributions to cardiopulmonary resuscitation and emergency cardiovascular care in general; authorship of books, articles, and education and teaching materials in the field of emergency cardiovascular care; national and international recognition for their contributions to the field; and development of innovative technological and scientific breakthroughs. The 2005 Honorees are listed in the front of this supplement. Past honorees are listed here:

1985
James Elam, MD
Archer Gordon, MD
James Jude, MD
Guy Knickerbocker, PhD
Peter Safar, MD

1992
Stephen Carveth, MD
Leonard Cobb, MD
Frank Pantridge, MD
Joseph Redding, MD
Paul Zoll, MD

2000
Douglas Chamberlain, MD
Leon Chameides, MD
Mickey Eisenberg, MD, PhD
Gordon Ewy, MD
Laerdal Foundation represented by Tore Laerdal
Richard Kerber, MD
William Montgomery, MD
Joseph Ornato, MD
Leonard Scherlis, MD
Max Harry Weil, MD, PhD.

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Appendix 3: Conflict of interest for editors, editorial board, special contributors and reviewers, and honorees

International Liaison Committee on Resuscitation

See details in following pages.

doi:10.1016/j.resuscitation.2005.10.023

Writing group	Member	Employment	Research grant	Other research support	Speakers bureau/ honoraria	Ownership interest	Consultant/advisory board	Other
Editors								
1	Hazinski, Mary Fran	Vanderbilt Children's Hospital	Served as uncompensated principle investigator for Medtronic Physio Control sponsored study of AEDS in children (1996—99, published in 2003).	None	None	None	Senior Science Editor, American Heart Association; consultation fees for AHA ECC programs	None
2	Nolan, Jerry	Royal United Hospital NHS Trust, Bath, UK	None	None	None	None	None	None
Editorial board								
1	Montgomery, William H.	Straub Clinic and Hospital	None	None	None	None	None	None
2	Zaritsky, Arno	University of Florida, College of Medicine	None	None	None	None	None	None
3	Morley, Peter	Melbourne Health	None	None	None	None	Deputy Chair, Australian Resuscitation Council; Evidence worksheet expert, American Heart Association	None
4	Nadkarni, Vinay	University of Pennsylvania, School of Medicine; Children's Anesthesiology Assoc., Division Critical Care	Research grants, NIH/NICHD; Ross/Abbott; Sensormedics; Drager Medical	None	None	None	Unpaid Education Consultant: Laerdal, Medical Education Technologies, Inc.	None
5	O'Connor, Robert	Christiana Care Health Systems	None	Research funding from AED manufacturers, Astra-Zeneca, McNeil, Pfizer; No salary support	None	None	President, National Association EMS Physicians; Chair, ACEP EMS Committee; Board, National Registry of EMTs	None
6	Deakin, Charles	Southhampton University Hospital, UK	None	None	None	None	None	None
7	Nichol, Graham	University of Washington	None	Sponsor, Investigational Device Exemption; Wearable Cardioverter Defibrillator trial; RAFT Trial investigator (CIHR, Medtronic Inc.); Sponsor, Wenabk Defibrillator IDF.	None	None	None	None

8	Handley, Anthony	Essex Rivers Healthcare Trust	None	None	None	None	Part-time Consultant, Laerdal Sophus, Copenhagen; Executive member, Resuscitation Council, London	None
9	Hickey, Robert	University of Pittsburgh	None	None	None	None	None	None
10	Zideman, David	Hammersmith Hospitals NHS Trust, UK	None	None	None	None	Chairman, European Resuscitation Council; Chairman, British Association for Immediate Care; District Medical Officer, St. John Ambulance	None
11	Perlman, Jeffrey	Weill—Cornell Medical Center, New York, USA	None	None	None	None	None	None
12	Richmond, Sam	City Hospitals, Sunderland NHS Trust	None	None	None	None	Chairman, Newborn Life Support, Resuscitation Council (UK); Editor, Newborn Life Support Provider Course Material (no royalties or financial gain)	None
13	Mattes, Mark	Clarian Health Partners, Inc.	None	None	None	None	Assistant Chief, Sugar Creek TWA Fire Department, New Palestine, IN, USA	None
14	Finn, Judith	University of Western Australia	None	None	None	None	None	None
15	Morrison, Laurie	University of Toronto, Sunnybrook and Women's University, Academic Health Science Center	Grantee, Zoll Medical, Boston, MA, USA; Grantee, Advantis, Grantee, Hoffman La Roche, Toronto, CA, USA	None	None	None	None	None
16	Arntz, Hans-Richard	Benjamin Franklin University Hospital, Berlin	None	Travel Reimbursement, Bristol Myers Squibb	Paid Speaker, Hoffmann La Roche, Boehringer Ingelheim, Merck—Sharp Dohme, Lilly, Germany	None	None	None
17	Billi, John	University of Michigan Medical School	None	None	None	None	Editorial Board, Resuscitation	None

Writing group	Member	Employment	Research grant	Other research support	Speakers bureau/ honoraria	Ownership interest	Consultant/advisory board	Other
18	Shuster, Michael	Self-employed	None	None	None	None	Chair: ECC Policy Advisory Committee, Heart and Stroke Foundation of Canada	None
19	Jacobs, Ian	University of Western Australia	Research grant, National Health and Medical	Untied Research Support, Laerdal Australia; Research Support, CA, USA; Registry, St. John Ambulance	None	None	Chairman, Australian Resuscitation Council	None
20	Kloeck, Walter	Academy of Advanced Life Support	None	None	None	None	Chairman, Resuscitation Council of Southern Africa	Distributor of AHA ECC Training Materials
21	Timerman, Sergio	Heart Institute, St. Paul University, School of Medicine	None	None	None	None	Consultant, Baldacci; Paid Consultant, Boehringer Ingelhem	None
22	Bossaert, Leo	None	None	None	None	None	Executive Director, European Resuscitation Council	None
23	Sayre, Michael	The Ohio State University	Research grant, Zoll/Revivant	Travel Reimbursement, Medtronic	None	None	Ohio Board of EMS, NHTSA Department of Transportation	None
24	Hammill, William	University of Virginia	None	Partner: IEC Inc.—contract with Cardioconcepts to develop web-based BLS products for healthcare providers, Cardioconcepts has partnered with the AHA to develop this product line	None	None	None	None
Special contributors and reviewers								
1	Chameides, Leon	Retired, Paid Editor/Writer 2005 AHA ECC Guidelines	None	None	None	None	None	None
2	Becker, Lance	University of Chicago	None	Fellowship Funds, Alsius Corp., Irvine, CA, USA	None	None	Consultant/Research Funds, Philips Medical, Seattle, WA, USA; Consultant, Abbott Labs, Abbot Park, IL, USA; Consultant, DSMB/PRC NIH, Bethesda, MD, USA	None

3	Steen, Petter Andreas	University of Oslo	None	None	None	None	Member, Laerdal Executive Board; Member, Norwegian Air Ambulance Executive Board; Member, Cardiodigital Medical Advisory Board	None
4	Baskett, Peter	Retired, Editor-in-Chief, Resuscitation	None	None	None	None	None	None
5	Kattwinkel, John	University of Virginia	None	None	None	None	None	None
6	Verbeek, Rick	The Ottawa Hospital, University of Toronto, and the Ottawa Health Research Institute	None	None	None	None	None	None
2005 Honorees								
1	Baskett, Peter	Retired, Editor-in-Chief, Resuscitation	None	None	None	None	None	None
2	Cummins, Richard	University of Washington Medical Center	None	None	None	None	None	None
3	Keenan, William	St. Louis University	None	None	None	None	None	None
4	Steen, Petter Andreas	University of Oslo	None	None	None	None	Member, Laerdal Executive Board; Member, Norwegian Air Ambulance Executive Board; Member, Cardiodigital Medical Advisory Board	None
5	Weisfeldt, Myron	Johns Hopkins University	Grantee, NIH, Bethesda, MD, USA	None	None	None	None	None