Key Principles in the Application of ACLS

The Importance of Time

The passage of time drives all aspects of ECC. The final outcomes are determined by the intervals between collapse or onset of the emergency and the delivery of basic and advanced interventions. The probability of survival declines sharply with each passing minute of cardiopulmonary compromise. Some interventions, like basic CPR, slow the rate at which this decline in probability occurs. CPR makes this contribution by supplying some blood flow to the heart and brain. Some single interventions, such as tracheal intubation, clearing an obstructed airway, or defibrillating a heart in VF, are sufficient alone to restore a beating heart. For all of these interventions, independently sufficient or simply contributory, the longer it takes to administer these therapies, the lower the chances of benefit.

The “Periarrest” Period

Emergency cardiovascular care no longer focuses only on the patient in cardiac arrest. Emergency care providers cannot narrow their objectives to only the arrest state. They must recognize and treat effectively those patients “on their way to a cardiac arrest” and those recovering in the immediate postresuscitation period. Once these patients are identified, ECC personnel must be able to rapidly initiate appropriate therapy. If responders treat critical conditions properly in this “periarrest” or “prearrest” period, they can prevent a full cardiopulmonary arrest from occurring.

Consequently, the international ACLS recommendations present the science-based clinical guidelines and some educational material for these periarrest conditions:

- Acute coronary syndromes
- Acute pulmonary edema, hypotension, and shock
- Symptomatic bradycardias
- Stable and unstable tachycardias
- Acute ischemic stroke
- Impairments of rate, rhythm, or cardiac function in the postresuscitation period (by definition a periarrest/prearrest condition)

Other parts of the ECC and CPR guidelines present guidelines for more specific causes of cardiac arrest, such as electrolyte abnormalities, drug toxicity or overdoses, and toxic ingestions.

Never Forget the Patient

Resuscitation challenges care providers to make decisions quickly and under pressure. Providers must occasionally limit their focus for a brief time to a specific aspect of the resuscitative attempt: getting the IV infusion line started, placing the tracheal tube, identifying the rhythm, and remembering the “right” medication to order. But rescuers constantly must return to an overall view of each resuscitative attempt. The flow diagrams or algorithms focus the learner on the most important aspects of a resuscitative effort: airway and ventilation, basic CPR, defibrillation of VF, and medications suitable for a particular patient under specific conditions.

Code Organization: Using the Primary and Secondary ABCD Surveys

The International Perspective

Many approaches to code organization exist. The section that follows describes the approach taught in AHA courses for ACLS and pediatric resuscitation. This does not imply that methods of code organization used in other countries are incorrect or less successful.

Why Is Training in ACLS Intentionally Multidisciplinary?

An understandable tendency exists internationally to separate the highly trained professional from less skilled personnel during ACLS training. Such a practice, however, would jeopardize one of the most important objectives of resuscitation training. This objective is to have each member of the multidisciplinary response team know and understand the skills and roles of each of the other team members. An accomplished senior physician may claim, “I already know the resuscitation guidelines and already possess the psychomotor skills. Why must I attend a learning session with less trained responders who are not authorized to perform tracheal intubation, start an IV drip, or order medications?”

An experienced instructor might respond in several ways, but the response should remind the expert that he or she must still work with the entire responding team. The expert must know what the other team members can and cannot perform so that attempted resuscitation proceeds smoothly, quietly, and effectively.

Of even greater importance, the ACLS team member who possesses the lowest level of professional training will attend future resuscitative attempts as a critical quality control agent. Nurses, for example, who work in critical care and emergency care areas may not perform intubation or defibrillation in some settings, but they can detect with surprising speed...
and accuracy when other team members attempt the procedure incorrectly! In American hospitals, particularly academic teaching centers, nurses prevent innumerable medical mishaps during resuscitative attempts. They gently (and sometimes not so gently) point out when the tracheal tube is misplaced, the IV line has become a subcutaneous line, CPR is inadequate, or the medication ordered was incorrect or wrongly dosed.

While emergency personnel are encouraged to know and experience the role of team leader, training should concentrate on the team aspects of resuscitative efforts. The course of resuscitative attempts may be complex and unpredictable. Indeed, a good resuscitation team has been likened to a fine symphony orchestra. The team recognizes the team leader for broad skills of organization and performance. They recognize the individual team member for specific performance skills. Like an orchestra, all are performing the same piece, polished by practice and experience, with attention to both detail and outcome. There is no excuse for a disorganized and frenetic code scene.

The team leader should be decisive and composed. The team should stick to the ABCs (airway, breathing, and circulation) and keep the resuscitation room quiet so that all personnel can hear without repetitious commands. Team members should

- State the vital signs every 5 minutes or with any change in the monitored parameters
- State when procedures and medications are completed
- Request clarification of any orders
- Provide primary and secondary assessment information

The team leader should communicate her or his observations and should actively seek suggestions from team members. Evaluation of airway, breathing, and circulation should guide the efforts whenever the vital signs are unstable, when treatment appears to be failing, before procedures, and for periodic clinical updates.

The next section describes the Primary and Secondary ABCD Surveys. This aide-mémoire provides an easily remembered listing of the content and sequence of the specific assessment and management steps of a resuscitative attempt.

The Primary and Secondary ABCD Surveys
All who respond to cardiorespiratory emergencies should arrive well trained in a simple, easy-to-remember approach. The ACLS Provider Course teaches the Primary and Secondary Survey Approach to emergency cardiovascular care. This memory aid describes 2 sets of 4 steps: A-B-C-D (8 total steps). With each step the responder performs an assessment and then, if the assessment so indicates, a management.

Conduct the Primary ABCD Survey
The Primary ABCD Survey requires your hands (gloved!), a barrier device for CPR, and an AED for defibrillation. The Primary ABCD Survey assesses and manages most immediate life threats:

- Airway: Assess and manage the Airway with noninvasive techniques.
- Breathing: Assess and manage Breathing with positive-pressure ventilations.
- Circulation: Assess and manage the Circulation, performing CPR until an AED is brought to the scene.
- Defibrillation: Assess and manage Defibrillation, assessing the cardiac rhythm for VF/VT and providing defibrillatory shocks in a safe and effective manner if needed.

Conduct the Secondary ABCD Survey
This survey requires medically advanced, invasive techniques to again assess and manage the patient. The rescuer attempts to restore spontaneous respirations and circulation to the patient and when successful, continues to assess and manage the patient until relieved by appropriate emergency professionals. In brief: resuscitate, stabilize, and transfer to higher-level care.

- Airway: Assess and manage. Advanced rescuers manage a compromised airway by placing a tracheal tube.
- Breathing: Assess and manage. Assess adequacy of breathing and ventilation by checking tube placement and performance; correct all problems detected. Manage breathing by treating inadequate ventilation with positive-pressure ventilations through the tube.
- Circulation: Assess and manage the circulation of blood and delivery of medications by
  —Starting a peripheral IV line
  —Attaching ECG leads to examine the ECG for the most frequent cardiac arrest rhythms (VF, pulseless VT, asystole, and PEA)
  —Administering appropriate rhythm-based medications
- Differential Diagnosis: Assess and manage the differential diagnoses that you develop as you search for, find, and treat reversible causes.

The Resuscitation Attempt as a “Critical Incident”: Code Critique and Debriefing
After any resuscitation attempt team members should perform a code critique. In busy emergency or casualty departments, carving out the necessary few minutes can be difficult. The lead physician, however, should assume responsibility to gather as many team members as possible for at least a pause to reflect. This debriefing provides feedback to prehospital and in-hospital personnel, gives a safe venue to express grieving, and provides an opportunity for education. Table 1 provides information on critical incident stress debriefing.

An alternative approach to critical stress debriefing is presented by Kenneth V. Iserson, MD, in his book Pocket Protocols: Notifying Survivors About Sudden, Unexpected Deaths, from which the excerpt in Table 2 is adapted.

Family Presence in the Resuscitation Area
In a number of countries, hospitals have begun to allow family members and loved ones to remain in the resuscitation suite during procedures and actual resuscitative efforts. Evaluations of these programs, pioneered by critical care and emergency nurses, have confirmed a remarkable level of approval and gratitude by participating family members.
TABLE 1. Recommendations for Resuscitation Team Critique and Debriefing

Ask team members to assemble soon after the event. With few exceptions all team members should be present.

Gather the group in a private place if possible. Use the resuscitation room if available. State the purpose: “We want to have a brief review (debriefing) of our resuscitative attempt.”

Start with a review of the events and conduct of the code. “Let’s start from the arrival of the paramedics. Could (nurse) review our sequence of interventions?”

State the algorithm or protocol that should have been followed; discuss what was actually done; discuss why there were any variations. “So this was an out-of-hospital VF arrest treated by the medics. When we assumed care, what protocol was indicated? How well did we do?”

Analyze the decisions and actions that were done correctly and effectively. Discuss decisions that may have been incorrect; discuss any actions that were performed less than optimally. Allow free discussion. “When the patient’s pulse was restored it seemed like everyone left the room. Only (nurse) was in the room when Mr. (patient) rearrested. Who wants to explain that delay?”

All team members should share their feelings, anxieties, anger, and possible guilt. “I feel upset because when the admitting team arrived they were really obnoxious, demanding a lot of tests and x-rays. They made me feel that we had done a bad job.”

Ask for recommendations or suggestions for future resuscitative attempts. “How can we do this better the next time?” (Nurse:) “I think we should not call the admitting team until the patient is completely stable and ready to go upstairs.”

Inform team members unable to attend the debriefing of the process followed, the discussion generated, and the recommendations made. “Chuck, we are going to implement that plan to allow family members in the code room during the resuscitation. I know you have been opposed to that. What if we designate our social worker to stay at the side of the family members the entire time they are near the resuscitation?”

The team leader should encourage team members to contact him or her if questions arise later.

These evaluations, mostly in pediatric cases, have noted significant reduction in posttraumatic stress and self-reports of a greater sense of resolution and fulfillment. In the 2000 pediatric resuscitation guidelines, family presence in the resuscitation area has a Class IIb positive recommendation. Provision must be made for a professional to accompany the family members during these observed attempts, to direct positioning, to answer questions, and to explain procedures. In addition, the accompanying professional can observe for signs of acute discomfort in the family members and can end the observations.

We lack sufficient evidence about family presence during adult resuscitations, but this is simply due to an absence of research in adults. Success in such programs for adults is predictable, provided that the professionals involved demonstrate the same high level of care and concern as shown by nurses and social workers involved in pediatric resuscitative attempts.

Ethics and the Clinical Practice of BLS and ACLS: Do Resuscitation Efforts “Fail”?

Of major importance, but often neglected in the rush to learn all of advanced resuscitation training, we must not forget the

TABLE 2. Critical Incident Stress Debriefing of Professionals: A Simplified Protocol

There are 4 sequential aspects to critical incident stress debriefing (CISD). These are the on-scene debriefing, the initial defusing, the formal CISD, and the follow-up CISD. Not all 4 aspects are always used, however.

On-Scene or Near-Scene Debriefing

This is performed by an officer, chaplain, or health professional knowledgeable in both CISD and the operations of the team. This individual primarily watches for the development of any signs of acute stress reactions. Rather than a formal debriefing, it is mainly a period of aware observation.

Initial Defusing

Performed within a few hours of the incident, this is a situation in which participants have an opportunity to discuss their feelings and reactions in a positive and supportive atmosphere. This discussion may be led by a senior officer or health professional familiar with CISD who has good interpersonal skills, or it may have no leader at all and be a spontaneous interaction among team members. It is best done through a mandatory team meeting.

The key to success at this stage is to maintain a supportive rather than a critical atmosphere, to keep comments confidential, and to ban comments that are tough, insensitive, or could be construed as “gallows humor.” If this is not done, it will quickly end any sharing feelings among team members.

Formal CISD

Typically led by a mental health professional familiar with CISD, these formal sessions are held within 24 to 48 hours after the incident. Specially trained public and private CISD teams now exist throughout the United States, Canada, and in many other countries. Many of these are associated with local or regional police or fire departments (who can also be contacted to locate other competent teams).

These sessions often follow a standard format by first laying out the noncritique and confidentiality ground rules. Then the participants are asked to describe themselves and key activities during the incident, their feelings during the incident and at present, and any unusual symptoms they experienced or are experiencing.

Participants may be asked to explore linkages between the event and past events, nonjudgmentally describe others’ actions (to help describe their own actions), and describe their own and the group’s successes during the incident. The facilitator then describes typical posttraumatic stress disorder (PTSD) symptoms and finally suggests an activity to help them regain a sense of purpose and unity (such as attending the memorial service for the victims). During this session, the leader also tries to identify those who may need more intensive counseling.

Follow-Up CISD

Not always or even frequently done, these sessions are held from several weeks to months after the incident. They can be held to resolve specific group issues or more often to help specific individuals. (When held on an individual basis, these are essentially psychological counseling sessions.)

Groups who will encounter events triggering PTSD in the course of their work must have this service available before it is needed. Individuals providing these counseling services may themselves be subject to PTSD, and, if so, should also undergo debriefing.

Additional Resources

For more information to assist professionals working with PTSD victims, contact the National Center of PTSD at telephone 802-296-5132, e-mail ptsd@dartmouth.edu, or website http://www.dartmouth.edu/dms/ptsd/Clinicians.htm or the Post Traumatic Stress Resources web page at http://www.long-beach-va-gov/ptsd/stress.htm.

TABLE 3. Conveying News of a Sudden Death to Family Members

| Call the family if they have not been notified. Explain that their relative has been admitted to the Emergency Department and that the situation is serious. Survivors should not be told of the death over the telephone. |
| Obtain as much information as possible about the patient and the circumstances surrounding the death. Carefully go over the events as they happened in the Emergency Department. |
| Ask someone to take family members to a private area. Walk in, introduce yourself, and sit down. Address the closest relative. |
| Briefly describe the circumstances leading to the death. Go over the sequence of events in the Emergency Department. Avoid euphemisms such as “he’s passed on,” “she’s no longer with us,” or “he’s left us.” Instead, use specific phrases and words such as “death,” “dying,” or “dead.” “Your mother died quietly, without suffering. . . .” “Her death was quiet and peaceful. . . .” |
| Allow time for the shock to be absorbed. Make eye contact, touch, and share. Convey your feelings with a phrase such as “You have my (our) sincere sympathy” rather than “I (we) are sorry.” |
| Allow as much time as necessary for questions and discussion. Go over the events several times to make sure everything is understood and to facilitate further questions. |
| Allow the family the opportunity to see their relative. If equipment is still connected, let the family know. |
| Know in advance what happens next and who will sign the death certificate. Physicians may impose burdens on staff and family if they fail to understand policies about death certification and disposition of the body. |
| Know the answers to these questions before meeting the family. |
| Enlist the aid of a social worker or the clergy if not already present. |
| Offer to contact the patient’s attending or family physician and to be available if there are further questions. Arrange for follow-up and continued support during the grieving period. |

resuscitation team and team members, as well as the surviving friends and relatives. As soon as you declare death for the arrest victim, you immediately acquire a new set of patients—the family, friends, and loved ones of the person who dies (see Table 3).

Remember that when the heart or brain of a person in arrest cannot be restarted, do not use the word fail. The team did not fail to restore the heartbeat, nor did the heart itself fail to respond to the efforts. Instead think in terms of an attempt to restore a “heart too good to die”5 rather than a “heart too sick to live.”6 At the start, however, the clinical reality is unknown; caregivers have no way of knowing the status of suddenly arrested hearts when they arrive on the scene of a cardiac emergency.

In the past we used the phrase “give a trial of CPR”; the only way to recognize “too good to die” versus “too sick to live” was to give the patient a rapid, aggressive evaluation period of BLS and ACLS. If spontaneous circulation did not return quickly, then we assumed that the verdict in the trial of CPR was “person at the end of his or her life.” In such a situation continued resuscitative efforts are inappropriate, futile, undignified, and demeaning to both patient and rescuers. “Part 2: Ethical Aspects of CPR and ECC” provides an ethical framework with which to consider resuscitative efforts and presents specific recommendations for prehospital and in-hospital care providers.

References

7B: Understanding the Algorithm Approach to ACLS

Origin of the ACLS Algorithms
The first ACLS “algorithms” appeared in the 1986 ECC and CPR Guidelines.1 These outlines of the 4 algorithms presented the interventions for the 4 arrest rhythms, using double-spaced lines of type connected by vertical arrows. Since those first primitive algorithms, diagrams have been a major tool to depict critical observations, critical actions, and critical decision points in resuscitation. Since 1986 similar algorithms have been published by the Resuscitation Councils of Europe (1992)2-3 and in southern Africa (1995).4-7 In the years since 1986 a variety of algorithmic approaches emerged. Differences have been in design and detail, not in science or clinical recommendations. Each set of ACLS algorithms contained information on the same general principles of resuscitation but presented it in a unique style with varying amounts of detail and very different target audiences.4,5

The Structure of the Algorithms
All resuscitation algorithms depict both observation and action steps. These steps typically alternate. The observation steps serve as a series of decision-making points or “decision nodes.” You identify the problems present at the decision node and then select the proper action to take. The alternating observation and decision steps in the algorithms closely resemble the alternating “assess-manage” steps fundamental to emergency care and resuscitation.

In 2000 in this set of algorithms, we have given all observational boxes curved corners and all action boxes square corners. The treatment of every resuscitation emergency can be mapped into this assess-manage series of steps, with repeating loops and reassessment.

The Philosophy of the Algorithms
The algorithms have grown to mean different things to different resuscitation councils around the world. They mean different things to the training networks within those resuscitation councils. In some resuscitation councils the algorithms were designed to distill essential information about identification and treatment of a problem to its essence—such a concise display targets the novice practitioner and encourages the expert to provide his or her own detail or additional information. Such an approach was favored by Dr Walter Kloeck, National Chairman of the Resuscitation Councils of Southern Africa. Dr Kloeck’s sparse, clean design aimed to depict the most common assessments and actions performed for the vast majority of patients. These algorithms were designed for the beginning or student learner of CPR, ECC, and ACLS. This elegantly simple style of teaching materials has come to dominate the teaching materials of many international resuscitation councils.7

At the same time, within the AHA the algorithms came to be used by instructors and experienced clinicians as teaching tools. The training network began to request inclusion of more and more detail to address a wider variety of clinical situations with more and more information for the clinician and for the ACLS instructor. These algorithms, although more complex, were thought to be more useful during actual resuscitations and more useful for teaching the scope of resuscitation practice.

Clearly, each approach—concise versus complex—has its merits. When the first international algorithms were developed for the ILCOR Advisory Statements, the differences in algorithmic approach became apparent.8 Because the ILCOR advisory statements were evidence-based consensus documents, the algorithms that emerged there were spare. They were limited to those points of assessment and care on which there was absolute agreement.

The algorithms contained in the International Guidelines 2000 represent the second iteration of international algorithms, developed by science experts at the Evidence Evaluation Conference and at the Guidelines 2000 Conference. They represent a compromise between the concise approach of many international resuscitation councils and the detailed approach favored within the AHA. Details are provided within the text and in boxes pulled out of the main body of the algorithm.

Application of the Algorithms
These algorithms are designed to serve as an aide-mémoire, to remind the clinician of important aspects of assessment and therapy. They are not designed to be either comprehensive or limiting. The clinician should always determine whether the algorithm is appropriate for the patient and should be prepared to deviate from the algorithm if the patient’s condition warrants. The algorithms should be thought of as a general recipe from a cherished grandmother—the general guiding principles are there, but the richness will be in the individual application. The algorithms may provide the recipe but they still require a “thinking cook” (see Table 4).

The algorithms are depicted in a sequential format. This is misleading, however, because in most resuscitation situations multiple care providers are present, and many assessments and interventions are accomplished simultaneously. Many of these algorithms contain notes on assessment and evaluation that should be considered throughout the resuscitation (eg, verify proper tracheal tube placement, identify and treat reversible causes).
Learners and clinicians are not expected to memorize the algorithms in full detail. They are expected to consult the algorithms. It is hoped that copies of these algorithms will be available at courses and written evaluations for courses. The rationale for this approach is realistic and professional, based on established principles of adult education. The clinician should know where to find appropriate information and how to apply it. The algorithms are intended to lead the clinician along the path of assessment and intervention during the resuscitation experience.

References

7C: A Guide to the International ACLS Algorithms

Summary/Overview
The ILCOR algorithm presents the actions to take and decisions to face for all people who appear to be in cardiac arrest—unconscious, unresponsive, without signs of life. The victim is not breathing normally, and no rescuer can feel a carotid pulse within 10 to 15 seconds. Since 1992 the resuscitation community has examined and reconfirmed the wisdom of most recommendations formulated by international groups through the 1990s. Sophisticated clinical trials provided high-level evidence on which to base several new drugs and interventions. Finally, we have learned that we should continue to place a strong emphasis after 2000 on building a base of critically appraised, international scientific evidence. Evidence-based review opened many eyes; only a small proportion of resuscitation care rests on a base of solid evidence.

Note: The numbers below, such as “1 (Figure 1),” match numbers in the algorithms.

Figure 1: ILCOR Universal/International ACLS Algorithm
Figure 1, the ILCOR Universal/International ACLS Algorithm, and Figure 2, the Comprehensive ECC Algorithm, are groundbreaking efforts to unify and simplify the essential information of adult ACLS. They demonstrate the integration of the steps of BLS, early defibrillation, and ACLS.

The ILCOR algorithm (Figure 1) shows how simply the overall approach can be presented, with minimum elaboration of separate steps. The Comprehensive ECC Algorithm (Figure 2) provides more details, particularly to support the AHA teaching approach based on the Primary and Secondary ABCD Surveys. Both algorithms depict many of the concepts and interventions that are new since 1992.

Notes to the ILCOR Universal/International ACLS Algorithm

1 (Figure 1)
BLS algorithm. The simple instruction “BLS algorithm” directs the rescuers to start the 6 basic steps of the international BLS algorithm:

1. Check responsiveness
2. Open the airway
3. Check breathing
4. Give 2 effective breaths
5. Assess circulation
6. Compress chest (no signs of circulation detected)

Note that step 6 does not use the term “pulse.” In their 1998 BLS guidelines, the European Resuscitation Council and several ILCOR councils dropped a specific reference in their algorithms to “check the carotid pulse.” They replaced the pulse check with a direction to “check for signs of circulation,” namely, “look for any movement, including swallowing or breathing (more than an occasional gasp).” Their guidelines instruct rescuers to “check for the carotid pulse” as one of the “signs of circulation,” but the pulse check does not receive the prominent emphasis that comes from inclusion in the algorithm. By 2000 many locations had confirmed the success of this European approach. Additional evidence had accumulated that the pulse check was not a good diagnostic test for the presence or absence of a beating heart. After international panels of experts reviewed the evidence at the Guidelines 2000 Conference, they also endorsed the approach of omitting the pulse check for lay responders from the International Guidelines 2000.

2 (Figure 1)
Attach defibrillator/monitor; assess rhythm. Once the responders start the BLS algorithm, they are directed to attach the defibrillator/monitor and assess the rhythm.

3 (Figure 1)
VF/pulseless VT. If they are using a conventional defibrillator and the monitor displays VF, the rescuers attempt defibrillation, up to 3 times as necessary. If using an AED, the rescuers follow the signal and voice prompts of the device, attempting defibrillation with up to 3 shocks. After 3 shocks they should immediately resume CPR for at least 1 minute. At the end of the minute, they should repeat rhythm assessment and shock when appropriate.

4 (Figure 1)
Non-VF rhythm. If the conventional defibrillator/monitor displays a non-VF tracing or the AED signals “no shock indicated,” the responders should immediately check the pulse to determine whether the nonshockable rhythm is producing a spontaneous circulation. If not, then start CPR; continue CPR for approximately 3 minutes. With a non-VF rhythm the rescuer needs to return and recheck the rhythm for recurrent VF or for spontaneous return of an organized rhythm in a beating heart. At this point the algorithm enters the central column of comments.

5 (Figure 1)
During CPR: tracheal tube placement; IV access. In this period the rescuers have many tasks to accomplish. The central column includes the major interventions of ACLS: placing and confirming a tracheal tube, starting an IV, giving appropriate medications for the rhythm, and searching for and correcting reversible causes. Note that the ECC Comprehensive Algorithm (Figure 2) conveys this same approach using the memory aid of the Secondary ABCD
Figure 1. ILCOR Universal/International ACLS Algorithm.
Figure 2. Comprehensive ECC Algorithm.

- Person collapses
- Possible cardiac arrest
- Assess responsiveness

Unresponsive

Begin Primary ABCD Survey (Begin BLS Algorithm)
- Activate emergency response system
- Call for defibrillator
- A Assess breathing (open airway, look, listen, and feel)

Not Breathing

- B Give 2 slow breaths
- C Assess pulse, if no pulse →
- C Start chest compressions
- D Attach monitor/defibrillator when available

No Pulse

- CPR continues
- Assess rhythm

VF/VT

Attempt defibrillation (up to 3 shocks if VF persists)

Non-VF/VT

- Non-VF/VT patients:
  - Epinephrine 1 mg IV, repeat every 3 to 5 minutes
- VF/VT patients:
  - Vasopressin 40 U IV, single dose, 1 time only or
  - Epinephrine 1 mg IV, repeat every 3 to 5 minutes (if no response after single dose of vasopressin, may resume epinephrine 1 mg IV push; repeat every 3 to 5 minutes)

Secondary ABCD Survey

- Airway: attempt to place airway device
- Breathing: confirm and secure airway device, ventilation, oxygenation
- Circulation: gain intravenous access; give adrenergic agent; consider → antiarythmics, buffer agents, pacing

Non-VF/VT (asystole or PEA)

CPR for 1 minute

CPR up to 3 minutes

4,5

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Therefore, there is no critical need to separate the subjects into VF, pulseless VT, PEA, or asystole. Both rhythms have a “differential diagnosis” in terms of what entities can produce a PEA and an asystolic rhythm. Responders must aggressively evaluate PEA victims to discover a potential reversible cause. There is a narrow diagnostic interval of just a few minutes at the discovery of PEA. Asystole, on the other hand, is rarely salvaged unless a reversible cause (eg, severe hyperkalemia, overdose of phenothiazine) is found. Only occasionally does asystole respond to epinephrine in higher doses, atropine, or pacing, because the patient is simply destined to die, given the nature of the original precipitating event.

The only distinguishing treatment for arrest victims is that rescuers treat VF/VT patients with defibrillatory shocks.

Both the ILCOR Universal Algorithm and the Comprehensive ECC Algorithm (Figure 2) convey the concept that all cardiac arrest victims are in 1 of 2 “rhythms”: VF/VT rhythms and non-VF rhythms.

- Non-VF comprises asystole and PEA, which are treated alike.
- Therefore, there is no critical need to separate the subjects into VF, pulseless VT, PEA, or asystole.

All cardiac arrest victims receive the same 4 treatments

- CPR
- Tracheal intubation
- Vasopressors
- Antiarrhythmics

End of Algorithm Notes

Notes to the Comprehensive ECC Algorithm
1 (Figure 2) Begin Primary ABCD Survey. Unresponsive; not breathing. Boxes 1 and 2 cover the steps of the BLS Algorithm and cover the Primary ABCD Survey. The survey is a memory aid and conveys no therapeutic value as stated and displayed. The Primary and Secondary ABCD Surveys are simple mnemonics that assist initial learning. They also provide a useful mental “hook” for later review and recall. Listing more details within the algorithm provides easy review of the steps, especially when the learner has not participated routinely in actual resuscitation attempts.

2 (Figure 2) VF/VT: attempt defibrillation (up to 3 shocks if VF persists). Rhythm assessment and continued CPR are at the center of the Comprehensive ECC Algorithm. The metaphor of a clock ticking away for a cardiac arrest victim in VF is overused but accurate. With each minute of persistent VF, the probability of survival declines. Two clocks are racing. One is the clock that measures the 

therapeutic interval (from collapse to arrival of the defibrillator). One is the clock that measures the 

irreversible damage interval (from cessation of blood flow to the brain to the start of permanent, irreversible brain death).

Here is an observation that will put the racing clocks into perspective. Several experts have observed that great amounts of time and money are spent on the development of new defibrillation waveforms, novel antiarrhythmics, innovative vasopressors, and fresh approaches to ventilation and oxygenation. The total combined effect on survival of these interventions is equivalent to nothing more than cutting the interval from collapse to defibrillatory shock by 2 minutes.

3 (Figure 2) Non-VF/VT. The ILCOR recommendation is to consider the non-VF/VT rhythms as one rhythm when the patient is in cardiac arrest. Consider non-VF/VT as either asystole or PEA. The treatment in the algorithm is the same for both: epinephrine, atropine, transcutaneous pacing. Electrical activity on the monitor screen is a more positive rhythm than asystole. Later in this discussion PEA and asystole are presented in much greater detail.

Both rhythms have a “differential diagnosis” in terms of what entities can produce a PEA and an asystolic rhythm. Responders must aggressively evaluate PEA victims to discover a potential reversible cause. There is a narrow diagnostic interval of just a few minutes at the discovery of PEA. Asystole, on the other hand, is rarely salvaged unless a reversible cause (eg, severe hyperkalemia, overdose of phenothiazine) is found. Only occasionally does asystole respond to epinephrine in higher doses, atropine, or pacing, because the patient is simply destined to die, given the nature of the original precipitating event.

4 (Figure 2) Secondary ABCD Survey. Use of a vasopressor: epinephrine for non-VF/VT, vasopressin for refractory VF. This section of the algorithm makes the same points about persistent arrest from VF/VT and non-VF/VT as the ILCOR Universal Algorithm. The ECC Comprehensive Algorithm, however, uses the memory aid of the Secondary ABCD Survey, a device repeated in all the cardiac arrest algorithms. The algorithm notes expand on these concepts.
Primary ABCD Survey
(Begin BLS Algorithm)
Activate emergency response system
Call for defibrillator
A Airway: open airway; assess breathing (open airway, look, listen, feel)
B Breathing: give 2 slow breaths
C CPR: check pulse; if no pulse →
C Start Chest Compressions
D Defibrillator: attach AED or monitor/defibrillator when available

Secondary ABCD Survey
A Intubate as soon as possible
B Confirm tube placement; use 2 methods to confirm
  • Primary physical examination criteria plus
  • Secondary confirmation device (qualitative and quantitative measures of end-tidal CO2)
B Secure tracheal tube
  • Prevent dislodgment; purpose-made tracheal tube holders recommended over tie-and-tape approaches

Potentially reversible causes. Sudden VF/VT arrests are straightforward in their management. Management consists of early defibrillation, which can succeed independently of other interventions and independently of discovery of the cause of the arrhythmia. With non-VF/VT arrest, however, successful restoration of a spontaneous pulse depends almost entirely on recognizing and treating a potentially reversible cause. As an aide mémoire, Figure 1 places the following list, referred to as “the 5 H’s and 5 T’s,” in the algorithm layout:

The “5 H’s”
• Hypovolemia
• Hypoxia
• Hydrogen ion (acidosis)
• Hyperkalemia/hypokalemia and metabolic disorders
• Hypothermia/hyperthermia

The “5 T’s”
• Toxins/tablets (drug overdose, illicit drugs)
• Tamponade, cardiac
• Tension pneumothorax
• Thrombosis, coronary
• Thrombosis, pulmonary

Figure 2, the Comprehensive ECC Algorithm, expands the table of reversible causes by listing possible therapeutic interventions next to each of the potential causes.

Consider: Is one of the following conditions playing a role?
Hypovolemia (volume infusion)
Hypoxia (oxygen, ventilation)
Hydrogen ion—acidosis (buffer, ventilation)
Hyperkalemia (CaCl plus others)
Hypothermia (see Hypothermia Algorithm in Part 8)
“Tablets” (drug overdoses, accidents)

Tamponade, cardiac (pericardiocentesis)
Tension pneumothorax (decompress—needle decompression)
Thrombosis, coronary (fibrinolytics)
Thrombosis, pulmonary (fibrinolytics, surgical evacuation)

End of Algorithm Notes

Newly Recommended Agent:
Vasopressin for VF/VT
People knowledgeable about the ACLS recommendations during the 1990s will immediately notice that the recommendations for the requisite vasoconstrictor, epinephrine, have changed. The first 3 algorithms—the ILCOR Universal Algorithm, the Comprehensive ECC Algorithm, and Ventricular Fibrillation—each contain the same recommendation for vasopressin as an adrenergic agent equivalent to epinephrine for VF/VT cardiac arrest.

This is one of the most important new recommendations in the International Guidelines 2000. Vasopressin, the natural substance antidiuretic hormone, becomes a powerful vasoconstrictor when used at much higher doses than normally present in the body. Vasopressin possesses positive effects that duplicate the positive effects of epinephrine. Vasopressin does not duplicate the adverse effects of epinephrine. (See “Pharmacology II: Agents to Optimize Cardiac Output and Blood Pressure” for more detailed material on vasopressin.)

Vasopressin received a Class IIb recommendation (acceptable, not harmful, supported by fair evidence) from the panel of international experts on adrenergics. Notice that vasopressin is recommended as a single, 1-time dose in humans. Vasopressin requires less frequent administration because the 10- to 20-minute half-life of vasopressin is much greater than the 3- to 5-minute half-life of epinephrine.
**Primary ABCD Survey**

*Focus:* basic CPR and defibrillation

- Check responsiveness
- Activate emergency response system
- Call for defibrillator

**Airway:** open the airway

**Breathing:** provide positive-pressure ventilations

**Circulation:** give chest compressions

**Defibrillation:** assess for and shock VF/pulseless VT, up to 3 times (200 J, 200 to 300 J, 360 J, or equivalent biphasic) if necessary

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**Rhythm after first 3 shocks?**

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**Persistent or recurrent VF/VT**

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**Secondary ABCD Survey**

*Focus:* more advanced assessments and treatments

**Airway:** place airway device as soon as possible

**Breathing:** confirm airway device placement by exam plus confirmation device

**Breathing:** secure airway device; purpose-made tube holders preferred

**Breathing:** confirm effective oxygenation and ventilation

**Circulation:** establish IV access

**Circulation:** identify rhythm → monitor

**Circulation:** administer drugs appropriate for rhythm and condition

**Differential Diagnosis:** search for and treat identified reversible causes

---

- **Epinephrine** 1 mg IV push, repeat every 3 to 5 minutes or

- **Vasopressin** 40 U IV, single dose, 1 time only

---

**Resume attempts to defibrillate**

1 × 360 J (or equivalent biphasic) within 30 to 60 seconds

---

**Consider antiarrhythmics:**

- amiodarone (I lb), lidocaine (Indeterminate),
- magnesium (I lb if hypomagnesemic state),
- procainamide (I lb for intermittent/recurrent VF/VT).

**Consider buffers.**

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**Resume attempts to defibrillate**

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*Figure 3.* Ventricular Fibrillation/Pulseless VT Algorithm.
After the single dose of vasopressin, the algorithms allow a return to epinephrine if there is no clinical response to vasopressin. This return to epinephrine has no specific human evidence to provide support, although at least 1 clinical trial in Europe is under way. In an informal poll of the experts on the adrenergic panel, every person accepted this recommendation to return to epinephrine after 10 to 20 minutes. (The possibility of a second dose of vasopressin in 10 to 20 minutes was discussed and seems rational. However, this was listed as a Class Indeterminate recommendation because we lack research in humans that addresses this question.)

The rather imprecise time range between the dose of vasopressin and the administration of subsequent epinephrine allows flexibility in the decisions about when to give subsequent adrenergics. The dilemma is: give too soon and cause adverse effects from excessive vasopressin; give too late and the chances of a positive outcome vanish.

Primary and Secondary ABCD Surveys

In some locations, particularly in courses for ACLS providers, the learners are taught a memory aid called the Primary and Secondary ABCD Surveys. These 8 steps apply to all cardiovascular-cardiopulmonary emergencies. Course directors crafted the ABCD surveys to help ACLS providers remember the specific action steps. By memorizing the 2 surveys, ACLS students learn specific actions in a specific sequence. The surveys use the familiar mnemonic of the first 4 letters of the alphabet, and they maintain the traditional actions associated with those 4 letters:

- A = Airway
- B = Breathing
- C = Circulation
- D = Defibrillation (or Differential Diagnosis in the Secondary ABCD Survey)

Because repetition is a well-documented aid to learning, the elements of the Primary and Secondary ABCD Surveys are repeated in several other algorithms: VF/VT, PEA, and asystole.

Figure 3, VF/pulseless VT, conveys more details about the Secondary ABCD Survey:

- A = Airway control with endotracheal intubation
- B = Breathing effectively: verify with primary and secondary confirmation of proper tube placement
- C = Circulation, which incorporates vital signs, ECG monitoring, access to the circulation via IV lines, and then administration of rhythm-appropriate medications
- D = Differential Diagnosis

A directive to “consider the differential diagnoses” improves the resuscitation protocols, because this is a recommendation to stop and think: What caused this arrest? With the addition of this step, resuscitation teams will identify more cardiac arrests with reversible causes. Although we lack evidence that supports use of this memory aid, its use has the strong appeal of common sense.

The Secondary ABCD Survey in Figure 2 states perhaps the most important new recommendations for out-of-hospital care providers.

- We make stronger and more explicit recommendations to confirm tracheal tube placement.
- We recommend that resuscitation personnel take specific actions to prevent tube dislodgment after an initial correct placement.

During the years 1999 to 2000, publications about out-of-hospital pediatric resuscitation documented high rates of tube dislodgment. The researchers discovered that on arrival and evaluation in the Emergency Department, 8% to 12% of tracheal tubes were in the esophagus or hypopharynx. Given the study design, researchers were unable to determine whether these possibly lethal mishaps were due to incorrect initial tube placement or dislodgment after placement. This information has heightened concerns that ACLS providers may be committing undetected harm while performing our most critical interventions.

Figure 3: VF/Pulseless VT

Figure 3 covers the treatment of VF/pulseless VT in more depth than Figures 1 and 2. Figure 3 was created as a teaching aid to convey specific details about the Primary and Secondary ABCD Surveys. The treatments outlined in Figures 1, 2, and 3 are identical: CPR, defibrillation if VF/VT, advanced airway control, intravenous access, rhythm-appropriate medications.

Always Assume VF (Figures 1 Through 3)

Note that Figure 1, the ILCOR Universal ACLS Algorithm, Figure 2, the ECC Comprehensive Algorithm, and Figure 3, the Ventricular Fibrillation/Pulseless VT Algorithm, state this precept unequivocally: rescuers must assume that all adult sudden cardiac arrests are caused by VF/pulseless VT. All training efforts therefore place a strong emphasis on immediate recognition and treatment of VF/pulseless VT. Proper treatment with early defibrillatory shocks allows VF/pulseless VT to provide the majority of adult cardiac arrest survivors. Several mature EMS systems, such as Seattle/King County, Washington, USA, have collected data for >25 years. Year after year VF/VT contributes 85% to 95% of the survivors.

Energy Levels for Shock and Defibrillation Waveforms

The appearance of biphasic waveform defibrillators has generated great enthusiasm in the resuscitation community. Reaching EMS organizations in 1996, the first biphasic defibrillator approved for market shocked at only 1 energy level, approximately 170 J. Competitive market forces stirred up considerable controversy over the efficacy of biphasic waveform shocks in general and nonescalating energy levels in particular. This unseemly chapter in the history of medical device manufacturers has been reviewed in detail in a Medical Scientific Statement from the Senior Science Editors and the chairs of the ECC subcommittees. Biphasic waveform defibrillators are conditionally acceptable—regardless...
of initial shock energy level and regardless of the energy level of subsequent shocks (nonescatalizing). The condition that must be met is clinical data that confirms equivalent or superior effectiveness to monophasic defibrillators when used in the same clinical setting. For example, to meet this condition manufacturers cannot compare rescue defibrillatory shocks delivered to a fibrillating heart in the Electro physiology Stimulation Laboratory versus defibrillatory shocks delivered to patients with 12-minute-old VF in the absence of CPR efforts from bystanders. (See “Defibrillation” in Part 6 for more detail on waveforms and energy levels.) The International Guidelines 2000 panel experts, the ILCOR representatives, and other delegates thought that the class of recommendation for biphasic shocks, nonescalating energy levels, should be upgraded from Class IIb in 1998 to Class IIa in 2000.

CPR, VF, and Defibrillation

After 3 unsuccessful attempts to achieve defibrillation, the first 3 algorithms instruct rescuers to provide approximately 1 minute of CPR. This produces some reoxygenation of the blood and some circulation of this blood to the heart and brain. The precise effect of this minute of CPR on refractory VF is unclear.

Stimulated by the 1999 publication of a retrospective analysis of out-of-hospital cardiac arrest data from the Seattle, Washington, EMS system, the Evidence Evaluation Conference (September 1999) included this topic on its agenda. The EMS personnel initially used a protocol in which arriving EMTs attached an AED and analyzed and shocked any VF rhythms as quickly as possible. Later the protocol directed the EMTs to perform 60 to 90 seconds of CPR before attaching the AED and shocking VF. The survival rates to hospital discharge were significantly higher during the period of prescribed preshock CPR. Other experts argued that a fibrillating myocardium suffers unrelenting deterioration as long as VF continues, CPR or no CPR. A minute or so of preshock CPR does not prevent this deterioration. This guideline recommendation was classed as Indeterminate because the quality and amount of evidence, on both sides of the question, were at lower levels: retrospective data (Level 5) and extrapolation of data from other sources (Level 7), particularly animal studies (Level 6).

Diminishing Roles for Drugs in VF Arrest

The ILCOR Universal Algorithm, the Comprehensive ECC Algorithm, and similar comments in Figure 3 relegate adrenergic agents, antiarrhythmic agents, and buffer therapy to secondary roles for both VF and non-VF patients. This secondary role applies to time-honored agents such as epinephrine, lidocaine, procainamide, and buffer agents and to newly available agents such as amiodarone. Meticulous, systematic review reveals that relevant, valid, and credible evidence to confirm a benefit due to these agents simply does not exist. This does not mean that resuscitation drugs were selected capriciously by the pioneers of resuscitation decades ago. They applied common sense, rational conjecture, and extrapolations from animal studies to arrive at the antiarrhythmics used over the past decade. If an agent is shown in animal models to raise the fibrillation threshold and lower the defibrillation threshold, then a reasonable assumption would be that the drug would facilitate defibrillation of the human heart to a perfusing rhythm. This sort of rational conjecture produced the rather eclectic groups of drugs that have stocked resuscitation kits for more than a decade.

In addition, it was not until the 1990s that researchers discovered the dismal truth that antiarrhythmic drugs were acting more like proarrhythmic agents. Drugs given to prevent VFVT arrest appear to generate VFVT arrest. With critical reappraisal these disturbing discoveries undermined the validity and credibility of scores of excellently designed and executed studies. Through the use of critical appraisal, most researchers in this area realized that the only proper evaluation of new resuscitation agents had to be prospective, randomized clinical trials in which the only acceptable control group had to be placebo.

Designs of studies of new drugs versus standard therapy were unacceptable for the obvious reason—if both standard therapy and the new drug made cardiac arrest victims worse, we could never obtain valid results. The adverse effects would not be recognized unless one agent was significantly worse than the other. Ironically, the researchers would conclude that the less worse drug was actually a superior agent of positive benefit to patients. See “Pharmacology I: Agents for Arrhythmias” and “Pharmacology II: Agents to Optimize Cardiac Output and Blood Pressure” for more detailed material that supports these observations.

New Class of Recommendation for Epinephrine and Lidocaine: Indeterminate

An immense amount of animal research and lower-level human research exists on epinephrine in cardiac arrest. These projects are remarkable in the homogeneity of results—the findings are consistently and invariably positive. But almost no valid, consistent, and relevant human evidence exists to support epinephrine over placebo in human cardiac arrest. Clinical researchers have not conducted prospective, placebo-controlled, clinical trials in humans on this topic. Consequently, the international, evidence-based guidelines had to conclude that epinephrine was Class Indeterminate.

Similarly, no study has shown that lidocaine is effective as an agent to use in human arrest from refractory, shock-resistant VF. Our growing awareness of the proarrrhythmic effects of antiarrhythmics now requires that researchers evaluate lidocaine and other antiarrhythmics against placebo, and not against some other antiarrhythmics. No clinical differences will be observed if 2 antiarrhythmics are equally ineffective or even equally harmful. At this time, therefore, lidocaine receives a Class Indeterminate recommendation.
Notes to Figure 3: VF/Pulseless VT Algorithm
Assume that VF/VT persists after each intervention.

1 (Figure 3)
Defibrillatory shock waveforms
- Use monophasic shocks at listed energy levels (300 J, 300 to 360 J, 360 J) or biphasic shocks at energy levels documented to be clinically equivalent (or superior) to the monophasic shocks.

2 (Figure 3)
2A Confirm tube placement with
- Primary physical examination criteria plus
- Secondary confirmation device (end-tidal CO₂, end-diastolic diameter) (Class IIa)

2B Secure tracheal tube
- To prevent dislodgment, especially in patients at risk for movement, use purpose-made (commercially available) tracheal tube holders, which are superior to tie-and-tape methods (Class IIb)
- Consider cervical collar and backboard for transport (Class Indeterminate)
- Consider continuous, quantitative end-tidal CO₂ monitor (Class IIa)

2C Confirm oxygenation and ventilation with
- End-tidal CO₂ monitor and
- Oxygen saturation monitor

3 (Figure 3)
3A Epinephrine (Class Indeterminate) 1 mg IV push every 3 to 5 minutes. If this fails, higher doses of epinephrine (up to 0.2 mg/kg) are acceptable but not recommended (there is growing evidence that it may be harmful).

3B Vasopressin is recommended only for VF/VT; there is no evidence to support its use in asystole or PEA. There is no evidence about the value of repeat vasopressin doses. There is no evidence about the best approach if there is no response after a single bolus of vasopressin. The following Class Indeterminate action is acceptable, but only on the basis of rational conjecture. If there is no response 5 to 10 minutes after a single IV dose of vasopressin, it is acceptable to resume epinephrine 1 mg IV push every 3 to 5 minutes.

4 (Figure 3)
4A Antiarrhythmics are indeterminate or Class IIb: acceptable; only fair evidence supports possible benefit of antiarrhythmics for shock-refractory VF/VT.
- Amiodarone (Class IIb) 300 mg IV push (cardiac arrest dose). If VF/pulseless VT recurs, consider administration of a second dose of 150 mg IV. Maximum cumulative dose: 2.2 g over 24 hours.
- Lidocaine (Class Indeterminate) 1.0 to 1.5 mg/kg IV push. Consider repeat in 3 to 5 minutes to a maximum cumulative dose of 3 mg/kg. A single dose of 1.5 mg/kg in cardiac arrest is acceptable.
- Magnesium sulfate 1 to 2 g IV in polymorphic VT (torsades de pointes) and suspected hypomagnesemic state.
- Procainamide 30 mg/min in refractory VF (maximum total dose: 17 mg/kg) is acceptable but not recommended because prolonged administration time is unsuitable for cardiac arrest.

4B Sodium bicarbonate 1 mEq/kg IV is indicated for several conditions known to provoke sudden cardiac arrest. See Notes in the Asystole and PEA Algorithms for details.

5 (Figure 3)
Resume defibrillation attempts: use 360-J (or equivalent biphasic) shocks after each medication or after each minute of CPR. Acceptable patterns: CPR-drug-shock (repeat) or CPR-drug-shock-shock-shock (repeat).

Figure 4: Pulseless Electrical Activity
The absence of a detectable pulse and the presence of some type of electrical activity other than VT or VF defines this group of arrhythmias. When electrical activity is organized and no pulse is detectable, clinicians traditionally have used the term electro-mechanical dissociation (EMD). This term, however, is too specific and narrow. Strictly speaking, EMD means that organized electrical depolarization occurs throughout the myocardium, but no synchronous shortening of the myocardial fiber occurs and mechanical contractions are absent.

In the early 1990s the international resuscitation community began to adopt the summary term pulseless electrical activity (PEA). PEA would more accurately embrace a heterogeneous group of rhythms that includes pseudo-EMD, idioventricular rhythms, ventricular escape rhythms, postdefibrillation idioventricular rhythms, and bradyasystolic rhythms. Additional research with cardiac ultrasonography and indwelling pressure catheters has confirmed that often a pulseless patient with electrical activity also has associated mechanical contractions. These contractions are too weak to produce a blood pressure detectable by the usual methods of palpation or sphygmomanometry. Of utmost importance, ACLS providers must know that PEA is often associated with specific clinical states that can be reversed when identified early and treated appropriately.

Notes to Figure 4: Pulseless Electrical Activity
Both VF/VT and PEA are “rhythms of survival.” People in VF/VT can be resuscitated by timely arrival of a defibrillator, and people in PEA can be resuscitated if a reversible cause of PEA is identified and treated appropriately. The PEA algorithm puts great emphasis on searching for specific, reversible causes of PEA. The algorithm features a table of the top 10 causes of PEA, arranged as the “5 H’s and 5 T’s.” If reversible causes are not considered, rescuers will have little chance of recognition and successful treatment. Sodium bicarbonate provides a good example of how the cause of the PEA relates to the therapy. Sodium bicarbonate can vary between being a Class I intervention and being a Class III intervention, depending on the cause.
Figure 4. Pulseless Electrical Activity Algorithm.

**Pulseless Electrical Activity**
(PEA = rhythm on monitor, without detectable pulse)

**Primary ABCD Survey**
Focus: basic CPR and defibrillation
- Check responsiveness
- Activate emergency response system
- Call for defibrillator
A Airway: open the airway
B Breathing: provide positive-pressure ventilations
C Circulation: give chest compressions
D Defibrillation: assess for and shock VF/pulseless VT

**Secondary ABCD Survey**
Focus: more advanced assessments and treatments
A Airway: place airway device as soon as possible
B Breathing: confirm airway device placement by exam plus confirmation device
B Breathing: secure airway device; purpose-made tube holders preferred
B Breathing: confirm effective oxygenation and ventilation
C Circulation: establish IV access
C Circulation: identify rhythm → monitor
C Circulation: administer drugs appropriate for rhythm and condition
C Circulation: assess for occult blood flow ("pseudo-EMT")
D Differential Diagnosis: search for and treat identified reversible causes

**Review for most frequent causes**
- Hypovolemia
- Hypoxia
- Hydrogen ion — acidosis
- Hyper-/hypokalemia
- Hypothermia
- "Tablets" (drug OD, accidents)
- Tamponade, cardiac
- Tension pneumothorax
- Thrombosis, coronary (ACS)
- Thrombosis, pulmonary (embolism)

**Epinephrine** 1 mg IV push, repeat every 3 to 5 minutes

**Atropine** 1 mg IV (if PEA rate is slow), repeat every 3 to 5 minutes as needed, to a total dose of 0.04 mg/kg
1 (Figure 4)
Sodium bicarbonate 1 mEq/kg is used as follows:
- If patient has known, preexisting hyperkalemia
- If known, preexisting bicarbonate-responsive acidosis
- To alkalinate urine in aspirin or other drug overdoses

Class I (acceptable, supported by definitive evidence)
- If patient has known, preexisting hyperkalemia
- In tricyclic antidepressant overdose
- To alkalinate urine in aspirin or other drug overdoses

Class IIa (acceptable, good evidence supports)
- If known, preexisting bicarbonate-responsive acidosis
- In tricyclic antidepressant overdose

Class IIb (acceptable, only fair evidence provides support)
- In intubated and ventilated patients with long arrest interval
- On return of circulation, after long arrest interval

May be harmful (Class III) in hypercarbic acidosis

2 (Figure 4)
Epinephrine: recommended dose is 1 mg IV push every 3 to 5 minutes (Class Indeterminate).
- If this approach fails, higher doses of epinephrine (up to 0.2 mg/kg) may be used but are not recommended.
- (Although one dose of vasopressin is acceptable for persistent or shock-refractory VF, we currently lack evidence to support routine use of vasopressin in victims of PEA or asystole.)

3 (Figure 4)
Atropine: the shorter atropine dose interval (every 3 to 5 minutes) is possibly helpful in cardiac arrest.
- Atropine 1 mg IV if electrical activity is slow (absolute bradycardia=rate <60 bpm or
- Relatively slow (relative bradycardia=rate less than expected, relative to underlying condition)

End of Algorithm Notes

Other observed pulseless cardiac arrest arrhythmias are those in which the electrical activity (QRS complex) is wide versus narrow and fast versus slow. Most clinical studies have observed poor survival rates from PEA that is wide-complex and slow. These rhythms often indicate malfunction of the myocardium or the cardiac conduction system, such as occurs with massive AMI. These rhythms can represent the last electrical activity of a dying myocardium, or they may indicate specific critical rhythm disturbances. For example, severe hyperkalemia, hypothermia, hypoxia, preexisting acidosis, and a large variety of drug overdoses can be wide-complex PEs. Overdoses of tricyclic antidepressants, β-blockers, calcium channel blockers, and digitalis will produce a slow, wide-complex PEA.

In contrast, a fast, narrow-complex PEA indicates a relatively normal heart responding exactly as it should for severe hypovolemia, infections, pulmonary emboli, or cardiac tamponade. These conditions have specific interventions.

The major action to take for a cardiac arrest victim in PEA is to search for possible causes. These rhythms are often a response to a specific condition, and helpful clues can appear if one simply looks at the electrical activity width and rate.

Hypovolemia is the most common cause of electrical activity without measurable blood pressure. Through prompt recognition and appropriate therapy, the many causes of hypovolemia can often be corrected, including hypovolemia from hemorrhage or from anaphylaxis-induced vasodilation. Other causes of PEA are cardiac tamponade, tension pneumothorax, and massive pulmonary embolism.

Non-specific therapeutic interventions for PEA include epinephrine and (if the rate is slow) atropine, administered as presented in Figure 4. In addition, personnel should provide proper airway management and aggressive hyperventilation because hypoventilation and hypoxemia are frequent causes of PEA. Clinicians can give a fluid challenge because the PEA may be due to hypovolemia.

Immediate assessment of blood flow by Doppler ultrasound may reveal an actively contracting heart and significant blood flow. The blood pressure and flow, however, may fall below the threshold of detection by simple arterial palpation. Any PEA patient with a Doppler-detectable blood flow should be aggressively treated. These patients need volume expansion, norepinephrine, dopamine, or some combination of the three. They might benefit from early transcutaneous pacing because a healthy myocardium exists and only a temporarily disturbed cardiac conduction system stands between survival and death. Although in general PEA has poor outcomes, reversible causes should always be targeted and never missed when present.

Figure 5: Asystole: The Silent Heart Algorithm

Patients in cardiac arrest discovered on the defibrillator’s monitor screen to be in asystole have a dismal rate of survival—usually as low as 1 or 2 people out of 100 cardiac arrests. During a resuscitation attempt, brief periods of an organized complex may appear on the monitor screen, but spontaneous circulation rarely emerges. As with PEA, the only hope for resuscitation of a person in asystole is to identify and treat a reversible cause.

Figure 5, the Asystole Algorithm, outlines an approach much more in keeping with our current understanding of the issues surrounding asystole. The Asystole Algorithm focuses on “not starting” and “when to stop.” With prolonged, refractory asystole the patient is making the transition from life to death. ACLS providers who try to make that transition as sensitive and dignified as possible serve their patients well.

Notes to Figure 5: Asystole

1 (Figure 5)
Scene Survey: DNAR patient?
- Any clinical indicators that resuscitation attempts are not indicated, eg, signs of death? If Yes: do not start/attempt resuscitation.
Asystole

Primary ABCD Survey
Focus: basic CPR and defibrillation
- Check responsiveness
- Activate emergency response system
- Call for defibrillator
A Airway: open the airway
B Breathing: provide positive-pressure ventilations
C Circulation: give chest compressions
C Confirm true asystole
D Defibrillation: assess for VF/pulseless VT; shock if indicated

Rapid scene survey: any evidence personnel should not attempt resuscitation?

Secondary ABCD Survey
Focus: more advanced assessments and treatments
A Airway: place airway device as soon as possible
B Breathing: confirm airway device placement by exam plus confirmation device
B Breathing: secure airway device; purpose-made tube holders preferred
B Breathing: confirm effective oxygenation and ventilation
C Circulation: confirm true asystole
C Circulation: establish IV access
C Circulation: identify rhythm → monitor
C Circulation: give medications appropriate for rhythm and condition
D Differential Diagnosis: search for and treat identified reversible causes

Transcutaneous pacing
If considered, perform immediately

Epinephrine 1 mg IV push, repeat every 3 to 5 minutes

Atropine 1 mg IV, repeat every 3 to 5 minutes up to a total of 0.04 mg/kg

Asystole persists
Withhold or cease resuscitation efforts?
- Consider quality of resuscitation?
- Atypical clinical features present?
- Support for cease-efforts protocols in place?

Figure 5. Asystole: The Silent Heart Algorithm.
2 (Figure 5)
**Confirm true asystole**
- Check lead and cable connections
- Monitor power on?
- Monitor gain up?
- Verify asystole in another lead?

3 (Figure 5)
**Sodium bicarbonate** 1 mEq/kg
- Indications for use include the following: overdose of tricyclic antidepressants; to alkalinize urine in overdoses; patients with tracheal intubation plus long arrest intervals; on return of spontaneous circulation if there is a long arrest interval.
- Ineffective or harmful in hypercarbic acidosis.

4 (Figure 5)
**Transcutaneous pacing**
- To be effective, must be performed early, combined with drug therapy. Evidence does not support routine use of transcutaneous pacing for asystole.

5 (Figure 5)
**Epinephrine**
- Recommended dose is 1 mg IV push every 3 to 5 minutes. If this approach fails, higher doses of epinephrine (up to 0.2 mg/kg) may be used but are not recommended.
- We currently lack evidence to support routine use of vasopressin in treatment of asystole.

6 (Figure 5)
**Atropine**
- Use the shorter dosing interval (every 3 to 5 minutes) in asystolic arrest.

7 (Figure 5)
**Review the quality of the resuscitation attempt**
- Was there an adequate trial of BLS? of ACLS? Has the team done the following:
  - Achieved tracheal intubation?
  - Performed effective ventilation?
  - Shocked VF if present?
  - Obtained IV access?
  - Given epinephrine IV? atropine IV?
  - Ruled out or corrected reversible causes?
  - Continuously documented asystole >5 to 10 minutes after all of the above have been accomplished?

8 (Figure 5)
**Reviewed for atypical clinical features?**
- Not a victim of drowning or hypothermia?
- No reversible therapeutic or illicit drug overdose?
  —“Yes” to the questions in Notes 7 and 8 means the resuscitation team complies with recommended criteria to terminate resuscitative efforts where the patient lies (Class IIa)
  —If the response team and patient meet the above criteria, then withhold urgent field-to-hospital transport with continuing CPR=Class III (harmful; no benefit)

9 (Figure 5)
**Withholding or stopping resuscitative efforts out-of-hospital**

If criteria in 7 and 8 are fulfilled:
- Field personnel, in jurisdictions where authorized, should start protocols to cease resuscitative efforts or to pronounce death outside the hospital (Class IIa).
- In most US settings, the medical control official must give direct voice-to-voice or on-scene authorization.
- Advance planning for these protocols must occur. The planning should include specific directions for
  —Leaving the body at scene
  —Death certification
  —Transfer to funeral service
  —On-scene family advocate
  —Religious or nondenominational counseling

**End of Algorithm Notes**

Asystole most often represents a confirmation of death rather than a “rhythm” to be treated. Team leaders can cease efforts to resuscitate the patient from confirmed and persistent asystole when the resuscitation team has done the following:

- Provided suitable basic CPR
- Eliminated VF
- Achieved successful tracheal intubation with primary and secondary confirmation of tube placement
- Confirmed throughout the efforts that the tube was secure and had not been dislodged
- Monitored oxygen saturation and end-tidal CO₂ to ensure that the best possible oxygenation and ventilation were achieved
- Established successful IV access
- Maintained these interventions for ≥10 minutes, during which time the confirmed rhythm was asystole
- Administered all rhythm-appropriate medications
- Updated waiting family members, spouses, or available friends about the severity of the patient’s condition and lack of response to interventions
- Discussed the concept of programs to support family presence during resuscitative attempts and offered that option to appropriate family members. Note that family presence at resuscitative efforts is not a spur-of-the-moment offer, extended or not extended at the whim of supervising physicians. Rather, family presence at resuscitative efforts requires a formal program, with advance planning, assigned roles, and even rehearsals.

**When to Stop?**
Is it possible to state a specific time interval beyond which rescuers have never resuscitated patients? Does every resuscitation attempt have to continue for that length of time to guarantee that every salvageable person will be identified and saved? As outlined in the algorithm notes, the resuscitation team must make a conscientious and competent effort to give patients “a trial of CPR and ACLS,” provided that the person had not expressed a
decision to forego resuscitative efforts. The final decision to stop efforts can never be as simple as an isolated time interval, but clinical judgment and a respect for human dignity must enter the decision making. Many people among the resuscitation community strongly believe that we have erred greatly in the tendency to try prolonged, excessive resuscitative efforts.

Emergency medical response systems should not require the field personnel to transport every victim of cardiac arrest back to a hospital or Emergency Department (ED). In European countries most out-of-hospital ALS care is provided by medical doctors, so decisions about stopping CPR, transportation back to the ED, and pronouncing death are handled by an authorized medical doctor in the field. Transportation with continuing CPR is justified if there are interventions available in the ED that cannot be performed in the field (such as central core rewarming equipment) or field interventions (such as tracheal intubation) that were unsuccessful in the field.

In the United States, outdated concepts of EMS care can linger for years. For example, many systems still dictate the practice of “scoop and run” on all major medical patients, not just major trauma. For nontraumatic cardiac arrest solid evidence confirms that ACLS care in the ED offers no advantage over ACLS care in the field. Stated succinctly—if ACLS care in the field cannot resuscitate the victim, neither will ED care. Civil rules, administrative concerns, medical insurance requirements, and even reimbursement enhancement have frequently led to requirements to transport all cardiac arrest victims back to a hospital or ED. If these are unselective requirements, they are inappropriate, futile, and ethically unacceptable. There should be no requirements for ambulance transport of all patients who suffer an out-of-hospital cardiac arrest. This is especially true when the patient is pulseless and CPR is continued during transport. Researchers and EMS experts continue to publish observational studies on this practice of transporting all field resuscitations back to an ED, most often for pronouncement of death. To have the resuscitation team succeed with one of these victims and then have the victim survive to hospital discharge is extremely rare—usually <1%.

Likewise, it is inappropriate for clinicians to apply routine “stopping rules” without thinking about the particular situation. “Part 2: Ethical Aspects of ECC and CPR” provides a more detailed discussion of these issues. Cessation of efforts in the prehospital setting, following system-specific criteria and under direct medical control, should be standard practice in all EMS systems.

- Symptoms (chest pain, shortness of breath, decreased level of consciousness)
- Signs (low blood pressure, shock, pulmonary congestion, congestive heart failure)

3 (Figure 6)
If the patient is symptomatic, do not delay transcutaneous pacing while awaiting IV access or for atropine to take effect.

4 (Figure 6)
Denervated transplanted hearts will not respond to atropine. Go at once to pacing, catecholamine infusion, or both.

5 (Figure 6)
Atropine should be given in repeat doses every 3 to 5 minutes up to a total of 0.03 to 0.04 mg/kg. Use the shorter dosing interval (3 minutes) in severe clinical conditions.

6 (Figure 6)
Never treat the combination of third-degree heart block and ventricular escape beats with lidocaine (or any agent that suppresses ventricular escape rhythms).

7 (Figure 6)
Verify patient tolerance and mechanical capture. Use analgesia and sedation as needed.

End of Algorithm Notes

Transcutaneous pacing is a Class I intervention for all symptomatic bradycardias. If clinicians are concerned about the use of atropine in higher-level blocks, they should remember that transcutaneous pacing is always appropriate, although not as readily available as atropine. If the bradycardia is severe and the clinical condition is unstable, implement transcutaneous pacing immediately.

There are several other cautions to remember about treatment of symptomatic bradycardias. Lidocaine may be lethal if the bradycardia is a ventricular escape rhythm and unwary clinicians think they are treating preventricular contractions or slow VT. In addition, transcutaneous pacing can be painful and may fail to produce effective mechanical contractions. Sometimes the patient’s “symptom” is not due to the bradycardia. For example, hypotension, associated with bradycardia, may be due to myocardial dysfunction or hypovolemia rather than to conducting system or autonomic problems.

Figure 6 lists interventions in a sequence based on the assumption of worsening clinical severity. Give patients who are “precardiac arrest,” or moving in that direction, multiple interventions in rapid sequence. Begin preparations for pacing, IV atropine, and administration of an epinephrine infusion. If the patient displays only mild problems due to the bradycardia, then atropine 0.5 to 1.0 mg IV can be given in a repeat dose every 3 to 5 minutes, to a total of 0.03 mg/kg. (For severe bradycardia or asystole, a maximum dose of 0.04 mg/kg is advisable.) Selection of the dosing interval (3 to 5 minutes) requires judgment about the severity of the patient’s symptoms. The provider should repeat atropine at shorter intervals for more distressed patients. Dopamine (at rates of 2

Figure 6: Bradycardia

Notes to Figure 6: Bradycardia

1 (Figure 6)
If the patient has serious signs or symptoms, make sure they are related to the slow rate.

2 (Figure 6)
Clinical manifestations include
Bradycardia
- **Slow** (absolute bradycardia = rate <60 bpm)
- **Relatively slow** (rate less than expected relative to underlying condition or cause)

**Primary ABCD Survey**
- Assess ABCs
- Secure airway noninvasively
- Ensure monitor/defibrillator is available

**Secondary ABCD Survey**
- Assess secondary ABCs (invasive airway management needed?)
- Oxygen–IV access–monitor–fluids
- Vital signs, pulse oximeter, monitor BP
- Obtain and review 12-lead ECG
- Obtain and review portable chest x-ray
- Problem-focused history
- Problem-focused physical examination
- Consider causes (differential diagnoses)

**Serious signs or symptoms?**
Due to the bradycardia?

---

No

- **Type II second-degree AV block**
- **Third-degree AV block?**

---

No

- Observe

---

Yes

- Intervention sequence
  - **Atropine** 0.5 to 1.0 mg
  - **Transcutaneous pacing** if available
  - **Dopamine** 5 to 20 μg/kg per minute
  - **Epinephrine** 2 to 10 μg/min

---

Yes

- Prepare for transvenous pacer
- If symptoms develop, use transcutaneous pacemaker until transvenous pacer placed

---

**Figure 6. Bradycardia Algorithm.**
to 5 \( \mu g/kg \) per minute) can be added and increased quickly to 5 to 20 \( \mu g/kg \) per minute if low blood pressure is associated with the bradycardia. If the patient displays severe symptoms, clinicians can go directly to an epinephrine infusion.

Transcutaneous pacing should be initiated quickly in patients who do not respond to atropine or who are severely symptomatic, especially when the block is at or below the His-Purkinje level. Newer defibrillator/monitors have the capability to perform transcutaneous pacing. This intervention, unlike insertion of transvenous pacemakers, is available to and can be performed by almost all ECC providers. This gives transcutaneous pacing enormous advantages over transvenous pacing because transcutaneous pacing can be started quickly and conveniently at the bedside.

Reference
7D: The Tachycardia Algorithms

Major New Concepts for the International Guidelines 2000

At the 1999 Evidence Evaluation Conference and the Guidelines 2000 Conference, experienced electrophysiologists, arrhythmia experts, and clinical cardiologists led the evidence review and discussions on the tachycardias (Figures 7, 8, and 9). They brought their expertise, their experience using new antiarrhythmics, and their knowledge of the incumbent tachycardia algorithm. The evidence reviews and discussions contributed many insights, revisions, and new medications. The tachycardia algorithm from the early 1990s forced an immediate decision in regard to “stable versus unstable.” That same emphasis remains in 2000—the most important clinical decision to make when a rapid heart rate is noted is whether the patient is also experiencing signs and symptoms due to the rapid heart rate. In such situations the universal recommendation is immediate cardioversion rather than a trial of antiarrhythmics (see Figure 10, Electrical Cardioversion Algorithm).

For tachycardic patients not in need of immediate cardioversion, the International Guidelines 2000 place much more emphasis on 2 themes not previously highlighted:

- Making a specific rhythm diagnosis
- Recognizing those tachycardic patients who have significantly impaired cardiac function (ejection fraction <40%; overt signs of heart failure)

Emphasis on these themes has resulted in not just 1 complicated algorithm for tachycardias but 3 new algorithms and 1 table. The Guidelines 2000 Conference experts, clinicians, and teachers resisted any activity that would make ACLS training and learning more complicated. On learning of the background rationale, however, clinical leaders in resuscitation have accepted the need for more expansive algorithms, in return for providing better and safer acute care.

Antiarrhythmics or Proarrhythmics?

The other new concept that dominates the 2000 approach to tachycardias comes from continued evidence that antiarrhythmics are just as likely to be proarrhythmic agents as they are to be antiarrhythmic agents. Their tendency to induce arrhythmias becomes particularly acute in damaged or impaired hearts. In damaged hearts normal functional myocardium is interlaced with scarred and damaged tissue. These areas become the source of reentry arrhythmias, irritable foci, and blocked conduction.

Regardless of their Vaughn-Williams classification, all antiarrhythmics are capable of a proarrhythmic effect. When a second antiarrhythmic is added to this milieu the negative consequences escalate exponentially. Consequently—with rare exceptions—the International Guidelines 2000 recommend 1 and only 1 antiarrhythmic per patient. This should lead to far fewer events in which >1 antiarrhythmic causes significant worsening of the patient’s condition and a much lower threshold to cardiovert patients nonurgently, before they become significantly more symptomatic.

Specific Rhythm Diagnosis

Adenosine, first approved for marketing shortly before the publication of the 1992 guidelines, has been a highly successful agent for supraventricular arrhythmias. Teaching has emphasized its safety and its diagnostic capabilities. If a patient’s monitor displayed a wide-complex tachycardia, clinicians became almost cavalier about pushing in higher and higher amounts of adenosine to see whether the rhythm converted out of a presumed supraventricular tachycardia with aberrancy. Most such patients are in VT with wide complexes. Faced with persistent wide-complex tachycardia, after 3 or 4 boluses of adenosine the clinician faced a dawning realization that treatment with adenosine or multiple calcium channel blockers for 30 to 45 minutes of persistent tachycardia was not helpful. This approach also exposes the patient to unpleasant side affects of adenosine, the possibility of worse rhythms, and a destabilization of heart rate and blood pressure. Consequently the International Guidelines 2000 attempt to avoid the simplistic approach of overuse of adenosine for diagnostic purposes. Instead, the clinicians are expected to devote more attention to making explicit diagnoses, within the scope of their available resources.

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Figure 7: The Tachycardia Overview Algorithm

The classic “chicken or egg” dilemma occurs often in symptomatic tachycardia events. Did the stress and discomfort of acute, severe, substernal chest pain lead to a cardiac response of tachycardia? or did the marginally compromised heart develop ischemia and chest pain as the sequelae of a paroxysmal tachycardia? The patients diagnosed as “unstable” will immediately be treated with urgent, electric, synchronized cardioversion.

Note: The numbers of notes, such as “Note 1,” match numbers in the algorithms.
Figure 7. The Tachycardia Overview Algorithm.
Control of Rate and Rhythm (Continued From Tachycardia Overview)

<table>
<thead>
<tr>
<th>Atrial fibrillation/atrial flutter with</th>
<th>1. Control Rate</th>
<th>2. Convert Rhythm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal cardiac function</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal cardiac function</td>
<td>Note: If AF &gt;48 hours' duration, use agents to convert rhythm with extreme caution in patients not receiving adequate anticoagulation because of possible embolic complications. Use only 1 of the following agents (see note below): • Calcium channel blockers (Class Ia) • β-Blockers (Class I) • For additional drugs that are Class Ila recommendations, see Guidelines or ACLS text</td>
<td>Consider • DC cardioversion Use only 1 of the following agents (see note below): • Amiodarone (Class Ila) • Ibutilide (Class Ila) • Flecaïnine (Class Ila) • Propafenone (Class Ila) • Procainamide (Class Ila) • For additional drugs that are Class Iib recommendations, see Guidelines or ACLS text</td>
</tr>
<tr>
<td>Impaired heart (EF &lt;40% or CHF)</td>
<td>(Does not apply)</td>
<td></td>
</tr>
</tbody>
</table>

### Note to Figure 7: The Tachycardia Overview Algorithm
Unstable condition must be related to the tachycardia. Signs and symptoms may include chest pain, shortness of breath, decreased level of consciousness, low blood pressure, shock, pulmonary congestion, congestive heart failure, and AMI.

### End of Algorithm Note

The Tachycardia Overview Algorithm (Figure 7) divides tachycardias into 4 diagnostic categories. (In the algorithm the 4 columns have a number at the top to better orient the reader):

1. Atrial fibrillation/flutter
2. Narrow-complex tachycardias
3. Wide-complex tachycardias of unknown type
4. Stable monomorphic and polymorphic tachycardia

### 1. Atrial Fibrillation/atrial Flutter (Column 1)

Figure 7 reminds the ACLS provider of the focus for evaluation:

1. Is the patient clinically unstable?
2. Is cardiac function impaired?
3. Is Wolff-Parkinson-White syndrome present?
4. Was the onset of the atrial fibrillation or atrial flutter clearly recognized by the patient? Has the atrial fibrillation/flutter been present for more than or less than 48 hours?

The treatment focus for atrial fibrillation/flutter is on 4 areas as well:

1. Unstable versus stable? Treat urgently.
2. Control rate with agents that reduce the rate of conduction across the AV node.
3. Convert the rhythm with either medications or cardioversion when indications are present and urgent.
4. Provide anticoagulation if indicated.

See “Section 5: Pharmacology I” and “Section 6: Pharmacology II” for more detailed presentations on these topics.

The table accompanying Figure 7 provides treatment details for atrial fibrillation and flutter. The table displays the
## Control of Rate and Rhythm (Continued From Tachycardia Overview)

<table>
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<td></td>
<td></td>
</tr>
<tr>
<td>WPW</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**WPW**

### Note:
- For AF >48 hours' duration, use agents to convert rhythm with extreme caution in patients not receiving adequate anticoagulation because of possible embolic complications.
- **Primary anti-arrhythmic agents**
  - Use only 1 of the following agents (see note below):
    - Amiodarone (Class IIb)
    - Flecainide (Class IIb)
    - Propranolol (Class IIb)
    - Sotalol (Class IIb)
- **Class III (can be harmful)**
  - Adenosine
  - β-blockers
  - Calcium blockers
  - Digoxin

**Duration <48 Hours**

- DC cardioversion
- Amiodarone (Class IIb)

**Duration >48 Hours or Unknown**

- **Primary anti-arrhythmic agents**
  - Use only 1 of the following agents (see note below):
    - Amiodarone (Class IIb)
    - Flecainide (Class IIb)
    - Procainamide (Class IIb)
    - Propafenone (Class IIb)
    - Sotalol (Class IIb)
- **Class III (can be harmful)**
  - Adenosine
  - β-blockers
  - Calcium blockers
  - Digoxin

**Note:** Occasionally 2 of the named antiarrhythmic agents may be used, but use of these agents in combination may have proarrhythmic potential. The classes listed represent a Class of Recommendation rather than the Vaughan-Williams classification of antiarrhythmics.

---

### Major questions to ask and factors to consider when formulating a treatment plan for atrial fibrillation/flutter:
- What is the cardiac status? normal? impaired?
- Does the person have Wolff-Parkinson-White syndrome?
- What is the duration of the atrial fibrillation? Can you date and time the onset unequivocally? Is the duration less than or more than 48 hours? Is anticoagulation indicated?
- Electrical cardioversion?
- Would pharmacological conversion pose higher risk of emboli?
- Is the rate too high?

### 2. Tachycardia (Atrial Fibrillation and Flutter)

See the table accompanying Figure 7.

---

**Narrow-Complex Tachycardias**

The ACLS provider needs to move from the end of column 2, Narrow-Complex Tachycardias (Figure 7), to Figure 8: Narrow-Complex Supraventricular Tachycardia.

Figure 7, under Narrow-Complex Tachycardias, demonstrates the emphasis in 2000 on establishing a specific diagnosis first by close ECG analysis, then by consulting cardiology specialists if available. The consultants may choose to use esophageal-lead ECGs or echocardiograms as well as serial 12-lead ECGs. In narrow-complex, stable supraventricular tachycardias, the algorithm recommends diagnostic use of vagal maneuvers and adenosine. These diagnostic efforts should yield a diagnosis such as PSVT, ectopic atrial tachycardia, or MAT.

The overview algorithm then directs the clinician to Figure 8, Narrow-Complex Supraventricular Tachycardia. Here clinicians make the clinical assessment of cardiac function, with ejection fractions <40% qualifying as enough compromise to alter the therapeutic approach.

**Figure 8: Narrow-Complex Supraventricular Tachycardia**

In general, narrow-complex supraventricular tachycardias can be treated with amiodarone, β-blockers, or calcium channel blockers if cardiac function is preserved. (This list is in alphabetical order and does not imply that 1 of these 3 is better than another.) If cardiac function is compro-
Figure 8. Narrow-Complex Supraventricular Tachycardia Algorithm.

Narrow-Complex Supraventricular Tachycardia, Stable

Attempt therapeutic diagnostic maneuver
- Vagal stimulation
- Adenosine

Junctional tachycardia

Preserved
- No DC cardioversion!
- Amiodarone
- β-Blocker
- Ca²⁺ channel blocker

EF <40%, CHF
- No DC cardioversion!
- Amiodarone

Paroxysmal supraventricular tachycardia

Preserved
- Priority order:
  - Ca²⁺ channel blocker
  - β-Blocker
  - Digoxin
  - DC cardioversion
  - Consider procainamide, amiodarone, sotalol

EF <40%, CHF
- Priority order:
  - No DC cardioversion!
  - Digoxin
  - Amiodarone
  - Diltiazem

Ectopic or multifocal atrial tachycardia

Preserved
- No DC cardioversion!
- Ca²⁺ channel blocker
- β-Blocker
- Amiodarone

EF <40%, CHF
- No DC cardioversion!
- Amiodarone
- Diltiazem
mised, then the drug options narrow to only amiodarone, the agent with the best balance between side effects and effectiveness in heart failure patients. Note the prohibition to DC cardioversion in all patients with impaired cardiac function.

3. Stable Wide-Complex Tachycardias: Unknown Type
Figure 7, Column 3, recommends the same diagnostic/therapeutic approach as recommended for narrow-complex tachycardias. After a series of diagnostic efforts clinicians
Steps for Synchronized Cardioversion

1. Consider sedation.
2. Turn on defibrillator (monophasic or biphasic).
3. Attach monitor leads to the patient ("white to right, red to ribs, what's left over to the left shoulder") and ensure proper display of the patient's rhythm.
4. Engage the synchronization mode by pressing the "sync" control button.
5. Look for markers on R waves indicating sync mode.
6. If necessary, adjust monitor gain until sync markers occur with each R wave.
7. Select appropriate energy level.
8. Position conductor pads on patient (or apply gel to paddles).
10. Announce to team members: "Charging defibrillator—stand clear!"
11. Press "charge" button on apex paddle (right hand).
12. When the defibrillator is charged, begin the final clearing chant. State firmly in a forceful voice the following chant before each shock:
   - "I am going to shock on three. One, I'm clear." (Check to make sure you are clear of contact with the patient, the stretcher, and the equipment.)
   - "Two, you are clear." (Make a visual check to ensure that no one continues to touch the patient or stretcher. In particular, do not forget about the person providing ventilations. That person's hands should not be touching the ventilatory adjuncts, including the tracheal tube!)
   - "Three, everybody's clear." (Check yourself one more time before pressing the "shock" buttons.)
13. Apply 25 lb pressure on both paddles.
14. Press the "discharge" buttons simultaneously.
15. Check the monitor. If tachycardia persists, increase the joules according to the electrical cardioversion algorithm.
16. Reset the sync mode after each synchronized cardioversion because most defibrillators default back to unsynchronized mode. This default allows an immediate defibrillation if the cardioversion produces VF.

Figure 10. Synchronized Cardioversion Algorithm.
should be able to establish into which of 3 arrhythmic categories the wide-complex tachycardia belongs:

1. Narrow-complex tachycardia with aberrancy obscuring the narrow QRS (subsequently these patients are referred to Figure 8: Narrow-Complex Supraventricular Tachycardia)
2. Stable monomorphic or polymorphic VT (subsequently these patients are referred to Figure 9: Stable VT: Monomorphic or Polymorphic)
3. Stable wide-complex tachycardia: if this is the most likely rhythm diagnosis for you and your patient, proceed to evaluate the cardiac functional status. Figure 7 shows that DC cardioversion and amiodarone are recommended treatments for those tachycardic patients who have clinical heart failure. Be careful with adding anything else to the treatment for these patients. For the patient with well-preserved cardiac function, choose either DC cardioversion (Figure 10) or procainamide or amiodarone. Most experts choose DC cardioversion as their first treatment and treatment of choice for all wide-complex tachycardias regardless of cardiac function. That way they can easily add another antiarrhythmic if the cardioversion fails; however, the converse (use cardioversion if antiarrhythmic fails) does not always hold true.

Figure 9: Stable Ventricular Tachycardia: Monomorphic or Polymorphic?

4. Monomorphic and Polymorphic VT
Figure 7, Column 4, directs the clinician to Figure 9 for more details on the treatment of those stable VTs that fall into 2 subsets: monomorphic VT and polymorphic VT. If the polymorphic VT has a prolonged QT interval, it becomes a question of whether the patient is suffering from torsades de pointes. With such an exotic name it is only right that the rhythm of polymorphic VT looks distinctive and unusual, with the entire rhythm tracing resembling an up-and-down, thick-and-thin pattern characterized as a “spindle and node” pattern.

Polymorphic VT merits special attention because it is a common arrest rhythm suffered by drug overdose patients and toxin patients from exposures to nonmedicinal drugs.

Figure 9 provides directions for the immediate treatment of these arrhythmias, with the cardiac function status of primary importance. Notice that amiodarone is becoming the only antiarrhythmic agent acceptable for the elderly and those with progressive decline in cardiac function.

See the callout notes for stable VTs for more details.

Notes to Figure 9: Stable VT: Monomorphic or Polymorphic?

1 (Figure 9)
Monomorphic VF with normal cardiac function
Use just 1 agent (to avoid proarrhythmic effects of combination therapy).

This reduces adverse side effects. Choose 1 agent from these lists:

Top agents
- Procainamide (IIa)
- Sotalol (IIa)

Others acceptable
- Amiodarone (IIb)
- Lidocaine (IIb)

Monomorphic or polymorphic VT with impaired cardiac function
If clinical signs are suggestive of impaired LV function (ejection fraction <40% or congestive heart failure) in either long- or normal-QRS tachycardias, use
- Amiodarone (IIb)
- Lidocaine (IIb)

then use
- Synchronized cardioversion

3 (Figure 9)
Detailed dosing of amiodarone (Class IIb) in patients with impaired cardiac function
- 150 mg IV bolus over 10 minutes (international dose: 5 mg/kg)
- Repeat 150 mg IV (over 10 minutes) every 10 to 15 minutes as needed
- Alternative infusion: 360 mg over 6 hours (1 mg/min over 6 hours), then 540 mg over the remaining 18 hours (0.5 mg/min)
- Maximum total dose: 2.2 g in 24 hours. This means that all doses (including those used in resuscitation) should be added together, so the total cumulative dose per 24 hours is limited to 2.2 g
- See guidelines or ECC Handbook Drug Table

4 (Figure 9)
Detailed dosing of lidocaine (Class Indeterminate) in patients with impaired cardiac function
- 0.5 to 0.75 mg/kg IV push
- Repeat every 5 to 10 minutes
- Then infuse 1 to 4 mg/min
- Maximum total dose: 3 mg/kg (over 1 hour)

5 (Figure 9)
If rhythm is suggestive of torsades de pointes
- Stop/avoid treatments that prolong QT
- Identify and treat abnormal electrolytes

Medications (all Class Indeterminate):
- Magnesium
- Overdrive pacing (with or without β-blocker)
- Isoproterenol (as temporizing measure to overdrive pacing)
- Phenytoin or lidocaine

End of Algorithm Notes