

Cardioversion Past, Present, and Future

Ivan Cakulev, MD; Igor R. Efimov, PhD; Albert L. Waldo, MD

Simplicity is the ultimate sophistication.

—Leonardo da Vinci

Recent years have seen rapid proliferation of ablative and antiarrhythmic therapies for treating various ventricular and supraventricular arrhythmias. Yet cardioversion and defibrillation remain the main modalities to restore normal sinus rhythm. Their simplicity, reliability, safety, and, most important, their efficacy in promptly restoring normal sinus rhythm are unmatched in our current treatment armamentarium.

History

The Early Work

Contemporary cardiology has been significantly affected by the ready availability of this simple method for terminating atrial and ventricular tachyarrhythmias. However, fascination with electricity and its use in biological systems is hardly contemporary. The first capacitor that was able to store electric energy in a glass container was discovered in 1745. It was named the Leyden jar, and its use was shortly thereafter tested in the electrocution of small animals. There is a large body of literature in Italy, France, and England on biological and medical application of electricity dating from the 17th and 18th centuries. Although physicians across Europe started using electricity as an experimental treatment, the earliest recorded scientific approach with the use of electric shocks was that of Peter Abildgaard in 1775.¹ He systematically shocked hens, delivering electric charges in different parts of their body. Electric stimuli applied anywhere across the body of the hen, particularly in the head, could render the animal lifeless, but subsequent shocks delivered to the chest could revive the heart.

Abildgaard was only one of the several scientists who studied the effects of electricity on animals. Some reported similar findings, and others could not reproduce his results. However, Luigi Galvani in 1781 first clearly described the link between electricity and its presence in biological systems.² He was the first to use the term *animal electricity*, coined after his famous experiments in which he caused the legs of a skinned frog to kick when touched with a pair of scissors during an electric storm. The recognition of electricity in living organisms sparked intense interest and excitement

and led to application of electricity to revive the dead. Possibly the first description of successful resuscitation with the use of electric shock was reported by Charles Kite in 1788, when a 3-year-old girl, a victim of a fall, was shocked through the chest by an electric generator and a Leyden jar by a Mr Squires of London.³ A similar report by Fell appeared in *Gentlemen's Magazine* in 1792, with the description of this first prototype of a modern defibrillator (Figure 1).

In 1792, the British scientist James Curry published a review of resuscitation cases and recommended that "moderate shocks be passed through the chest in different directions in order, if possible, to rouse the heart to act."⁴ Several other successful attempts at resuscitation led the Royal Humane Society in England to publish a report in 1802 suggesting the application of electric shock to distinguish "real from apparent death" and praising the potential of electric resuscitation.

Scientists at that time were unaware that, at least in some cases, revival with electricity was perhaps due to successful termination of ventricular fibrillation (VF). Ludwig and Hoffa were the first to describe this arrhythmia in 1849 when they observed bizarre and chaotic action of the ventricles when exposed directly to electric current.⁵ The nature of this arrhythmia was subjected to speculation. Neurogenic theory that explained VF as a consequence of abnormal generation and conduction within the neural network was favored. A French neurophysiologist, Edme Vulpian, coined the term *fibrillation* and first suggested that the heart itself was responsible for originating and sustaining this irregular rhythm that results in mechanical disarray.⁶

In 1889, John McWilliam of Aberdeen, Scotland, was the first to suggest that VF, and not cardiac standstill, was the mechanism of sudden death in humans.⁷ Previously, he had experimented with mammalian animal hearts and was able to induce VF by applying electricity directly to the heart.⁸ Two physicians, Jean-Louis Prevost, a former trainee of Vulpian, and Frederic Battelli, worked together at the University of Geneva, Switzerland, on the mechanism of electrically induced VF.⁹ They confirmed the observations of Ludwig, Vulpian, and McWilliam in 1899 by showing that a small amount of electricity delivered across the chest can induce VF.⁹ It is fascinating that their secondary observation, mentioned only in a footnote, that larger electric shocks success-

From the Department of Medicine, Case Western Reserve University/University Hospitals of Cleveland Case Medical Center, Cleveland, Ohio (I.C., A.L.W.); and Department of Biomedical Engineering, Washington University in St Louis, St Louis, Mo (I.R.E.).

Correspondence to Ivan Cakulev, MD, Division of Cardiology, MS LKS 5038, University Hospitals of Cleveland Case Medical Center, 11100 Euclid Ave, Cleveland, OH 44106-5038. E-mail ivan.cakulev@case.edu

(*Circulation*. 2009;120:1623-1632.)

© 2009 American Heart Association, Inc.

Circulation is available at <http://circ.ahajournals.org>

DOI: 10.1161/CIRCULATIONAHA.109.865535

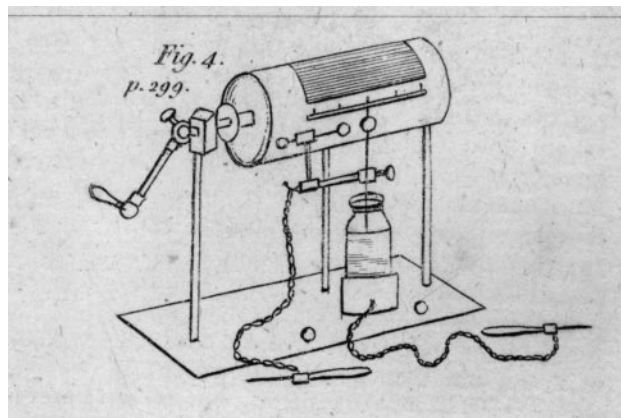


Figure 1. An apparatus similar to Charles Kite's, built and successfully used by Fell, as described in the 1792 issue of the *Gentleman's Magazine*. Courtesy of Mark Gulezian, Takoma Park.

fully restored normal sinus rhythm stirred little interest until the first defibrillation experiments some 30 years later. Even well-respected figures, giants in the field like Carl Wiggers, who later made significant contributions to the theory of fibrillation and defibrillation, were skeptical of the report of Prevost and Battelli and did not find "their claims worthy of the time, effort or expense."¹⁰

Work in the First Half of the 20th Century in the West

Nevertheless, Prevost and Battelli proposed the so-called incapacitation theory, whereby VF is terminated by complete electromechanical incapacitation of the myocardium established by the electric shock that also stopped and abolished the return of normal electric and mechanical work of the heart. Consequently, direct massage of the heart was suggested to support the circulation until electromechanical function of the heart was restored. This method was perfected by Carl Wiggers¹⁰ and used later during the pioneering studies with defibrillation in humans by Claude Beck.¹¹

The late 19th and early 20th centuries brought rapid expansion of commercially available electric power. This progress was followed by a growing number of accidents involving electrocution. It soon became apparent that most of the deaths were due to VF. Orthello Langworthy and Donald Hooker, both physicians at Johns Hopkins University, and William Kouwenhoven, an electrical engineer, were funded by the Consolidated Edison Electric Company of New York City to investigate the possible remedies for these frequent accidents. They studied both alternating current (AC) and direct current (DC) shocks and concluded that AC shock was more effective in terminating VF.¹² In 1933, the Johns Hopkins group succeeded in terminating VF in a dog when they accidentally applied a second shock, hence the term *countershock*.¹³ In 1936, Ferris and colleagues, another team composed of engineers and cardiologists, reported the first closed-chest defibrillation in sheep with the use of an AC shock.¹⁴

All of these experiments culminated with the first reported defibrillation of the exposed human heart performed by



Figure 2. Claude S. Beck, MD. Courtesy of the Dittrick Medical History Center, Case Western Reserve University, Cleveland, Ohio.

Claude Beck (Figure 2), a cardiothoracic surgeon at Western Reserve University/University Hospitals of Cleveland, Ohio, in 1947.¹¹ Beck was aware of Carl Wiggers' work on the mechanisms of fibrillation and defibrillation. Wiggers, also of Western Reserve University, had described the induction of VF through the concept of the vulnerable period.¹⁵ He was also a proponent of defibrillation, although he did not believe in transthoracic delivery of electric shocks. These conclusions influenced Beck when he performed the first known defibrillation of VF in humans. He was operating on a 14-year-old boy. During the closure of the wound, the pulse stopped, at which time the wound was reopened, and cardiac massage was performed for the next 45 minutes. An ECG confirmed VF, and seeing no other option, Beck delivered a single shock that failed to defibrillate the VF. After intracardiac administration of procaine hydrochloride, he delivered the second shock that restored sinus rhythm. This success triggered immediate acceptance of defibrillation across the world. Beck's defibrillator used AC directly from the wall socket (Figure 3). He built it together with his friend James Rand III of the RAND Development Corporation. The most significant drawback, however, was that it could be used only to defibrillate exposed hearts. Therefore, for years it was used only in operating rooms.

Work in the Soviet Union

Concurrent with the studies in the 1930s and 1940s in the West, a different approach to defibrillation was being developed in the Soviet Union. The latter provided further insight into the mechanisms of defibrillation and paved the way for development of modern defibrillation waveforms and the use of DC shock. The director of the Institute of Physiology at the Second Medical University in Moscow was Professor Lina



Figure 3. Beck's defibrillator. Courtesy of the Dittrick Medical History Center, Case Western Reserve University, Cleveland, Ohio.

Stern, who, as a former trainee and then associate of Prevost and Battelli, had studied VF and defibrillation. She assigned a PhD project on the study of arrhythmogenesis and defibrillation to Naum Gurvich (Figure 4), a young physician member of her laboratory. Gurvich later became a key figure and made fundamental discoveries in the fields of fibrillation and defibrillation. In 1939, in their classic work,^{16,17} Gurvich and



Figure 4. Naum L. Gurvich, MD. Courtesy of Margarita Bogush-evich, MD.



Figure 5. The first DC defibrillator ID-1-VEI for external transthoracic and internal use made in the USSR in 1952. Paddles and cords were stored in the separate metal box, which is leaning on the device. The defibrillator in this picture was given in 1958 to Dr Robert Hosler, an associate of Dr Claude Beck, by Dr Vladimir Negovsky in Moscow during Dr Hosler's visit to Russia. Courtesy of the Dittrick Medical History Center, Case Western Reserve University, Cleveland, Ohio.

Yuniev proposed using a single discharge from a capacitor to defibrillate VF, thus effectively introducing DC shock for defibrillation purposes. Until then, an AC shock was favored and was being developed as the most effective way to defibrillate VF. Parenthetically, in the West, AC shock continued to be used exclusively until the early 1960s. During his doctoral research (1933–1939), Gurvich found that an AC shock at a frequency of 50 to 500 Hz could not be tolerated and, in fact, led to VF. However, he also showed that a single discharge from a capacitor with a DC shock terminated VF. Another advantage of a DC shock was that large amounts of energy could be delivered in a relatively short period of time. In the 1940s, combining his studies with the Wiggers-Wegria model of the vulnerable period, he proposed a completely new concept in the field of defibrillation that was based on using biphasic defibrillation waveforms. Gurvich first reported using rounded biphasic waveforms, produced by a capacitor and inductor, for defibrillation as early as 1939, although at that time he was unaware of the superiority of this waveform over the monophasic waveform. More importantly, these advances allowed Gurvich¹⁸ to propose his “excitatory” theory of defibrillation, which suggested that direct excitation of the myocardium prevents further propagation of fibrillatory waves without preventing resumption of normal sinus rhythm. He also introduced the concept of the mother-reentrant circuit as a foundation for the development and sustainability of VF.¹⁹ In the United States, MacKay and Leeds in 1953 reported on their first experience with DC shock in dogs.²⁰ Their conclusion was similar to that of Gurvich: They pointed out that DC shock is more efficacious and safer than AC shock, and they also suggested the use of DC shock in humans. All of these reports had opened the way to explore the use of DC or capacitor shocks. In 1952, Gurvich designed the first commercially available transthoracic DC defibrillator (Figure 5) in the world.^{19,21,22} The application of this device was described in great detail in the

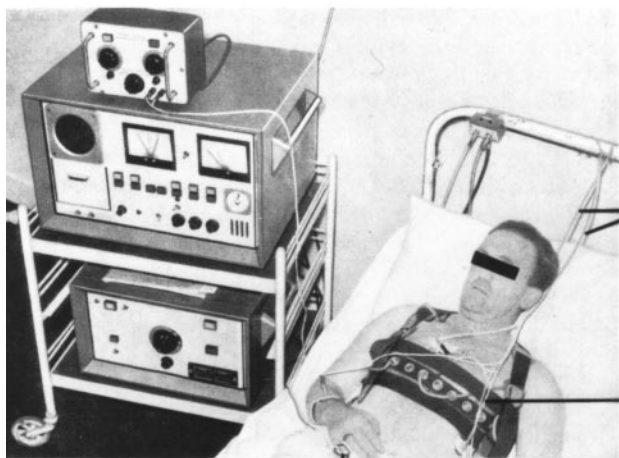


Figure 6. Bloc Réanimateur, the first automatic external defibrillator/pacer designed by Fred Zacouto, MD. Courtesy of Fred Zacouto, MD.

Soviet Ministry of Health resuscitation guidelines, published first in 1952.²³ The guidelines required every operating room of a major hospital to have a defibrillator. This first DC defibrillator, ID-1-VEI, used a monophasic waveform that, 10 years later, became known as the Lown waveform.

Following the work of Gurvich in Moscow, another physician-scientist behind the Iron Curtain made the next important defibrillation contribution. In 1957, Bohumil Peleška, from Prague, Czechoslovakia, reported on both direct and transthoracic use of DC shock for defibrillation purposes.²⁴ He constructed his own DC defibrillator, modifying Gurvich's design by including an iron core in the inductor,²⁵ and is credited with improving the procedure of cardioversion by using lower voltage and describing the effects of DC shock. Thus, the original work on biphasic defibrillation waveforms and DC cardioversion and defibrillation had originated initially in the East.

It was again in the Soviet Union in February 1959 that Vishnevskii and Tsukerman performed the first reported cardioversion of atrial fibrillation (AF) using a DC shock.^{26,27} The patient had AF for 3 years, and the restoration of normal sinus rhythm took place during mitral valve surgery. The same team reported the first successful transthoracic cardioversion of atrial arrhythmias in 20 patients using DC cardioversion in 1960.²⁸ In 1970, Gurvich introduced the first biphasic transthoracic defibrillator, which became standard in Soviet medical practice from that time, preceding Western analogs by at least 2 decades.²⁹

Of note, as part of "an international trip to further international cooperation in medical research for the good of people," in 1958, the well-known and influential senator Hubert H. Humphrey visited Moscow.³⁰ During that trip, Humphrey visited the Research Laboratory of General Resuscitology (Resuscitation), where he met with its director, Vladimir Negovsky, and the laboratory's leading defibrillation researcher, Naum Gurvich. "There, I saw his successful animal experiments on the reversibility of death, that is, on the revival of 'clinically dead' animals through massive electric shocks. When I returned to our country, I reported publicly on his experiments."³¹ Later, Humphrey urged the

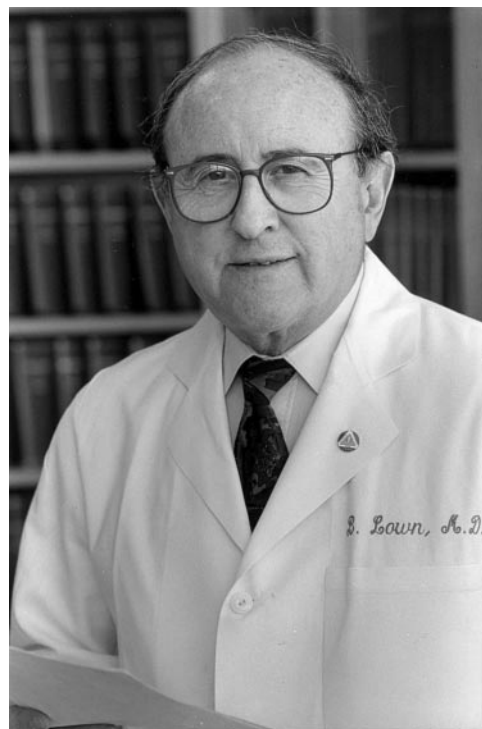


Figure 7. Bernard Lown, MD. Courtesy of Lown Cardiovascular Research Foundation.

development of programs through the National Institutes of Health "on the physiology of death, on resuscitation, and related topics."³¹ Nevertheless, the work behind the Iron Curtain remained virtually unrecognized in the West. However, as we shall see, the work became known to an electrical engineer working for the American Optical Company, and this had a profound impact on the field.

Work in the Western World After 1950

In 1956, Paul Zoll of Beth Israel Hospital and Harvard Medical School in Boston, Mass, demonstrated successful closed-chest defibrillation in humans, again using an AC shock.³² Not long after, in 1960, working at Lariboisiere Hospital in Paris, France, an electrical engineer and physician, Fred Zacouto, completed the design of the first external automatic defibrillator/pacer³³ (Figure 6). He had invented it in March 1953 and filed the related patent in July 1953 in Paris. His "Bloc Réanimateur" was able to sense a slow pulse from an infrared device attached to different parts of patient's body (ear lobe and a finger) and provide transcutaneous pacing until spontaneous return of heart activity. At the same time, it could detect VF from an ECG and deliver an AC shock of adjustable voltage and duration with the ability to redetect VF and redeliver a shock if needed. It was first used to successfully defibrillate a patient in November 1960. A total of 68 devices were produced and sold by 1968, first by Zacouto's Savita company and later by Thomson-CFTH. The device was used in hospitals in France, Switzerland, and Germany.

Bernard Lown (Figure 7) of the Peter Bent Brigham Hospital in Boston, Mass, is credited in the Western world with initiating the modern era of cardioversion. He was the



Figure 8. Barouh V. Berkovits. Courtesy of the Heart Rhythm Foundation.

first in the West to combine defibrillation and cardioversion with portability and safety. In 1959, in a patient with recurrent bouts of ventricular tachycardia (VT), Lown was the first to transthoracically apply AC shock using the Zoll defibrillator to successfully terminate an arrhythmia other than VF.³⁴ This event is notable because intravenous administration of procainamide had failed to terminate the patient's VT, and application of the transthoracic shock became a dire necessity to try to save a human life.³⁵ Because the procedure was unplanned and on an urgent basis and because there was not any information of which Lown's team was aware to provide data on the safety and efficacy of the procedure, it was done despite the hospital's resistance and only after Lown took sole responsibility.³⁵ Lown later recalled the following: "Never having seen an AC defibrillator, I hadn't the remotest idea how to use one. A host of questions needed prompt answers: Was the shock painful? Was the anesthesia required? Was there an appropriate voltage setting to reverse ventricular tachycardia? If the shock failed, how many additional ones could be delivered? Did the electric discharge traumatize the heart or injure the nervous system? Could it burn the skin? Were there any hazards for bystanders? Was it explosive for the patients receiving oxygen? My head was migrainous from the avalanche of questions."³⁵ At that time, clearly, Lown knew little about defibrillation and the intricacies of AC versus DC shock.

In early 1961, Lown "fortunately, and quite accidentally, met a brilliant young electrical engineer, Baruch [*sic*] Berkowitz [*sic*]"³⁵ (Figure 8), who was helping Lown's laboratory with instruments for research projects unrelated to the problem of cardioversion and defibrillation. Barouh Berkovits had been developing a DC defibrillator while working for the American Optical Corporation as the Director

of Cardiovascular Research. Although the American Optical Corporation manufactured an AC defibrillator, Berkovits was very aware of its shortcomings because he was familiar with the previous work of Gurvich.³⁶ Thus, aware that DC shock was safer and more effective, Berkovits had decided to build a DC defibrillator for possible commercial use. After the "accidental" meeting of Berkovits with Lown, when they learned of each other's interests, Berkovits asked Lown if he would be interested in testing his device. In April 1961, Lown formally asked Berkovits to study his DC defibrillator in canines and for possible clinical application.³⁷ A series of intense experiments followed that involved testing the efficacy of multiple waveforms and evaluating the safety of DC shock in a very large number of canines. During these experiments, the Lown-Berkovits investigation group, aware of the importance of avoiding the vulnerable period, introduced for the first time the novel concept of synchronizing delivery of the shock with the QRS complex sensed from the ECG. During these studies, they also developed a monophasic waveform, later known as the "Lown waveform," with high efficacy and safety for shock delivery during a rhythm other than VF. These studies culminated with the use of the DC cardioverter-defibrillator in patients. Lown is also credited with coining the term *cardioversion* for delivery of a synchronized shock during an arrhythmia other than VF. Noting the previous work with DC defibrillation in humans by Gurvich in the Soviet Union and Peleška in Czechoslovakia, as well as the adverse effects of AC shock, in 1962 Lown et al reported their success in terminating VT with a single DC monophasic shock in 9 patients.³⁸ Lown subsequently went on to expand DC cardioversion to successfully convert both atrial and ventricular arrhythmias using the monophasic DC shock.³⁹⁻⁴¹ This success promptly resulted in the acceptance and worldwide spread of DC cardioversion. One result of the success of the DC cardioverter-defibrillator was the development of the modern cardiac care unit, where Lown again played an important role. In 1962, Berkovits patented the DC defibrillator for the American Optical Corporation.

The impact of this "new technique" was indeed profound. The ability to "reverse death" with a simple shock had dramatically improved in-hospital cardiac arrest outcomes. However, it was widely known that the highest mortality was taking place in the immediate period after an individual suffered a heart attack, mainly outside hospital premises.

This problem was boldly addressed by J. Frank Pantridge, who, working together with John Geddes at the Royal Victoria Hospital in Belfast, UK, created the first Mobile Coronary Care Unit, which began operation on January 1, 1966.⁴² The initial assembly of the defibrillator for this mobile unit, which consisted of 2 car batteries, a static inverter, and an American Optical defibrillator, weighed 70 kg. Any initial skepticism that defibrillation out of the hospital would not be feasible, and may even be detrimental, disappeared when the initial 15-month experience with the "flying squad" was published.⁴³ Aware of the work of Peleška, Pantridge's team made further improvements in the design of the defibrillator. A key stage in the development of the mobile intensive care unit came with the design of a small, portable defibrillator. Using the miniature capacitor

developed for the US National Aeronautics and Space Administration, Pantridge, together with John Anderson, a biomedical engineer, developed a 3.2-kg portable defibrillator that became available in 1971.

With great passion, Pantridge advocated his approach of making early defibrillation readily available everywhere. His ideas first became widely accepted in the United States. Subsequently, Anderson and Jennifer Adgey, another physician from the Belfast group, were among the first to develop the semiautomatic and automatic portable external defibrillator in the late 1970s and early 1980s. With continued development, the portable defibrillator gradually evolved from exclusive use by physicians and was given to paramedics, then to firemen, and finally to members of the public. The benefits of this approach are more than obvious today.⁴⁴

The Implantable Cardioverter-Defibrillator

Although external transthoracic DC cardioversion gained wide acceptance and radically improved patient outcomes, the work on defibrillation did not stop here. Defibrillation from an implantable device was the next major achievement that dramatically changed our approach to treat sudden cardiac death. Michel Mirowski conceived the idea for an implantable cardiac defibrillator while working in Israel. Mirowski trained at Tel Hashomer Hospital in Israel, where his mentor was Harry Heller.⁴⁵ Heller had developed repetitive bouts of VT that were treated with quinidine or procainamide. However, Mirowski was very aware that, sooner or later, this arrhythmia would take Heller's life. It was the sudden death of his mentor in 1966 and the recognition that sudden arrhythmic death was a major problem without, at that time, a solution that influenced Mirowski to dedicate his career to design and develop the implantable cardiac defibrillator. Mirowski recognized that it would be very difficult to accomplish his goal in Israel. In 1968, he accepted a position at Sinai Hospital of Baltimore, Md, as a director of the Coronary Care Unit, with 50% of his time for research. He arrived there in the summer of 1969, and in November 1969 he began working toward his goal with Morton Mower, a young cardiologist and a vital coinvestigator. Together, they produced and tested in dogs the first prototype of an automatic defibrillator⁴⁶ (Figure 9). Virtually simultaneously and independently, John Schuder, a PhD in Electrical Engineering and then an Associate Professor of Biophysics and Surgery at the University of Missouri in Columbia, also began work on an implantable defibrillator.⁴⁷ While contemplating future projects during an American Heart Association meeting in 1969, and having been steeped in "transthoracic defibrillation, knowledge about waveform efficacy, and an appreciation of circuit design and component problems," Schuder later commented, "it was almost immediately apparent that the automatic implantable defibrillator was a doable project. I decided to go home and do it."⁴⁷ In fact, Schuder was the first to implant and successfully use a cardiac defibrillator in a dog in January 1970.⁴⁸ He subsequently abandoned his work on the implantable defibrillator, instead concentrating his work on optimization of shocking waveforms. Schuder's continued contributions laid the foundation for the miniature, low-energy, reliable, high-voltage, biphasic waveform, which



Figure 9. Drs Morton Mower (left) and Michel Mirowski (right) with their first prototype of an automatic defibrillator. Courtesy of Ariella Rosengard, MD.

ultimately made contemporary implantable cardioverter-defibrillator (ICD) therapy possible.

The continued path to the first implantable cardiac defibrillator in humans was anything but simple or short. As stated by William Staewen, the Director of the Biomedical Engineering Department at Sinai Hospital of Baltimore, Md, and Morton Mower, "The design had to be virtually unfailured. It had to reliably sense ventricular defibrillation and deliver a high energy electric shock to correct the arrhythmia in less than one minute. This had to be accomplished with a device placed remotely in the hostile environment of the body. It had to function as designed for years and must not, if it would fail for any reason, cause injury to the patient."⁴⁹ When one considers the technical challenges with the potential for both harmful effects and lack of clinical benefit, it comes as no surprise that many leading medical and engineering authorities, including Lown himself, challenged this novel and original idea.⁵⁰ Nevertheless, Mirowski and Mower, ultimately working with Dr Stephen Heilman and his small company, Medrad (later, Intec Systems, a subsidiary of Medrad), persevered in their project, overcoming many obstacles, from the enormous to the small. They finally achieved their goal. In February 1980, after 11 years of development, the first internal cardiac defibrillator was implanted in a patient at the Johns Hopkins Hospital in Baltimore by Levi Watkins, the cardiothoracic surgeon, and Philip Reid, the cardiac electrophysiologist. After the third patient implantation, the device also included cardioversion. The cardioversion-defibrillation device obtained Food and Drug Administration approval in 1985. Soon after, antitachycardia pacing was added. The Food and Drug Administration approval ended a century-long era of investigation, description of basic mechanisms of arrhythmias, and attempts at resuscitation of the dead that finally culminated in an implantable device that safely and effectively aborted sudden cardiac death. The ICD device continued to improve and has now been developed to the point that it can be used virtually at any time and in any place to treat ventricular arrhythmias, if needed. The dedication of many individuals and groups has made this possible. Unfortunately, the space limitation for this article prevents us from mentioning all those who have

and still are contributing to the developments in this field. Finally, we should note that an implantable atrial defibrillator was also developed,⁵¹ but its use is limited by the pain associated with delivered therapy.

Present

Little has changed in the technique of cardioversion since Lown's article in the early 1960s. Progress has been made in reducing the already low associated complication rate and in understanding the factors responsible for success. Successful cardioversion or defibrillation occurs when a shock with sufficient current density reaches the myocardium. Because the maximum energy stored in the capacitor is fixed, the principal determinant of current density is transthoracic impedance to DC discharge. The factors influencing transthoracic impedance that can be modified by the technique of cardioversion include the interface between the electrode and skin, the electrode size, and the electrode placement. Although a variety of chest placements have been used, there are 2 conventional positions for the electrode paddles: anteroposterior and anterolateral. In the anterolateral position, paddles are placed between the ventricular apex and the right infraclavicular area, whereas in the anteroposterior position, one paddle is placed over the sternum and the second interscapularly. Lown originally advocated that the anteroposterior position is superior because it requires less energy to reverse AF.⁵² Some studies have confirmed this notion,^{53,54} whereas others have shown no advantage to either paddle position.^{55,56} Because only $\approx 4\%$ to 5% of the shocking energy actually reaches the heart,⁵⁷ minor deviation of this electric field probably has little effect on the final outcome. In today's era of biphasic waveforms, the position of the paddles most likely plays an even smaller role.

The size of the electrodes through which the shock is delivered has been shown to significantly influence the transthoracic impedance.^{58–60} A larger electrode leads to lower impedance and higher current, but an increase in size of the electrode beyond the optimal size leads to a decrease in current density.⁶¹ In humans, paddle electrode size with a diameter between 8 and 12 cm appears to be optimal.^{62,63} Improved skin-to-electrode contact also leads to reduction of transthoracic impedance and an increase in the success rate. Hand-held paddles may be more effective than self-adhesive patch electrodes, perhaps because of better skin contact.⁶⁴ In addition, the usage of non-salt-containing gels has been associated with an increase in transthoracic resistance.⁶⁵

The Biphasic Waveform

Gurvich was the first to demonstrate the superiority of the biphasic waveform over the monophasic in dogs in 1967.⁶⁶ Most of the external defibrillators in the Soviet Union from the early 1970s used biphasic waveforms,⁶⁷ which are known in Russia as the Gurvich-Venin waveform. It took much longer for the West to realize the benefit of the biphasic waveform over the original Lown monophasic waveform. The first experiments comparing the monophasic and biphasic waveforms for transthoracic defibrillation were done independently by Schuder et al in the 1980s.^{68,69} Ventritex's Cadence V-100, approved by the Food and Drug Adminis-

tration in 1993, was the first ICD that used a biphasic waveform. Curiously, this waveform was first used in ICDs and only a few years later in external defibrillators. The efficacy of an ICD is limited by the maximum stored energy. In their attempt to limit the device size, manufacturers of the ICD finally chose the more effective biphasic waveform. Although the Gurvich-Venin biphasic waveform was superior to the monophasic waveform, its requirement for an inductor precluded major reduction in size for use in ICDs. It was the work of John Schuder⁴⁷ and also Raymond Ideker,⁷⁰ then at Duke University, on optimization of biphasic waveforms that made miniaturization of implantable defibrillators possible. After 2000, most defibrillators developed for either external or internal use were "biphasic" devices, meaning that they reverse polarity 5 to 10 ms after the discharge begins. The biphasic waveform has been shown in humans to defibrillate both AF and VF more effectively than monophasic waveform.^{71–75} Despite the clear superiority of the biphasic waveform, the recommended initial shock energy remains unclear. The 2006 American College of Cardiology/American Heart Association/European Society of Cardiology guidelines on the management of AF recommend starting at 200 J with a monophasic waveform. "A similar recommendation to start with 200 J applies to biphasic waveforms, particularly when cardioverting patients with AF of long duration."⁷⁶ The American Heart Association Advanced Cardiac Life Support guidelines recommend initially defibrillating VF with the use of a 360-J monophasic shock or a default 200-J biphasic shock if the type of the biphasic waveform is unknown.

Besides the waveform shape, success in restoring normal sinus rhythm is related directly to the type and duration of the arrhythmia. Successful termination of organized tachycardias requires less energy than disorganized rhythms such as polymorphic VT, AF, or VF. Similarly, tachycardias of shorter duration have higher immediate conversion success rates. For instance, the overall success rate in restoring sinus rhythm in patients with AF is $\geq 90\%$ when the arrhythmia is of < 1 year's duration compared with 50% when AF has been present for > 5 years.⁷⁷

The risks associated with DC cardioversion are related mainly to inadvertent initiation of new tachyarrhythmias, the unmasking of bradycardia, and postshock thromboembolism. More than 25% of patients have bradycardia immediately after cardioversion, and this incidence is higher in patients with underlying sinus node dysfunction.⁷⁸ Ventricular arrhythmias are uncommon after cardioversion unless an unsynchronized shock was applied, VT previously existed,^{79,80} or digitalis toxicity was present. In the latter instance, DC cardioversion is contraindicated. A major risk associated with cardioversion is thromboembolism. Thromboembolic events are more likely to occur in patients with AF who have not been anticoagulated adequately before cardioversion. The incidence varies and has been reported to be between 1% and 7% .^{81,82} In a large series, the incidence was reduced to 0.8% from 5.3% with proper anticoagulation.⁸¹

Nevertheless, the efficacy and safety of cardioversion in its current form have withstood the test of time, and it continues to be used widely by clinicians as the most frequent approach to restoring sinus rhythm. This success, associated with a

very favorable risk profile, has initiated a trend toward wider use of cardioversion/defibrillation not only by medical personnel but also by the general public. Although portable automatic external defibrillators have existed since 1979,⁸³ the accumulation of clinical studies confirming their safety, efficacy, and diagnostic accuracy has recently prompted several US federal initiatives to expand public access to defibrillators.^{84–87}

Future Directions

It is hard to imagine the changes that the future may bring to a technique that has changed so little over the last several decades. Progress usually occurs when light is shed on the unknown. Clearly, as we more fully understand all the intricacies of fibrillation and defibrillation, advances in this field will be made.

We ultimately need to prevent sudden cardiac death in a more effective manner. Currently, we are only partially successful in this task.⁸⁸ By far, the vast majority of sudden cardiac death episodes occur in subjects without any identifiable or recognized heart disease. Our current attention is focused only on the relatively small percentage of patients with identifiable or recognized risk factors for sudden cardiac death, mainly subjects with structural heart disease. It is obvious that we do not have an effective solution for the largest part of the population at risk. Further expansion of defibrillation in public spaces is needed. Early warning systems detecting the location of cardiac arrest victims and rapid use of a nearby defibrillator should be developed.⁸⁹

Our success in preventing sudden cardiac death will depend on our ability to identify the subjects at risk for future events and/or to reduce the adverse effects and risks that are associated with our current treatment strategies. At the present time, in subjects without clear and identifiable risk markers, we are unable to predict who will suffer from sudden cardiac death. Hence, it is necessary to focus our attention on improving the risk profile of our most effective available treatment for sudden cardiac death: defibrillation. For these subjects, only by reducing the risk of the therapy without affecting the quality of life can we improve the risk-benefit ratio and expand the use of cardioversion/defibrillation to combat this serious problem effectively. Eventually, the use of defibrillation may be similar to the current use of seat belts. If the risks are sufficiently low and major inconveniences are avoided, there would be a good reason to expand their use to populations at much lower relative risk for sudden cardiac death.

In this regard, several areas of potential improvement can be identified. The continued development of a less invasive initial implantation procedure that can also avoid intravascular housing of the leads and the device should be pursued. Already, prototypes of an ICD with subcutaneous leads whose implantation does not require intravenous access have been designed.⁹⁰ Their approval is currently under review. Further improvement in the technology of wearable vest defibrillators can result in even better outcomes with less risk. Having the device on the human body rather than in it will eliminate the risks associated with implantation and will avoid all future complications associated with maintaining

the device in the intravascular and intracardiac space. The device will have to be much smaller and less cumbersome than currently available wearable vests to avoid interference with daily activities. It would still have to provide accurate diagnosis and safe and effective treatment of lethal arrhythmias. The benefits of obviating invasive implantation and having a device that will completely eliminate known adverse issues associated with the presence of leads would be indispensable.

Another very important area for future improvement would be to further reduce the defibrillation threshold. This would serve the ultimate goal of eventually eliminating pain, anesthesia, and sedation during shocks, if possible. To achieve this, several different strategies, perhaps in combination with each other, will be used. Current research points toward the direction this is already taking. In all likelihood, more effective cardioversion/defibrillation waveforms will be used. Shocks from ≥ 2 sites simultaneously or sequentially will further improve cardioversion/defibrillation effectiveness. In addition, combination of shocks with cardiac pacing may prove particularly useful. We already know that pacing can influence and terminate reentrant or triggered arrhythmias.⁹¹ Work on animal models and humans on the mechanisms of VF and AF suggests the presence of 1 or more drivers^{92–95} that may make the strategy of combining shocks with pacing plausible. The hope would be that this combination will result in the need for less energy to restore normal sinus rhythm. This would certainly benefit internal as well as external cardioversion/defibrillation. Clearly, this approach requires more work on the mechanisms of these arrhythmias and the technology used to cardiovert and defibrillate them. Just as in the past, dedicated individuals and teams will be needed to fully solve the puzzles of fibrillation and defibrillation. It may be a while before we come close to this goal, but if the past is the harbinger of the future, then we look forward to the future with great optimism.

Epilogue

As we reviewed the beginnings and subsequent development of defibrillation and cardioversion for this article, we were surprised to learn how much seminal work had been done behind the Iron Curtain that was almost completely unknown in the West. We can only speculate on the reasons for this, but the final result was that for too many years, humanity was deprived of life-saving treatment that should have been available much earlier. It was fortunate that Barouh Berkovits bridged the gap between East and the West by making the DC transthoracic cardioverter/defibrillator available to Dr Bernard Lown, an interested and dedicated clinician. However, it took another 4 decades for both West and East to merge on the uniform use of biphasic shock waveforms for external as well as internal defibrillators. It seems obvious to us that if we can learn anything from this history, it would be to facilitate cooperation and avoid the barriers that have existed and even still exist among us. Whatever those barriers may be, they are, after all, only made by humans.

Acknowledgments

The authors wish to thank the following individuals with whom personal communication by 1 or more of the authors made this article

possible: A.A. Jennifer Adgey; Sidney Alexander; Raghavan Amarasingham; Barouh Berkovits; Amy Beeman; Betsy Bogdanský; Ian Clement; Leonard Dreifus; Edwin Duffin, Jr; John Fisher; Gregory Flaker; Bruce Fye; John Geddes; Boris Gorbunov; Robert J. Hall; M. Stephen Heilman; Raymond Ideker; James Jude; Alan Kadish; Claudia Kenney; Richard Kerber; G. Guy Knickerbocker; Bernard Kosowsky; Peter Kowey; Mark Kroll; Samuel Levy; Bernard Lown; Frank Marcus; Morton Mower; John Muller; Michael Orlov; Phillip Podrid; Christine Riegel; Jeremy Ruskin; Ariella Rosengard; Vikas Saini; John Schuder; Hein Wellens; Roger Winkle; Fred Zacouto; and special thanks to Jayakumar Sahadevan.

Sources of Funding

This work was supported in part by grants from the Jennie Zoline Foundation, Blue Dot Foundation, Gemstone Foundation, and National Institutes of Health/National Heart, Lung, and Blood Institute grants HL067322 and HL074283.

Disclosures

None.

References

- Abildgaard PC. Tentamina electrica in animalibus instituta. *Societas Medical Havniensis Collectanea*. 1775;2:157.
- Cajavilca C, Varon J, Sternbach GL. Resuscitation great: Luigi Galvani and the foundations of electrophysiology. *Resuscitation*. 2009;80:159–162.
- Annual Reports of the Royal Humane Society for the Recovery of the Apparently Drowned (London)*. 1774:1–31.
- Stillings D. The first defibrillator? *Med Prog Technol*. 1974;2:205–206.
- Hoffa M, Ludwig C. Einige neue Versuche uber Herzbewegung. *Zeitschrift Rationelle Medizin*. 1850;9:107–144.
- Vulpian EFA. Notes sur les effets de la faradisation directe des ventricules du coeur chez le chien. *Arch Physiol Norm Path*. 1874;6:975–982.
- McWilliam J. Electrical stimulation of the heart in man. *BMJ*. 1889;1:348–350.
- McWilliam J. Fibrillatory contraction of the heart. *J Physiol*. 1887;8:296–310.
- Prevost JL, Battelli F. Le mort par les descharges électrique. *J Physiol*. 1899;1:1085–1100.
- Wiggers CJ. The mechanism and nature of ventricular fibrillation. *Am Heart J*. 1940;20:399–412.
- Beck CS, Pritchard WH, Feil HS. Ventricular fibrillation of long duration abolished by electric shock. *JAMA*. 1947;135:985.
- Kouwenhoven WB. Current flowing through heart under conditions of electric shock. *Am J Physiol*. 1932;100:344–350.
- Kouwenhoven WHD. Resuscitation by countershock. *Electrical Eng*. 1933;52:475–477.
- Ferris LP, King BH, Spence PW. Effects of electric shock on the heart. *Electrical Eng (NY)*. 1936;55:498.
- Wiggers CJ, Wégria R. Ventricular fibrillation due to single, localized induction and condenser shocks applied during the vulnerable phase of ventricular systole. *Am J Physiol*. 1940;128:500–505.
- Gurvich N, Yuniev G. Restoration of regular rhythm in the mammalian fibrillating heart [in Russian]. *Byull Eksper Biol Med*. 1939;8:55–58.
- Gurvich N, Yuniev G. Restoration of regular rhythm in the mammalian fibrillating heart [in English]. *Am Rev Sov Med*. 1947;3:236–239.
- Gurvich N. *The Main Principles of Cardiac Defibrillation*. Moscow, USSR: Medicine; 1975.
- Gurvich N. Fibrillation and defibrillation of the heart. *Medgiz Moscow*. 1957.
- MacKay RS, Leeds SE. Physiological effects of condenser discharges with application to tissue stimulation and ventricular defibrillation. *J Appl Physiol*. 1953;6:67–75.
- Negovsky V. Method to terminate heart fibrillation. In: Negovsky V, ed. *Pathophysiology and Therapy of Agony and Clinical Death*. Moscow, USSR: Medgiz; 1954:41–45.
- Gurvich NL. Restoration of vital functions of the organism following fatal electric shock. [in Russian]. *Klin Med (Mosk)*. 1952;30:66–70.
- Instructions on the application of methods to resuscitate life functions in terminally ill. USSR Ministry of Health. Moscow, USSR: Medgiz; 1959.
- Peleška B. Transthoracic & direct defibrillation [in Czechoslovakian]. *Rozhl Chir*. 1957;36:731–755.
- Peleška B. Cardiac arrhythmias following condenser discharges led through an inductance: comparison with effects of pure condenser discharges. *Circ Res*. 1965;16:11–18.
- Vishnevskii AA, Tsukerman BM, Smelovskii SI. Control of fibrillating arrhythmia by the method of electrical defibrillation of the atrium [in Russian]. *Klin Med (Mosk)*. 1959;37:26–29.
- Vishnevsky AA, Tsukerman BM. Some debatable problems on electroimpulse therapy of cardiac arrhythmias. *J Cardiovasc Surg (Torino)*. 1966;7:402–407.
- Vishnevskii A, Tsukerman B. Atrial defibrillation in 20 patients with mitral valve disease. *J USSR Acad Med Sci*. 1961;8:32–35.
- Deleted in proof.
- Humphrey H. Eight hours with Khrushchev. *Life Magazine*. January 12, 1959:80–91.
- Humphrey HH. *The Congressional Record*. October 13, 1962: A7837–A7839.
- Zoll PM, Linenthal AJ, Gibson W, Paul MH, Norman LR. Termination of ventricular fibrillation in man by externally applied electric countershock. *N Engl J Med*. 1956;254:727–732.
- Effert S. Automatic monitoring equipment and indication for implantation of electrical pacemakers [in German]. *Thoraxchir Vask Chir*. 1963;11:158–166.
- Alexander S, Kleiger R, Lown B. Use of external electric countershock in the treatment of ventricular tachycardia. *JAMA*. 1961;177:916–918.
- Lown B. The shock that cures: DC and cardioversion. In: Lown B, ed. *The Lost Art of Healing*. New York, NY: Houghton Mifflin Co; 1996:188–201.
- Oral History Collection Heart Rhythm Society: Interview With Barouh Berkovits by Seymour Furman*. Natick, Mass; Heart Rhythm Society; January 21, 1996.
- Personal Communication with Barouh Berkovits: Letter to Barouh Berkovits from Bernard Lown. April 10, 1961.
- Lown B, Amarasingham R, Neuman J. New method for terminating cardiac arrhythmias: use of synchronized capacitor discharge. *JAMA*. 1962;182:548–555.
- Lown B, Perloth MG, Kaidbey S, Abe T, Harken DE. “Cardioversion” of atrial fibrillation: a report on the treatment of 65 episodes in 50 patients. *N Engl J Med*. 1963;269:325–331.
- Lown B, Bey SK, Perloth MG, Abe T. Cardioversion of ectopic tachycardias. *Am J Med Sci*. 1963;246:257–264.
- Guiney TE, Lown B. Electrical conversion of atrial flutter to atrial fibrillation: flutter mechanism in man. *Br Heart J*. 1972;34:1215–1224.
- Pantridge JF, Geddes JS. Cardiac arrest after myocardial infarction. *Lancet*. 1966;1:807–808.
- Pantridge JF, Geddes JS. A mobile intensive-care unit in the management of myocardial infarction. *Lancet*. 1967;2:271–273.
- Rho RW, Page RL. The automated external defibrillator. *J Cardiovasc Electrophysiol*. 2007;18:896–899.
- Kastor JA. Michel Mirowski and the automatic implantable defibrillator. *Am J Cardiol*. 1989;63:977–982.
- Mirowski M, Mower MM, Staewen WS, Tabatnick B, Mendeloff AI. Standby automatic defibrillator: an approach to prevention of sudden coronary death. *Arch Intern Med*. 1970;126:158–161.
- Schuder JC. The role of an engineering oriented medical research group in developing improved methods and devices for achieving ventricular defibrillation: the University of Missouri experience. *Pacing Clin Electrophysiol*. 1993;16(pt 1):95–124.
- Schuder JC. Completely implanted defibrillator. *JAMA*. 1970;214:1123.
- Staewen WS, Mower MM. History of ICD. In: Kroll MW, Lehmann MH, eds. *Implantable Cardioverter Defibrillator Therapy: The Engineering-Clinical Interface*. New York, NY: Springer; 1996:22.
- Lown B, Axelrod P. Implanted standby defibrillators. *Circulation*. 1972;46:637–639.
- Wellens HJ, Lau CP, Luderitz B, Akhtar M, Waldo AL, Camm AJ, Timmermans C, Tse HF, Jung W, Jordaens L, Ayers G. Atrioverter: an implantable device for the treatment of atrial fibrillation. *Circulation*. 1998;98:1651–1656.
- Lown B, Kleiger R, Wolff G. The technique of cardioversion. *Am Heart J*. 1964;67:282–284.
- Kirchhof P, Eckardt L, Loh P, Weber K, Fischer RJ, Seidl KH, Bocker D, Breithardt G, Haverkamp W, Borggrefe M. Anterior-posterior versus anterior-lateral electrode positions for external cardioversion of atrial fibrillation: a randomised trial. *Lancet*. 2002;360:1275–1279.

54. Botto GL, Politi A, Bonini W, Broffoni T, Bonatti R. External cardioversion of atrial fibrillation: role of paddle position on technical efficacy and energy requirements. *Heart*. 1999;82:726–730.
55. Siaplaouras S, Buob A, Rotter C, Bohm M, Jung J. Randomized comparison of anterolateral versus anteroposterior electrode position for biphasic external cardioversion of atrial fibrillation. *Am Heart J*. 2005;150:150–152.
56. Mathew TP MA, McIntyre M, Harbinson MT, Campbell NP, Adgey AA, Dalzell GW. Randomised comparison of electrode positions for cardioversion of atrial fibrillation. *Heart*. 1999;81:576–579.
57. Lerman BB, Deale OC. Relation between transcardiac and transthoracic current during defibrillation in humans. *Circ Res*. 1990;67:1420–1426.
58. Kerber RE, Jensen SR, Grayzel J, Kennedy J, Hoyt R. Elective cardioversion: influence of paddle-electrode location and size on success rates and energy requirements. *N Engl J Med*. 1981;305:658–662.
59. Ewy GA, Horan WJ. Effectiveness of direct current defibrillation: role of paddle electrode size, II. *Am Heart J*. 1977;93:674–675.
60. Dalzell GW, Cunningham SR, Anderson J, Adgey AA. Electrode pad size, transthoracic impedance and success of external ventricular defibrillation. *Am J Cardiol*. 1989;64:741–744.
61. Hoyt R, Grayzel J, Kerber RE. Determinants of intracardiac current in defibrillation: experimental studies in dogs. *Circulation*. 1981;64:818–823.
62. Sirna SJ, Ferguson DW, Charbonnier F, Kerber RE. Factors affecting transthoracic impedance during electrical cardioversion. *Am J Cardiol*. 1988;62:1048–1052.
63. Kerber RE, Martins JB, Kelly KJ, Ferguson DW, Kouba C, Jensen SR, Newman B, Parke JD, Kieso R, Melton J. Self-adhesive preapplied electrode pads for defibrillation and cardioversion. *J Am Coll Cardiol*. 1984;3:815–820.
64. Kirchhoff P, Monnig G, Wasmer K, Heinecke A, Breithardt G, Eckardt L, Bocker D. A trial of self-adhesive patch electrodes and hand-held paddle electrodes for external cardioversion of atrial fibrillation (MOBIPAPA). *Eur Heart J*. 2005;26:1292–1297.
65. Ewy GA, Taren D. Comparison of paddle electrode pastes used for defibrillation. *Heart Lung*. 1977;6:847–850.
66. Gurvich NL, Makarychev VA. Defibrillation of the heart with biphasic electric impulsion [in Russian]. *Kardiologia*. 1967;7:109–112.
67. Ussenko LV, Tsarev AV, Leschenko YA. Resuscitation great: Naum L Gurvich: a pioneer of defibrillation. *Resuscitation*. 2006;70:170–172.
68. Schuder JC, Gold JH, Stoeckle H, McDaniel WC, Cheung KN. Transthoracic ventricular defibrillation in the 100 kg calf with symmetrical one-cycle bidirectional rectangular wave stimuli. *IEEE Trans Biomed Eng*. 1983;30:415–422.
69. Schuder JC, McDaniel WC, Stoeckle H. Transthoracic defibrillation of 100 kg calves with bidirectional truncated exponential shocks. *Trans Am Soc Artif Intern Organs*. 1984;30:520–525.
70. Dixon EG, Tang AS, Wolf PD, Meador JT, Fine MJ, Calfee RV, Ideker RE. Improved defibrillation thresholds with large contoured epicardial electrodes and biphasic waveforms. *Circulation*. 1987;76:1176–1184.
71. Schneider T, Martens PR, Paschen H, Kuisma M, Wolcke B, Gliner BE, Russell JK, Weaver WD, Bossaert L, Chamberlain D; Optimized Response to Cardiac Arrest (ORCA) Investigators. Multicenter, randomized, controlled trial of 150-J biphasic shocks compared with 200- to 360-J monophasic shocks in the resuscitation of out-of-hospital cardiac arrest victims. *Circulation*. 2000;102:1780–1787.
72. Mittal S, Ayati S, Stein KM, Knight BP, Morady F, Schwartzman D, Cavlovich D, Platia EV, Calkins H, Tchou PJ, Miller JM, Wharton JM, Sung RJ, Slotwiner DJ, Markowitz SM, Lerman BB; ZOLL Investigators. Comparison of a novel rectilinear biphasic waveform with a damped sine wave monophasic waveform for transthoracic ventricular defibrillation. *J Am Coll Cardiol*. 1999;34:1595–1601.
73. Mortensen K, Risius T, Schwemer TF, Aydin MA, Koster R, Klemm HU, Lutomsky B, Meinertz T, Ventura R, Willems S. Biphasic versus monophasic shock for external cardioversion of atrial flutter: a prospective, randomized trial. *Cardiology*. 2008;111:57–62.
74. Mittal S, Ayati S, Stein KM, Schwartzman D, Cavlovich D, Tchou PJ, Markowitz SM, Slotwiner DJ, Schweiner MA, Lerman BB. Transthoracic cardioversion of atrial fibrillation: comparison of rectilinear biphasic versus damped sine wave monophasic shocks. *Circulation*. 2000;101:1282–1287.
75. Page RL, Kerber RE, Russell JK, Trouton T, Waktare J, Gallik D, Olgin JE, Ricard P, Dalzell GW, Reddy R, Lazzara R, Lee K, Carlson M, Halperin B, Bardy GH. Biphasic versus monophasic shock waveform for conversion of atrial fibrillation: the results of an international randomized, double-blind multicenter trial. *J Am Coll Cardiol*. 2002;39:1956–1963.
76. Fuster V, Ryden LE, Cannom DS, Crijns HJ, Curtis AB, Ellenbogen KA, Halperin JL, Le Heuzey JY, Kay GN, Lowe JE, Olsson SB, Prystowsky EN, Tamargo JL, Wann S, Priori SG, Blanc JJ, Budaj A, Camm AJ, Dean V, Deckers JW, Despres C, Dickstein K, Lekakis J, McGregor K, Metra M, Morais J, Osterspey A, Zamorano JL, Smith SC Jr, Jacobs AK, Adams CD, Anderson JL, Antman EM, Hunt SA, Nishimura R, Ornato JP, Page RL, Riegel B. ACC/AHA/ESC 2006 guidelines for the management of patients with atrial fibrillation: executive summary. *Circulation*. 2006;114:e257–e354.
77. Lown B. Electrical reversion of cardiac arrhythmias. *Br Heart J*. 1967;29:469–489.
78. Mehta PM, Reddy BR, Lesser J, Carson PE. Severe bradycardia following electrical cardioversion for atrial tachyarrhythmias in patients with acute myocardial infarction. *Chest*. 1990;97:241–242.
79. Eysmann SB, Marchlinski FE, Buxton AE, Josephson ME. Electrocardiographic changes after cardioversion of ventricular arrhythmias. *Circulation*. 1986;73:73–81.
80. DeSilva RA, Graboys TB, Podrid PJ, Lown B. Cardioversion and defibrillation. *Am Heart J*. 1980;100(pt 1):881–895.
81. Bjerkelund CJ, Orning OM. The efficacy of anticoagulant therapy in preventing embolism related to D.C. electrical conversion of atrial fibrillation. *Am J Cardiol*. 1969;23:208–216.
82. Arnold AZ, Mick MJ, Mazurek RP, Loop FD, Trohman RG. Role of prophylactic anticoagulation for direct current cardioversion in patients with atrial fibrillation or atrial flutter. *J Am Coll Cardiol*. 1992;19:851–855.
83. Diack AW, Welborn WS, Rullman RG, Walter CW, Wayne MA. An automatic cardiac resuscitator for emergency treatment of cardiac arrest. *Med Instrum*. 1979;13:78–83.
84. Guidelines for public access defibrillation programs in federal facilities. *Federal Register* 66FR28495; May 23, 2001.
85. Guidelines for public access defibrillation programs in federal facilities. *Federal Register* 66FR28495; May 23, 2001.
86. Emergency medical equipment. *Federal Register* 66FR19027; April 21, 2001.
87. National Conference of State Legislatures. State Laws on Cardiac Arrest and Defibrillators. Available at: <http://www.ncsl.org/default.aspx?tabid=14506>. Accessed January 1, 2009.
88. Myerburg RJ, Kessler KM, Castellanos A. Sudden cardiac death: epidemiology, transient risk, and intervention assessment. *Ann Intern Med*. 1993;119:1187–1197.
89. Wellens HJ, Gorgels AP, de Munter H. Cardiac arrest outside of a hospital: how can we improve results of resuscitation? *Circulation*. 2003;107:1948–1950.
90. Grace AA SW, Hood MA. A prospective, randomized comparison in humans of defibrillation efficacy of a standard transvenous ICD system with a totally subcutaneous ICD system (The S-ICD® System). Paper presented at: 2005 Heart Rhythm Society Scientific Sessions; May 4–7, 2005; New Orleans, La.
91. Waldo A, Wit A. Mechanisms of cardiac arrhythmias and conduction disturbances. In: Fuster V, Alexander RW, O'Rourke RA, eds. *Hurst's the Heart*. 10th ed. New York, NY: McGraw Hill; 2001:751–796.
92. Sahadevan J, Ryu K, Peltz L, Khrestian CM, Stewart RW, Markowitz AH, Waldo AL. Epicardial mapping of chronic atrial fibrillation in patients: preliminary observations. *Circulation*. 2004;110:3293–3299.
93. Jalife J, Berenfeld O, Mansour M. Mother rotors and fibrillatory conduction: a mechanism of atrial fibrillation. *Cardiovasc Res*. 2002;54:204–216.
94. Yoshida K, Chugh A, Ulfarsson M, Good E, Kuhne M, Crawford T, Sarrazin JF, Chalfoun N, Wells D, Boonyapisit W, Veerareddy S, Bilalakanty S, Wong WS, Jongnarangsin K, Pelosi F Jr, Bogun F, Morady F, Oral H. Relationship between the spectral characteristics of atrial fibrillation and atrial tachycardias that occur after catheter ablation of atrial fibrillation. *Heart Rhythm*. 2009;6:11–17.
95. Taberoux PB, Dossall DJ, Ideker RE. Mechanisms of VF maintenance: wandering wavelets, mother rotors, or foci. *Heart Rhythm*. 2009;6:405–415.

KEY WORDS: cardioversion ■ defibrillation ■ fibrillation ■ heart arrest ■ tachyarrhythmias

Cardioversion: Past, Present, and Future
Ivan Cakulev, Igor R. Efimov and Albert L. Waldo

Circulation. 2009;120:1623-1632

doi: 10.1161/CIRCULATIONAHA.109.865535

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

Copyright © 2009 American Heart Association, Inc. All rights reserved.

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

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

<http://circ.ahajournals.org/content/120/16/1623>

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

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

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