The Predictive Value of Ventricular Fibrillation Electrocardiogram Signal Frequency and Amplitude Variables in Patients with Out-Of-Hospital Cardiac Arrest

Hans-Ulrich Strohmenger, MD*, Trygve Eftestol, PhD†, Kjetil Sunde, MD, PhD‡, Volker Wenzel, MD*, Mechthild Mair, MD*, Hanno Ulmer, PhD§, Karl H. Lindner, MD*, and Petter A. Steen, MD, PhD‡

Departments of *Anesthesiology and Critical Care Medicine and §Biostatistics, Leopold-Franzens University, Innsbruck, Austria; †Department of Electrical and Computer Engineering Hogskolen i Stavanger, Stavanger, Norway; and ‡Department of Anesthesiology, Ulleval University Hospital, Oslo, Norway

We evaluated ventricular fibrillation frequency and amplitude variables to predict successful countershock, defined as pulse-generating electrical activity. We also elucidated whether bystander cardiopulmonary resuscitation (CPR) influences these electrocardiogram (ECG) variables. In 89 patients with out-of-hospital cardiac arrest, ECG recordings of 594 countershock attempts were collected and analyzed retrospectively. By using fast Fourier transformation analysis of the ventricular fibrillation ECG signal in the frequency range 0.333–15 Hz (median [range]), median frequency, dominant frequency, spectral edge frequency, and amplitude were as follows: 4.4 (2.4–7.5) Hz, 4.0 (0.7–7.0) Hz, 7.7 (3.7–13.7) Hz, and 0.94 (0.24–1.95) mV, respectively, before successful countershock ($n = 59$). These values were 3.8 (0.8–7.7) Hz ($P = 0.0002$), 3.0 (0.3–9.7) Hz ($P < 0.0001$), 7.3 (2.0–14.0) Hz ($P < 0.05$), and 0.53 (0.03–3.03) mV ($P < 0.0001$), respectively, before unsuccessful countershock ($n = 535$). In patients in whom bystander CPR was performed ($n = 51$), ventricular fibrillation frequency and amplitude before the first defibrillation attempt were higher than in patients without bystander CPR ($n = 38$) (median frequency, 4.4 [2.4–7.5] vs 3.7 [1.8–5.3] Hz, $P < 0.0001$; dominant frequency, 3.8 [0.9–7.7] vs 2.6 [0.8–5.9] Hz, $P < 0.0001$; spectral edge frequency, 8.4 [4.8–12.9] vs 7.2 [3.9–12.1] Hz, $P < 0.05$; amplitude, 0.79 [0.06–4.72] vs 0.67 [0.16–2.29] mV, $P = 0.0647$). Receiver operating characteristic curves demonstrate that successful countershocks will be best discriminated from unsuccessful countershocks by ventricular fibrillation amplitude (3000-ms epoch). At 73% sensitivity, a specificity of 67% was obtained with this variable.

(Anesth Analg 2001;93:1428–33)

Current international guidelines for management of ventricular fibrillation lack practical, reliable, real-time, and, especially, noninvasive variables to guide countershock strategies and concurrent drug therapy during cardiopulmonary resuscitation (CPR). In fact, the timing for defibrillation attempts recommended in the current CPR guidelines (1) is not based on scientific evidence and does not address individual response to therapy. Without any information on the situation in the myocardium, the window of opportunity to convert ventricular fibrillation into a supraventricular rhythm (SVR) may simply be missed and the patient dies, whereas in another case, a single lucky shock may be the key to survival. Thus, a noninvasive variable obtained during CPR that could reliably predict countershock success would be highly desirable.

In animal studies, the noninvasive variables median frequency, dominant frequency, and amplitude from the ventricular fibrillation electrocardiogram (ECG) signal correlate with myocardial blood flow during CPR and reliably predict defibrillation success (2–5). In patients undergoing open heart surgery or with out-of-hospital cardiac arrest, ventricular fibrillation median frequency, dominant frequency, and amplitude are predictive of successful countershock, which was, however, defined only as a stable SVR (6,7). Unfortunately, most studies evaluating defibrillation success lack fundamental, important information,

Supported in part by Laerdal Medical AS, Stavanger, Norway.
At the time of the study, Petter A. Steen was a Board member of Laerdal, and Trygve Eftestol was a part-time employee of Laerdal.
Accepted for publication August 7, 2001.
Address correspondence and reprint requests to Hans-Ulrich Strohmenger, MD, Department of Anesthesiology and Critical Care Medicine, Leopold-Franzens University, Anichstrasse 35, 6020 Innsbruck, Austria. Address e-mail to hans.strohmenger@uibk.ac.at.

0003-2999/01

namely, whether successful defibrillation resulted in an SVR with or without return of spontaneous circulation and stable arterial blood pressure. In our previous reports, for example, we were unable to obtain this information because either patients were on cardiopulmonary bypass so that individual cardiocirculatory variables could not be measured or exact arterial blood pressure data were not available in out-of-hospital cardiac arrest patients. Thus, to improve evaluation validity of defibrillation success, an end point that correlates not only with a certain ECG rhythm, but also with return of spontaneous circulation, would be strongly desirable. The purpose of this study, therefore, was to assess, in patients with out-of-hospital cardiac arrest, whether ventricular fibrillation median frequency, dominant frequency, spectral edge frequency, or amplitude variables are predictive of a successful countershock, defined as subsequent coordinated electrical activity with return of spontaneous circulation of any duration, as recorded in a regular Utstein registry (8). Our hypothesis was that there would be no difference between variables with regard to study end points.

Methods

Approval for this study was obtained from the Regional Committee for Research Ethics, Health Region III, Oslo, Norway, and the Norwegian Data Inspectorate. The data are part of an observational prospective study of patients with out-of-hospital cardiac arrest (9) between February 21, 1996, and April 4, 1997, when advanced cardiac life support management, patient ECG, and demographic data were documented for this study. Oslo, the capital of Norway, has a population of approximately 500,000 and an emergency medical service with an average response time of 8 min (9). Advanced cardiac life support was performed by paramedics and emergency physicians of the Oslo emergency medical service according to recommendations of the European Resuscitation Council (10). In brief, the first three countershocks were performed with an energy level of 200, 200, and 360 J; all subsequent countershocks were performed with an energy level of 360 J. A semiautomatic defibrillator (Heartstart 3000; Laerdal, Stavanger, Norway) was used for countershock therapy and simultaneous on-line recording.

The self-adhesive ECG/defibrillation electrodes were attached on the patient's skin to conform with standard lead II configuration. The ECG data were stored in medical control modules in digitized form (sampling rate, 100 Hz; frequency resolution, 0.33 Hz), including defibrillator-related log data up to a total duration of 20 min. A noncommercial software package (Graphical User Interface) developed in a MATLAB (Mathworks, Nantucket, MA) environment, was used for high-performance numeric computation and visualization. In particular, all ECG data were transformed into analog ECG tracings, including medical control module log data, in which cardiac rhythm before and after each countershock attempt was identified. After a CPR attempt, Utstein-style related demographic data of the cardiac arrest patient and advanced cardiac life support management were entered into a database by one of the authors (KS); subsequently, analog ECG tracings were systematically reviewed by three investigators. Thus, postcountershock ECG rhythms, return of spontaneous circulation, and advanced cardiac life support time intervals were interpreted. They were then linked with the medical control module and data of the exact advanced cardiac life support procedures of each individual.

The predominant rhythm within the first 10 s after each shock was categorized as follows: 1) persistent ventricular fibrillation, 2) SVR (narrow QRS rhythm or wide QRS rhythm with associated or disassociated atrial activity) with a pulse, 3) idioventricular rhythm/pulseless electrical activity (EMD) (wide QRS rhythms at any rate, but no atrial activity present), and 4) asystole. A countershock was regarded as successful when ventricular fibrillation was converted to an SVR, generating a pulse regardless of the duration without continuing CPR. The decision of whether a certain cardiac rhythm was generating a pulse was based on the Utstein guidelines. By use of the mathematical software package MATLAB, the fast Fourier transformation, a processing method that enables dividing a periodic signal into its frequency components, was used for analyzing frequency content for an arbitrary period of the ventricular fibrillation ECG signal just before a given countershock. Accordingly, median frequency, dominant frequency, and edge frequency are single-valued variables that describe the frequency distribution of the resulting power spectrum. Median frequency is the frequency at which half of the power of the spectrum is above and half below. Dominant frequency is the frequency corresponding to the power spectrum maximum, and edge frequency is the frequency below which 95% of the area under the power spectrum curve resides. In addition, measurement of amplitude was made from the original time domain ECG signal by calculating the difference between the maximum and the minimum amplitude for each segment of the ECG just before the countershock. Frequency and amplitude content for each 3000- and 9000-ms period immediately before a given countershock were analyzed. Each countershock was analyzed as an independent event, because the purpose of this study was to predict the result of each countershock independent of clinical variables that may affect outcome. Because a witnessed collapse, bystander CPR, or both may have an important effect on ECG variables and subsequent outcome, these variables were determined throughout the 3000-ms interval before the very first defibrillation attempt and analyzed separately.

Variables were checked for normal distribution by using a Kolmogorov-Smirnov test with the Lilliefors correction. Because most variables showed small but significant deviations from normal distribution, we used nonparametric testing and median (range) for describing the data. A Kolmogorov-Smirnov *z*-test was used to assess significant differences in these variables regarding the result of the countershock, witnessed collapse, and bystander CPR. For investigating correlations between ventricular fibrillation ECG variables and arrest time, the correlation coefficient of Spearman (r) was used. Statistical difference was considered at $P < 0.05$. Receiver operating characteristic (ROC) curves, including the areas under the curves (AUC), were performed to evaluate sensitivity and specificity for different thresholds of the ECG variables with regard to successful versus unsuccessful countershocks. An ROC curve demonstrates the relationship of sensitivity and specificity at various points and shows how well successful countershocks will be discriminated from unsuccessful countershocks by each ECG variable. An AUC of 1.0 would represent a perfect discrimination; an AUC of 0.5 refers to a case with no discrimination at all.

Results

During the study period, 594 countershocks were performed in 89 patients; 59 (9.9%) shocks resulted in an SVR with a pulse (Table 1). With a frequency range of 0.333–15 Hz, values of median frequency, dominant frequency, and amplitude during the 3000- and 9000-ms period before the 59 successful countershocks were significantly higher than those before the 535 unsuccessful countershocks (Table 2). The areas under the ROC curves for each ECG variable are shown in Table 3 and demonstrate that successful countershocks will be best discriminated from unsuccessful countershocks by the ventricular fibrillation amplitude (3000-ms epoch). At 73% sensitivity, a specificity of 67% was obtained with this variable at a threshold value of 0.73 mV (Table 4).

Fifty-nine shocks resulted in an SVR, 214 shocks in EMD, and 79 shocks in asystole, whereas ventricular fibrillation persisted after 242 shocks. Median (range) of median frequency, dominant frequency, spectral edge frequency, and amplitude of ventricular fibrillation during the 3000-ms epoch before the first defibrillation attempt resulting in an SVR were 4.4 (2.4–7.5) Hz, 4.0 (0.7–7.0) Hz, 7.7 (3.7–13.7) Hz, and 0.94 (0.26–1.95) mV, respectively. In contrast, median frequency, dominant frequency, spectral edge frequency, and amplitude before unsuccessful defibrillations were 3.9 (1.4–7.7) Hz ($P < 0.001$ versus SVR), 3.0 (0.3–8.0) Hz ($P < 0.0001$ versus SVR), 7.7 (2.8–12.4) Hz (not significant), and 0.64 (0.05–3.04) mV ($P < 0.0001$ versus SVR), respectively, when persistent ventricular fibrillation resulted; 3.8 (0.9–7.3) Hz ($P < 0.0001$ versus SVR), 3.2 (0.3–9.7) Hz ($P < 0.0001$ versus SVR), 7.3 (2.0–13.3) Hz (not significant), and 0.55 (0.03–2.80) mV ($P < 0.0001$ versus SVR), respectively, when EMD resulted; and 3.8 (0.9–6.4) Hz ($P < 0.01$ versus SVR), 3.0 (0.3–8.3) Hz ($P < 0.001$ versus SVR), 7.3 (3.0–11.7) Hz (not significant), and 0.48 (0.05–1.96) mV ($P < 0.0001$ versus SVR, $P < 0.05$ versus ventricular fibrillation and EMD), respectively, when asystole resulted.

Table 1. Patient Characteristics of 89 Patients Undergoing Advanced Cardiac Life Support in the Oslo, Norway, Emergency Medical Service Between February 21, 1996, and April 4, 1997

Variable	Data
Age (yr)	68.5 (18–96)
Male sex (*n*)	74 (83%)
Arrest because of cardiac cause (*n*)	69 (78%)
Arrest because of noncardiac cause (*n*)	20 (22%)
Collapse witnessed (*n*)	71 (80%)
Collapse unwitnessed (*n*)	18 (20%)
Bystander CPR performed (*n*)	51 (57%)
No bystander CPR performed (*n*)	38 (43%)
Presenting ECG rhythm ventricular fibrillation (*n*)	59 (66%)
Presenting ECG rhythm asystole (*n*)	25 (28%)
Presenting ECG rhythm other or unknown (*n*)	5 (6%)
Interval collapse first defibrillation (min)	14 (0–34)
Interval CPR initiation first defibrillation (min)	6.0 (0–32.5)
Successful defibrillation: rhythm and pulse (*n*)	59 (9.9%)
Unsuccessful defibrillation: no rhythm, no pulse (*n*)	535 (90.1%)

All data are given in median (range) or *n* (%). CPR = cardiopulmonary resuscitation; ECG = electrocardiogram.

In patients for whom bystander CPR was performed ($n = 51$), ventricular fibrillation frequency variables during the 3000-ms epoch before the first defibrillation attempt were significantly higher than in patients without bystander CPR ($n = 38$) (median frequency, 4.4 [2.4–7.5] vs 3.7 [1.8–5.3] Hz, $P < 0.0001$; dominant frequency, 3.8 [0.9–7.7] vs 2.6 [0.8–5.9] Hz, $P < 0.0001$; spectral edge frequency, 8.4 [4.8–12.9] vs 7.2 [3.9–12.1] Hz, $P < 0.05$). With respect to ventricular fibrillation amplitude, there was a marked trend toward increased values in patients with bystander CPR (0.79 [0.06–4.72] vs 0.67 [0.16–2.29] mV; $P = 0.0647$).

In comparison to patients with unwitnessed collapse, ventricular fibrillation amplitude values in patients with witnessed arrest were significantly higher (0.83 [0.18–4.27] mV vs 0.34 [0.06–2.94] mV; $P < 0.01$),

Table 2. Ventricular Fibrillation Frequency and Amplitude Variables During a 3000- and 9000-ms Period Before Successful and Unsuccessful Countershocks in the Frequency Range 0.333 to 15 Hz

Electrocardiogram variable	Successful (n = 59)		Unsuccessful (n = 535)		
	Median	Range	Median	Range	P value
3000-ms period					
MF (Hz)	4.4	2.4–7.5	3.8	0.8–7.7	0.0002
DF (Hz)	4.0	0.7–7.0	3.0	0.3–9.7	<0.0001
EF (Hz)	7.7	3.7–13.7	7.3	2.0–14.0	0.0228
A (mV)	0.94	0.24–1.95	0.53	0.03–3.03	<0.0001
9000-ms period					
MF (Hz)	4.6	2.1–6.7	3.9	1.5–7.5	0.0004
DF (Hz)	4.0	0.7–7.3	3.1	0.2–9.1	<0.0001
EF (Hz)	8.1	3.6–12.9	7.7	2.4–12.6	0.4505
A (mV)	1.08	0.34–2.09	0.70	0.06–4.72	<0.0001

MF = median frequency; DF = dominant frequency; EF = edge frequency; A = amplitude.

Table 3. Area Under the Receiver Operating Characteristic (ROC) Curve of Electrocardiogram Variables for Prediction of Countershock Success

ECG variable	Area under the ROC curve (95% confidence interval)
3000-ms period	
MF (Hz)	0.664 (0.598–0.730)
DF (Hz)	0.662 (0.600–0.724)
EF (Hz)	0.578 (0.498–0.659)
A (mV)	0.738 (0.678–0.798)
9000-ms period	
MF (Hz)	0.663 (0.593–0.733)
DF (Hz)	0.692 (0.631–0.752)
EF (Hz)	0.567 (0.487–0.646)
A (mV)	0.730 (0.670–0.791)

ECG = electrocardiogram; MF = median frequency; DF = dominant frequency; EF = edge frequency; A = amplitude.

Table 4. Optimal Electrocardiogram Variable Thresholds with Regard to Countershock Evaluated by Receiver Operating Characteristic Analysis

Electrocardiogram variable	Threshold	Sensitivity (%)	Specificity (%)
3000-ms period			
MF (Hz)	3.9	78	52
DF (Hz)	3.2	81	51
EF (Hz)	8.8	44	76
A (mV)	0.73	73	67
9000-ms period			
MF (Hz)	4.1	71	57
DF (Hz)	2.8	92	42
EF (Hz)	9.4	29	82
A (mV)	0.83	80	63

MF = median frequency; DF = dominant frequency; EF = edge frequency; A = amplitude.

whereas no significant difference in ventricular fibrillation frequency variables was found between these groups (data not presented). In patients with witnessed collapse, there was a weak but significant negative correlation between cardiac arrest time and dominant frequency ($r = -0.23$; $P < 0.05$; $n = 71$) and ventricular fibrillation amplitude ($r = -0.47$; $P < 0.0001$; $n = 71$), but not between cardiac arrest time and median frequency ($r = -0.14$; $P = 0.17$; $n = 71$) or spectral edge frequency ($r = -0.14$; $P = 0.18$; $n = 71$) before the first countershock attempt. In patients with witnessed arrest, there was a trend toward increased success rate of the first countershock (restoration of spontaneous circulation rate, 10 of 71 vs 0 of 18) in comparison to patients with unwitnessed collapse ($\chi^2 = 0.09$).

Discussion

Analysis of 594 shocks performed in 89 patients with out-of-hospital cardiac arrest indicates that successful countershocks will be best discriminated from unsuccessful countershocks by ventricular fibrillation amplitude; namely, a 73% sensitivity and a specificity of 67% were obtained with this variable. Furthermore, bystander CPR increased ventricular fibrillation frequency and amplitude variables. It is interesting that there were significant differences in ventricular fibrillation amplitude, but not in frequency variables, between witnessed and unwitnessed collapse.

The results of our study are in full agreement with the results of several previous laboratory investigations that have demonstrated that ventricular fibrillation amplitude, median frequency, and dominant frequency before successful defibrillation attempts were significantly higher than those before unsuccessful countershocks. This study further confirms clinical observations that in patients with coarse ventricular fibrillation, countershock success is more likely than in patients with fine ventricular fibrillation (11). The burden of evidence for a given CPR intervention is usually at least hospital admission, but hospital discharge should not be lost as the true goal of CPR (1). It is interesting that these rules have not been applied in some recent defibrillation studies, in which the end point of several studies of mono- versus biphasic defibrillation, for example, has been termination of ventricular fibrillation, but not return of spontaneous circulation (12). Thus, EMD would be interpreted as a success, which clinically certainly would be false and could cause a significant statistical error. Results of some human studies evaluating ventricular fibrillation wave forms therefore remain open to discussion, because it is not known whether a postshock SVR was associated with spontaneous circulation or not. In this article, we defined successful defibrillation as an SVR combined with a palpable pulse, arterial blood pressure, or both; as such, both median and dominant frequencies before successful versus unsuccessful countershocks were significantly higher. Accordingly, evaluation of ventricular fibrillation variables alone or in combination can serve as an objective noninvasive measurement for predicting not only electrical success as termination of ventricular fibrillation (2,4,5), but also return of spontaneous circulation. Although these results are promising, the predictive values of median frequency and amplitude variables in this study sample were disappointingly low and clearly inferior to results reported by our working group (7) and others (13). It could be that besides outcome definitions, frequency band measurements, study design, or data set size, the more sophisticated data acquisition possible in the laboratory setting as compared with that in clinical studies, such as sampling rates of 1000 vs 100 Hz, and 12- vs 8-bit resolution, is the main underlying reason for this discrepancy. Because of the large sample size in our database, the results of our study nevertheless seem reliable.

Most of the previous studies evaluating ventricular fibrillation wave forms have not addressed the duration of basic or advanced cardiac life support. This may be of fundamental importance, because bystander CPR or interventions administered by the emergency medical service may, of themselves, have an important effect on defibrillation success or failure. For example, defibrillating a nonperfused fibrillating heart is an established model to produce postcountershock EMD (14), whereas three minutes of basic life support before countershock, instead of immediate defibrillation upon arrival of rescuers at the scene, correlated with significantly improved short-term survival in a recent large clinical study (15). This could indicate that basic life support contributes more to defibrillation success than previously thought. In fact, in a recent laboratory study, we have shown that three minutes of basic life support improved median frequency to a level that usually correlates with successful defibrillation (16). The data of the present study are in agreement with these observations, because bystander CPR increased ventricular fibrillation median and dominant frequency from 3.7 (1.8–5.3) Hz to 4.4 (2.4–7.5) Hz ($P < 0.0001$) and from 2.6 (0.8–5.9) Hz to 3.8 (0.9–7.7) Hz ($P < 0.0001$), respectively, and increased the return of spontaneous circulation rates from ~8% to ~14% ($P = 0.09$), but unfortunately lacked power to yield statistical significance.

Progressive ischemia caused by prolonged cardiac arrest results in rapid depletion of high-energy myocardial phosphate stores (17,18), intracellular calcium overload (19), decrease in ventricular fibrillation frequency and amplitude variables (11,20–22), and increased likelihood of asystole or EMD after countershock (11,23). Patients with witnessed collapse in our study had a weak but significant negative correlation between cardiac arrest time and both dominant frequency and ventricular fibrillation amplitude; this indicates that these variables are related to the duration of untreated cardiac arrest. Because of the characteristic variation of median frequency over time with two frequency peaks (21), it is not surprising that there was no significant correlation between median frequency and arrest time.

The good news in this study is that 28 (31%) of 89 patients had return of spontaneous circulation, but the bad news is that 535 (90%) of 594 countershocks did not result in spontaneous circulation, but were useless and probably harmful in causing thermal damage to the heart (24). This is actually of considerable clinical relevance, because in this study alone, a cumulative energy of 535 unsuccessful shocks with an estimated power of ~200 J each would be a stunning ~107.000 J, causing possible iatrogenic myocardial injury. Thus, as suggested by a laboratory study (24), defibrillation has to be considered as an intervention that not only saves lives, but may also be an underlying mechanism for cardiac failure in the postresuscitation phase. Accordingly, it is even more important not to defibrillate during CPR when rescuers guess that the time is right, but, rather, when scientifically proven variables indicate optimal timing. It is hoped that, in a not too distant future, microprocessor-based technology for ventricular fibrillation ECG signal analysis could be permanently integrated with a defibrillator for guiding countershock therapy by using predetermined frequency/amplitude values.

There are several limitations of this study that should be noted. First, the predictive power of variables such as ventricular fibrillation median frequency is clearly inferior in humans compared with data from laboratory experiments, because absolute values in humans, but not in animals, are closer to a range that is heavily disturbed by oscillatory overtones of chest compression variables. However, it is remarkable that the variation of amplitude variables in our study was small, despite a significant influence of electrode positions on ventricular fibrillation amplitude. Second, we are unable to determine whether defibrillation success would have been better had we not used the shock option of the automatic external defibrillators being used in the study. Third, all defibrillators administered monophasic, exponential wave form shocks; therefore, we cannot say whether outcome with biphasic wave form defibrillators would have been superior, as suggested by recent clinical studies (25). Fourth, the fact that a noninvasive variable could predict countershock success does not necessarily mean that the chance of surviving sudden cardiac arrest improves as well. Finally, we analyzed several ventricular fibrillation wave form variables in regard to their predictive power alone, but not in combination. In fact, it is possible that combining, for example, the most powerful predictors into a new denominator would yield better results. However, similar to developing a pharmacologic CPR "cocktail," this may be much more difficult than anticipated because of multiple permutations between variables. The most desirable algorithm would be a strategy that greatly enhances predictive power compared with current variables, and it would need to be less prone to being disturbed by CPR-related artifacts.

In conclusion, the results of this study of 594 shocks in 89 patients with out-of-hospital cardiac arrest indicate that median frequency, dominant frequency, and ventricular fibrillation amplitude values before successful countershocks were significantly higher than those before unsuccessful countershocks. This study further demonstrates that successful countershocks will be best discriminated from unsuccessful countershocks by ventricular fibrillation amplitude; at 73% sensitivity, a specificity of 67% was obtained with this variable. However, because of the low predictive power of the variables used, further studies that include alternative variables (e.g., new feature-extraction techniques such as nonlinear methods) should be performed before this method can be applied to the treatment of human cardiac arrest.

References

1. Guidelines for cardiopulmonary resuscitation and emergency cardiovascular care: International Consensus on Science. Circulation 2000;102:I1–I284.
2. Noc M, Weil MH, Tang W, et al. Electrocardiographic prediction of the success of cardiac resuscitation. Crit Care Med 1999;27:708–14.
3. Noc M, Weil MH, Gazmuri RJ, et al. Ventricular fibrillation voltage as a monitor of the effectiveness of cardiopulmonary resuscitation. J Lab Clin Med 1994;124:421–6.
4. Strohmenger HU, Lindner KH, Keller A, et al. Effects of graded doses of vasopressin on median fibrillation frequency in a porcine model of cardiopulmonary resuscitation: results of a prospective randomized controlled trial. Crit Care Med 1996;24:1360–5.
5. Brown CG, Griffith RF, Lighten PV, et al. Median frequency: a new parameter for predicting the defibrillation success rate. Ann Emerg Med 1991;20:787–9.
6. Strohmenger HU, Lindner KH, Lurie KG, et al. Frequency of ventricular fibrillation as a predictor of defibrillation success during cardiac surgery. Anesth Analg 1994;79:434–8.
7. Strohmenger HU, Lindner KH, Brown CG. Analysis of the ventricular fibrillation ECG signal amplitude and frequency parameters as predictors of countershock success in humans. Chest 1997;111:584–9.
8. Recommended guidelines for uniform reporting of data from out-of-hospital cardiac arrest: the 'Utstein style'—prepared by a Task Force of Representatives from the European Resuscitation Council, American Heart Association, Heart and Stroke Foundation of Canada, Australian Resuscitation Council. Resuscitation 1991;22:1–26.
9. Sunde K, Eftestol T, Askenberg C, Steen PA. Quality assessment of defibrillation and advanced life support using data from the medical control module of the defibrillator. Resuscitation 1999;41:237–47.
10. Robertson C, Stehen P, Adgey J, et al. The European Resuscitation Council guidelines for adult advanced life support. Resuscitation 1998;37:81–90.
11. Weaver WD, Cobb LA, Dennis L, et al. Amplitude of ventricular fibrillation waveform and outcome after cardiac arrest. Ann Intern Med 1985;102:53–5.
12. Bardy GH, Marchlinski FE, Sharma AD, et al. Multicenter comparison of truncated biphasic shocks and standard damped sine wave monophasic shocks for transthoracic ventricular defibrillation: Transthoracic Investigators. Circulation 1996;94:2507–14.
13. Brown CG, Dzwonczyk R. Signal analysis of human electrocardiogram during ventricular fibrillation: frequency and amplitude parameters as predictors of successful shock. Ann Emerg Med 1996;17:426–37.
14. Wenzel V, Lindner KH, Prengel AW, et al. Vasopressin improves vital organ blood flow after prolonged cardiac arrest with postcountershock pulseless electrical activity in pigs. Crit Care Med 1999;27:486–92.
15. Cobb LA, Fahrenbruch CE, Walsh TR, et al. Influence of cardiopulmonary resuscitation prior to defibrillation in patients with out-of-hospital ventricular fibrillation. JAMA 1999;281:1182–8.
16. Achleitner U, Wenzel V, Strohmenger HU, et al. Assessment of basic life support efficacy performing ventricular fibrillation waveform analysis. Resuscitation. In press.
17. Neumar RW, Brown CG, Robitaille RL, et al. Myocardial high energy phosphate metabolism during ventricular fibrillation with total circulatory arrest. Resuscitation 1990;19:199–226.
18. Angelos MG, Torres CA, Waite MD, et al. Left ventricular myocardial adenosine triphosphate changes during reperfusion of ventricular fibrillation: the influence of flow and epinephrine. Crit Care Med 2000;28:1503–8.
19. Clusin WT, Buchbinder M, Ellis AK, et al. Reduction of ischemic depolarization by the calcium channel blocker diltiazem. Circ Res 1984;54:10–20.
20. Brown CG, Dwonczyk HA, Werman HA, et al. Estimation the duration of ventricular fibrillation. Ann Emerg Med 1989;18:1181–5.
21. Martin RD, Brown CG, Dzwonczyk R. Frequency analysis of the human and swine electrocardiogram during ventricular fibrillation. Resuscitation 1991;22:85–91.
22. Achleitner U, Wenzel V, Strohmenger HU, et al. The effects of repeated doses of vasopressin or epinephrine on ventricular fibrillation in a porcine model of prolonged cardiopulmonary resuscitation. Anesth Analg 2000;90:1067–75.
23. Martin G, Cosin J, Such M, et al. Relation between power spectrum time course during ventricular fibrillation and electromechanical dissociation: effects of coronary perfusion and nifedipine. Eur Heart J 1986;7:560–9.
24. Xie J, Weil MH, Tang W, et al. High-energy defibrillation increases the severity of postresuscitation myocardial dysfunction. Circulation 1997;96:683–8.
25. Schneider T, Martens PR, Paschen H, et al. 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;102:1780–7.