

Adverse Outcomes of Interrupted Precordial Compression During Automated Defibrillation

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Background—Current versions of automated external defibrillators (AEDs) require frequent stopping of chest compression for rhythm analyses and capacitor charging. The present study was undertaken to evaluate the effects of these interruptions during the operation of AEDs.

Methods and Results—Ventricular fibrillation was electrically induced in 20 male domestic swine weighing between 37.5 and 43 kg that were untreated for 7 minutes before CPR was started. Defibrillation was attempted with up to 3 sequential 150-J biphasic shocks, but each was preceded by 3-, 10-, 15-, or 20-second interruptions of chest compression. The interruptions corresponded to those that were mandated by commercially marketed AEDs for rhythm analyses and capacitor charge. The sequence of up to 3 electrical shocks and delays were repeated at 1-minute intervals until the animals were successfully resuscitated or for a total of 15 minutes. Spontaneous circulation was restored in each of 5 animals in which precordial compression was delayed for 3 seconds before the delivery of the first and subsequent shocks but in none of the animals in which the delay was >15 seconds before the delivery of the first and subsequent shocks. Longer intervals of CPR interventions were required, and there was correspondingly greater failure of resuscitation in close relationship to increasing delays. The durations of interruptions were inversely related to the durations of subthreshold levels of coronary perfusion pressure. Postresuscitation arterial pressure and left ventricular ejection fraction were more severely impaired with increasing delays.

Conclusions—Interruptions of precordial compression for rhythm analyses that exceed 15 seconds before each shock compromise the outcome of CPR and increase the severity of postresuscitation myocardial dysfunction. (*Circulation*. 2002;106:368-372.)

Key Words: cardiopulmonary resuscitation ■ fibrillation ■ defibrillation ■ compression ■ myocardium

Automated external defibrillators (AEDs) provide the single best option for improving the currently poor outcomes of out-of-hospital cardiopulmonary resuscitation (CPR) performed by bystanders. Fundamental to the operation of AEDs is the capability of automated ECG rhythm analyses, during which CPR is interrupted. Previous experimental studies had demonstrated both decreased resuscitability and greater impairment of postresuscitation myocardial function and survival when precordial compression was interrupted for 20 seconds in small animals.¹⁻³ Because current versions of AEDs prompt “hands-off” intervals of >10 seconds for rhythm analyses before advising the rescuer to deliver an electrical shock, as documented below, we hypothesized that such interruptions would adversely affect the outcomes of CPR.

Threshold levels of coronary perfusion pressure (CPP) are identified as major determinants of successful cardiac resuscitation.⁴⁻⁷ Interruptions in precordial compression predictably result in declines in CPP and therefore delays in

restoring threshold values of CPP.^{5,6} We therefore anticipated that increasing the length of intervals of interrupted chest compression results in corresponding decreases in the success of CPR and greater severity of postresuscitation myocardial dysfunction.

These considerations prompted our group to quantify the effects of interruptions in chest compression comparable to or even less than those that are mandated for the operation of current versions of AEDs.

The experiments were performed on an established porcine model of prolonged cardiac arrest. Our hypothesis was that the initial success of attempted defibrillation, the preservation of myocardial function in resuscitated animals, and postresuscitation survival were each compromised by interruption of chest compressions of durations equal to or less than the interruptions mandated by currently used AEDs.

Methods

Initial trials were performed to document the durations of “hands-off” intervals mandated by current versions of AEDs. The time

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intervals extending from the verbal prompt “do not touch the patient” until the advisory prompts to “shock” or “do not shock” for each of 3 sequential shocks were measured with a conventional stopwatch. Three measurements were obtained by 3 independent observers for each of 3 commercially marketed AEDs with fully charged batteries.

The experiments were performed in an established porcine model of cardiac arrest. This pig model has consistently demonstrated cardiorespiratory alterations during CPR that were subsequently confirmed on human patients.^{8–12} All animals received humane care in compliance with the “Principles of Laboratory Animal Care” formulated by the National Society for Medical Research and the *Guide for the Care and Use of Laboratory Animals* prepared by the National Institutes of Health (Publication 86-32, revised 1985). Our Institute’s Animal Care and Use Committee approved the protocol. The Institute is fully accredited by the American Association for Accreditation of Laboratory Animal Care.

Animal Preparation

Male domestic pigs weighing 40 ± 2 kg (S & S Farms, Ranchita, Calif) were fasted overnight except for free access to water. Anesthesia was induced by injection of ketamine (20 mg/kg IM) and completed after injection of sodium pentobarbital (30 mg/kg IV). A cuffed endotracheal tube was advanced into the trachea. Animals were mechanically ventilated with a volume-controlled ventilator (model MA-1, Puritan-Bennett). End-tidal PCO_2 (PETCO_2) was monitored with an infrared capnometer (model 01R-7101A, Nihon Kohden Corp). Respiratory frequency was adjusted to maintain PETCO_2 between 35 and 40 mm Hg before cardiac arrest and after resuscitation. Additional doses of sodium pentobarbital (8 mg/kg) were administered at intervals of ≈ 1 hour or when necessary to maintain anesthesia.

For the measurement of left ventricular function, a 5-MHz echocardiographic biplane transducer with a 5-MHz continuous-wave Doppler transesophageal echocardiography sensor with 4-way flexure (model 21363A, Hewlett-Packard Co, Medical Products Group) was introduced into the esophagus for a distance of ≈ 35 cm from the incisor teeth. For the measurement of aortic pressure, a fluid-filled catheter was advanced from the surgically exposed right femoral artery into the thoracic aorta. For the measurement of right atrial pressure and for measurements of cardiac output by the thermodilution method, together with measurement of core (blood) temperature, a 7F, pentalum, thermodilution-tipped catheter (Abbott Critical Care 41216) was advanced from the surgically exposed right femoral vein and flow-directed into the pulmonary artery. For inducing ventricular fibrillation, a 5F pacing catheter (EP Technologies, Inc) was advanced from the right cephalic vein into the right ventricle until an ECG current of injury indicated endocardial contact. The catheter was subsequently advanced into the apex of the ventricle with the aid of an image intensifier. For the measurement of scalar ECG signals, 3 adhesive electrodes were applied to the shaved skin of the right and left infraclavicular areas and the left thigh.

Experimental Procedures

Just before cardiac arrest was induced, the animal was randomized to 1 of 4 time delays of interrupted precordial compression by the sealed-envelope method. Cardiac arrest was induced with 1-mA AC delivered to the endocardium of the right ventricle. The pacing wire was then withdrawn to avoid injury to the endocardium during chest compression. Seven minutes after onset of ventricular fibrillation, a fixed 150-J biphasic shock was delivered between adult external defibrillation pads with a Codemaster defibrillator (Codemaster XL, Hewlett-Packard Co). The electrodes were applied to the infraclavicular area and apex. Each shock was delivered after a delay of 3, 10, 15, or 20 seconds, contingent on the outcome of randomization. If spontaneous circulation was not restored, up to 3 shocks were delivered, each preceded by the specified interval of interrupted precordial compression. Precordial compression was then started with a mechanical chest compressor (Thumper, model 1000, Michigan Instruments) at 100 compressions per minute. Coincident with start of precordial compression, the animal was mechanically ventilated with a tidal volume of 15 mL/kg and an FIO_2 of 1.0. Chest

compression was synchronized to provide a compression/ventilation ratio of 5:1 with equal compression-relaxation intervals, ie, a 50% duty cycle. The compression force was adjusted to decrease the anteroposterior diameter of the chest by 25% so as to maintain CPP within the range of 15 ± 2 mm Hg. The sequence of shocks was repeated after 1 minute of chest compression until the animal was resuscitated or for a maximum of 15 minutes. The animal was regarded as successfully resuscitated when defibrillation resulted in (1) an organized cardiac rhythm, (2) return of spontaneous circulation (ROSC) with a mean aortic pressure of ≥ 60 mm Hg, and (3) maintaining mean 60 mm Hg arterial pressure at ≥ 60 mm Hg for an interval of ≥ 5 minutes. Postresuscitation cardiorespiratory measurements were continued for a duration of 4 hours after ROSC. At the end of 4 hours, catheters were removed and the wounds were surgically repaired. The animal was then returned to its cage and observed for a total of 72 hours. Except for the administration of butorphanol in doses of 0.1 mg/kg at intervals of 4 hours, no other supportive treatment was provided. After 72 hours, the animal was euthanized with an injection of 150 mg/kg pentobarbital IV. Autopsy was routinely performed for gross documentation of injuries to the bony thorax and the thoracic and abdominal viscera to identify injuries caused by CPR interventions or by obfuscating disease as previously described.¹³

Measurements

Hemodynamic data, including aortic, right atrial, and pulmonary artery pressure and PETCO_2 , together with the ECG, were continuously measured and recorded on a PC-based data acquisition system supported by CODAS hardware/software as previously described.^{13–15} The CPP was digitally computed from the differences in time-coincident diastolic aortic and right atrial pressures. Cardiac output was measured by conventional thermodilution techniques after injection of 5 mL of saline maintained between 0°C and 5°C. Echocardiographic measurements were obtained with the aid of a Hewlett-Packard Sonos 2500 ECG system with a 5-MHz biplane transducer with 4-way flexure (model 21363A, Hewlett-Packard Co, Medical Products Group). A long-axis 2- or 4-chamber view was obtained. Left ventricular end-diastolic and end-systolic volumes were calculated by the method of disks (Acoustic Quantification Technology, Hewlett-Packard). From these, ejection fractions, stroke volumes, and the rate of change of ventricular volumes were computed. These measurements served as quantifiers of myocardial function. Cardiac output was also calculated as the product of the flow-time-velocity integral at the aortic valve, valve diameter, and heart rate for comparison with the results obtained by the ventricular volume measurements and by the thermodilution method. These procedures have been described previously.^{13–16}

Statistical Analyses

The independent variables included the time delays represented by the 4 groups. The dependent variables included the duration and outcome of CPR, the total energy delivered before ROSC, and the measurements that quantified myocardial function. Data are presented as mean \pm SD. For measurements between groups, 1-way ANOVA was used. Comparisons among time-based measurements within each group were performed with ANOVA for repeated measurements. When the dependent variable was categorical, χ^2 and Fisher’s exact tests were used. A value of $P < 0.05$ was regarded as statistically significant.

Results

The measured “hands-off” intervals mandated for rhythm analyses and capacitor charging for each of these currently available AEDs ranged from 10 to 19.5 seconds.

Baseline hemodynamic, blood gas, and arterial lactate measurements were within the physiological ranges previously reported for this porcine model.^{2,14} No significant differences in heart rate, mean aortic pressure, arterial and mixed venous pH, PCO_2 , PO_2 , and arterial blood lactate

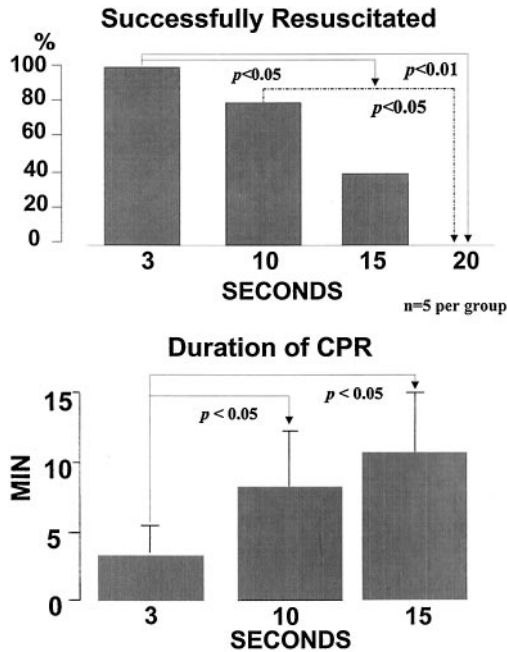


Figure 1. Top, Decreased success of CPR with increasing durations of interrupted chest compression. Bottom, Duration of CPR before ROSC was prolonged in relationship to increasing intervals of interrupted chest compression.

concentration were observed among baseline measurements in the 4 groups.

A progressive fall in the success of initial resuscitation was observed (Figure 1). ROSC was restored in all animals in which the duration of interruption was 3 seconds. This contrasted with 10-second and 15-second interruptions, in which the success of CPR was significantly reduced. No animals in which precordial compression was interrupted for 20 seconds were resuscitated. The differences were statistically significant between groups with the χ^2 test. With Fisher's exact test, a statistically significant difference was demonstrated only between the 3-second-interruption and the 20-second-interruption groups (Figure 1). The duration of the resuscitation effort before ROSC averaged 3.3 minutes after 3-second interruptions, 8.2 minutes after 10-second interruptions, and 10.8 minutes after 15-second interruptions. The differences were statistically significant (Figure 1).

Significantly higher postresuscitation mean arterial pressure values were observed in animals after 3 seconds of interruption than after longer interruptions (Figure 2). The ejection fraction was significantly reduced in all resuscitated animals but disproportionately so when interruptions exceeded 3 seconds. At the end of 72 hours, the ejection fraction was only 70% of baseline in animals after 15 seconds of interruption (Figure 3). Cardiac output measured during the 4 hours that followed successful resuscitation was maintained at baseline levels of 5 L/min. Disproportionate increases in heart rate, however, followed the longer intervals of interrupted chest compression (Figure 4). The greater heart rate explained the lack of differences in cardiac output. Each of the initially resuscitated animals survived and was fully alert at the end of 72 hours.

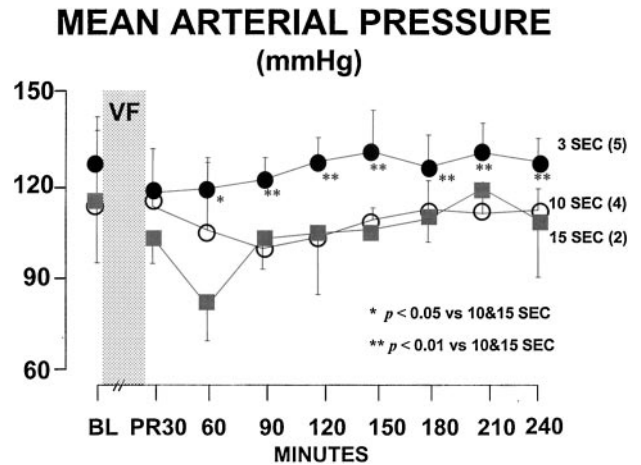


Figure 2. Decreases in postresuscitation mean aortic pressure associated with prolongation of interrupted of chest compression. (n) represents numbers of resuscitated animals. BL indicates baseline; PR, postresuscitation intervals.

The CPP was maintained below the threshold value of 12 mm Hg² for significantly longer intervals after 10-, 15-, and 20-second delays. Each of the differences among the 4 groups was significant (Figure 5). The reduced number of chest compressions connected linearly with failure to achieve threshold levels of CPP (Figure 6).

Discussion

Improvements in outcomes of cardiac resuscitation followed introduction of AEDs for use by minimally trained rescuers, especially in airports and casinos. At the same time, the priorities of CPR were reassessed such that defibrillation, airway, and breathing took priority over initiation of precordial compression.¹⁷⁻¹⁹ Yet, the priority of ventilation over precordial compression was challenged and defined as of questionable benefit in settings of "sudden death" based on both experimental and clinical investigations.^{2,20-23} In addition, there is increasing evidence that after 4 minutes of untreated cardiac arrest, precordial compression may best

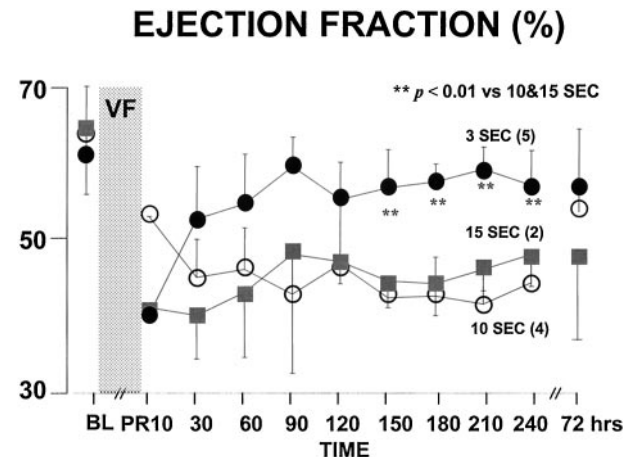


Figure 3. Comparison of ejection fractions among 3 groups. (n) represents numbers of resuscitated animals. BL indicates baseline; PR, postresuscitation intervals.

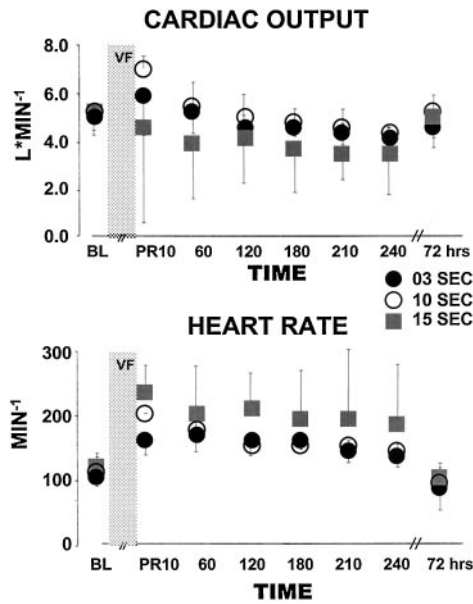


Figure 4. Comparisons between baseline and postresuscitation measurements of cardiac output and heart rate. BL indicates baseline; PR, postresuscitation intervals.

precede defibrillation.²⁴ This prompted us to conduct controlled preclinical studies with a focus on the benefits of chest compression and specifically the possibility that interruptions in chest compression may prove detrimental.

Chest compression was introduced to modern medicine in the 19th century but was widely adopted only after 1960.^{25,26} Chest compressions during cardiac arrest provide forward blood flow such that critical levels of myocardial and cerebral blood flows are preserved. CPP, as a surrogate for coronary blood flow, represents an indirect indicator of myocardial perfusion.⁸ Threshold values of CPP are maintained with uninterrupted chest compression. Delays for rhythm analyses and capacitor charging necessarily reduced the number of compressions over time and the capability to maintain threshold levels of CPP, as shown in Figure 5. This was previously demonstrated by both Berg et al²⁷ and Kern et al.²⁸ These investigators demonstrated that interrupting chest compressions for rescue breathing decreases CPP, myocardial blood flow, and 24-hour survival. The interruptions in

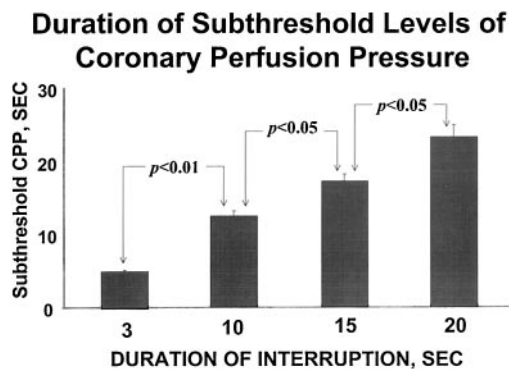


Figure 5. Relationship between durations of interruptions and subthreshold levels of CPP during initial 3 minutes of CPR.



Figure 6. Relationship between numbers of chest compressions delivered over first 3 minutes of CPR and either ROSC or failure of resuscitation (NR).

chest compression and their effects on myocardial perfusion translated into correspondingly lower success of the initial resuscitation effort, as shown in Figure 6. The critical impact of interruption and the resulting decreases in coronary perfusion are further reflected in postresuscitation myocardial function. Postresuscitation reductions in arterial pressure and ejection fractions provide additional evidence that reduced myocardial perfusion was associated with greater severity of postresuscitation myocardial dysfunction. Cardiac output was minimally affected after successful resuscitation because disproportionate increases in heart rate compensated for the observed decreases in stroke volumes. In confirmation of earlier investigations, postresuscitation myocardial dysfunction was largely reversed after 72 hours in the absence of interruption other than for <3 seconds during delivery of the shock.^{2,29} We therefore implicate interruptions in chest compression, resulting in critical reductions in myocardial perfusion and with exacerbation of global ischemic injury of the heart, as explanations for the outcomes, especially in settings in which ventricular fibrillation consumes disproportionately large myocardial oxygen demands.^{30,31} To this extent, the present studies only extend the well-established understanding that the longer the duration of myocardial ischemia, the greater is the impairment in myocardial function, whether it be a result of regional or global ischemia.³²⁻³⁴

We recognize that the design and operation of current AEDs that mandate “hands-off” and therefore interruptions in precordial compression compromise outcomes. Optimally, chest compressions would be uninterrupted and rhythm analyses would proceed without invalidation by artifacts. We therefore recognize an important incentive for perfecting methods of ECG analyses that proceed without loss of sensitivity and specificity, using advanced methods of signal processing to remove the artifacts produced by chest compression. We have partially achieved this.^{35,36} We also recognize the need for methods by which rescuers are protected from electrical shock when chest compression is uninterrupted. Our group has also addressed this need by development of methods of shielding rescuers without compromise to the capability to maintain effective chest compression.

We acknowledge several limitations in the present study and in the interpretation of the results. First, the number of animals enrolled and the experimental design precluded

statistical significance with respect to long-term survival. Because the majority of animals failed resuscitation efforts when interruptions exceeded 10 seconds, no data on survival or postresuscitation myocardial function was obtained in nonsurvivors. Second, our studies were performed on young, healthy, anesthetized animals free of underlying disease, and under conditions of optimal airway control and mechanical ventilation, conditions that are unlikely to occur in out-of-hospital settings of cardiac arrest. Finally, our studies were performed without any pharmacological interventions.

Within these constraints, the hypothesis is supported that interruptions of chest compression during CPR, as mandated by current versions of AEDs, compromise outcomes of CPR.

Acknowledgments

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