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Heart failure

GW23-e2650 **CONTINUOUS COMPRESSION WITHOUT DEFIBRILLATION FAVOURED NO SHORT-TERM SURVIVAL IN PROLONGED VENTRICULAR FIBRILLATION**

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Objectives Aims: During the 2005 American Heart Association (AHA) Consensus Conference, compression first versus defibrillation first for sudden cardiac arrest with ventricular fibrillation (VF) had drawn much interest. Some data challenged the standard

practice of providing defibrillation first, especially when 4–5 min or longer had elapsed between collapse and rescuer intervention. To allow rescuers the option of providing CPR first, particularly for out-of-hospital cardiac arrest when the response interval is estimated to be longer than 4–5 min, 1.5–3 min of CPR before defibrillation attempt could be considered for the purpose that heart can respond more favourably to a defibrillation attempt. However, the ideal duration of CPR before attempted defibrillation hasn't determined because of lacking sufficient data. Investigators had drawn conflicting conclusions on the durations of CPR before offering first defibrillation attempt. In this study, we aimed to compare strategies of 2- with 4-min CPR before defibrillation in a canine model of 12-min VF to evaluate efficacy on hemodynamic parameters and survival outcomes, then determine the optimal CPR duration prior to the first defibrillation attempt for prolong VF.

Methods Twenty adult mongrel dogs (12–18 kg) of either sex were bred more than 1 week by the anesthaesiologist, so that when anesthetised, they would not be anxious. All animals were then fasted overnight, but had free access to water. Anesthetised were induced with 3% sodium pentobarbital (30 mg/kg IV). The degree of anesthaesia was assessed by respiration rate, pulse rate and animal movement, and additional sodium pentobarbital was administered as necessary. The anesthaesia was used in all surgical interventions and unnecessary suffering was avoided. After anesthaesia, animals were placed in a supine position and incubated with a 5.5–6.0 cuffed endotracheal tube via direct laryngoscope. Dogs were mechanically ventilated to maintain normocapnia by use of a ventilator (Newport E-100M, Newport Medical Instruments, Costa Mesa, California, USA). Ventilation was begun at a tidal volume of 10–15 ml/kg, a ventilatory rate of 16–20 breaths/min and a ratio of inspiration to expiration of 1:1.5–2.0. Three surface electrodes configured to correspond to standard-lead II electrocardiography (ECG) were placed on shaven areas of the thorax and limbs. Continuous ECG was attained by use of an automated external biphasic waveform defibrillator (M4735, Philips, Eindhoven, Netherlands) and a multipurpose polygraph (PowerLab/16sp, AD instruments, Sydney, Australia). Two 6-F catheters (Cordis Corp, Miami, Florida, USA) were positioned in the ascending aorta and right atrium through the right femoral artery and vein, and catheter positions were verified by x-ray (CGO-3000, Beijing Wandong Medical Equipment, China). Arterial and venous pressures were monitored continuously by use of the multipurpose polygraph. Both ECG measurements and pressures were acquired digitally at a sampling rate of 1000 points/sec with use of commercially available polygraphy (Chart for Windows V.5.5, AD Instruments, Castle Hill, Australia). The aortic pressure (AOP) and right atrial pressure (RAP) were recorded at baseline and during CPR, com-AOP and com-RAP as compression pressures while decom-AOP and decom-RAP as decompression pressures. Coronary perfusion pressure (CPP) was calculated as AOP–RAP simultaneously during the decompression phase of CPR. Typically, 10 steady individual compressions within each minute of the initial 4 min of CPR were collected for mean pressure analysis. Every peak AOP was defined as apex of the compression phase and the lowest decompression point as the AOP trough. VF was induced by delivering a 5-sec alternating current at 50 Hz, 80-V externally across the thorax through two subcutaneous needle electrodes and was evaluated as (1) characteristic ECG waveform and (2) AOP <20 mm Hg. Assisted ventilation was discontinued when VF was established, which was untreated and allowed to persist for 12 min before experimental interventions began. After 12-min untreated VF, dogs were then randomised to manual CPR for 2 min or 4 min (figure 1). For the 2-min group (N=10), animals received 2 min of standard, closed-chest, manual CPR in the anterior/posterior position, a rate of 100–120 compressions/min demonstrated on the defibrillator and the polygraphy. Chest compressions were synchronised to provide a compression: ventilation ratio of 30:2. Ventilations were delivered by use of a conventional bag-valve technique. The interruptions to deliver rescue breaths were eliminated because the artificial air passage was established. After this first CPR cycle, dogs were quickly assessed for subsequent treatment: (1) if VF persisted, a biphasic 150 J countershock was delivered, followed by another 2 min of CPR without any post-shock rhythm or pulse assessment; (2) another 2 min of CPR begun immediately if ECG showed asystole or pulse-less electromechanical dissociation; (3) 1 mg/kg epinephrine was administered intravenously with AOP <80 mm Hg; (4) if ROSC was achieved (AOP≥80 mm Hg sustained for at least 1 min), advanced life support, as recommended by the 2005 AHA guidelines, was then applied. Resuscitation sequence was restarted immediately until ROSC was achieved or with a 30-min total resuscitation attempt. For the 4-min group (N=10), all interventions were identical except for 4-min CPR initiated immediately after 12-min untreated VF. During 4-min CPR, the first 1 mg epinephrine was administered in the second minute without interrupting CPR. For both groups, 1 mg epinephrine was administered intravenously after each assessment until ROSC was achieved or the 30-min resuscitation attempt was terminated. During the whole process of experiment, animals were given intravenous fluids to restore third-space fluid losses. After successful resuscitation, anesthaesia was maintained, ventilation was reconnected and advanced life support was continued to maintain AO≥80 mm Hg, then animals were monitored for another 2 h. Animals were euthanatised with an IV injection of 10% KCl.

Results AOP in the first 3 min were comparable between the two groups, but in the 4th minute of CPR, AOP was prominent higher in the 2-min group than the 4-min group (97.75±46.22 mm Hg vs 60.74±23.45 mm Hg, p=0.04). With comparable RAPs in both groups during the 4 min of CPR, CPP demonstrated a similar change to AOP, of which 2-min group also became prominent higher than the 4-min group in the 4th minute of CPR (44.03 ±27.21 mm Hg vs 10.92±23.28 mm Hg, p=0.01). In 2-min group, a tendency of more elevated AOP during CPR was observed, especially when necessary epinephrine and defibrillation were promptly administrated. Similar trend was also shown in CPP. However in the 4-min group, a steady AOP and a consequent CPP were demonstrated. With comparable epinephrine administration and defibrillation attempt in each group, six of ten dogs (60%) in the 2-min group achieved immediate ROSC, five of which survived for 2 h, while one dog failed to respond to resuscitation effort after rhythm restoration for 45 min. In the 4-min group, four of ten dogs (40%) achieved immediate ROSC with three survived for 2 h.

Conclusions Conclusion: Increasing the conventional 2 min CPR to 4 min before the first defibrillation attempt for prolonged VF did not improve survival rate in a 12-min canine VF model. As longer compression might compromise resuscitation effectiveness, 2 min of CPR prior to initial defibrillation was recommended.

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