Effects of Incomplete Chest Wall Decompression During Cardiopulmonary Resuscitation on Coronary and Cerebral Perfusion Pressures in a Porcine Model of Cardiac Arrest

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BACKGROUND: Recent data suggest that generation of negative intra-thoracic pressure during the decompression phase of CPR improves hemodynamics, organ perfusion and survival. We hypothesize that incomplete chest wall recoil during the decompression phase of standard CPR increases intrathoracic pressures and intracranial pressures resulting in a decrease in venous return in the chest cavity and therefore a decrease in coronary perfusion pressure, an increase in intracranial pressure and a decrease in cerebral perfusion pressures.

METHODS: 9 pigs in ventricular fibrillation for 6 min, were treated with an automated compression/decompression device with compressions at 100/min and a depth of 25% of the antero-postero diameter and a compression to ventilation ratio of 15:2 with 100% decompression (standard CPR) for 3 min. Compression depth was reduced to 75% of complete decompression for 1 min of CPR and then restored for another 1 min of CPR to 100% decompression. Coronary perfusion pressure (CPP) was calculated as the diastolic (aortic minus intracranial pressure). Cerebral perfusion pressure (CAPP) was calculated as diastolic (Aortic minus intracranial pressure). Unpaired t-test was used for statistical analysis. All pressures expressed as mmHg.

Results: With 100% - 75% - 100% chest wall recoil, the CPP was 23.5 ± 2.0, 15.0±1, 15.5±1 (P<0.01); CAPP was 8.5±3.4-9.2±4.7 (P<0.03); diastolic aortic pressure was 26.5±2.5, 19.3±2.1; Intracranial pressure during decompression was 18.1±2.4, 20.2±2.4, 16.6±2.3; RA diastolic pressure 3±1.8, 4.2±1.7, 3.17±1.4; and MAP was 41.6±3.3, 34.2±2.7, 38.6±2.9.

CONCLUSIONS: Incomplete chest wall recoil during the decompression phase of CPR improves hemodynamics, organ perfusion and survival.

A New Method for Detecting Cardiac Pulmonary Edema Before the Appearance of Clinical Signs and for the Evaluation of Treatment Efficacy

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The aim of this study was to investigate whether patients with heart disease can be effectively monitored by a new index, ITI (internal thoracic impedance), so that evolving cardiopulmonary edema (CPE) can be detected at its pre-clinical stage. If reliable, such monitoring could enable the early treatment of CPE, enhance its efficacy, and possibly abort the process of worsening pulmonary congestion. A new impedance monitor (RS-205) was used in this study. This monitor measures ITI by subtracting the skin-electrode contact impedance from total transthoracic impedance. The clinical onset of CPE was determined according to accepted clinical and radiographic criteria. ITI of the right side of the chest was measured every 30 minutes in 264 consecutive patients hospitalized for an acute coronary syndrome. In all 264 patients that comprised study population monitoring was initiated when clinical symptoms and signs of CPE were absent. 38 of these patients developed CPE during monitoring. In all 38 patients, ITI decreased by more than 12% from the initial value 30 minutes and in 23 of these patients it occurred one hour before the clinical onset of CPE, respectively (p<0.001). In light of these findings therapy was initiated in 4 patients when an ITI decrease of 11-12% from initial value was recorded. In these 4 patients, clinical CPE did not develop and ITI returned to initial value after treatment. In contrast, ITI did not decrease by more than 10% in any of the remaining 222 patients who did not develop CPE. Further evaluation of the impact of ITI monitoring on the clinical course of patients at risk to develop CPE seems warranted.

Conclusion: ITI monitoring was found to be an accurate, practical and reliable method to detect CPE at its pre-clinical stage and to gauge of the effectiveness of the treatment. The preliminary data in four patients show that treatment of CPE in its pre-clinical stage may abort CPE evolution.

Electrocardiogram Morphology Predictors of Pharmacologic Termination of Ventricular Tachycardia


Background: Pharmacologic termination of ventricular tachycardia (VT) is variable with different degrees of efficacy with different agents. This study was undertaken to determine if there are morphologic characteristics of VT that may predict drug efficacy.

Methods: 172 patients with sustained VT were randomized to receive either a 150 mg bolus of a new formulation of intravenous amiodarone (A) or a bolus of 100 mg lidocaine (L). If the first bolus failed to terminate the VT, a second bolus was administered. If the VT persisted, electroshock was delivered. In each patient, an electrocardiogram (EKG) was recorded before drug administration during VT and was analyzed for VT morphology: polymorphic vs monomorphic VT, QRS width, QRS amplitude, as well as ventricular rate. Multivariate analysis (ANOVA) was performed to assess the possible predictors of VT termination.

Results: Of the 172 patients, 29 had shock resistant VT. 143 had sustained hemodynamically unstable VT. Of the 94 patients who received A, 85 had monomorphic VT with 50 terminating pharmacologically (59%) and 9 had polymorphic VT with a 33% termination (NS). Of the 78 patient who received L, 74 had monomorphic VT with 44 (59%) pharmacologic VT termination and 4 had polymorphic VT with a 25% termination (p=0.05). For shock resistant monomorphic VT, the cumulative VT termination was 75% with A vs. 33% with L (p<0.05) and for shock resistant polymorphic VT 83% with A vs. 0% with L (p<0.05). The QRS was narrower in converters vs. non converters (141±27 ms vs. 175±54 ms, p=0.001). There was a trend of slower VT rate between converters vs. non converters (175±31 vs. 182±35 beats/minute, NS), but there was no difference in VT amplitude. In the 172 patients, the cumulative VT terminations (pharmacologic and electroshock) for A vs. L were 91% vs. 85% in monomorphic VT (NS) and 89% vs. 25% in polymorphic VT (p<0.01).

Conclusions: Amiodarone was more effective than L in converting VT in patients with...