Mechanical Response of the Left Ventricle During AC Induced Hemodynamic Collapse

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Abstract

Medical equipment can unintentionally allow the flow of small amounts of AC current through the patient causing hemodynamic collapse without fibrillation. This study examines the mechanical response of the left ventricle during AC induced hemodynamic collapse.

Six dogs received 5 seconds of AC current stimulation ranging from 4-160 Hz and 10-1000 μA to the right ventricle. A quadripolar catheter was placed in the apex of the left ventricle to measure left ventricular volume. Short-axis ultrasound images were recorded to measure left ventricular cross sectional area and wall thickness.

Our results showed that the mean volume of the left ventricle during collapse was significantly smaller ($p < 0.05$) than the mean volume preceding collapse. Cross sectional area also decreased significantly and wall thickness increased. This suggests that the heart assumes a contracted, systole-like state during collapse.

1. Introduction

Medical equipment can unintentionally allow electrical "leakage" current to flow through the patient, posing a risk of ventricular fibrillation (VF) or complete hemodynamic collapse without fibrillation (collapse). Recent studies have shown that the current threshold for inducing collapse is below that of VF in both humans [1] and canines [2]. This study examines the volume, cross sectional area and wall thickness of the LV during these collapse episodes in order to gain an understanding of the physical mechanisms by which collapse occurs.

2. Methodology

This study followed all applicable institutional guidelines in the care and treatment of laboratory animals, including guidelines of the American Heart Association and the Public Health Service

Six mongrel dogs were anesthetized with intravenous pentobarbital (30 to 35 mg/kg initially with maintenance doses of approximately 3 mg/kg/hour). The dogs were intubated with a cuffed endotracheal tube and ventilated with room air (Harvard Apparatus, Dover, MA, USA). A femoral artery line was inserted to display systemic pressure. Saline was infused continuously through a peripheral intravenous line. Electrodes for the lead II ECG were applied, and monitored continuously. When necessary, a hot water blanket was used to maintain body temperature.

A quadripolar pacing catheter (Cardio-rhythm, Medtronic, Inc., Minneapolis, MN, USA) was placed in the right ventricular (RV) apex via the jugular vein under fluoroscopic guidance. The tip of the catheter (17 mm^2) carried the AC stimulating current. The return for the AC stimulation was a patch (12 mm diameter) of stainless steel 2 cm caudal and lateral (left) of the clavicle at the sternum. The patch was used to emulate a typical ECG recording electrode, a possible return path for leakage currents. Electrode paste (Spectra 260, Parker, NJ, USA) was used between the metal and the shaved skin.

A steerable, quadrapolar volume catheter (RF Performr 5523, Medtronic, Minneapolis, MN) was placed in the LV apex via the femoral artery and deflected towards the basal region under fluoroscopic guidance. The intraventricular impedance, which is inversely proportional to volume, was determined by passing a 1.2-1.3 mA, 10 kHz current through the proximal and distal electrodes of the quadrapolar catheter and measuring the voltage between the second and third electrodes [3]. Intraventricular volume measurements were recorded at a rate of 1000 samples per second.

The pacing catheter delivered five seconds of AC stimulation between 10-1000 μA. AC stimulation was given randomly at 4, 5, 6, 7, 8, 9, 20, 40, 80 and 160 Hz. Stimulation was generated by a PCI-1200 DAQ card (National Instruments, Austin, TX, USA) passed through a calibrated, transimpedance (voltage to current converting) stimulus isolation unit (model 2200; A-M Systems, Everett, WA, USA). Stimulation strength was increased until VF was induced, at which point no additional stimuli were given at that frequency for that repetition. Stimulation resulted in episodes categorized as no effect, effect, collapse or VF. When VF was induced, defibrillation was accomplished within 10 seconds with an external defibrillator (DC-190; Burdick, Redmond, WA, USA). Following VF, a minimum of three minutes

were allowed to elapse, providing enough time to allow blood pressure, oxygen saturation and heart rate to return to normal levels. Collapse data only was analyzed in this study.

Two dimensional ultrasound was used to record short axis images of the LV. For each collapse episode, 6 images were digitized. These consisted of a diastolic and systolic image taken approximately 1 second before the delivery of the stimulus, and four collapse images, taken at one second intervals beginning exactly one second after the delivery of the stimulus. Ventricular area was traced using the freehand tool in NIH image, and area was obtained in square pixels. Thickness was determined by drawing a line from the center of the LV cavity (as measured by NIH Image) through the left ventricular free wall. This line was drawn at an angle of 48 +/- 1 degrees (as measured by NIH Image) for all images. Ventricular wall thickness was measured in pixels along this line.

3. Results

A total of 199 collapse episodes were analyzed from the ultrasound data. Figures 1 and 2 illustrate our results for thickness and area averaged over all test subjects and all frequencies. Paired, two-tailed t-tests were performed to compare measurements before and during collapse. Differences are considered significant for $p < 0.05$. LV thickness during collapse was significantly greater than diastolic thickness, and greater than systolic thickness for the fourth second of collapse. LV cross sectional area during collapse was significantly smaller than diastolic area, and smaller than systolic area for the second, third and fourth seconds of collapse.

A total of 764 collapse episodes were analyzed from the volume catheter data. Mean volume was computed during a one second interval immediately preceding the stimulus, and during a one second interval between the third and fourth seconds of collapse. Intraventricular impedance showed a significant ($p = 0.028$) increase of 54%, indicating a decrease in LV cavity volume during collapse.

4. Discussion

This study investigates the mechanical response of the left ventricle during weak AC current induced hemodynamic collapse. During collapse the LV cavity is smaller than normal and the wall thickness is larger than normal. This suggests that the heart assumes a systole-like state during collapse.

Limitations of this study include the indirect nature of the volume measurements (inferred from intraventricular impedance) and potential human error in the analysis of ultrasound images to measure LV area and wall thickness.

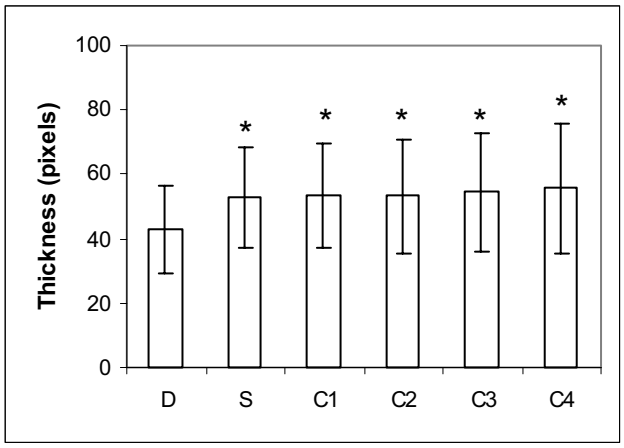

Figure 1. Mean thickness for diastole (D), systole (S), and first through fourth second after the initiation of collapse (C1-C4). Error bars represent standard deviation. A single asterisk indicates significant difference from diastole, and a double asterisk indicates a significant difference from systole and diastole.

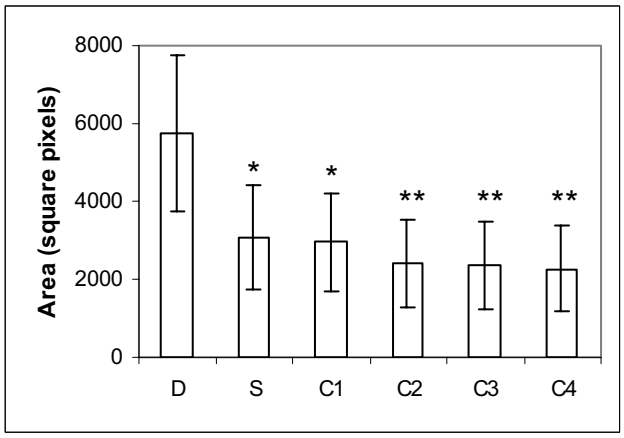

Figure 2. Mean area for diastole (D), systole (S), and first through fourth second after the initiation of collapse (C1-C4). Error bars represent standard deviation. A single asterisk indicates significant difference from diastole, and a double asterisk indicates a significant difference from systole and diastole.

To determine the degree to which human error could have influenced our analysis of the ultrasound data, two volunteers were given 2 sample sets of 12 images each and asked to perform the procedure outlined in the methodology section to obtain thickness and area. The thickness and area measurements from these 24 total images were then compared to the original measurements of those same 24 images. It was found, for area measurements, that the average coefficient of variation over all three measurers (original measurer and 2 volunteers) over all 24 images was 17%. For thickness,

the average coefficient of variation was 5%. Significant differences between mean area measurements ranged between 23-68%, and 5-30% for mean thickness. Thus, we conclude that subjectivity involved in the image analysis would not profoundly affect our results.

5. Conclusion

AC induced hemodynamic collapse is characterized by a reduction in ventricular volume and cross sectional area, and an increase in ventricular wall thickness. This suggests that the heart assumes a contracted, systole-like state during collapse.

Acknowledgements

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References

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