Cardiac Pacing in a Chronically Instrumented Non-Human Primate Model During Centrifuge

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The Physiology Research Branch has developed a chronically instrumented non-human primate model for evaluating cardiac function during exposure to altered gravitational environments. This model has been used to measure cardiovascular hemodynamics and electrical activity. We have expanded the model to include cardiac pacing for evaluation of responses and mechanisms in normal and dysrhythmic states. In particular, we have been able to produce constant rates by means of atrial, ventricular, and dual chamber pacing during centrifugation. Pre-ventricular contractions, bigeminal, and trigeminal rhythms have also been invoked using the same pacing model.
CARDIAC PACING IN A CHRONICALLY INSTRUMENTED NON-HUMAN PRIMATE MODEL DURING CENTRIFUGATION

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Abstract -- The Physiology Research Branch has developed a chronically instrumented non-human primate model for evaluating cardiac function during exposure to altered gravitational environments. This model has been used to measure cardiovascular hemodynamics and electrical activity. We have expanded the model to include cardiac pacing for evaluation of responses and mechanisms in normal and dysrhythmic states. In particular, we have been able to produce constant rates by means of atrial, ventricular, and dual chamber pacing during centrifugation. Premature contractions, bigeminal, and trigeminal rhythms have also been invoked using this same pacing model.

I. INTRODUCTION

The Physiology Research Branch conducts research designed to elucidate basic physiological mechanisms in aerospace environments which provide a clinical basis for development and application of the most effective procedures to protect or prevent aircrew from exposure to risk factors that may compromise their health, safety, and performance in Air Force Systems. A chronically instrumented non-human primate model was developed for evaluating cardiac function during altered gravitational environments [1-3]. This model has been used to simultaneously measure internal ECG, cardiovascular hemodynamic data (left atrial pressure, aortic pressure, left ventricular pressure, right atrial pressure, pulmonary artery pressure, right ventricular pressure, and aortic flow), and cardiac dimensions (left and right ventricular volumes). These data have been used for calculating beat-to-beat systemic arterial compliance (SAC) and total peripheral resistance (TPR) using a two-element windkessel model [4]. Due to the transient nature of this model and the reliance of the calculations on heart rate, it would be advantageous to be able to clamp heart rate and produce a controlled, constant rate.

The primary objective of our study was to incorporate cardiac pacing into the existing human surrogate model. A secondary objective was to develop the capability to produce arrhythmia's (isolated PVCs, bigeminy, trigeminy, and increased P-R intervals) during hypergravity.

II. METHODS

Instrumentation, implanted via a midsternal thoracotomy, included pacing leads, pacemaker, flow probe, and pressure sensors. Two bipolar pacing leads (Cardiac Pacemakers Inc., St. Paul, MN) were implanted on the right atrium and four unipolar pacing leads (Cardiac Pacemakers Inc., St. Paul, MN) were attached to the left ventricle. A Triumph DR model 1224 dual chamber, adaptive rate pacemaker (Cardiac Pacemakers Inc., St. Paul, MN) was implanted subcutaneously anterior to the umbilicus. One bipolar and two unipolar leads were connected to the internal pacemaker and the remaining leads were exteriorized (see below). An active redirection transit-time (ART') flow transducer (Triton Technology, San Diego, CA) was implanted around the ascending aorta. Electrical activity was measured internally using medical-grade stainless steel wire with sensing sites located in the pericardium near the right atrium and left ventricular free wall and in the musculature caudal to the xyphoid process. Polyurethane vascular access ports were implanted in the right atrium, left ventricle, and right ventricle. All cabling, wires, and tubing were then tunneled subcutaneously and exteriorized from the skin in the midscapular region of the back. Instrumented subjects were then given a one month post-operative recovery period.

Prior to each experimental procedure, 3-Fr micromanometer-tipped catheters (Millar Instruments Inc., Houston, TX) were calibrated and inserted into the appropriate chambers via the vascular access ports. The catheters that were inserted in the ventricles have a pressure transducer at the tip and one 3 cm proximal from the tip. The distal transducers were inserted through the ventricles into the aorta and pulmonary artery with the proximal transducers remaining in the left and right ventricles, respectively. External pacing and arrhythmia induction was accomplished with a model EP-2 clinical stimulator (Hi-tronics Designs Inc., Budd Lake, NJ) designed for cardiac pacing research.

III. RESULTS

A rhesus monkey and a goat, selected for pilot studies, have been completed and we currently have a fully instrumented baboon. Data have been successfully obtained from the first two subjects during studies conducted in the
laboratory under normal conditions. Atrial, ventricular, and dual chamber pacing (Fig. 1.) were accomplished along with isolated PVCs, bigeminy (Fig. 2.), trigeminy, and decreased P-R intervals. Data from the currently instrumented baboon were obtained during experimental procedures conducted at the Brooks Air Force Base centrifuge facility.

IV. DISCUSSION

Presently, we are able to produce a controlled, constant rate using atrial, ventricular, or dual chamber pacing with a slightly decreased P-R interval. Further, we are able to produce isolated PVCs, bigeminy, and trigeminy.

We have scheduled a radiofrequency AV-node ablation procedure that will enable us to produce dual chamber pacing with increased P-R intervals and also to lower ventricular rate. In the future, we would like to enhance our model with the ability to lower heart rate while maintaining the integrity of the conduction path in the heart. This would give us a decreased heart rate and would not alter the normal hemodynamics of the system.

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