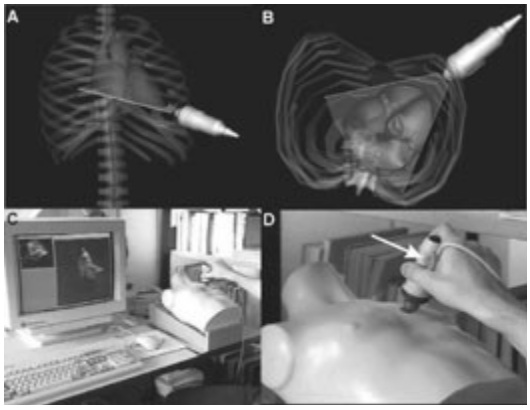
for orientation and guiding. The demonstrator provides an excellent training tool to become acquainted with echocardiography.



1058-69 Modeling and Prediction of Acute Hemodynamic Effects of an Arterial Assist Device

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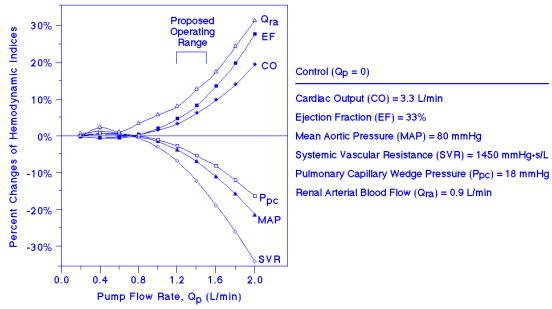
Background: The concept of utilizing an arterial assist device in the form of a femoral-aortic flow pump has been proposed to be of benefit in patients with severe refractory congestive heart failure by reducing afterload and is currently under investigation. Mechanisms for putative improved hemodynamics as suggested in preliminary studies are not well understood. Method: A previously validated computer model is used to simulate hemodynamics and acute effects of the assist pump. The pump is modeled as a constant flow source (Qp) from femoral artery to proximal descending aorta. The pressure head loss in the descending aorta due to outflow velocity (V) is modeled by use of the Bernoulli's equation: (dVV/2)(Qp/CO), where d is density of blood and CO is the intrinsic cardiac output. Result: The control was set for congestive heart failure with hemodynamic indices defined and shown in figure. The model maintained constant preload and intrinsic myocardial contractility, and did not include effects due to change in coronary perfusion. The percent changes of these indices were plotted as Qp varied from 0.2 to 2 L/min. Conclusion: Based on our computer simulation, superimposing an additional constant flow on pulsatile aortic flow produces moderate improvement of hemodynamic indices in the proposed operant range of pump flow (1.2-1.5 L/min). A variety of system parameters could shift this range. Observed increase in renal blood flow and secondary neurohumoral actions could yield additional hemodynamic benefit.



1058-70 Changes in Left Ventricular Activation Sequence in a Finite Element Model of Single Site Right Ventricular and Dual Site Biventricular Pacing

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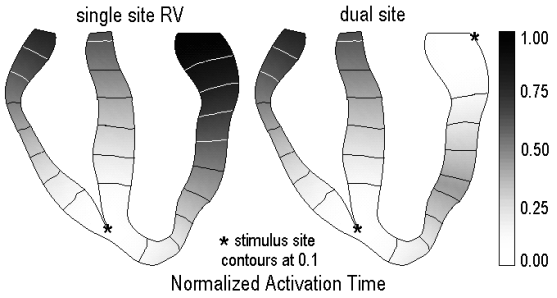
Background: Dual site biventricular pacing, where the left ventricle (LV) is paced with the right ventricle (RV), improves outcome in some heart failure patients. Single site RV pacing may lead to worse outcomes than dual site pacing, which may be related to asynchronous LV activation. **Methods**: A two-dimensional finite element model of a realistic ventricular geometry was used to compute changes in the LV activation sequence using single site RV pacing and dual site biventricular pacing protocols. The RV protocol activated apical cavity nodes to simulate an endocardial lead stimulus. The dual site protocol added a lead in the coronary sinus vein by activating nodes at the base of the LV free wall. The cable equation, governing the spread of excitation, was solved in each case to obtain the activation time of the propagated wave. **Results**: The upper midventricle of the LV free wall was activated 67% earlier with dual site pacing relative to RV pacing; the entire free wall was activated 55% earlier. Dual site pacing showed the activation sequence reversal observed in patients benefiting from biventricular pacing, and more closely resembles normal activation by reducing LV activation asynchrony. **Conclusion**: Realistic simulations showed markedly different LV free wall activation between single site RV and dual site pacing. This tool is useful to investigate how altered activation enhances cardiac output and improves outcome, and may suggest new beneficial pacing strategies. Supported by NCRR-NIH Grant P20 RR 16457.



1058-71 Visualization of the Effect of Atrial-Ventricular and Right-Left Delay on Cardiac Output During Biventricular Pacing

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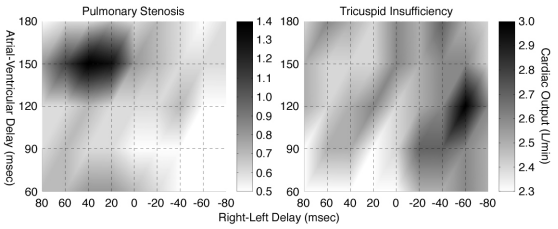
Background: Biventricular pacing (BiVP) has great potential as an adjunctive treatment for patients with acute and chronic heart failure. Acute optimization of BiVP requires determining the appropriate atrial-ventricular delay (AVD) and right-left delay (RLD) on a patient-to-patient basis. Presently there is no good method for optimization of AVD and RLD in BiVP for improved cardiac output (CO), so we therefore examined the utility of CO surface plots for optimization of BiVP.
Methods: In a study of pulmonary stenosis and tricuspid insufficiency in anesthetized pigs with induced heart block, AVD and RLD were varied during BiVP and CO measured using an ultrasonic flow probe. Surface plots displaying CO with variations in AVD and RLD were generated. CO, represented by a red-to-blue color map (the vertical bar), is plotted against varying AVD (60 to 180 msec) on the ordinate and RLD (80 to –80 msec) on the abscissa and linearly interpolated between measured values.
Results: The figure below shows representative CO surface plots during BiVP in a pig with pulmonary stenosis (left) and tricuspid insufficiency (right). (Note: Here the color spectrum is displayed as a gray scale map, with black representing the highest CO). The plots show the optimal settings of AVD and RLD, indicated by the darkest area of the surface.
Conclusion: CO surface plots allow visualization of the effects of AVD and RLD on CO during BiVP. By use of emerging technologies, CO maps may be useful for determining the optimal AVD and RLD settings for BiVP.



1058-72 Computer Model of the Human Atrium as a Study Tool for Atrial Arrhythmias

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Background: Atrial fibrillation (AF) affects 5% of people older than 60 years of age. The design of a successful strategy against AF depends on an understanding of the fundamental mechanisms creating this arrhythmia. An accurate anatomic and electrophysiologic model of the normal atrium is an essential experimental system for the study of AF.
Methods: Our model of the normal human atrium includes fast conducting regions (pectinate muscles, the crista terminalis, superior and inferior interatrial connections), slow conducting regions (isthmus, limbus of fossa ovalis), and the pulmonary veins (Figure). It uses a model of the atrial myocyte that accurately reproduces the human transmembrane potential and the effective refractory period (ERP). Pacing simulations in this model were used to evaluate how closely our model resembles the known human atrial behavior.
Results: The accuracy of the interatrial connections results in simulations of atrial depolarization and refractoriness (ERP ~235 ms) that closely resembles those reported for the human atrium. AF can be induced in this model by cross-field stimulation; this AF is self-terminating, as is also seen in real human atrium.
Conclusion: A mathematical model of the human atrium that resembles human atrial physiology has been developed and may prove to be an indispensable research tool for the study of atrial arrhythmias.