

A closed-chest animal model was developed in which an "acoustical window" permits the entire heart to be irradiated with the transducer moved slowly on the chest wall. The model is formed by removal of a portion of the fifth rib, incision of the pericardium, and attachment of the incised edges to the thoracic wall to retain the intervening lobes of the left lung. A catheter filled with heparinized Ringer's solution is inserted into the left atrium via the atrial appendage and exteriorized for microsphere injections. The left anterior descending coronary artery or one or more of its branches are ligated to produce an anterior wall infarct. The chest is then closed and the pneumothorax carefully evacuated to remove as much air as possible. Prior to surgery, a catheter-tip pressure transducer is advanced through a common carotid to the left ventricle for measurement of ventricular pressure and dp/dt. Both femoral arteries are catheterized to the level of the descending aorta for simultaneous reference blood flow collections. Lead II EKG and heart rate are recorded continuously.

Studies of the effects of therapeutic ultrasound on blood flow in skeletal muscle have indicated that an increase in flow results from the hyperthermia associated with tissue absorption of the acoustical energy. Some preliminary studies in our laboratories have suggested that coronary blood flow distribution in normal myocardium may be altered by low levels of therapeutic ultrasound. A study is in progress to evaluate the effect of a therapeutic level of ultrasound on regional coronary blood flow in normal and acutely ischemic areas of the canine myocardium. A mechanical effect associated with microacoustical streaming would be an attractive mechanism to provide potential benefit.

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EFFECT OF ULTRASOUND ON REGIONAL CORONARY BLOOD FLOW IN NORMAL
 AND ISCHEMIC CANINE MYOCARDIUM

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Flow determinations are done in a control state and during ultrasound therapy at 1, 2 and 4 hours after coronary artery ligation. The control session includes a placebo massage that is given for the same length of time as the therapeutic ultrasound. An experimental protocol has been established to investigate several variables including duration of irradiation, average intensities, and time of therapy after coronary ligation. The treatment of each animal consists of a slow massage over the heart with 870 KHz of continuous wave ultrasound at 1, 2 and 3 watts/cm² (average intensities) given in random order at 1, 2 and 4 hours post-ligation. Microspheres (3M Co., 9-1µ) with one of six randomly chosen labels are injected as a bolus at the 5th, 10th or 15th minute of each treatment session and flushed in the 5 cc heparinized Ringer's solution. The reference blood flow samples are collected by a Harvard infusion-withdrawal pump operated at 2.72 ml/minute started 30 seconds before microsphere injection and continuing for 2 minutes.

After all six sessions are completed, the animal is heparinized and terminated with sodium pentobarbital, and the heart excised. The heart is flushed with Ringer's solution, 2, 3, 5-triphenyl tetrazolium chloride (TTC) stain and 10% buffered formalin. After the left ventricle is isolated and weighed, the heart is sliced in 1 cm sections apex to base through the infarct and perpendicular to the long axis of the heart. The valves and chordae tendinae are removed and the sections divided into eight subsections which are further divided into endocardial and epicardial segments. Each segment is weighed and counted along with the blood tubes overnight in a Packard Automatic spectrometer interfaced with a Packard multi-channel analyzer. Isotope separation and regional coronary blood flows are calculated with standard techniques using a PDP-11/45 computer.

Preliminary results show an increase in coronary blood flow in both normal and ischemic myocardium. The irradiations performed at 1 hour post-ligation have given the greatest increase in coronary flow thus far. The myocardium from the core of the ischemic area has shown least effects, whereas the myocardium from the ischemic border zones shows an increase in flow during the treatment periods. Conclusions at this stage would be premature, but there do seem to be some alterations in the coronary blood flow distribution either through thermal and/or mechanical mechanisms.

(Work supported by NIH Contract #N01-HV-53004 of the Cardiac Diseases Branch of the NHLBI and the Indianapolis Center for Advanced Research, Inc.)

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