Effect of arterial counterpulsation on left ventricular volume and pressure

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The effect of arterial counterpulsation on coronary blood flow and the tension-time index (TTI) has been reported by several investigators (11, 13, 20). However, since myocardial oxygen consumption is dependent, in part, upon the total wall tension of the left ventricle (17), the volume of this chamber must be considered as well as its pressure during ejection, ejection time, and contractile state (9). Left ventricular contractility has been reported to increase during arterial counterpulsation (21). Thus, directly measured myocardial oxygen consumption may be at variance when compared with only the TTI (13, 20).

The purpose of this investigation was to study the effects of arterial counterpulsation, produced by the SIMAS circulatory assist device, on the volume, pressure, and contractile state of the left ventricle.

METHODS

Nine mongrel dogs weighing between 19 and 38 kg were anesthetized with chloralose, 50–100 mg/kg, with respiration maintained by a Harvard respiratory pump. A thoracotomy was performed and the heart was suspended in a pericardial cradle. The animals were anticoagulated with 100–200 mg heparin given intravenously. Left ventricular pressure was measured through a large-bore cannula inserted via the ventricular apex and connected to a Statham P23Db strain gauge. The diastolic pressure tracings were recorded on a high gain in centimeters H2O as well as in millimeters Hg in order to accurately determine left ventricular end-diastolic pressure. Also, the maximal rate of left ventricular pressure rise (max dp/dt) was determined in 11 studies on 6 dogs. Aortic pressure was measured through a second large-bore metal cannula inserted via the left carotid artery to the aortic arch and connected to a Statham P23Db strain gauge. Recordings were taken on an Electronics for Medicine DR-8 photographic recorder at paper speeds of 200 mm/sec. Heart rate was held constant by electrical pacing with a Grass impulse generator (model S4) through a bipolar pacing electrode sutured to the right atrium.

Cutdowns were made over each femoral artery just below the inguinal ligament and a no. 18 French catheter was inserted into each vessel after ligation of the distal end. The catheters were sutured in place and attached to a SIMAS circulatory assist device (SIMAS pump, model 60EH53, Hamilton Standard Division of United Aircraft Corp., Windsor Locks, Conn.) which produced the arterial counterpulsation. A pressure bottle, filled with blood, was attached between the pump and the catheters. The inlet to the pumping device from the pressure bottle was equipped with a one-way valve allowing blood to be infused into the system without a regurgitant flow during the pumping action. The tubing from the pressure bottle was clamped upon equilibration of mean aortic pressure during the time actual measurements were recorded. This system permitted the
maintenance of a constant mean aortic pressure during the control and counterpulsation studies.

A detailed description of the SIMAS unit and its use has been reported previously (22, 25). In brief, the SIMAS circulatory assist device is a computer-automated myocardial augmentation system consisting of an electronic controller, actuator, and hydraulic supply and pressure-pulse generator. It is controlled by synchronization with the electrocardiogram. The actuator and hydraulic supply module generate and physically direct hydraulic power through a fluid-filled tube to the pressure pulse generator upon commands from the electronic controller. Its connections to the arterial tree are made through catheters inserted into the femoral arteries. During systole, a quantity of blood is withdrawn from the arterial tree and during diastole it is returned. This tends to decrease the aortic pressure during ventricular systole and increase it during diastole, thereby augmenting coronary perfusion pressure. In this study, 20-ml pump volumes were used during arterial counterpulsation. The pump output was checked by an electromagnetic flowmeter connected to the tubing into which the catheters used in the animals were inserted. The pump was found to range between 22.8- and 23.5-ml stroke output when set at 20 ml with the no. 18 French catheters at the pumping rates used, 120-170/min.

Left ventricular volumes were analyzed by a method previously described from this laboratory (14, 15). Briefly, six lead beads were placed close to the endocardium of the left ventricle so as to define the three axes of a nonprolate ellipsoidal shell of ventricular muscle. The base bead was sutured at the level of the aortic valve and the apex bead was sutured to the apical dimple. These two beads formed the long axis of the ellipsoid. Four beads were implanted through a needle equidistant around the left ventricular midplane in order to form the two minor axes of the ellipsoid. These three axes defined an ellipsoidal figure which was filmed at 60 frames/sec with biplane (90° cinefluorography). Left ventricular ellipsoidal cavity volume was calculated from the equation: \[ LVV = \frac{4}{3\pi abc}, \]
where \( a \), \( b \), and \( c \) represent the three calculated axes divided by 2. This volume includes the papillary muscles and trabecular carnaecae and does not represent the absolute left ventricular blood volume. However, the changes in volume of the ellipsoid represent a change in the volume of blood contained in the ventricle. The accuracy of this method has been reported previously (14).

The measurement of the six beads used to determine the absolute values of the three lengths was taken from each pair of matched 35-mm cinemuls with a LARR-V digitizer (Computer Industries, Inc., Van Nuys, Calif.) and was corrected for X-ray magnification and distortion from a simultaneously filmed steel sphere of known size. The paired biplane film measurements were matched up with the simultaneously recorded pressure measurements by means of a film frame marker on the Electronics for Medicine oscillographic recorder.

Three cardiac cycles were superimposed in time and a composite cycle was obtained. The end-diastolic volumes were obtained at 0.06 sec after the onset of the QRS wave of the simultaneously recorded electrocardiogram. This time corresponded to the "notch" produced by atrial contraction, which was seen in the left ventricular diastolic pressure tracing. The diastolic pressure tracings were recorded on a high gain in centimeters H2O as well as in millimeters Hg in order to accurately derive left ventricular end-diastolic pressure.

Systolic volumes were taken as the smallest volumes in the composite cycle. These were matched with their corresponding left ventricular pressures and were almost always simultaneous with the dicrotic notch in the aortic pressure tracing, both with and without arterial counterpulsation.

The volume measurements from the control studies were compared with 20-ml SIMAS arterial counterpulsation. The paired studies were taken within 3 mm of each other at the same heart rates and at constant mean aortic pressures.

RESULTS

The effects of arterial counterpulsation on left ventricular volume and pressure were determined in 15 studies on 9 dogs. In all studies the stroke output of the SIMAS unit was set at 20 ml. Typical pressure tracings during the control study are shown in Fig. 1A and during arterial counterpulsation in Fig. 1B. The diastolic pressure wave from the pump was timed to begin at the dicrotic notch of the aortic pressure pulse. This gives the maximal allowable time for diastolic augmentation and allows the positive wave to dissipate before the beginning of ventricular ejection, thereby having little effect on aortic pressure at the time of aortic valve opening. In this study (Fig. 1, A and B) the maximal rate of left ventricular pressure rise (max dp/dt) fell 30% during counterpulsation without a significant change in aortic diastolic pressure or left ventricular end-diastolic pressure. The maximal rate of rise of left ventricular pressure fell an average of 16% during counterpulsation in 11 studies on 6 dogs.

The pressure findings in the 15 studies are given in Table 1 and illustrated in Fig. 2. The mean aortic pressures were almost identical, 104 ± 10 mm Hg control as compared with 103 ± 9 mm Hg during counterpulsation. The left ventricular end-diastolic pressures were the same, 13 cm H2O. The peak left ventricular pressure averaged 119 ± 13 mm Hg in the controls and 103 ± 13 mm Hg in the pump study. This represents an average fall of peak systolic pressure of 16 mm Hg or a 14% decline. During arterial counterpulsation peak systolic pressure and mean aortic pressure were the same.

Ejection times taken from aortic valve opening to the dicrotic notch in the aortic pressure tracing were identical, 0.17 sec, representing no change in the duration of ejection with counterpulsation.

The ventricular volume studies are also given in Table 1 and illustrated in Fig. 3. End-diastolic volumes averaged 62 ± 16 ml control and 60 ± 14 ml during counterpulsion. End-systolic volumes averaged 45 ± 13 ml control and 44 ± 12 ml pump. These values are not significantly different. The difference between the systolic and diastolic volumes, or stroke volume, was 17 ± 12 ml control and 16 ± 9 ml pump. The ejection fraction, SV/EDV, was .26 in both studies.

Pressure-volume loops were constructed for both the control and pump studies. A typical pair of loops is shown in
Fig. 4. These loops define clearly end-systolic volume, end-diastolic volume, stroke volume, and their dynamic relationship with the corresponding pressure. The area of the loop represents the stroke work. The plot clearly shows a decrease in left ventricular stroke work during arterial counterpulsation which is due to the decrease in systolic pressure during ejection. The end-systolic, end-diastolic, and stroke volumes remained essentially constant between the control study and the counterpulsation study as seen from the parallel plots along the horizontal portions of the loops. There is no loop deformity to indicate aortic valvular insufficiency with the counterpulsation. The decrease in area of the work loop with counterpulsation is entirely due to a decrease in the pressure component of the work calculation. It should be noted that a decrease in left ventricular stroke work occurred during counterpulsation without a change in left ventricular end-diastolic volume and pressure.

DISCUSSION

Studies of ventricular pressure with counterpulsation devices have clearly shown a decrease in pressure in the left ventricle during ejection (2, 4, 8, 10, 24, 25) and a decrease in duration of ejection resulting in a decrease in tension-time index (11, 13, 20). Also arterial counterpulsation has been shown to reduce myocardial oxygen consumption in normotensive dogs (11, 13, 20), but this has not always correlated with the observed pressure changes (13, 20).
The effect of arterial counterpulsation on the contractile state of the left ventricle is of interest. Spotnitz et al. (21) have reported that this procedure may cause an increase in contractility. However, in the present study it was found that from the same left ventricular end-diastolic volume and pressure there was a decrease in stroke work and a decrease in the maximal rate of left ventricular pressure rise without a significant change in aortic diastolic pressure. This demonstrates a decrease in contractility of the left ventricle during counterpulsation. This conclusion is further reinforced by the fact that with the decrease in systolic pressure during ejection (afterload) there was no change in the ejection fraction.

Two possible mechanisms may be postulated to explain the finding of a decrease in contractility of the left ventricle during counterpulsation. The level of developed systolic pressure by the left ventricle is one of the determinants of its contractile state (1, 3, 19). Sarnoff has demonstrated an increase in contractility of the left ventricle by an increase in aortic pressure (afterload) (19). During counterpulsation the reduced pressure during systole which results from the withdrawal of blood from the arterial tree during this time should cause a decrease in contractility of the left ventricle.

Another determinant of the contractile state of the left ventricle is the level of arterial baroreceptor activity, since it is an important regulator of cardiac sympathetic efferent activity and of catecholamine release from the adrenal medulla (5, 18). During counterpulsation there is a “double” stimulation of the arterial baroreceptors during each cardiac cycle. This increased stimulation of the arterial baroreceptors should cause a decrease in cardiac sympathetic efferent discharge and a decrease in release of catecholamines from the adrenal medulla. Both of these mechanisms would cause a decrease in contractility of the left ventricle.

Of further interest was the difficulty maintaining aortic pressure during the initial phase of augmentation. Arterial baroreceptor stimulation during arterial counterpulsation would also cause peripheral vasodilation and a decrease in mean aortic pressure. The fall in mean pressure was in the range of 15 mm Hg, despite a diastolic augmented pressure wave equal to the previous control systolic ejection wave. The pressure would tend to correct itself in 5–15 min after the onset of counterpulsation. However, mean aortic pressure could be easily maintained by increasing infusion from the pressure bottle immediately upon initiating counterpulsation.

The importance of pressure work of the left ventricle was emphasized by Sarnoff, Braunwald, and co-workers (17) who stated that myocardial oxygen consumption depends primarily on the pressure generated during systole and its
duration (tension-time index). However, these authors stated that the relation is only valid to the extent to which changes in ventricular radius occur since considerations including Laplace's law apply and the "same intraventricular TTI will require a greater myocardial fiber tension in a large heart than in a small one" (17). Studies by Monroe and French (16) have demonstrated that the development of large heart than in a small one during arterial counterpulsation is less than predicted from the induced changes in the TTI (13, 20).

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