THE PUMPING AND LEFT VENTRICULAR UNLOADING CAPABILITIES OF THE VENTRICULAR SYNCHRONOUS SKELETAL-MUSCLE VENTRICLE

The pumping and left ventricular unloading capabilities of the left ventricular, ventricular synchronous skeletal-muscle ventricle were determined in nine anesthetized dogs ranging in weight from 20.7 to 31.8 kg. The ventricular synchronous skeletal-muscle ventricle consists of the left rectus abdominis muscle wrapped around a 4-mil-thick polyethylene pouch (wrapped volume 80 to 100 ml) connected to the left ventricular apex with no valve and to the aorta via a prosthetic heart valve. The rectus muscle is timed to contract tetanically and relax during left ventricular ejection. This arrangement provides a high precontraction pressure for the rectus muscle and a high muscle capillary blood flow during skeletal muscle relaxation. The timing signal for initiation of the train of stimulating pulses (40/sec) was derived from the ventricular electrogram. The delay for the stimulus train determines the preload for the rectus muscle and along with the stimulus train duration determines ventricular synchronous skeletal-muscle ventricle stroke volume, which was measured by electric impedance. With unconditioned rectus muscles (70 to 120 gm) and with a pumping ratio of 1:3, ventricular synchronous skeletal-muscle ventricle stroke volume averaged 26.1 ml, which provided an average output of 876 ml/min. The normalized ventricular synchronous skeletal-muscle ventricle output was 35.6 ml/min per kilogram of body weight. In a typical resting dog (and man), the normalized cardiac output is 70 ml/min per kilogram. Therefore the ventricular synchronous skeletal-muscle ventricle is capable of pumping 52% of the cardiac output (with a pumping ratio of 1:3). The optimum train delay from the apex of the ventricular electrogram ranged from 10 to 100 msec. The left ventricular ejection period averaged 309 msec, and this determines the time available for the rectus muscle to contract and relax. Evidence for unloading the left ventricle is shown by the reduced left ventricular diastolic pressure and stroke volume for the postassisted beats. (J THORAC CARDIOVASC SURG 1995;109:1127-37)

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There are two critical requirements for the optimum use of electrically stimulated skeletal muscle, wrapped around a blood-filled pouch, to assist the circulation. First, a precontraction pressure on the order of aortic pressure is needed to obtain a

forceful contraction.^{1,2} Second, a high muscle capillary blood flow is necessary to avoid muscle fatigue.^{3,4} The ventricular synchronous skeletal-muscle ventricle (VS-SMV) described herein satisfies both requirements.⁵ The VS-SMV consists of a muscle-wrapped pouch connected directly to the apex of the left ventricle (LV) with no valve and to the aorta via a prosthetic heart valve. Therefore LV and VS-SMV pouch pressure are always the same: low during ventricular diastole (ensuring a high muscle capillary and myocardial blood flow) and high during ventricular systole when the skeletal muscle is timed to contract, thereby ensuring a high preload and forceful contraction. The objective of this report is to demonstrate the importance of timing to obtain LV unloading and circulatory assistance provided by the VS-SMV in a short-term animal model with the use of unconditioned rectus abdominis muscles.

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Fig. 1. VS-SMV consisting of a muscle-wrapped pouch connected to the LV apex (with no valve) and to the aorta via a conduit with a prosthetic heart valve.

Theory of operation. The VS-SMV is applied as shown in Fig. 1. There is no valve between the LV and the pouch, which is wrapped with the left rectus abdominis muscle. At the outlet of the pouch is a prosthetic heart valve at the entrance of the conduit that leads to the aorta. The method used to attain the dual goal of a low pouch diastolic pressure, followed by a high precontraction pressure, is based on precise timing of the tetanic contraction of the rectus muscle relative to LV systole. When the desired pouch precontraction pressure is reached, the rectus muscle is contracted tetanically by an appropriately delayed train of stimuli after the R wave of the electrocardiogram (ECG). The pulse train duration determines the duration of muscle contraction and is chosen so that the skeletal muscle is completely relaxed at the end of the LV ejection period. The pulse train delay determines the pre-

contraction pressure (preload) and is selected so that the skeletal muscle starts to contract at the beginning of LV ejection.

Ranvier⁶ was the first to point out that there are two types of skeletal muscle: (1) fast twitch (pale) and (2) slow twitch (red), with the latency between the stimulus and maximum force of contraction being longer for slow-twitch (fatigue-resistant) muscle. However, when a muscle is contracted by nerve electrodes there are additional delays. First, there is a propagation time for the nerve action potential to reach the myoneural junctions. Second, there is a myoneural junction delay. Third, there is a propagation time for the muscle action potential. Fourth, there is a latent period between muscle excitation and the development of force, this latter time being known as the latent period, which is typically 40 to 100 msec for slow-twitch muscles and 5 to 10 msec

Fig. 2. A, Timing diagram showing the R wave of the ECG, pressure in the LV, ejection period, stimulus train, and muscle contraction, identifying contraction latency (70 msec) and relaxation latency (100 msec). TD, train duration. B, LV cycle of an animal in which LV ejection starts at B (140 msec from R wave) and ejection period (B-E) is 350 msec.

for fast-twitch muscles.⁷ Of all of these delays, the latent period is the longest.

Not only is there a latency between the onset of the stimulus train and the development of muscle force, there is also a relaxation latency, that is, the time between termination of the stimulus train and complete muscle relaxation. In preliminary dog

studies with muscle-wrapped pouches in which the motor nerves were stimulated, we found that the contraction latency is on the order of 70 msec and the relaxation latency is about 100 msec. These two latencies have an important bearing on selection of the onset and termination of the VS-SMV stimulus train so that the skeletal muscle contracts only

Fig. 3. Frank-Starling law of the heart. *Inset* shows force of each contraction as resting tension is increased continuously. *A* is operating point and increased loading increases force of contraction; decreased loading decreases force of contraction.

during the LV ejection period and is relaxed by the end of the ejection period.

As illustrated in Fig. 2, *A*, for the skeletal muscle to start contracting at the onset of LV ejection, which occurs about 100 msec after the peak of the first ventricular excitation wave (Q or R) in the dog, the stimulus train must be initiated 100 – 70 (30) msec (delay) after the Q or R wave, whichever is earliest. Likewise, for the skeletal muscle to be fully relaxed at the end of LV ejection, which occurs about 400 msec after the Q or R wave (whichever is first), the stimulus train must be terminated at 400 – 100 (300) msec. Therefore the maximum duration of the stimulus train, under these circumstances, is 300 – 30 (270) msec, as shown in Fig. 2, *A*.

Fig. 2, *B*, shows a typical ventricular electrogram, LV pressure, and aortic root flow velocity. In this animal, LV ejection began at *B*, 140 msec after the R wave, and the ejection period ended at *E*. The duration of the ejection period was 350 msec.

From the foregoing it is clear that the VS-SMV stroke volume depends on the train delay, which determines the preload on the skeletal muscle, and on the train duration, which controls the duration of muscle contraction. Reducing train duration reduces VS-SMV stroke volume and increasing train duration increases VS-SMV stroke volume; but there is a long-duration limit for train duration, namely one that permits complete relaxation of the skeletal muscle by the end of the LV ejection period to avoid restriction of LV filling.

When any LV unloading technique is evaluated, it is necessary to recognize that the Frank-Starling law is bidirectional, that is, increasing preload increases the force of contraction and decreasing preload decreases the force of contraction. Fig. 3 (*inset*) shows LV pressure of an isolated coronary-perfused canine heart as the preload is increased with each beat. The graph of force of contraction versus resting tension identifies two regions: (1) the Frank-Starling region (0–*B*) and (2) a failure region (beyond *B*). Choosing an operating point *A*, increasing the preload increases the force of contraction. However, if the preload is decreased, the force of contraction decreases, that is, unloading decreases the force of contraction as shown. Therefore, when an LV-unloading device is evaluated, the force of contraction of the postassisted beat will be less if the LV precontraction pressure is lower. Consequently the stroke volume of the postassisted (unloaded) beat will be less than that for the preassisted beat.

Material and methods

All studies described herein were done in accordance with the Guide for Care and Use of Laboratory Animals, published by the U.S. Department of Health, Education and Welfare (DHHS Publication No. NIH 85-23, revised 1985). In addition, this study was approved by the Purdue University Animal Care and Use Committee.

Nine mongrel dogs ranging in weight from 20.7 to 31.8 kg were anesthetized with intravenous thiopental (10 mg/kg) and a cuffed endotracheal tube was inserted. Anesthesia was maintained with a mixture of isoflurane and oxygen. Lead II ECG and femoral artery pressure were monitored continuously.

Surgical procedure. A midline skin incision was made from the ventral midthorax to just inferior to the umbilicus to provide exposure of the left rectus abdominis muscle, which was dissected free from its attachments. The muscle was then transected just below the umbilicus and its aponeurotic origin divided. The anterior portion of the muscle is supplied by the inferior intercostal nerves and care was taken to preserve the five or six branches that supply the muscle belly. The vascular pedicle was preserved and the muscle remained attached to the body by only the intercostal nerves and the epigastric blood supply.

The rectus muscles (70 to 120 gm) were wrapped spirally around the empty cylindrical pouch (Fig. 4, *A*) made of 4-mil-thick polyethylene tubing. The muscle was trimmed so that a single layer encompassed the pouch. The unwrapped volume was typically 200 ml; the muscle-wrapped volume was 80 to 100 ml. Ribbed polytetrafluoroethylene (PTFE)* grafts (15 mm) were used as conduits to the pouch and a St. Jude Medical bileaflet valve

*Gore-Tex graft: Gore-Tex is a registered trademark of W. L. Gore & Associates, Inc., Newark, Del.

(St. Jude Medical, Inc., St. Paul, Minn.) (19 mm) was placed in an adapter at the outlet of the pouch facing the aorta. The length of the conduit from the LV to the pouch was made as short as possible to ensure rapid filling. A 12 mm diameter plastic stent was sewn to one end of a PTFE conduit.

The LV apical stent was placed without cardiopulmonary bypass in the following manner. A left lateral thoracotomy was done at the fifth interspace. Four quadrant sutures were preplaced around the apex of the LV encircling the site where the 12 mm stent was to be inserted. A stab ventriculotomy was made and a balloon-tipped urinary catheter was inserted into the apex through the ventriculotomy. The balloon on the catheter was then inflated. A cork borer was passed over the catheter and was used to remove a circular plug of the ventricular apex. The inflated balloon acted as a backstop for the borer to prevent injury to the intraventricular structures and to entrap the apical plug. The catheter, apical plug, and borer were then removed simultaneously and the occluded stent inserted quickly into the apical hole. The stay sutures were tightened immediately and horizontal mattress sutures, buttressed by Teflon pledgets, were placed to further secure the stent. Another 15 mm diameter ribbed PTFE graft was then anastomosed, end-to-side, to the aorta. The free ends of the two conduits were then attached to the ends of the pouch. Just after insertion of the stent, each dog was given 150 units/kg heparin intravenously; additional heparin was given as needed during each study.

Aortic flow velocity was measured with an ultrasonic perivascular flow probe (Transonics Systems, Inc., Ithaca, N.Y.) placed at the root of the aorta. LV pressure was measured with a catheter passed therein via the left atrial appendage. This method was used to avoid compromising aortic valve function.

Pouch volume measurement. Stroke volume of the VS-SMV was measured by the change in electric impedance (Z) appearing between hollow sleeve electrodes 1 and 2 in Fig. 1, within the ends of the pouch (Fig. 4, A), in the manner previously described by us.^{8,9} In brief, a direct-coupled, 50 kHz impedance recorder measured the pouch impedance and its increase with ejection. Calibration was achieved by crossclamping the two PTFE inlet and outlet conduits and blood was withdrawn in 5 ml increments from the pouch with a syringe while the pouch impedance was recorded. A calibration curve relating ohms change (ΔZ) to the volume of blood withdrawn was then constructed for each animal; Fig. 4, B, illustrates the calibration curve for each animal.

Stimulator. The trains of tetanic stimuli were delivered to the five or six rectus abdominis motor nerves via a stimulus isolation unit driven by two Grass stimulators (Grass Instruments, Quincy, Mass.). The motor-nerve electrodes were connected to the negative pole of the stimulus isolation unit. The positive pole was connected to the indifferent electrode, which was placed caudal to the rectus muscle to direct the return-path stimulating current away from the sensing electrodes on the right ventricle. The second stimulator generated each train of tetanic stimuli (40/sec, each pulse being 0.1 msec). The first stimulator gated the second stimulator on and off and

Fig. 4. A, VS-SMV pouch with metal-sleeve electrodes. B, Calibration curves for the increase in impedance (ΔZ) as blood is withdrawn.

thereby controlled the stimulus train duration, which determined the duration of muscle contraction. The first stimulator was activated by an electronic circuit that was triggered by the first deflection in the ventricular electrogram (Q or R) detected by two electrodes on the base of the right ventricle. The circuit allowed control of the delay between the R wave and the onset of the train of stimuli. The circuit also permitted delivery of a train of stimuli for each ventricular electrogram wave or a submultiple thereof, that is, 1:2 to 1:10.

Train delay studies. The stimulus train delay (d) determines the preload on the skeletal muscle and therefore the VS-SMV stroke volume. To ensure accurate measurement of LV stroke volume and LV plus VS-SMV stroke volume, the outlet (valve) end of the conduit was crossclamped. Therefore emptying of the VS-SMV was via the LV. With the use of a train duration of 150 msec, the augmentation in aortic root flow velocity was determined for delays ranging from 10 to 100 msec with a pumping ratio of 1:5. In all of the animals, the times between the apex of the first ventricular wave (Q or R) to the beginning and end of ejection were measured by the aortic root flow-velocity recording.

Train duration studies. After the optimum delay was determined, studies were done to determine the effect of stimulus train duration (TD) on VS-SMV stroke volume measured by the blood-calibrated pouch-impedance record.

Pumping capability studies. In a previous study,¹⁰ we showed that on a per-gram basis, unconditioned skeletal muscle can deliver about one third of the power of cardiac muscle. Therefore, for the pumping studies, we selected a pumping ratio of 1:3. Under this condition, the VS-SMV was contracted with each third heart beat and the volume pumped was the VS-SMV stroke volume (measured by pouch impedance), multiplied by the heart rate divided by three. Various train delays (d) and train durations (TD) were used.

Results

Fig. 5 is a record of the electrogram, LV/pouch pressure, pouch impedance (volume), and aortic root flow velocity from a 24 kg dog. To make this record the outlet (valve) end of the pouch was crossclamped so that all of the output passed through the flowmeter at the root of the aorta. On the left, the LV is unassisted and the stroke volume was 22.7 ml; the heart rate was 96 beats/min; therefore the control cardiac output was $96 \times 22.7 = 2179$ ml/min. In the center of the record the VS-SMV was activated with 200 msec trains of stimuli, delayed 75 msec from the R wave and activated with a pumping ratio of 1:3. The SV for the assisted beat (1) was 36.7 ml, 16 ml for the second beat (2), and 23.4 ml for the third beat (3). Therefore, during the period of cardiac assistance, the total blood flow was 32 (36.7 + 16 + 23.4) = 2435 ml/min, which is 12% more than the control value. However, during the period of assistance, the VS-SMV stroke volume was 37 ml, as shown by the impedance record; therefore the VS-SMV pumped $32 \times 37 = 1184$ ml/min, which is 48.6% of the total blood flow during the cardiac assistance period.

In Fig. 5, note that the LV diastolic pressure for the postassisted beat (2) is 12 mm Hg lower than for the assisted beat (1), providing evidence of unloading the LV. Consequently, the LV systolic pressure and stroke volume were less for the postassisted beat (2) because of the unloading.

The work performed is $\int PdV$, where P and V are pressure and volume. If, in Fig. 5, the $\int PdV$ for the preassisted beat is taken as 100%, the work done by the LV for the first postassisted beat (2) is 64% of that done by the LV before assistance, that is, the work unloading for this beat was 34%.

As discussed in the *Theory* section, two factors affect stroke volume: (1) the train delay and (2) the train duration. The train delay sets the precontraction load for the rectus muscle wrapped around the pouch. Fig. 6 illustrates the augmentation in aortic flow velocity (with the outlet end of the VS-SMV crossclamped) versus the train delay. Note that the

augmentation in aortic root flow velocity (about 160%) was maximum for a train delay from 20 to 80 msec for this 31.8 kg animal. The optimum train delays for the other animals are shown in Table I. The average time from the apex of the first ventricular excitation (Q or R) to the onset of LV ejection was 82 msec (52 to 127 msec) and the average LV ejection period was 309 msec (229 to 400 msec).

Fig. 7 illustrates the effect of train duration on VS-SMV stroke volume, measured by pouch impedance in this animal in which the optimum train delay was 50 msec. Fig. 7 shows that VS-SMV stroke volume can be controlled by varying train duration. At delays greater than 100 msec, there is little increase in VS-SMV stroke volume with increasing train duration. However, it must be recalled that the long-duration limit is that combination of train delay and train duration that causes the skeletal muscle to be relaxed at the end of the LV ejection period.

The effect of increasing train duration to unload the LV is shown in Fig. 8 in which LV/pouch pressure is recorded for train durations of 100, 150, 200, and 250 msec using a train delay of 20 msec. This short delay was used so that the rectus muscle would be relaxed before the end of the ejection period for the longest train duration. In Fig. 8, it is useful to focus on LV diastolic pressure for the preassisted and postassisted beats and systolic pressure for the same beats. For the train durations of 100, 150, 200, and 250 msec, the LV diastolic pressures for the postassisted beats were 2, 5, 12, and 16 mm Hg lower than those for the preassisted beats. The systolic pressures for the postassisted beats were 96%, 92%, 77%, and 34% of the preassisted beat pressures for the 100, 150, 200, and 250 msec pulse trains. Therefore by increasing the train duration, which increases stroke volume, there is the dual evidence of LV unloading.

Pumping capability. The pumping capability of the VS-SMV was determined with a 1:3 pumping ratio and consequently depends on heart rate. Therefore the VS-SMV output is the product of one third of the heart rate and the VS-SMV stroke volume, obtained from the blood-calibrated pouch-impedance recording. Table II presents the VS-SMV stroke volume and output (in milliliters per minute) along with the prevailing heart rate and the train duration used. Also shown are the dog weights. The average VS-SMV stroke volume was 26.1 ml and the average VS-SMV output was 876 ml/min. The normalized resting cardiac output for the dog (and man) is 70 ml/min per kilogram; therefore from

Fig. 5. Ventricular electrogram, LV/pouch pressure, pouch impedance, and aortic root flow velocity. On left and right, LV is unassisted. In center, LV is assisted by VS-SMV with pumping ratio of 1:3. To make this record, the outlet (valve) end of VS-SMV was clamped. Note reduction (12 mm Hg) in LV diastolic pressure for the postassisted beats (2).

Table II the VS-SMV was capable of providing from 12.6% to 106% of the normalized cardiac output with a pumping ratio of 1:3. The average normalized VS-SMV output was 35.6 ml/min per kilogram, that is, 52% of the normal resting cardiac output of 70 ml/min per kilogram.

Discussion

There are many reports on the use of electrically stimulated skeletal muscle to pump blood; the various techniques have been reviewed by Chiu.¹¹ However, there are only a few reports in which the blood pumping was via the LV-to-aorta route. LaFarge and associates¹² appear to have been the first to connect a doubly valved, air-compressed pouch between the LV apex and aorta in a long-term calf study. They reported a considerable reduction in LV work when the pouch was activated. The use of a skeletal muscle to compress a doubly valved pouch at the same site in dogs was reported by Drinkwa-

ter¹³ and Neilson¹⁴ and their colleagues. Stevens¹⁵ and Badylak¹⁶ and their colleagues investigated the doubly valved LV-to-aorta SMV in dogs with pharmacologically induced heart failure. In the study by Stevens and colleagues¹⁵, activation of the SMV during ventricular diastole improved the hypodynamic state considerably. In the study by Badylak and colleagues¹⁶, diastolic pumping with a ratio of 1:2 produced a blood flow equivalent to 45% of the cardiac output with normal ventricles. Lu and associates¹⁷ also reported the use of a doubly valved LV apex-to-aorta SMV, activated during LV diastole with 1:2 ratio in a six-dog study. After a 3-week vascular delay, followed by 6 weeks of muscle conditioning, in short-term studies, the SMVs were able to pump 40% of the blood flow at 3 hours with a pumping ratio of 1:2.

The use of a doubly valved LV apex-to-aorta SMV activated during LV diastole is characterized by a high pumping capability. However, when the

Fig. 6. Augmentation in aortic-root flow velocity (with outlet, valve-end conduit of the VS-SMV clamped) versus train delay (d) for typical dog with stimulus train duration (TD) of 200 msec.

Fig. 7. Relationship between VS-SMV stroke volume and train duration (TD), using a delay of 50 msec.

skeletal muscle is not contracting, the pressure in the pouch is high, and this sustained high pressure compresses the skeletal muscle capillaries and severely compromises muscle capillary blood flow,^{4,5} inviting early fatigue. In the study of Badylak and colleagues¹⁶ the lowest pouch pressure with the

Table I. Timing data

Dog No.	Optimum delay (msec)	R to LV ejection (msec)	Ejection period (msec)
1	—	65	300
2	50-150	73	307
3	10-100	70	330
4	30	127	360
5	10-60	100	400
6	50	60	280
7	20-100	104	256
8	10-40	90	229
9	15-75	52	317
Average	—	82	309

SMV not contracting was 39 mm Hg; in the study by Lu and associates¹⁷ the pressure was 57 mm Hg. Both pressures are more than enough to compress the skeletal muscle capillaries.^{3,4} The VS-SMV described herein eliminates this problem and provides the needed high precontraction pressure and high muscle capillary blood flow during the diastolic interval because pouch pressure is low and equal to LV end-diastolic pressure.

Fig. 8. LV pressure during VS-SMV assistance with pumping ratio of 1:5 for stimulus train durations (*TD*) of 100, 150, 200, and 250 msec with constant delay of 20 msec. Note that as train duration is increased, LV becomes more overempted, revealed by decrease in diastolic pressures (2, 5, 12, and 16 mm Hg) for the first postassisted beats.

In the present VS-SMV study, which used a pumping ratio of 1:3, the average VS-SMV output was 35.6 ml/min per kilogram of body weight. In the normal dog and man at rest, a typical cardiac output is 70 ml/min per kilogram. In other words, the VS-SMV, by itself, pumped $100 \times 36.7/70$, or 52%,

of the cardiac output. However, when one views the pumping capability of the VS-SMV, it is useful to remember that the output depends on the VS-SMV stroke volume and the pumping ratio.

When the VS-SMV is not activated, the LV ejects blood through the aortic valve and the pouch-outlet

Table II. *Pumping data*

Dog No.	Heart rate (beats/min)	VS-SMV stroke volume (ml)	VS-SMV output (ml/min)*	Train duration (msec)	VS-SMV output (ml/min per kg)	Dog weight (kg)
1	100					30
2	120	38.3	1532	250	74.0	20.7
3	108	8.2	224	150	9.29	24.1
4	72	11.7	281	150	10.5	26.8
5	66	17.0	375	200	15.0	25
6	96	38.3	1225	200	49.0	25
7	102	34.6	1177	250	37.0	31.8
8	132	30.8	1357	250	59.0	23
9	84	30.0	840	250	31.1	27
Average	—	26.1	876	212	35.6	25.4

*With a pumping ratio of 1:3.

valve, which can be heard to open and close. In a previous paper⁵ we showed that if the output end of the pouch is closed, blood does not stagnate in the pouch. Saline solution deposited therein was washed out as a result of stretching of the pouch by the LV pressure; the pouch recoiled during diastole thus washing out the saline solution. Of course the dead-end pouch washed out more quickly when the VS-SMV was activated.

Regarding activation of the VS-SMV, it is necessary to place stimulating electrodes on all five or six motor nerves that innervate the rectus muscle. A stimulus frequency of 35 to 40/sec provides a smooth, strong tetanic contraction. Pulses of 0.1 msec allow stimulation with the least electrical energy,¹⁸ because this duration is the chronaxie of a typical mammalian motor nerve. Timing of the stimulus train so that the onset of muscle contraction occurs after the onset of LV ejection is essential so that the skeletal muscle experiences a high preload. In the present study we found the optimum train delay from the R wave to be in the 10 to 100 msec range. In our previous dynamic cardiomyoplasty studies,¹⁹ we found that a train delay in the range of 40 to 80 msec caused the latissimus dorsi muscle to contract just before LV ejection when the ventricles bulge maximally and provided additional preload to the latissimus dorsi muscle. However, the optimum train delay from the Q or R wave of the ventricular electrogram cannot be specified a priori for all subjects; nonetheless, the optimum delay can be found quite easily. With a pumping ratio of 1:3 and selection of a short train duration (e.g., 150 msec), measurement of VS-SMV stroke volume as a function of train delay, as shown in Fig. 6, provides the desired information. Although pouch impedance provides the best indication of VS-SMV stroke volume, aortic root blood

flow velocity can be used provided the outlet of the VS-SMV is crossclamped so that all of the output is sensed by the aortic root flowmeter. After the optimum delay has been found, the train duration can be increased to increase VS-SMV stroke volume, as shown in Fig. 7.

The optimum train delay depends on the rectus contraction latency (Fig. 2, A), that is, the time between the onset of the stimulus train and the onset of muscle contraction. There is very little information on the range of time for unconditioned and conditioned muscle, although it has been found that the contraction latency time increases when skeletal muscle is conditioned. For example, Letson and colleagues²⁰ showed that the time from the onset of the stimulus train to 50% of maximum contraction force in the unconditioned latissimus dorsi muscle was 80 msec. After conditioning, the time to 50% of maximum force increased to 140 msec. Therefore, to accommodate the increase in contraction latency, it may be necessary to use the P wave of the ECG as the timing reference and to measure VS-SMV stroke volume as a function of stimulus train delay. There are no data on the effect of conditioning on the relaxation latency.

In conclusion, from the foregoing it is clear that the VS-SMV unloads the LV, evidence for which is the reduced diastolic pressure and reduced stroke volume for the postassisted beats. The high VS-SMV stroke volume results from the high precontraction pressure, secured by selection of the optimum train delay. Even with a pumping ratio of 1:3, it has been shown that the VS-SMV by itself is capable of pumping about one half of the cardiac output. Because LV and pouch pressure are always the same, muscle capillary blood flow occurs at the same time as coronary artery blood flow, thereby ensuring good muscle perfusion.

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