Power output of skeletal muscle ventricles in circulation: short-term studies

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ABSTRACT Skeletal muscle ventricles (SMVs) were constructed from preconditioned latissimus dorsi muscles in eight dogs and then connected to each animal’s systemic arterial circulation in short-term experiments. The lengths of time that SMVs could produce hemodynamic work as left ventricular assist devices were recorded. After 4 hr of continuous pumping at approximately 55 beats/min, six of eight SMVs were able to generate systolic pressures of 128 ± 23 mm Hg and flows of 340 ± 31 ml/min, representing 20 ± 4% of the animals’ cardiac output. After 8 hr of continuous pumping, five of the eight SMVs generated pressures of 110 ± 15 mm Hg and flows of 308 ± 88 ml/min, or 15 ± 7% of the animals’ cardiac output. The stroke work produced by the SMVs was intermediate between that of the animals’ left and right ventricles. Although the SMVs were stimulated to contract at only about one-third the heart rate, the power output of the SMVs approximated that of the right ventricles because of the greater stroke work of the SMVs. Two SMVs functioned as LVADs for 14 hr. Deterioration in SMV function eventually occurred. In each case, however, complications such as anemia, hypoxia, and hypotension, which are inherent to prolonged short-term experiments of this type, contributed to the deterioration of SMV function. The results presented here suggest that skeletal muscle has the potential to directly support the circulation; however, the length of time such muscle pumps are capable of functioning has yet to be determined.


IN A PREVIOUS EXPERIMENT, skeletal muscle ventricles (SMVs) constructed from canine latissimus dorsi muscles proved capable of generating significant pressures and flows while pumping fluid in an externalized hydraulic test circuit for a 4 hr period.1 Similarly constructed SMVs were capable of generating significant stroke work for prolonged periods (up to 9 weeks) when connected to totally implantable mock circulation devices.2 What is not known is how long SMVs can function as pumps in the systemic arterial circulation. The purpose of this experiment was to determine, in a short-term preparation, the length of time that SMVs could perform useful work as diastolic counterpulsators while pumping blood against an animal’s own systemic vascular resistance.

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Materials and methods

Eight adult male beagles weighing from 11 to 16 kg were studied. Skeletal muscle ventricles were constructed from left latissimus dorsi muscles that had first been subjected to a 3-week period of vascular delay and a subsequent 6-week period of electrical muscle preconditioning. The SMVs were connected to each animal’s own systemic arterial circulation and the lengths of time that the SMVs were able to perform hemodynamic work was calculated. This study conforms to the guiding principles for experimental animals of the American Physiological Society.

Vascular delay and electrical conditioning. At an initial operation, the animals were anesthetized with 30 mg/kg of sodium pentobarbital and placed on a Harvard ventilator. After preparation and draping in a sterile fashion, a 9 cm incision was made along the anterior axillary line, extending from the second rib to the lower costal cartilages. The collateral blood vessels to the latissimus dorsi from the chest wall were then divided. The insertions of the latissimus into the thoracolumbar fascia, pelvis, and tenth rib were identified. The tendons to the latissimus were not disturbed, but all vessels noted within the thoracolumbar fascia were electrocoagulated. The thoracodorsal pedicle was identified, and a specially designed platinum electrode with a silicone rubber cuff that had been joined to a Medtronic lead (Model 6901R) was placed around the thoracodorsal nerve, in a manner previously described.1,2 The thoracodorsal artery and vein were left undisturbed.

The Medtronic lead was then tunneled subcutaneously to a position under the upper right rectus muscle and connected to a
Medtronic unipolar Spectrax pulse generator (Model 5985) placed under the rectus muscle. The wounds were then closed.

The latissimus dorsi muscle was then left undisturbed for 3 weeks to permit the muscle to recover from interruption of its collateral blood supply. The pulse generator was then activated at a rate of 30 beats/min with a hand-held programmer (Medtronic, custom engineering). After 3 days, the pulse generator rate was increased to 60 beats/min, and after an additional 3 days the rate was increased to 120 beats/min (2 Hz). The voltage was set at 3.2 and the pulse width at 0.22 msec. Stimulation was continued at this frequency for 6 to 8 weeks. The stimulated latissimus dorsi muscles in all animals were palpitated at least twice weekly to ensure that they were contracting appropriately. The animals were able to move freely in cages and sustained no apparent disability or discomfort. No animal suffered a wound dehiscence, infection, or pulse generator failure.

Connection with systemic circulation. After the latissimus dorsi muscles had been electrically preconditioned for 6 to 8 weeks, the animals were prepared for the systemic circulation experiment. Anesthesia was induced with sodium pentobarbital (30 mg/kg). An endotracheal tube was positioned and they were placed on a Harvard ventilator. A 5 cm skin incision was made over the pulse generator pocket, and the pulse generator was disconnected from the electrode. A 9 cm incision was then made through the previous scar in the axilla, and the latissimus dorsi was reflected from the chest wall. The insertions of the latissimus into the thoracolumbar fascia, pelvis, and tenth rib were divided. A plane was made between the platysma and latissimus, and the latissimus was mobilized first distally and then left ventrally. The T-system, which itself was passed through the form of a conical spiral around a Teflon patch (Medtronic, custom engineering). A left thoracotomy was performed through the tenth intercostal space, and proximal and distal control of the thoracic aorta was obtained. Heparin was given (300 mg/kg) and the aorta was divided between vascular clamps. A 5/16 or 3/8 inch T-tube system, depending on the aortic size, was placed inside the lumen of the aorta through the cut ends and secured with umbilical tape. The T-tube was then connected to silicone rubber tubing in which a 19-mm Bjork-Shiley valve had been mounted (figure 1). A flowmeter (Biotronex Laboratories BL610) was incorporated and the entire device was connected to the SMV. A sidearm conduit with a screw clamp was positioned and bypassed the Bjork-Shiley valve. When the SMV was at rest, blood entered the SMV cavity through the sidearm around the valve and through leakage back through the closed Bjork-Shiley valve. When the SMV contracted, blood was ejected through the opened valve (and sidearm) into the descending aorta. The system permitted initial control of the filling pressure of the SMV in that, given a relatively constant SMV contraction rate, the diameter of the sidearm could be regulated to permit flow into the SMV cavity to a desired filling pressure. The system tested the ability of the SMVs to serve as diastolic counterpulsators, since the SMVs pumped blood against diastolic arterial pressure and peripheral resistance.

The SMV was made to contract in synchrony with the heart at a ratio of 1:2, 1:3, or 1:4, depending on the animal's heart rate, so that the SMVs pumped at a rate of between 40 and 60 beats/min. This was accomplished by amplifying arterial pressure waveforms with an AVCO intra-aortic balloon pump. The signals were sent to an IC4017 decade counter divider, which could provide selectable 1:1 through 1:9 synchronization with a stimulator. A WPI stimulator (World Precision Instruments 302 D-T) was triggered to provide pulse trains of 300 msec duration at 25 Hz frequency. In those animals with small SMV cavity volumes, pulse trains of shorter duration were used since those SMVs could be emptied with a shorter pulse train duration.

Carotid and femoral arterial catheters were placed and a No. 5F Swan-Ganz thermistor catheter was positioned in the pulmonary artery. External jugular and femoral venous catheters were inserted and the rectal temperature was maintained above 37°C with a heating blanket. Additional oxygen was given so that arterial oxygen saturation was maintained above 90%.

SMV pressures and flows were recorded on an Electronics for Medicine DR 12 recorder, equipped with a rapid-writer printout. Hemodynamic measurements were recorded every 10 min. The SMVs were allowed to contract until they could no longer produce flow. Heparin was given intravenously every hour (100 mg/kg). Arterial blood gases were obtained approximately every 15 min and the ventilator was adjusted when necessary. In five animals, blood transfusions were administered with blood obtained from donor animals.

At the termination of the procedure, the animals were killed with an overdose of anesthetic and the SMVs were inspected grossly.

Histochemistry. Just before the animals were killed, 1 g biopsy samples of the SMV were quickly frozen in liquid nitrogen for later histochemical analysis. Sections were cut on a cryostat and stained for myofibrillar ATPase activity, with acid and alkaline preincubation according to the method of Brooks.
TABLE 1
Length of time of SMV function with factors contributing to SMV failure

<table>
<thead>
<tr>
<th>Animal</th>
<th>Length of SMV function (hr)</th>
<th>Factors contributing to SMV failure</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>8 Anemia (Hct 18%)</td>
<td>Muscle appeared grossly ischemic</td>
</tr>
<tr>
<td>2</td>
<td>14 Hypotension, 2° bleeding from disconnected flow-meter at 4 hr</td>
<td>Anemia (Hct 12%)</td>
</tr>
<tr>
<td>3</td>
<td>2 Anemia</td>
<td>Area of SMV necrosis (outer layer)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hypoxia, 2° pulmonary edema</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(No transformation of muscle into predominantly type 1 fibers)</td>
</tr>
<tr>
<td>4</td>
<td>6 Anemia (Hct 20%)</td>
<td>Respirator failure at 4 hr; ventilator malfunction</td>
</tr>
<tr>
<td>5</td>
<td>9 Respirator failure at 4 hr; ventilator malfunction Cardiac arrest; resuscitation; epinephrine drip; hypoxia</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>12 Systemic hypotension</td>
<td>SMV appeared grossly ischemic</td>
</tr>
<tr>
<td>7</td>
<td>1.5 Sudden cardiac arrest</td>
<td>Ventilator malfunction (?)</td>
</tr>
<tr>
<td>8</td>
<td>14 Systemic hypotension</td>
<td>Hypoxia (Pao2 50 mm Hg)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Anemia (Hct 28%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pulmonary edema</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Transfusion reaction (?)</td>
</tr>
</tbody>
</table>

and Kaiser. The slides were reviewed, and representative muscle bundles were evaluated on both the acid and alkaline stain. The percentages of slow and fast fibers were calculated from each section by averaging the counts from the representative muscle bundles.

Results

The lengths of time that the eight SMVs performed useful work as diastolic counterpulsators ranged from 1.5 to 14 hr (table 1). Factors contributing to deterioration of SMV function are also listed, including bleeding with resultant anemia and hypotension, respirator

TABLE 2
Heart rates, pressures, and resistances (mean ± SD)

<table>
<thead>
<tr>
<th></th>
<th>Heart rate (beats/min)</th>
<th>MAP (mm Hg)</th>
<th>CVP (cm H2O)</th>
<th>PAW (cm H2O)</th>
<th>SVR (dyne-sec-cm⁻²)</th>
<th>PVR (dyne-sec-cm⁻²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Start (n = 8)</td>
<td>194 ± 18</td>
<td>109 ± 9</td>
<td>7 ± 3</td>
<td>6 ± 2</td>
<td>3216 ± 567</td>
<td>449 ± 218</td>
</tr>
<tr>
<td>2 hr (n = 7)</td>
<td>180 ± 19</td>
<td>109 ± 13</td>
<td>8 ± 2</td>
<td>7 ± 1</td>
<td>3494 ± 924</td>
<td>381 ± 96</td>
</tr>
<tr>
<td>4 hr (n = 6)</td>
<td>175 ± 26</td>
<td>104 ± 10</td>
<td>8 ± 2</td>
<td>8 ± 2</td>
<td>4375 ± 707</td>
<td>460 ± 58</td>
</tr>
<tr>
<td>6 hr (n = 6)</td>
<td>175 ± 17</td>
<td>97 ± 10</td>
<td>10 ± 3</td>
<td>8 ± 3</td>
<td>3949 ± 1050</td>
<td>468 ± 104</td>
</tr>
<tr>
<td>8 hr (n = 5)</td>
<td>173 ± 23</td>
<td>96 ± 16</td>
<td>10 ± 2</td>
<td>8 ± 3</td>
<td>3268 ± 1239</td>
<td>475 ± 145</td>
</tr>
</tbody>
</table>

MAP = mean arterial pressure; CVP = central venous pressure; PAW = pulmonary arterial wedge pressure; SVR = systemic vascular resistance; PVR = pulmonary vascular resistance.

malfunction, hypoxia, unexplained cardiac arrest, pulmonary edema, and a possible transfusion reaction.

In three animals, it appeared that ischemia of the SMV muscle layers contributed to deterioration of SMV function. All three SMVs were constructed with one and one-half muscle wraps; in two instances the wall of the SMV composed of the single layer of latisimus muscle looked ischemic. In a third SMV there was an obvious area of ischemic necrosis in the outer layer, while the inner layer appeared pink and contractile.

The mean temperatures, pH, and Pao2 values remained relatively constant over the 8 hr that the SMVs functioned as left ventricular assist devices (LVADs). At the start of the experiment, the mean rectal temperature was 38.5 ± 8°C and 38.6 ± 6°C after 8 hr. The mean blood pH was 7.32 ± 0.06 initially and 7.33 ± 0.05 after 8 hr. The mean arterial blood Pao2 was 119 ± 15 mm Hg initially and 190 ± 9.2 mm Hg after 8 hr. The mean hematocrits fell despite blood transfusions in five of the eight dogs. The mean hematocrit was 32 ± 4.7% initially and 26 ± 7.3% after 8 hr. The blood pressures and calculated vascular resistances are listed in table 2.

Measurements of cardiac output, ventricular stroke volume, and SMV function are listed in table 3. The stroke work of the SMVs was intermediate between the stroke work of the right and left ventricles in this experiment (figure 2, A). Although the rate of contraction of the SMVs was approximately one-third that of the right ventricles, the power output of the SMVs approximated that of the right ventricles because of the greater stroke work of the SMVs (figure 2, B). The mean amount of blood pumped by the SMVs (in ml/min) is depicted in table 3. SMV output is expressed as a percentage of the cardiac output in table 3 and was approximately 20% of the animals' cardiac output at 4 hr and 15% of the cardiac output at 8 hr.

Hemodynamic pressure tracings after 7.5 hr of continuous SMV pumping are shown in figure 3. Even
TABLE 3
Cardiac and SMV function (mean ± SD)

<table>
<thead>
<tr>
<th>Common LV and RV function</th>
<th>SMV function</th>
<th>Femoral diastolic augmentation of cardiac output</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac output (l/min)</td>
<td>SMV rate (beats/min)</td>
<td>SMV systolic pressure (mm Hg)</td>
</tr>
<tr>
<td>Stroke volume (ml)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Start (n = 8)</td>
<td>2.6 ±0.6</td>
<td>14 ±4</td>
</tr>
<tr>
<td>2 hr (n = 7)</td>
<td>2.5 ±0.6</td>
<td>14 ±3</td>
</tr>
<tr>
<td>4 hr (n = 6)</td>
<td>1.8 ±0.3</td>
<td>11 ±3</td>
</tr>
<tr>
<td>6 hr (n = 6)</td>
<td>1.9 ±0.5</td>
<td>14 ±3</td>
</tr>
<tr>
<td>8 hr (n = 5)</td>
<td>2.4 ±1.0</td>
<td>14 ±7</td>
</tr>
</tbody>
</table>

after 14 hr, one SMV still generated a flow of 211 ml/min, or 15% of that animal's cardiac output.

Histochemical analysis revealed the expected conversion from a muscle with a heterogeneous mixture of type I fatigue-resistant and type II fatigue-prone fibers to a muscle of predominantly type I fibers in seven of eight dogs (figure 4). In those seven dogs, greater than 85% of muscle cells examined contained evidence of slow myosin (type I fibers) by ATPase staining. Contralateral control latissimus was not routinely evaluated in this study but in a previous report was found to contain 45 ± 7.6% type I slow fibers and 57 ± 7.8% fast fibers. One animal (dog 3) had essentially no transformation of fibers observed on muscle biopsy specimens stained at alkaline and acid pH for ATPase.

Biopsy specimens were taken from four different areas of the latissimus dorsi skeletal muscle ventricle. The most proximal portion of the muscle (near the neurovascular bundle) was labeled zone A, the most distal area zone D. Zone D was therefore the innermost portion of the skeletal muscle wrap and zone A was the outermost portion. Zones A and B in most animals tended to have normal preservation of fiber architecture. There tended to be some degree of muscle damage in the zone D specimens, except for dog 1, in which no areas of muscle damage were observed. Muscle damage consisted of areas with vacuolization and loss of distinct fiber morphology. There was an increase in connective tissue in all SMVs. In some there was a large amount and in others there was much less.

Pilot study. Reasoning from the results of the other eight dogs that there are significant complications inherent to prolonged short-term experiments of this type, we conducted a pilot study with a ninth dog. The SMV was constructed in a similar manner to the other eight but it was constructed during the first operation so as to avoid interruption of newly formed collateral blood vessels with adjacent tissues, as possibly occurred in the other eight dogs. At a second operation, the SMV was connected to the aorta in a similar manner to the other eight, but a flowmeter was not incorpo-
rated into the circuit and a porcine valve was used instead of a mechanical valve. The SMV was activated via its motor nerve by a totally implantable prototype Medtronic R-wave synchronous burst stimulator. The electrical pulse train could be delayed so that it occurred during diastole, thereby allowing the SMV to function as a diastolic counterpulsator as with the other eight animals. If the animal’s heart rate was less than 100 beats/min, the pulse generator fired every other heart beat (1:2) but blocked down progressively as the heart rate increased above that.

After the SMV was connected to the dog’s circulation and the pulse generator activated, the animal’s chest was closed and the animal was allowed to recover. The dog was anticoagulated initially with intravenous heparin. This was followed by subcutaneous heparin every 8 hr. The dog was ambulatory the next day and was tether-free in that no tubes or wires crossed the skin except when measurements were being made. Arterial pressure tracings and an electrocardiogram with the superimposed electrical pulse train is shown in figure 5, recorded after 24 hr of continuous pumping. The animal’s heart rate was 200 beats/min at the time of the recording and therefore the pulse generator was discharging at a ratio of 1:5 with the heart rate.

On the second day after surgery, the SMV was noted to be contracting vigorously by palpation. The animal died suddenly that day, before arterial pressure measurements were made. The cause of death was not determined at autopsy.

Discussion
In these short-term experiments, the SMVs were capable of generating significant systolic pressures and outputs that were approximately 20% of the animals’ own left ventricles after 4 hr of continuous pumping. By 8 hr, three of the SMVs had failed. However, the remaining five were still capable of generating significant systolic pressures and outputs that were approximately 15% of the animals’ own hearts. Even after 14 hr, one of the two remaining SMVs could still generate 15% of the output of the animal’s own heart.

Previous attempts to use skeletal muscle as an LVAD have failed, sometimes in a matter of minutes. The failure of SMV function has usually been attributed to primary skeletal muscle fatigue. Neilson et al. have demonstrated diastolic augmentation with SMVs of their own design for hours but did not measure stroke work. Our SMVs differed from those reported by others in two important ways. First, we used a vascular delay period. In previous experiments we found that intramuscular blood flow during exercise is severely impaired in the latissimus dorsi muscle when the collateral blood supply is divided and the muscle is immediately exercised. We have shown, however, that a substantial exercise-induced increase in blood flow will return if the muscle is allowed to rest for 3 weeks after the collaterals are interrupted. A second way in which these SMVs differed from those reported in previous experiments by others is that after the vascular delay period, the muscles were electrically preconditioned for an additional 6 weeks to make them fatigue resistant. Long-term low-frequency stimulation has been shown by other investigators and more recently in our own laboratory to cause the muscle to change its contractile proteins, metabolic enzymes, and even the mitochondrial fraction, so that the muscle is better equipped to perform the long-term, relentless type of work required for cardiac augmentation. These changes were reflected in the histochemical analysis of the muscle in this experiment. Although it is not yet known if electrical preconditioning is a necessary prerequisite for long-term hemodynamic
work, in earlier short-term experiments we were able to demonstrate an advantage over SMVs that had not been electrically preconditioned.1

We have previously demonstrated that SMVs constructed from electrically preconditioned muscle can function over long periods when connected to a totally implantable mock circulation system.2 In that study, the SMVs pumped continuously against an afterload of 80 mm Hg with a preload of 40 to 50 mm Hg at a contraction rate of 54/min. Those preload and afterload pressures were similar to the pressures in this study. At the initiation of pumping, the SMVs connected to the mock circulation generated a mean systolic pressure of 135 mm Hg and a flow of 464 ml/min. The pressures and flows generated by the SMVs connected to the mock circulation were similar to those obtained in the present experiment. After 2 weeks of continuous pumping, the mean systolic pressure of the SMVs connected to the mock circulation system was 104 mm Hg and the mean flow was 206 ml/min. Two long-term SMVs pumped continuously for 5 and 9 weeks, respectively.

In a previous short-term experiment19 in which SMVs of a slightly different design were connected to the systemic circulation as diastolic counterpulsators for 4 hr, SMV stroke work was 0.65 ± 0.23 ergs × 10^6. The SMV stroke work was intermediate between that of the animals’ left and right ventricles. The mean stroke work of the left ventricles in that experiment was 1.9 ± 0.89 ergs × 10^6 and for the right ventricles 0.22 ± 0.13 ergs × 10^6. Similarly, after 1 week of continuous pumping against the mock circulation,2 SMV stroke work was 0.61 ± 0.28 ergs × 10^6, again intermediate between the left and right ventricles. Increasing the burst frequency to 85 Hz more than doubled the stroke work in that experiment. For example, the SMV of one dog after 1 week of continuous pumping was generating continuous stroke work of 1.2 ergs × 10^6, a flow of greater than 400 ml/min, and a systolic pressure of 143 mm Hg. When the burst frequency was increased to 85 Hz, the pressure obtained (preload 40 mm Hg, afterload 80 mm Hg) was 168 mm Hg, the flow 700 ml/min, and the stroke work 2.3 ergs × 10^6. A comparison of the stroke work and power out-
put of the SMVs in the present study (figure 2) with these data suggests that if the SMVs could function for prolonged periods in the circulation, they should be able to replace the entire function of the right ventricle and at least a portion of the function of the left ventricle.

The function, however, of each of the eight SMVs eventually deteriorated in this short-term experiment. In each preparation there was an identifiable problem, other than primary muscle fatigue, that may have contributed to or caused the failure of the LVADs function. Each animal was systemically anticoagulated, which led to a slow but steady loss of blood and eventual hypotension. Anemia (hematocrit less than 30%) ultimately proved to be a problem in all cases despite transfusions in five animals (table 1). Three animals became acutely hypoxic, due to respirator malfunction or suspected pulmonary edema, which in one animal was believed to be secondary to a transfusion reaction. In addition, pentobarbital was used intravenously as the anesthetic agent, with its potential ability to depress the activity of both the skeletal and cardiac muscle.

There were other factors that probably also contributed to the deterioration of SMV function during these experiments. The method of construction of the SMVs differed from that used in two previous studies. In those studies, SMVs were constructed during the first surgical procedure at the time the collateral blood supply to the SMV was divided. The SMVs were then rested for at least 3 weeks to permit an ingrowth of a new collateral blood supply. They were then electrically preconditioned before being used as a pump as in this study. In the present study, the collaterals to the latissimus dorsi were similarly divided during the first operation; however, the muscles were left in situ. At the second and final operation, the muscles were mobilized and the multilayered SMVs were constructed. During the same short-term and final experiment, the SMVs were connected to the systemic circulation. This method of delayed construction disrupted newly formed collateral blood vessels with adjacent tissue, which probably also contributed to the failure of the SMVs in this study. Three of the SMVs appeared grossly ischemic at the time the SMVs functioned as LVADs. Those muscles also appeared ischemic at the time the animals were killed. None of the SMVs in the earlier two studies appeared grossly ischemic at any time. In fact, in a previous short-term in-circulation study, transmural blood flow to the inner and outer layers of the SMV was measured while the SMVs functioned as LVADs. No evidence of muscle ischemia was detected by a microsphere technique to measure intramuscular blood flow. Those SMVs had been constructed during the first operation and then rested subcutaneously for 3 weeks before being electrically preconditioned and then used as a pump.

In summary, SMVs were capable of functioning as LVADs in the circulation for several hours. The stroke work and power output of the SMVs reported here compare favorably with those generated by the animal’s own heart. Each of the SMVs eventually deteriorated, but the deterioration was likely caused by problems inherent to prolonged experiments of this type. The results of these experiments suggest that skeletal muscle has the potential to directly support the circulation; however, the length of time such muscle pumps are capable of functioning has yet to be determined.
References

J D Mannion, M A Acker, R L Hammond, W Faltemeyer, S Duckett and L W Stephenson

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