Blood Flow Pattern in the Thoracodorsal Artery after Dynamic Cardiomyoplasty

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Abstract

To understand if a non invasive determination of blood flow to the Latissimus Dorsi Muscle (LDM) could be used as an indirect index of muscle viability, we postoperatively measured the thoracodorsal artery (IDA) blood flow variations during latissimus dorsi stimulation in 4 patients who had a cardiomyoplasty.

The TDA was in all cases identified by a characteristic biphasic blood flow pattern. We measured diameter (mm), mean velocity (cm/sec), systolic/diastolic ratio (S/D) and calculated blood flow (ml/min) during 5 minutes of latissimus dorsi stimulation at 2:1 and 1:1 synchronisation ratio while changing the voltage applied to the muscle so to obtain various strength of contraction. Mean blood flow was 33.75 ml/min in basal condition and increased to 42.83 ml/min during a synchronisation rate of 1:2 at a voltage of 150% of clinical setting. There was a further increase up to 50.99 ml/min for a synchronisation rate of 1:1 while, at the same time, a marked reduction of S/D ratio (from 4.29 to 1.97) was detected. In conclusion blood flow in the TDA appears to be directly related to metabolic needs of the stimulated muscle and this non invasive technique can be useful to follow-up patients and control long term muscle viability.

Key words: cardiomyoplasty, thoraco-dorsal artery, blood flow, heart failure, Doppler technique.

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In cardiomyoplasty the latissimus dorsi muscle is surgically wrapped around the heart and stimulated with a cardiomyostimulator to assist the failing heart.

The optimal performance of the latissimus dorsi muscle after cardiomyoplasty depends upon many factors such as: 1) an appropriate synchronization between the muscle and heart contractions so that the maximum work output is only obtained during cardiac systole, 2) the capacity of the Latissimus Dorsi Muscle (LDM) to withstand a chronic work strong enough to be effective in relieving the failing heart, 3) the necessity of avoiding an excessive stimulation possibly leading to muscle damage [5, 9], 4) the integrity of the muscle innervation and, last but not least, 5) a good blood supply to the muscle.

In fact, assessing blood flow characteristics and its modifications both in the initial training period and during the chronic ventricular assistance could be interesting and useful to avoid situations in which a muscular ischemia could be detrimental.

Materials and Methods

The study was conducted in 4 patients treated with the cardiomyoplasty procedure for heart failure between 1993 and 1995. All these patients, submitted to a Dynamic Cardiomyoplasty (DCMP) at least one year before, were reputed to have at the time of the study a still viable and well contracting muscle flap as seen both by transthoracic echocardiography and by palpatory methods. Patient characteristics are summarized in table 1.

To identify the thoracodorsal artery (TDA), patients were examined lying on their right side with the left arm slightly raised and abducted. A 7.0 MHz ultrasound probe was placed at the mid-axillary line in the third left intercostal space with a 60° angle and the probe was set for 40-mm depth. The TDA was identified, using the echo technique, just over the entrance site of the muscle wrap into the thoracic cavity. At this level the muscular flap is not very large and with a quick scanning the vascular pedicle is easily found and identified from other arteries (i.e. intercostal) for its distinct flow pattern [4]. In fact,
Blood flow pattern in the thoracodorsal artery after dynamic cardiomyoplasty

Table 1. Patient population.

<table>
<thead>
<tr>
<th></th>
<th>age</th>
<th>sex</th>
<th>Present NYHA</th>
<th>etiology of CHF</th>
<th>date of DCMP</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>57</td>
<td>m</td>
<td>I</td>
<td>idiopathic</td>
<td>Feb-93</td>
</tr>
<tr>
<td>2</td>
<td>53</td>
<td>m</td>
<td>I</td>
<td>ischaemic</td>
<td>Jun-93</td>
</tr>
<tr>
<td>3</td>
<td>67</td>
<td>f</td>
<td>I</td>
<td>idiopathic</td>
<td>Dec-93</td>
</tr>
<tr>
<td>4</td>
<td>66</td>
<td>m</td>
<td>II</td>
<td>ischaemic</td>
<td>Jan-95</td>
</tr>
</tbody>
</table>

while muscular arteries always show a triphasic pattern (forward-reverse-forward flow), the TDA in the transposed LDM has a distinct biphasic pattern constituted by a systolic peak followed by a low descending diastolic curve.

To study the chronic blood flow pattern and the blood supply to the LDM in patients submitted to DCMP and how these parameters could be modified by the stimulation applied, we designed a protocol in which all flow parameters were recorded varying both the stimulation ratio and the voltage applied to the LDM. As the clinical cardiomyostimulator settings were different for each patient, we considered standard for each patient the values set chronically in the last 3 months. Between patients, major differences in the stimulation of LDM were represented by the voltage applied (range 3.8 to 6 volts) and the synchronisation delay (range 16 to 55 msec), but these settings were kept untouched. At the time of the study all patients had 6 pulses per burst at a 1:2 synchronisation ratio.

Using transthoracic Color Doppler Echocardiography (Acuson 128 XP 10 Art) on B-mode imaging, diameter and cross-sectional area of the TDA were measured and simultaneously pulsed Doppler signals were recorded to study the blood flow pattern. The following parameters were derived or calculated from the shape of the Doppler signal curve: peak systolic velocity (Vmax), peak diastolic velocity (Vmin), average velocity (TAV) and systolic/diastolic ratio (S/D). Blood flow was computed as the product of TAV and the cross sectional area as described by De Bono and coll. [3].

While keeping the probe in the same place, with a synchronisation rate of 1:2, the muscle contracting force was incremented by stepwise increases in voltage, keeping each new voltage setting for three minutes before a 1 minute recording. The voltage was varied over a range from 50% of the "chronic" voltage (the voltage at which the muscle was clinically stimulated) up to a 150% of it. For each step the same parameters cited above were recorded. To minimise possible effects of the previous stimulation on parameter readings, the cardiomyostimulator was set to off for 5 minutes between each step (Table 2).

Results

The thoracodorsal artery of the transposed LDM was easily identified within 4 minutes in all patients. In basal condition, with the latissimus dorsi muscle not stimulated, total blood flow ranged from 15.26 ml/min to 50.24 ml/min with a mean of 33.75 ml/min. The lowest blood flow was measured in a patient with a cardiac index of 1.81/min. S/D ratio averaged 4.29 ± 1.51, and the thoracodorsal artery mean diameter was 2.03 mm (range 1.8-2.3 mm).

With a synchronisation ratio of 1:2 and increasing the voltage of stimulation from 50% of the "chronic voltage" up to a 150% of it, we observed a progressive blood flow increase (from 33.75 ml/min to 42.83 ml/min) and a simultaneous reduction of S/D ratio (from 4.29 to 2.74). Repeating the same steps with a synchronisation ratio of 1:1, we observed a further increase of blood flow (from 36.94 ml/min to 50.99 ml/min) and a marked reduction of S/D ratio (from 3.13 to 1.97). During the recovery period at the end of the protocol, we turned the cardiomyostimulator off and recorded a blood flow reduction and a S/D increase. However total blood flow was in every patient above basal.

Table 2. Study protocol.

<table>
<thead>
<tr>
<th>Step</th>
<th>Voltage to LDM</th>
<th>Stimu. ratio</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Chronic Voltage (CV)</td>
<td>1:2</td>
<td>Identify TDA (less than 4 minutes)</td>
</tr>
<tr>
<td>2</td>
<td>0</td>
<td>1:2</td>
<td>5' Recording of basal flow, artery diameter and cross sectional area</td>
</tr>
<tr>
<td>3</td>
<td>50% of the CV</td>
<td>1:2</td>
<td>After 3' stabilisation, 1' Recording</td>
</tr>
<tr>
<td>4</td>
<td>CV</td>
<td>1:2</td>
<td>After 3' stabilisation, 1' Recording</td>
</tr>
<tr>
<td>5</td>
<td>150% of CV</td>
<td>1:2</td>
<td>After 3' stabilisation, 1' Recording</td>
</tr>
<tr>
<td>6</td>
<td>0</td>
<td>1:2</td>
<td>5' Recording of basal flow, artery diameter and cross sectional area</td>
</tr>
<tr>
<td>7</td>
<td>50% of the CV</td>
<td>1:1</td>
<td>After 3' stabilisation, 1' Recording</td>
</tr>
<tr>
<td>8</td>
<td>CV</td>
<td>1:1</td>
<td>After 3' stabilisation, 1' Recording</td>
</tr>
<tr>
<td>9</td>
<td>150% of CV</td>
<td>1:1</td>
<td>After 3' stabilisation, 1' Recording</td>
</tr>
<tr>
<td>10</td>
<td>0</td>
<td>1:1</td>
<td>5' Recording of basal flow, artery diameter and cross sectional area</td>
</tr>
</tbody>
</table>

CV = Chronic voltage: the voltage at which the patient’s muscle is chronically stimulated; LDM = Latissimus Dorsi Muscle.
values for about five minutes, and S/D gradually increased towards the baseline values (Fig. 1-2).

Discussion

In skeletal muscle arteries, blood flow velocity during Doppler investigation displays a typical triphasic pattern characterised by a steep systolic rise followed by a lower and longer diastolic wave. This happens because during systole the heart muscle contracts and peripheral myocardial vessels, particularly at the subendocardial level, are compressed by the muscular fibers so blood can only flow mainly during diastole [7]. A similar biphasic flow pattern has also been described in patent coronary artery bypass grafts [2].

In patients we have studied, the same pattern of blood flow was recorded in the thoracodorsal artery after DCMP. This phenomenon may be partly explained by the similar phasic contraction and relaxation of the transposed and, electrically stimulated LDM. Nonetheless, when the synchronisation ratio was set to 1:2, we were not able to detect any major difference of blood flow pattern between the stimulated versus the unstimulated beat. When the cardiomyostimulator was switched off, the diastolic component of the flow was much lower. Possible explanations for this could be either a different behaviour of blood flow to muscle irrigated by a single terminal artery or the fact that the transposed LDM is so attached to the epicardium after DCMP that during stimulation the vascular bed is squeezed mimicking the effect of myocardial contraction on the coronary bed, but certainly more investigations are necessary to explain this modified pattern.

Increasing the voltage applied to the LDM causes more muscular fibers to be stimulated to simultaneously contract therefore increasing both vascular peripheral resistance in the muscle and its metabolic demands. In fact, in response to any increase in the voltage applied to LDM, we have observed a parallel increase of total blood flow and a decrease of the systo-diastolic ratio indicating that at higher voltages most of the flow happens during diastole.

As it has been shown that for a higher rate of stimulation, when the relaxation time between contractions shortens, the useful time of muscle perfusion lessens just when metabolic demands are higher [6, 10, 11]. It is possible to speculate that, in some cases, if the muscle were stimulated for long periods of time at high rates, blood supply to the muscle could be reduced enough to cause some degree of ischemia to the muscle and lead to chronic damage. This could partly explain the poor histological results found by Moreira et al. [9] in autopsy specimens of patients operated in the first years of DCMP experience when a 1:1 synchronisation ratio was the standard.

The use of a non-invasive method like color Doppler echocardiography to study blood flow variations in the thoracodorsal artery is to be considered a potentially important tool for patients' follow-up. Early in the postoperative days, it could be used to control the adequacy of blood flow to the muscle especially when a rise in CK enzymes poses doubts over flap vascularization. At long term, variation in blood flow in the TDA in response to any variation of the stimulation voltage could be used as an indirect but reliable method to check peripheral muscle viability.

Color Doppler echocardiography methods have some limitations. The error in vessels with diameters smaller than 2 mm approaches 20% [1]. An average diameter of 1.8 mm has been reported by Dominici and coll. [4] in a study of 104 patients examined to evaluate the latissimus dorsi vascular supply before and after surgical procedures for reconstructive surgery.

Simpson and associates [12] compared Doppler derived flow determinations with electromagnetic flowmeter recordings studied intraoperatively and found that the measurements obtained by ultrasound methods were overestimated. In fact the variations of absolute blood flow values with the Doppler ultrasound technique is not surprising because a minor variation in measured diameter of, for example, 0.3 mm in a vessel with an internal diameter smaller than 3 mm would cause a 36% difference in computed blood flow with the Doppler technique.

In our study we tried to minimize these errors. The vessel diameter was always measured by the same operator at the beginning of the study, and this measurement was used as a fixed value for the entire examination period. Moreover, even if vessel diameter and area were hand calculated and so subjected to operator error, we considered diameter values fixed for the entire duration of the study so to keep...
the error constant in each step and be able to study the absolute variations of flow.

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**References**


