INTRAMUSCULAR PRESSURE AND MUSCLE BLOOD FLOW DURING EXERCISE IN CHRONIC COMPARTMENT SYNDROME

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In nine patients with chronic compartment syndrome, the intramuscular pressure and muscle blood flow during constant dynamic exercise was studied by the microcapillary infusion method and by the 133-xenon clearance technique. Although muscle blood flow was normal at the start of exercise, pain and impaired muscle function eventually developed; muscle blood flow decreased while muscle relaxation pressure increased. The changes of muscle blood flow could not be correlated with any change of mean muscle pressure during exercise.

Eight months after fasciotomy the exercise test was repeated. Patients experienced no symptoms and the muscle relaxation pressure and blood flow during exercise were normal. It is suggested that chronic compartment syndrome is due to increased muscle relaxation pressure during exercise which causes decreased muscle blood flow, leading to ischaemic pain and impaired muscle function.

Chronic compartment syndrome of the lower leg, first described by Mavor in 1956, is characterised by pain induced by exercise, swelling and impaired muscle function (Reneman 1975). Different parameters have been used or considered for use in diagnosis, for example, intramuscular pressure at rest after exercise (French and Price 1962; Reneman 1975; Veith 1980; Wallensten 1983); the mean muscle pressure during exercise (Pur- anen and Alavaikko 1981; McDermott et al. 1982); and the muscle contraction pressure during exercise (McDer- mott et al. 1982). Although the pathogenesis of chronic compartment syndrome is related to increased intramuscular pressure, the critical level that elicits symptoms is incompletely understood.

Because skeletal muscle is not perfused during a contraction (Barcroft and Millen 1939), arterial inflow to its vascular bed during dynamic exercise occurs only between contractions (Folkow, Gaskell and Waaler 1970), that is, during muscle relaxation. An increase of the muscle relaxation pressure, which is the lowest value of intramuscular pressure between two muscle contractions, may therefore impede muscle blood flow during exercise and result in ischaemic pain and impaired muscle function. For this reason, it is of interest to study muscle blood flow during dynamic exercise as a function of muscle relaxation pressure in patients with chronic compartment syndrome.

PATIENTS AND METHODS

The criteria for diagnosing chronic compartment syndrome were the development of pain, swelling and impaired muscle function during exercise (described below); increased pressure (more than 35 mmHg) at rest after exercise; and a prolonged time (more than six minutes) for normalisation of the pressure after exercise (French and Price 1962; Reneman 1975). Twenty-two patients met these criteria and nine (six men and three women) volunteered to participate in our study, which was carried out between 1981 and 1984. Their mean age was 29 years (range 18 to 51). Chronic compartment syndrome was unilateral in five and bilateral in four of the patients, so that 13 affected legs and five symptom-free legs could be investigated. The study was approved by the Ethical Committee of the University of Göteborg.

Experimental set-up. Each patient was examined once before fasciotomy and once at a mean of eight months (range 3 to 9) after operation; the same examination procedure was followed on both occasions, and screening tests carried out before the first examination allowed all patients to become accustomed to the experimental situation. Intramuscular pressure was recorded at rest as well as during and after exercise. While the recordings were being made, the patient lay supine with the feet in shoes attached to an ergometer (Fig. 1) which kept the work constant and equal bilaterally. The pedals of the device were loaded with 4 to 6 kg, depending on the
patient's muscle strength. The patient was asked to dorsiflex both feet once a second for five minutes, this exercise being briefly interrupted to allow injection of the radio-isotope used to measure muscle blood flow; the patient was then asked to continue the exercise until muscle fatigue, pain or impaired muscle function developed.

Intramuscular pressure. This was measured in both legs at the same time by the microcapillary infusion method described in detail elsewhere (Styf and Körner 1986). The length of the teflon catheter (Myopress, Atos Medical, Höby, Sweden) was 30 cm and the outer diameter 1.05 mm. It was connected to an electromagnetic transducer (Siemens Elema 746) with a displacement of 0.06 mm³/0.014 MPa (=0.57 × 10⁻⁶ ml/mmHg) and a multichannel ink recorder (Siemens Elema, Mingograph 82). The reference pressure was chosen at a level of 5 cm below the manubrium sterni. The dynamic functioning of the system was tested by external compression and by active muscle contraction. An actual recording from an experiment is shown in Figure 2.

The pressure amplitude during exercise was calculated by subtraction of the muscle relaxation pressure (MRP) from the muscle contraction pressure (MCP). The mean muscle pressure (MMP) during exercise was calculated by adding the MRP to half the value of the pressure amplitude according to the formula:

\[ \text{MMP} = \frac{[(\text{MCP} - \text{MRP})/2] + \text{MRP}}{2} \]

Muscle blood flow. This was measured bilaterally in the anterior tibial muscle by the 133-xenon clearance technique (Lassen, Lindbjerg and Munck 1964), which gives reliable information on local muscle blood flow during standardised dynamic exercise (Tonnesen 1964). Approximately 3.7 MBq (=0.1 mCi) of 133-xenon in 0.1 ml of isotonic saline was injected into the anterior tibial muscle 1 cm to 2 cm from the tip of the catheter at a depth of 2 cm from the skin. Each detector was connected to a separate pulse-height analyser with a window set for the 80 keV photo peak of 133-xenon. The output from the rate meter (time constant = three seconds) was recorded on a logarithmic potentiometer writing on linear graph paper. The muscle blood flow was calculated from the slope of the clearance curve (Lassen et al. 1964). A recording from one of the experiments is shown in Figure 3. The muscle blood flow was determined three times during each investigation. The first reading was made during exercise three to five minutes after the injection; the second when the patient
Muscle relaxation pressure (MRP) and muscle blood flow (MBF) during exercise in the affected legs. A (pre-operative), after three to five minutes of submaximal work with no pain in the leg; B (pre-operative), at the end of exercise with severe pain and impaired muscle function; C (post-operative), at the end of exercise and after the onset of muscle fatigue. The bars indicate one standard deviation.

experienced pain and when muscle function was impaired at the end of the exercise; and the third immediately after the exercise had finished. No readings were accepted when the counting rate decreased to less than 20% of the initial value.

Fasciotomy. Fasciotomy of the anterior compartment in the 13 legs with symptoms was performed under general anaesthesia with tourniquet haemostasis. An incision 7 cm to 8 cm long was made at the lateral border of the anterior muscle compartment. The fascia was incised subcutaneously 2 cm laterally to the anterior ridge of the tibia along the whole muscle compartment from the fibular head to the anterior retinaculum just proximal to the ankle joint.

Significance. Pressure values were recorded as means with one standard deviation. Using Student's t-test, significance was accepted as p < 0.05.

RESULTS

These are summarised in Table I.

Pre-operative. The pre-operative muscle pressure at rest was 8.7 mmHg in the 13 affected legs compared with 6.7 mmHg in the symptom-free legs (p < 0.05). After three to five minutes of constant submaximal exercise no patient experienced discomfort, and the muscle relaxation pressure in all 18 legs had doubled. At the same time the muscle blood flow was 41.5 and 37.3 ml/100g/min in the two groups, respectively (Figs 4A and 5A).

After further constant submaximal dynamic exercise, pain in the anterior compartment eventually occurred in the affected legs; during this time, when the muscle relaxation pressure in these legs again more than doubled to 42.6 mmHg, the muscle blood flow had significantly decreased to 12.2 ml/100g/min. Changes for the five symptom-free legs, however, were less marked (p < 0.001; Table I, Figs 4B, 5B and 6).

Muscle relaxation pressure (MRP) and muscle blood flow (MBF) during exercise in the symptom-free legs. A (pre-operative), after three to five minutes, submaximal work with no pain; B (pre-operative), at the end of exercise when the patient experienced severe pain and impaired muscle function in the contralateral leg; C (post-operative), at the end of exercise after operation on the contralateral leg and after the onset of muscle fatigue.

After exercise the muscle blood flow in three patients with unilateral pain was monitored in both legs; this fell to resting values within three to five minutes in the symptom-free legs but, in the affected legs, there was a delay of about one minute before the onset of the hyperaemia, which lasted more than five minutes.

Postoperative. When the exercise test was repeated eight months after fasciotomy, the pain during exercise felt previously by all patients had disappeared. Other values for muscle relaxation pressure and muscle blood flow for both groups are shown in Table I.

In the 13 affected legs, the muscle contraction pressure had decreased from 150 mmHg before fascio-
Table I. Results of testing nine patients (18 legs) before and after fasciotomy

<table>
<thead>
<tr>
<th></th>
<th>13 legs with chronic compartment syndrome</th>
<th>5 painfree legs</th>
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<tbody>
<tr>
<td><strong>Pre-operative</strong></td>
<td></td>
<td></td>
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<tr>
<td>Muscle relaxation pressure (mmHg)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>At rest</td>
<td>8.7 ± 1.6</td>
<td>6.7 ± 1.0</td>
</tr>
<tr>
<td>After exercise (3 to 5 minutes, no discomfort)</td>
<td>19.1 ± 6.4</td>
<td>13.1 ± 2.8</td>
</tr>
<tr>
<td>After increased exercise (until pain in affected legs)</td>
<td>42.6 ± 6.8</td>
<td>20.8 ± 4.7</td>
</tr>
<tr>
<td>Muscle blood flow (ml/100g/min)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>After exercise (3 to 5 minutes, no discomfort)</td>
<td>41.5 ± 11.1</td>
<td>37.3 ± 9.5</td>
</tr>
<tr>
<td>After increased exercise</td>
<td>12.2 ± 8.3</td>
<td>39.2 ± 7.5</td>
</tr>
<tr>
<td>Muscle contraction pressure (mmHg)</td>
<td>150 ± 49</td>
<td>141 ± 50</td>
</tr>
<tr>
<td>Mean muscle pressure (mmHg)</td>
<td>97</td>
<td>81</td>
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| **Postoperative**                 |                                          |                |
| Muscle relaxation pressure (mmHg) |                                          |                |
| At rest                           | 5.5 ± 1.6                                | 6.0 ± 0.8      |
| After exercise                    | 14.5 ± 6.5                               | 24.9 ± 2.6     |
| Muscle blood flow (ml/100g/min)   | 49.9 ± 8.6                               | 47.0 ± 3.7     |
| Muscle contraction pressure (mmHg)| 141 ± 41                                 | 161 ± 41       |
| Mean muscle pressure (mmHg)       | 78                                       | 93             |

tomy to 141 mmHg after. In the non-operated, symptom-free legs, it had increased from 141 to 161 mmHg after operation but these differences were not statistically significant.

The mean muscle pressure had decreased from an average of 97 to 78 mmHg after operation in the affected legs, while in the others it had increased from 81 to 93. These pressure parameters were stable and did not increase during the test; at the end of exercise and before operation, when the legs became painful, these parameters even decreased slightly. The changes of muscle blood flow and the development of symptoms in the affected legs could not be correlated with any change of the mean muscle pressure during the exercise test.

The average systolic blood pressure was 138 mmHg and the diastolic pressure 77 mmHg at rest before exercise; the mean blood pressure was calculated to be 97 mmHg.

DISCUSSION

Our study showed that muscle blood flow had been normal in the anterior tibial muscle at the start of constant dynamic exercise in patients with chronic compartment syndrome; when they eventually experienced pain, swelling and impaired muscle function during the exercise test, the muscle blood flow had significantly decreased. In the five patients affected in one leg the muscle relaxation pressure had significantly increased compared with the symptom-free leg. The development of symptoms during exercise correlated well with the decrease of muscle blood flow and seemed to have been due to the relative muscle ischaemia that developed. The change of muscle blood flow during the pre-operative exercise test could not be related to any significant change of the muscle contraction pressure nor to the mean muscle pressure. Fasciotomy eliminated the pain and resulted in normal muscle blood flow and relaxation pressures during exercise.

The perfusion of skeletal muscle during constant submaximal dynamic exercise occurs only during the phase of muscle relaxation (Folkow et al. 1970). In our study the decreased blood flow during exercise correlated well with the pathological increase of muscle relaxation pressure in the symptomatic legs. This finding has not previously been reported, although the influence of increased muscle pressure on muscle blood flow at rest has been investigated by other authors (Ashton 1975; Sheridan and Matsen 1975; Ogata and Whiteside 1982). These studies indicate that muscle blood flow may be impeded when intramuscular pressure at rest exceeds 30 to 50 mmHg. Although we did not record blood pressure during exercise in our patients, these earlier reports seem to support our finding that the critical range of muscle relaxation pressure causing muscle ischaemia during exercise is between 34 and 55 mmHg (Fig. 6).

Our results showed that muscle relaxation pressure during exercise increased not only in the affected legs but also in the symptom-free legs; this is in line with our previous report (Styf and Körner 1986). The increase of muscle relaxation pressure during exercise is a normal process and depends upon an increase of muscle volume (Jacobsson and Kjellmer 1964; Lundvall et al. 1972). As the increase of muscle volume is load-dependent, muscle load and contraction frequency must be kept constant bilaterally during the exercise test so as to permit a correct interpretation of muscle blood flow and the different intramuscular pressure parameters.

In a closed compartment this increased volume may lead to a pathological increase of intramuscular pressure during exercise; this may affect the muscle blood flow in several ways, for example, by a decrease of the arteriovenous pressure gradient; by compression of the vessels at the capillary level; or by exceeding the critical closing pressure (Ashton 1975).

After fasciotomy the increase in muscle relaxation pressure is less dramatic during the exercise test and the muscle blood flow is constant and normal, even at the time of muscle fatigue (Fig. 2C). We believe this is because fasciotomy increases the space available for volume load, demonstrated by the fact that the patients had swollen legs after exercise but suffered no pain.

The intramuscular pressure during contraction has been shown to be related to the force output of skeletal muscle (Körner et al. 1984; Sejersted et al. 1984). In this study each subject tried to keep the amount of exercise
during the test before and after surgery at the same level in each leg. However, despite this effort, some patients unwittingly increased the muscle output on the ergometer after fasciotomy; this explains our non-significant increase in the muscle contraction pressure from 141 to 161 mmHg in the non-operated legs after fasciotomy on the symptomatic legs, and is also reflected by the non-significant increase of muscle blood flow in the same legs. A significant increase of muscle blood flow during maximal exercise in patients with chronic compartment syndrome after fasciotomy has been reported (Qvarfordt et al. 1983). However, in that study those values could not be related to either the muscle contraction or relaxation pressures because of the low dynamic properties of the pressure recording system. Our findings of decreased muscle contraction pressures in the operated legs are in line with the results of animal experiments (Garfin et al. 1981). Furthermore, intramuscular pressure also depends on the depth of the catheter in the muscle (Kirkebø and Wisnes 1982), a fact which may also influence the recordings of intramuscular pressure before and after surgery.

Obviously the mean muscle pressure depends on both the muscle contraction and relaxation pressures. In this study, the changes of muscle blood flow and the development of symptoms in the affected legs could not be related to any change in the mean muscle pressure, indicating that this may be an unreliable parameter for diagnosing chronic compartment syndrome due to its dependence on muscle force. At the end of exercise, when the legs became painful, the mean muscle pressure even decreased due to the pain-induced decrease of muscle force.

**Conclusion.** The results of this study indicate that the pathophysiology of chronic compartment syndrome includes a volume load of the muscle tissue in the closed compartment and a pathological increase of intramuscular pressure. As perfusion of a muscle during exercise occurs during the phase of muscle relaxation, this increased pressure (> 35 mmHg) produces progressive ischaemia, leading to pain and impaired muscle function. The process is reversible, but when exercise ends, the resting pressure does not return to normal within 20 minutes. Fasciotomy prevents the abnormal increase of pressure, permitting normal blood flow throughout exercise and thus eliminating symptoms. The mean muscle pressure is an unreliable parameter in the diagnosis of chronic compartment syndrome.

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**REFERENCES**


