Increase in Skeletal Muscle Performance during Emotional Stress in Man

By N. A. Berdina, O. L. Kolenko, I. M. Kotz, A. P. Kuznetzov, I. M. Rodionov, A. P. Savtchenko, and V. I. Thorevsky

ABSTRACT

An emotional stress arising as a result of intense mental arithmetic causes (in most subjects) an increase in blood flow through resting forearm muscle by an average of 85%. Combination of voluntary sustained handgrip contraction with mental arithmetic has shown that the duration of contraction increases by an average of 46%. This effect appears only in subjects who display an increase in blood flow as a result of mental arithmetic. A rise in muscle performance is also observed during mental arithmetic when contraction of forearm muscle is caused by direct stimulation of the motor nerve in the upper arm. Intra-arterial injection of atropine sharply reduces both the increase in blood flow through the resting muscle and the increase in muscle performance associated with mental arithmetic. Control performance (without mental arithmetic) is not altered by administration of atropine. Analysis of blood flow during muscle contraction leads to the conclusion that an increase in muscle performance during emotional stress is not dependent on an increase in muscle blood flow. The data suggest that during emotional stress the sympathetic nervous system causes metabolic changes in skeletal muscles that lead to an increase in muscle performance when oxygen deficiency exists.

KEY WORDS

cholinergic vasodilation atropine skeletal muscle blood flow mental arithmetic sustained contraction muscle metabolism

Vasomotor responses in skeletal muscles due to stimulation of sympathetic nerves differ from reactions observed in vessels of the skin, intestines, and other organs. After administration of α-receptor-blocking agents or reserpine, stimulation of sympathetic nerves causes an intense vasodilation. Since this dilation is suppressed by atropine and increased by cholinesterase-blocking substances, most investigators believe that it is mediated by cholinergic dilator fibers in the sympathetic nerves (1–5). Some authors ascribe these cholinergic fibers to the parasympathetic system (6).

Vasodilation in muscle may be produced without pharmacological interference by stimulation of some central structures—the anterior hypothalamus, the mesencephalon (7–9). These structures are regarded as a central representation of cholinergic vasodilator fibers. The muscle vasodilation is one of the vegetative components of the reaction of anxiety, alertness, or rage (10–14). It has been proposed that the functional significance of an increase in blood flow through the muscle is preparation for the expected physical exertion (12, 13). However, stimulation of the hypothalamus that is sufficient to produce significant vasodilation fails to alter the force of separate rhythmic muscle contractions (15, 16). Some increase in contraction force during stimulation of the hypothalamus has been observed only in a fatigued muscle (16).
Muscle Performance and Emotional Stress

Cholinergic vasodilation in skeletal muscle, contrary to other types of dilation, is accompanied by a decrease in oxygen consumption (17, 18) and an increase in lactic acid production (19). Hence one of us (Rodionov) thought that this form of vasodilation might be regarded as a result of sympathetic influence on muscle metabolism which leads to activation of anaerobic metabolism and inhibition of the aerobic phase. Hence an "emotional" increase in muscle performance, if it does occur, should be sought for under conditions of an inadequate muscle blood supply when anaerobic metabolism is the primary source of energy. Similar conditions are seen during prolonged sustained muscle contraction (20).

An emotional stress and the associated increase in rate of blood flow in muscle vessels are easily produced by intense mental arithmetic (21-23). Some data from our study have been published earlier (24, 25).

Methods

Thirteen healthy male subjects, age 18-30 years, participated in the experiment. Sustained muscle contraction of two types was examined—voluntary and induced. The latter was initiated by electrical stimulation of the nerve. In both cases the maximal handgrip effort for contraction which could possibly be developed by the subject was determined. Subsequently the investigation was carried out at tensions that were 30% of the maximal effort. In the case of voluntary work the subject was asked to make an all-out effort to hold the handgrip dynamometer. Each effort was followed by 25-30 minutes of rest, which allowed sufficient recovery to avoid diminution of contraction time in the next attempt. The principal experiments were preceded by thorough training which lasted until the variations of contraction time did not exceed 10 seconds. The induced contraction was achieved by stimulating the nerve with superficial electrodes placed on the internal brachial sulcus. The frequency of stimulation was 50/sec. The induced contraction was registered with a tensometric handgrip dynamometer and was recorded on a loop oscillograph.

Muscle performance was estimated by two parameters—the time necessary for the contraction force to decrease to zero and the total amount of work, calculated according to the area under a recorded curve of the contraction force. The blood flow rate in the forearm was determined by venous occlusion plethysmography (26). The blood flow rates were calculated by dividing the area under the curve of the blood flow rate by the time of registration. Blood pressure was measured with a tonometer. Atropine, 0.4-0.6 mg in 8 ml of saline, was injected during 1.5 minutes into the brachial artery above the elbow.

The emotional stress was created by intense mental arithmetic, carried out against a disturbing background of critical remarks by experimenters, periodic flashes of a photostimulator and clicks of a metronome.

Results

Mental Arithmetic—The average blood flow rate at rest in nine experiments was 3.3 ml

<table>
<thead>
<tr>
<th>Subject</th>
<th>Blood flow at rest (ml 100 ml tissue⁻¹ min⁻¹)</th>
<th>Mental arithmetic (% col. 1)</th>
<th>Duration (sec)</th>
<th>Blood flow (ml 100 ml tissue⁻¹ min⁻¹)</th>
<th>Duration (% col. 3)</th>
<th>Mental arithmetic (%) col. 4</th>
<th>Blood flow (% col. 4)</th>
</tr>
</thead>
<tbody>
<tr>
<td>B.K.</td>
<td>3.2</td>
<td>175</td>
<td>270</td>
<td>4.2</td>
<td>130</td>
<td></td>
<td></td>
</tr>
<tr>
<td>B.K.</td>
<td>3.3</td>
<td>200</td>
<td>300</td>
<td>3.8</td>
<td>120</td>
<td></td>
<td></td>
</tr>
<tr>
<td>V.K.</td>
<td>3.0</td>
<td>190</td>
<td>300</td>
<td>4.0</td>
<td>120</td>
<td></td>
<td></td>
</tr>
<tr>
<td>V.A.</td>
<td>2.4</td>
<td>187</td>
<td>105</td>
<td>3.4</td>
<td>186</td>
<td></td>
<td>80</td>
</tr>
<tr>
<td>M.B.</td>
<td>4.0</td>
<td>182</td>
<td>120</td>
<td>5.4</td>
<td>142</td>
<td></td>
<td></td>
</tr>
<tr>
<td>M.B.</td>
<td>3.5</td>
<td>186</td>
<td>115</td>
<td>4.0</td>
<td>148</td>
<td></td>
<td>140</td>
</tr>
<tr>
<td>A.K.</td>
<td>3.2</td>
<td>175</td>
<td>120</td>
<td>4.1</td>
<td>150</td>
<td></td>
<td>122</td>
</tr>
<tr>
<td>A.K.</td>
<td>3.6</td>
<td>150</td>
<td>120</td>
<td>4.1</td>
<td>150</td>
<td></td>
<td>120</td>
</tr>
<tr>
<td>G.P.</td>
<td>3.5</td>
<td>220</td>
<td>90</td>
<td>4.7</td>
<td>167</td>
<td></td>
<td>100</td>
</tr>
<tr>
<td><strong>Mean</strong></td>
<td>3.3</td>
<td>185</td>
<td>171</td>
<td>4.3</td>
<td>146</td>
<td></td>
<td>111</td>
</tr>
<tr>
<td><strong>P</strong></td>
<td>&lt;0.001</td>
<td></td>
<td></td>
<td></td>
<td>&lt;0.001</td>
<td></td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

*From t-test on paired observations (experiment with mental arithmetic and without it).
Influence of mental arithmetic on blood flow in a resting forearm (left), during voluntary contraction (right, open circles and broken lines), and during voluntary contraction accompanied by mental arithmetic (right, dots and solid lines) in subject G.P. (Table 1). Arrows show the beginning and the end of mental arithmetic (left) and the beginning and the end of contraction (right).

100 ml⁻¹ min⁻¹ (Table 1). In seven of the eight subjects tested, blood flow increased 85% during mental arithmetic (Fig. 1), and arterial pressure increased an average of 17%. One subject showed no increase in blood flow through forearm muscles though he handled the calculations as well as the others.

**Voluntary Sustained (Static) Contraction.**—The average duration of handgrip contraction in nine experiments was 171 seconds (Table 1). During contraction, blood flow rose slightly from the resting level (Table 1). The limited increase in flow was due to the high intramuscular pressure (27) that constricted vessels and opposed the effect of exercise hyperemia, which was revealed only after muscle relaxation. The blood flow returned to the resting level 15-20 minutes after muscle relaxation. During voluntary contraction, an increase in arterial blood pressure and pulse rate was observed. In six experiments the average arterial pressure during contraction rose 47% and the pulse rate increased 50% (P < 0.001) (Fig. 2). These changes in pulse rate and arterial pressure suggest that the flow increase in a working muscle is due to the increase in perfusion pressure.

**Sustained Contraction and Mental Arithmetic.**—The average duration of handgrip contraction increased in nine experiments from 171 seconds without mental arithmetic to 235 seconds with mental arithmetic, i.e., by 46% (P < 0.001) (Table 1). Prolongation of the handgrip was observed in six subjects, who showed an increase of blood flow during mental arithmetic at rest. In one subject who showed no increase in blood flow with mental arithmetic, prolongation of sustained contraction was not observed when accompanied by mental arithmetic.

Since blood flow during contraction combined with mental arithmetic was higher than with contraction alone, the increase in muscle performance might be ascribed to a greater blood flow. However, this was not the case. The average increase in blood flow during contraction with mental arithmetic was only 11% higher than during contraction without mental arithmetic (P < 0.05), and this small increment in flow does not correlate with the increase in muscle performance. For example, in subject G.P. prolongation of contraction during mental arithmetic of 67% was not accompanied by an increase in blood flow.
In subject V.A. there was actually a decrease in blood flow during contraction with mental arithmetic as compared with contraction alone, whereas the duration of contraction was 86% higher with mental arithmetic than without it. These data suggest that prolongation of sustained contraction during mental arithmetic does not depend on the increase in blood flow during contraction. The greater muscle performance during mental arithmetic was only observed in the subjects in whom mental arithmetic at rest caused an increase of the blood flow rate. Hence, the question arises whether the increase in muscle performance and the increase of blood flow at rest are brought about by the same mechanism. To answer this question a series of experiments with the administration of atropine was performed.

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Effect of Atropine on Vasodilation and the Increase of Muscle Performance during Mental Arithmetic.—Atropine greatly reduces the vasodilation during mental arithmetic (22). Intra-arterial injection of atropine was performed in five subjects. The results obtained in a single experiment are shown in Figure 3. The results of all experiments are summarized in Table 2.

Atropine significantly decreased the effect of mental arithmetic on the rate of blood flow at rest (Table 2 and Fig. 3, right). It is worth noting that atropine at the concentration used failed to influence the duration of sustained contraction not associated with mental arithmetic \((P > 0.05)\). However, it significantly shortened the duration of contraction associated with mental arithmetic \((P < 0.01)\) (Table 2, Fig. 3). The average relative prolongation of contraction combined with mental arithmetic compared with the duration of contraction without mental arithmetic was 57% prior to the administration of atropine and 24% after drug administration \((P < 0.01)\). These data suggest that the increase in blood flow in the resting forearm muscles and the increase in muscle performance during mental arithmetic are of a similar nature, since both were reduced by atropine.

Influence of Mental Arithmetic on the Duration of Induced Contraction.—Prolongation of a sustained voluntary contraction combined with mental arithmetic might be due to an increase in the work capacity of the muscles or the subject might be distracted from the unpleasant sensations in the fatigued muscle by the mental arithmetic and consequently be able to maintain a state of sustained contraction for a longer time. To distinguish between these two possibilities a series of experiments with induced muscle contraction (IMC) was performed. The development of IMC during the stimulation of a motor nerve cannot be regulated at will and therefore reflects the muscle performance of the contractile mechanism. The enhancement...
TABLE 3

Blood Flow and Muscle Performance during Induced Contraction Combined with Mental Arithmetic

<table>
<thead>
<tr>
<th>Subject</th>
<th>Blood flow</th>
<th>Induced contraction</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>At rest (ml 100 ml tissue⁻¹ min⁻¹)</td>
<td>Mental arithmetic (% col. 1)</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>V.K.</td>
<td>2.2</td>
<td>195</td>
</tr>
<tr>
<td>V.K.</td>
<td>2.5</td>
<td>184</td>
</tr>
<tr>
<td>S.K.</td>
<td>2.7</td>
<td>189</td>
</tr>
<tr>
<td>V.I.</td>
<td>3.4</td>
<td>197</td>
</tr>
<tr>
<td>V.Z.</td>
<td>2.7</td>
<td>191</td>
</tr>
<tr>
<td><strong>MEAN</strong></td>
<td><strong>2.7</strong></td>
<td><strong>191</strong></td>
</tr>
</tbody>
</table>

Notations as in Table 1.

of muscle performance by mental arithmetic with IMC is shown in Figure 4.

The average duration of IMC in seven experiments was 70 seconds (Table 3). The blood flow pattern with IMC was similar to that seen with voluntary sustained contraction (compare Fig. 5 and Fig. 2). In seven experiments performed on five subjects, IMC was combined with mental arithmetic. When IMC was accompanied by mental arithmetic, muscle performance improved significantly in four of the five subjects. The average duration of IMC in six experiments performed on four subjects increased by 153% (P<0.001) and the total amount of "work" by 187% (P<0.001). It is of interest that although mental arithmetic caused an increase in the contraction duration it failed to affect the initial force of induced contraction (Fig. 4).

In this series of experiments (as in the previous one), one subject failed to show an increase in forearm blood flow during mental arithmetic. This subject also showed no increase of muscle performance during IMC accompanied by mental arithmetic.

It appears that an increase in muscle performance can occur not only during mental arithmetic but also with other types of emotional stress. For example, on one occasion subject V.K. had been irritated by an unpleasant conversation prior to the experiment. The duration of induced contraction was observed to be 4 minutes instead of the expected 1.5 minutes usually observed in this subject. Somewhat later the subject recovered from the emotional stress and the duration of contraction returned to 1.5 minutes. Subsequently, IMC with mental arithmetic resulted in an increase in the duration of contraction (Table 3).

Discussion

Our results indicate that a subject is capable of maintaining sustained contraction for a longer period of time provided that the effort is accompanied by emotional stress such as that caused by intense mental arithmetic. This
observation may be explained in two ways. It may be postulated that the efficiency of the peripheral contractile machinery is increased. However, it is also possible that mental arithmetic distracts the subject from the unpleasant sensations that arise in a fatigued muscle, and consequently he is capable of maintaining a contraction for a longer time. There are two arguments in favor of the first suggestion. First, an increase of muscle performance is significantly reduced after an intra-arterial injection of atropine administered in such a way that the drug is pharmacologically effective only on tissues situated distal to the site of injection, i.e., the forearm muscle. Second, the duration of muscle contraction and the amount of work increases during emotional stress even if the contraction is caused by stimulation of a motor nerve and cannot be regulated at will.

Can the increase in muscle performance during emotional stress be ascribed to an increase in the rate of blood flow to an active muscle? An increase in muscle performance is observed only in subjects who have an increase in blood flow in resting muscles as a result of mental arithmetic. The relationship between these two phenomena is strengthened by the fact that both the increase in muscle performance and the vasodilation in a resting muscle are significantly reduced by atropine. Such an explanation seems to be quite reasonable. The sympathetic nervous system increases the blood flow during mental arithmetic in resting and active muscle and, therefore, improves the blood supply and increases performance. However, the experiments indicate that such an explanation is untenable since the blood flow during control contraction does not greatly differ from the blood flow during contraction accompanied by mental arithmetic. Hence supplementary emotional dilation observed during mental arithmetic fails to change the situation. Furthermore, it has been shown that the sympathetic dilator effect on muscle is not observed during exercise hyperemia (15).

If an increase in muscle performance during mental arithmetic cannot be explained by an increase in muscle blood supply, it may be due to the influence of the sympathetic nervous system on the contracting muscles. If this is so, then where does the effect take place? Is it due to an improvement of excitation conduction through the synapse or does it directly affect muscle fibers? There is no reason to assume that the synapse is uninvolved. However, it appears that the contractile capacity of the muscle is likely to change. First, an increase in muscle performance during emotional stress is significantly reduced after the administration of atropine. Since it is known that atropine in low concentrations does not influence the conductivity in the synapse (28) it seems more likely that atropine acts directly on the muscle fiber. Second, the evidence cited above permits the assumption that vasodilation in a resting muscle and an increase of its contractile capacity during mental arithmetic may be regarded as two manifestations of the same phenomenon. If this is the case, this effect cannot be due merely to the neuromuscular synapse since an increase in blood flow through a resting muscle cannot be explained by an influence on the somatic synapse.

The vasodilating effect and the increase of muscle performance may have a common cause, namely sympathetic influence on the metabolism of striated muscle fibers. A change in metabolism in a resting muscle may cause vasodilation as well as an increase in performance of the contractile machinery during sustained contraction. If the sympathetic system tends to activate anaerobic metabolism (19) one may assume that the physiological significance of this type of sympathetic influence lies in preparing the muscle for work under conditions of limited oxygen supply.

Orbeli claims that the sympathetic system can affect the efficiency of skeletal muscle (29). This concept has been confirmed in experiments on cold-blooded and warm-blooded animals (30–32) and is known as the phenomenon of Orbeli-Ginetzinsky. That the
sympathetic system and norepinephrine facilitate conductivity in the neuromuscular synapse (33) is well known. An increase in the end-plate potential, the frequency of miniature end-plate potentials, and the sensitivity of the postsynaptic membrane have been described by a number of authors (33-38). The sympathetic nervous system of cold-blooded animals can influence the contractile capacity of muscle fiber itself. The experiments of Sinitzin seem to be most convincing in this respect (39). However, there are some indications that an increase in the contraction force in fatigued muscle of cold-blooded animals during stimulation of the sympathetic fibers is completely eliminated by ergonovine and is apparently caused only by release of norepinephrine (40, 41). The data obtained in warm-blooded animals are different. For example, Baetjer (42) showed that an increase in the contraction force in skeletal muscles of warm-blooded animals during stimulation of the sympathetic chain does not weaken but, on the contrary, increases after the administration of ergonovine. The increase in muscle performance that we observed is apparently not mediated by catecholamines either, since (1) it is reduced by atropine and (2) it is closely related to the neurogenic vasodilation that occurs in a resting muscle without any participation of catecholamines (1-5). These data suggest that the sympathetic nervous system in warm-blooded animals, contrary to the picture observed in cold-blooded ones, can influence a striated muscle fiber by a means other than via catecholamines, and the mediator may be acetylcholine.

References


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