Effects of systemic arterial blood pressure on the contractile force of a human hand muscle

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Wright, Julie R., D. I. McCloskey and Richard C. Fitzpatrick. Effects of systemic arterial blood pressure on the contractile force of a human hand muscle. J. Appl. Physiol. 88: 1390–1396, 2000.—The effect of physiological changes in systemic blood pressure on the force output of working adductor pollicis (AP) muscle was studied in six normal subjects. Supramaximal tetanic stimulation at the ulnar nerve produced repeated isometric contractions at 1-s intervals. Force output declined gradually with time. During the train of contractions, subjects voluntarily contracted the knee extensors for 1 min; this raised systemic blood pressure by 29%. Force output from AP rose in parallel with blood pressure so that 18% of the contraction force lost through fatigue was recovered for each 10% increase in blood pressure. When blood pressure in the hand was kept constant despite the increased systemic pressure, force output did not rise. The results show that muscle performance is strongly affected by physiological changes in central blood pressure and suggest that sensory input concerning the adequacy of muscle performance exerts a feedback control over the increase in systemic blood pressure during muscular activity.

WHEN A MOTOR COMMAND IS SENT to a muscle, a parallel or collateral command is sent to cardiovascular centers in the brainstem, and this acts to increase cardiac output and central blood pressure (4, 10). For moderate-intensity isometric exercise, this central command explains most of the pressor response (3, 11, 12, 14). In addition to the central pressor response, chemoreceptors within the muscle detect the buildup of metabolites and act through cardiovascular centers to produce a muscle reflex that increases blood pressure (1, 6). A component of this muscle reflex may also arise from muscle mechanoreceptor afferents (9). The relative effect of the muscle reflex is likely to be greater for higher-intensity exercise (3, 15).

Previous studies from this laboratory have investigated the effect of changes in muscle perfusion pressure on the performance of human adductor pollicis (AP) muscle comprised primarily of slowly fatiguing type I fibers (7). At low workloads, muscle performance is sensitive to changes in perfusion pressure across the physiological range (75–125 mmHg). Force output decreases as perfusion pressure falls and increases as perfusion pressure rises (2). At higher workloads, the rate of fatigue of AP increases as perfusion pressure is reduced within the physiological range (17).

The dependence of muscle performance on muscle perfusion suggests a feedback control that could regulate the central pressor response. As force output falls through fatigue or changes in perfusion, centrally generated motor command signals would increase if a voluntary attempt were made to maintain force output. Muscle performance could be restored if these centrally generated commands evoked an increase in central blood pressure and if that resulted in an increase in local perfusion pressure and blood flow to the active muscle. The increase in local perfusion pressure and blood flow will improve muscle performance (2). The present study tests the hypothesis that an increase in central arterial blood pressure will increase force output from AP. A train of intermittent contractions of AP was elicited by tetanic stimulation of the ulnar nerve. During this train of contractions, central systemic blood pressure was increased by having subjects contract a leg muscle. Because AP is a small muscle working at relatively low workload, the muscle reflex contribution of AP to the increase in central blood pressure is very small and therefore negligible (2, 11, 12). This fact permits the effect of an increase in central blood pressure on muscle performance to be determined without the muscle contributing significantly to a central pressor response or a muscle reflex.

METHODS

The subjects were six normal adults (two women and four men; age 27–55 yr). The institutional Human Ethics Committee approved the experiments, and subjects gave written, informed consent. Tetanic force from AP elicited by electrical stimulation was compared when subjects were at rest and when central blood pressure was raised by having subjects contract the leg muscles.

Setup. Subjects sat with the right arm extended and resting on a stable support (Fig. 1). A cord around the palm held the hand against the support, and strings kept the fingers extended. The hand was set at eye level, an average of 17.5 ± 1.2 cm above the jugular notch. The thumb was abducted ~45° from the palm, internally rotated, and flexed to be perpendicular to the plane of the hand. In this position, the flexors of the thumb cannot assist adduction, and AP provides almost all of the adduction force (13). To measure adduction force of the thumb, a metal ring was secured around the interphalangeal joint of the thumb and attached to an isometric strain gauge oriented in the plane perpendicular to the thumb (Fig. 1). Contraction of AP were elicited by

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the leg contraction, to minimize changes in blood pressure
instructed to breathe normally, particularly while beginning
stimulated arm relaxed during the leg exercise and the
between contractions, subjects were instructed to keep the

target force. To minimize drifts in the baseline of force of AP

the knee extensors for 1 min by viewing the oscilloscope that

experimenter, the subject produced the 50% MVC force with

ously for 2 min (Fig. 1). At that time, on a cue from the

quired level was reached. Stimuli were delivered continu-

ation voltage was rapidly increased from zero until the re-

subject.

of MVC was set on an oscilloscope that was viewed by the

that was attached via a strain gauge to a chair leg. To raise

contraction (MVC) of knee extension force was measured as

in each subject, and this voltage was then increased by 20% to

provide a safety margin that assured maximal activation.

Arterial blood pressure was measured in the middle finger

and stimulation of AP continued for a further 2 min. Thus

the leg muscle contraction, subjects were told to relax the leg,

that could arise through a Valsalva maneuver. After 1 min of

the leg muscle contraction, subjects were told to relax the leg,

and stimulation of AP continued for a further 2 min. Thus

each trial lasted for 5 min.

Control. In a control experiment, perfusion pressure of the

hand muscles was kept constant despite the increase in

central blood pressure. This experiment was performed at

least 1 wk later to eliminate residual effects. The protocol was

identical to the main experiment except that, during the leg

contraction, the hand was gradually raised to offset the

increase in central arterial pressure and to keep the perfusion

pressure in the hand constant. The support for the arm was

configured so that the hand could be raised by rotation of the

arm about the shoulder without any voluntary muscle activ-

ity by the subject (2). Perfusion pressure in the hand was

monitored by the plethysmograph recording of blood pressure

in the nonstimulated left hand, which was elevated along

with the stimulated hand. The averaging delay of the plethys-

mograph meant that there was usually some lag in the

experimenter tracking the blood pressure changes, particu-

larly at the end of the leg exercise when blood pressure fell

rapidly. The height of the hands above the jugular notch was

continuously recorded and was used to calculate central blood

pressure by subtracting the equivalent hydrostatic pressure.

Voluntary contraction. The increase in blood pressure
during voluntary contractions of AP was measured to com-
pare it with that during electrically stimulated contractions.

While positioned in the same apparatus used in the experi-
m ents that used electrical stimulation, subjects made brief

isometric contractions of thumb adduction. Subjects timed

the contractions at 1 Hz to a metronome and reached the

same force output as achieved with the electrical tetanic

contractions by bringing an oscilloscope display of force up to

a target mark. The hand was set at heart level. Subjects

produced a regular rhythm of contractions with a force profile

similar to that obtained by electrical stimulation (i.e., duty

cycle = 1:3).

Measurement and analysis. Data were sampled and re-
corded on computer (DAQ, National Instruments AT-MIO-

16XE-50). The thumb adduction and knee extension forces

and arterial blood pressure were sampled continuously at 50

Hz. EMG was band-pass filtered (160 Hz–1 kHz). The EMG

and force of each twitch were sampled at 5 kHz for 300 ms.

The tetanic force amplitude was calculated as the difference

between the prestimulus force and the maximal tetanic force.

The twitch force amplitude was calculated in a similar

manner.

RESULTS

Blood pressure. Central arterial blood pressure re-
mained essentially constant for the first 2 min of each
trial while subjects were at rest. At that time, subjects

tertained electrical stimulations. Cross sections of

muscles operating on the thumb. Contractions were trains of
tetani delivered once every second, each consisting of five
bipolar supramaximal stimuli of 1-ms duration at 40-ms
intervals. These contractions were delivered in a 10-s cycle,
with ninetetani followed by a single twitch at the 10th second
(Fig. 1). Electromyographic activity (EMG) of AP was re-
corded from surface electrodes over the belly of the muscle
and adjacent to the first metacarpophalangeal joint, with a
reference electrode on the metacarpophalangeal joint of the
thumb. The stimulus voltage that produced a maximal compo-

mixture of action potential AP (1 ms at 60–100 V) was determined

in each subject, and this voltage was then increased by 20% to

provide a safety margin that assured maximal activation.

Arterial blood pressure was measured in the middle finger

of the left hand, which was rested on the knee, by using a

servo pulse plethysmograph (Ohmeda Finapres 2300). Cen-

tral arterial blood pressure was calculated from the blood pressure

measured in the left hand by subtracting the hydrostatic

pressure of a column of blood between the jugular notch and

the hand.

Before the experiment, the force of a maximal voluntary
contraction (MVC) of knee extension force was measured as

the subject made a brief contraction against an ankle strap

that was attached via a strain gauge to a chair leg. To raise
central blood pressure during the experiment, a target of 50% of
MVC was set on an oscilloscope that was viewed by the

subject.

Protocol. When the experiment commenced, the stimula-
tion voltage was rapidly increased from zero until the re-
quired level was reached. Stimuli were delivered continu-
ously for 2 min (Fig. 1). At that time, on a cue from the

experimenter, the subject produced the 50% MVC force with

the knee extensors for 1 min by viewing the oscilloscope that

showed traces indicating the leg force and the 50% MVC

target force. To minimize drifts in the baseline of force of AP

between contractions, subjects were instructed to keep the

stimulated arm relaxed during the leg exercise and the

experimenter monitored EMG from the hand. Subjects

were instructed to breathe normally, particularly while begin-

ning the leg contraction, to minimize changes in blood pressure

that could arise through a Valsalva maneuver. After 1 min of

the leg muscle contraction, subjects were told to relax the leg,

and stimulation of AP continued for a further 2 min. Thus

each trial lasted for 5 min.

Control. In a control experiment, perfusion pressure of the

hand muscles was kept constant despite the increase in

central blood pressure. This experiment was performed at

least 1 wk later to eliminate residual effects. The protocol was

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and force of each twitch were sampled at 5 kHz for 300 ms.

The tetanic force amplitude was calculated as the difference

between the prestimulus force and the maximal tetanic force.

The twitch force amplitude was calculated in a similar

manner.

RESULTS

Blood pressure. Central arterial blood pressure re-
mained essentially constant for the first 2 min of each
trial while subjects were at rest. At that time, subjects

began to exert the leg force, and central blood pressure

immediately rose. This is illustrated for a typical
subject in Fig. 2, and for the overall group of subjects in

Fig. 3. In all subjects, central blood pressure continued

to increase throughout the period of the leg contraction,

and after 1 min central arterial blood pressure had

risen by 29.0 ± 2.9 mmHg (means ± SE) or 29.1 ± 2.3% when

normalized to the blood pressure at the start of

leg contraction. After cessation of the leg contraction,

blood pressure fell to within 6 mmHg of its initial level

within 10 s.

Contraction force. A typical set of results from one

subject is shown in Fig. 2. At the beginning of each trial,
twitch force (11.2 ± 1.5 N) and tetanic force (43.1 ± 6.2 N) were maximal. All subjects showed a similar gradual decay in force output during the first 2 min until the start of leg contraction. During this period, force output fell by an average of 14.8 ± 1.8% of the initial maximum. For all subjects, after the leg contraction commenced and blood pressure started to rise, tetanic force output and twitch force from AP rose above the baseline of slow decay and continued to rise throughout the period of the leg contraction in parallel with the increasing blood pressure. The mean maximal rise in force during the period of leg contraction was 6.7 ± 0.9%. On average, 38.9 ± 5.8% of the decrease in force after the start of contractions was recovered. After the leg contraction ceased and blood pressure fell, tetanic force declined again and returned to its initial pattern of slow decay. Average data for the group are shown in Fig. 3. Every 10th cycle was a single twitch rather than a tetanus, and twitch force followed a similar pattern to tetanic force in all subjects.

Throughout the experiments, the compound muscle action potentials (CMAP) from the single twitches remained constant in all subjects. Figure 4 shows an example of three CMAP from one subject overlaid, one before the period of elevated blood pressure, one during, and one after that period. The corresponding force profiles from these three twitches are shown. Although the CMAP remain unchanged during leg contraction, force amplitude increases.

Tetanic force never rose to above its initial maximum level in any subject. To investigate whether this could be achieved, for one subject a trial was undertaken in which the leg contraction commenced 45 s after the maximal tetanic force rather than the usual 2 min. In this situation there was a similar increase in blood pressure to that shown in the main experiment but a
smaller increase in tetanic force. However, the force never rose above the initial maximal force, even when the leg contraction and period of increased blood pressure persisted for 90 s.

Control. In control experiments, perfusion pressure in the hand was kept approximately constant during the period of leg contraction as the experimenter raised the hand to offset the increase in central blood pressure. As with the main experiments, central blood pressure again remained essentially constant for the first 2 min of the experiment and rose a similar amount during the period of leg contraction (Figs. 2 and 3). The maximal increase in central blood pressure was $25.0 \pm 2.2$ mmHg or $25.3 \pm 2.6\%$, and it was not significantly different from the increase during the experimental trials (paired t-test on normalized maximum rise, $P = 0.09$). There was a small increase ($4.3 \pm 2.0$ mmHg) in muscle perfusion pressure in this situation because the experimenter could not precisely track the changes in central blood pressure.

During the first 2 min of the control experiments, the decline in tetanic force was $16.4 \pm 2.5\%$, similar to that when the hand was kept level (Figs. 2 and 3). During the period of the leg muscle contraction, neither tetanic force nor twitch force increased. In addition, there was no significant difference between the time constants of the steady exponential decline in tetanic force for the periods before and during the leg contraction (paired t-test, $P = 0.12$).

The increase in AP contraction force above that at the beginning of the period of the leg muscle contraction underestimates the true effect because of the declining baseline contraction force. To account for this, the difference between the experimental and control data for each subject was used to calculate the proportion of contraction force recovered after the initial maximum. For each subject and for the group mean, this proportion is plotted against the percent increase in blood pressure for the period of the leg muscle contraction (Fig. 5). The slope of this relationship indicates that, on average, 18% of the contraction force lost after the initial maximum was recovered for each 10% increase in central blood pressure for the range of blood pressure produced here.

Voluntary contraction. In all subjects, blood pressure rose when they made a train of voluntary contractions with the same pattern as the electrically elicited forces in the main experiments. The mean increase in normal-

![Fig. 4. Single twitches. Overlaid compound muscle action potentials (CMAP) and twitch force profiles for 1 subject for single twitches occurring before (B), during (D), and after (A) period of leg contraction.](http://jap.physiology.org/)

![Fig. 5. Relationship between force recovery and BP. Percentage of force decline after start of stimulated contractions that has been recovered is shown for six 10-s epochs during period of leg contraction for group mean data and for each subject (slopes indicated). Difference between experimental and control data for each subject was used to calculate proportion of contraction force recovered after initial maximum.](http://jap.physiology.org/)

Fig. 4. Single twitches. Overlaid compound muscle action potentials (CMAP) and twitch force profiles for 1 subject for single twitches occurring before (B), during (D), and after (A) period of leg contraction.
ized blood pressure during 2 min was 19.3 ± 3.3%. The increase is shown in Fig. 3 (voluntary graph) and contrasts with the near constant blood pressure during the 2 min of contractions produced by electrical stimulation (experiment and control graphs).

DISCUSSION

This study shows that force output from human AP, a muscle comprised largely of fatigue-resistant muscle fibers, is sensitive to physiologically evoked changes in central arterial blood pressure. The protocol of electrical stimulation used here produced a gradual decline of the tetanic force output from AP over the initial 2 min. At the same time, there was little or no increase in central blood pressure due to the electrical stimulation and the contraction of AP. When subjects made a voluntary isometric contraction of the knee extensors to produce an increase in central arterial blood pressure, the decline in force output from AP was arrested, and muscle force increased in parallel with the rising blood pressure. The force output increased toward, but never exceeded, the initial maximal level. When the leg contraction ceased, blood pressure and AP force output fell rapidly to their initial levels.

It is conceivable that factors associated with the voluntary contraction of the leg muscles other than the increase in central blood pressure could have caused the increase in muscle force. Such an interaction could occur within the muscle. For example, there could be autonomic effects on the vascular bed of AP or the muscle fibers themselves associated with the motor command to the leg muscles, and this could increase muscle contractile force. Any such interactions appear to have had negligible effect, as there was no increase in force associated with the contraction of the leg muscles in control experiments in which the perfusion pressure in the hand muscle was prevented from rising by elevating the hand as central arterial pressure rose. It is also possible that interactions within the central nervous system between the voluntary contraction of the leg muscles and the nerve stimulation could have affected force output from AP. Afferent impulses arising from the nerve stimulation provide an input to the motor neuron pool, and potentiation associated with the voluntary contraction of the leg muscles could increase force output from the hand muscles. Such potentiation would be reflected as altered late F-waves in the compound action potential of AP, but these were not observed (Fig. 4). Additionally, if this generated any significant effect, it would be expected to appear simultaneous with the onset of the leg contraction, but that was not seen. Instead, muscle force rose progressively in parallel with the increase in central blood pressure. These considerations, and previous demonstrations of muscle blood flow on force output (5), indicate that changes in blood pressure are responsible for the changes in force output.

Using the same stimulation protocol, Fitzpatrick et al. (2) showed a relatively large decrease in muscle force output when the hand was elevated to reduce perfusion pressure (6.3% per 10 mmHg). In contrast, there was only a small increase in force when the hand was lowered to increase arterial inflow (2.2% per 10 mmHg). It was suggested that this occurred because both arterial and venous pressures increase when the arm is lowered and this diminishes the net change in perfusion pressure across the muscle vascular bed. In the present experiment, where the central arterial blood pressure was elevated with presumably only small, if any, changes in venous pressure, there was a much larger effect on muscle force output (5.2 ± 0.6% per 10 mmHg). Thus the directional effect of increased perfusion pressure producing only small changes in muscle performance is likely to be a consequence of lowering the hand to increase perfusion pressure. It appears that both increases and decreases in arterial perfusion pressure have effects of similar magnitude on muscle performance.

There was no steady increase in blood pressure during the 2-min period of electrical stimulation and contraction of AP, although there was a small initial increase in blood pressure (mean = 7%) similar to that described in Fitzpatrick et al. (2). When subjects made the same level of contraction voluntarily, there was a progressive increase in blood pressure, reaching 19.3% ± 3.3 N after 2 min (Fig. 3). Thus, for this muscle and contraction level, there is a small muscle reflex and a large central pressor response.

The leg-muscle contraction used to raise central blood pressure increases systemic sympathetic nervous activity through the central pressor response and the muscle chemoreflex (8). Any vasoconstriction in the hand muscles arising through this sympathetic activity would be expected to decrease muscle blood flow and muscle force output. It is therefore of note that in the control experiments there was no obvious increase in the rate of decline in force (Fig. 3, control). This observation agrees with the recent finding that chemoreflex-mediated sympathetic activity increases vascular resistance in the resting forearm but has no effect in the exercising forearm (16). Thus local factors are likely to override the generalized sympathetic-mediated vaso-

![Fig. 6. Motor and BP control. Afferent feedback (F) about muscle performance is compared centrally with desired motor outcome to adjust motor command (M) to muscle. A collateral signal to cardiovascular centers (C) generates central pressor response. This study shows that change in BP affects muscle performance (X) and thereby ultimately provides a feedback control on BP.](http://jap.physiology.org/)

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constriction and thereby allow blood flow to reflect the changes in perfusion pressure.

Recently, Wright et al. (17) showed that, within normal physiological levels, a reduction in local muscle perfusion pressure increases the rate of fatigue of AP. This study also showed an increase in blood pressure that is proportional to the extent of muscle fatigue, independent of the level of muscle perfusion. This finding suggests that muscle performance exerts a control over the increase in blood pressure during exercise. The present study shows that central blood pressure, in turn, exerts a control on muscle performance. Thus a feedback loop can be conceived in which sensory feedback of information about a muscle’s performance is centrally compared with the desired motor outcome to produce an appropriate adjustment of the motor command to the muscle (Fig. 6). Along with the motor command, a parallel or collateral signal is sent to cardiovascular centers that generate the central pressure response. The present study shows that the resulting changes in central blood pressure have an effect on muscle performance and thereby on the adequacy of the motor command. An interesting aspect of this control on blood pressure is that the feedback is via afferents that provide somatosensory information about motor performance rather than afferents providing direct information about cardiovascular status, as occurs, for example, with the cardiovascular regulatory baroreceptor reflex.

Muscle force output did not rise above the initial maximum, even when the increase in blood pressure was early in the sequence of contractions, indicating that muscle performance was already optimal at the start of the contractions. It therefore appears that the increase in blood pressure acts to restore a proportion of the decline in force produced by the previous history of muscle contractions. It has yet to be determined whether an increase in central blood pressure before the initial muscle contraction would increase the initial force output, although the considerations above suggest that such an effect would be small.

When subjects made isometric contractions of AP at the same force level produced by the electrical stimulation, there was a large increase in central blood pressure, similar to that produced by the leg contraction. Therefore, the strength of the effect of blood pressure on muscle performance can be estimated from the slope of the relationship between the proportional restoration in force and the proportional increase in blood pressure (Fig. 5). For this range of blood pressures, each 10% increase in central blood pressure restored, on average, 18% of the lost force. The protocol used here of contraction of the knee extensors is likely to increase vasconstrictor tone as a means of raising blood pressure. This could result in a relative restriction of muscle perfusion compared with a situation in which the increase in blood pressure arose through a voluntary contraction of AP itself. Thus this protocol may underestimate the effect of central blood pressure on muscle force output. We can conclude that systemic arterial blood pressure exerts strong effects on muscle contractile forces and that sensory input regarding muscle performance also provides afferent information used for the regulation of blood pressure during muscular activity.

In the earlier study by Wright et al. (17), in which subjects made fatiguing voluntary contractions of AP, the slope of the relationship between the increase in blood pressure and the force lost was 0.55 ± 0.02; that is, a 10% loss of force produced a 5.5% increase in blood pressure. In the present study, the slope of the inverse relationship of blood pressure increase to force recovery is 1.72 ± 0.22. With these values, it is possible to speculate on the gain of the feedback effect of blood pressure on muscle performance if we assume that a simple negative feedback loop is operating. The product of these gains, force lost to blood pressure × blood pressure to force recovered, is 0.94 ± 0.09 and a measure of the gain of the feedback loop. Loop gain is a dimensionless quantity that defines the strength of the feedback effect. Therefore, it can be estimated that the rise in blood pressure that results from muscle fatigue reduces that muscle fatigue by approximately one-half [1/(1 + loop gain)]. In other words, the muscle would fatigue twice as fast if blood pressure did not rise.

In summary, these results show that muscle performance is sensitive to changes in systemic arterial blood pressure, and a previous study demonstrated that systemic arterial pressure increases in proportion to muscle fatigue. Sensory input about motor performance provides information concerning the adequacy of the rise in blood pressure so that that the blood pressure regulation based on the motor command is not an open loop but operates as a feedback control. These findings have important implications for our understanding of blood pressure control during everyday activity, but, in addition, they have important implications for our understanding of motor control. Motor output is very sensitive to cardiovascular responses. Indeed, with gains as high as those shown here, cardiovascular reflexes appear to have stronger effects on motor performance than do the array of motor reflexes that have so long been the particular interest of motor physiologists.

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