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THE REFLEX NATURE OF THE PRESSOR RESPONSE TO MUSCULAR EXERCISE

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SUMMARY

1. In anaesthetized cats tetanic contraction of the hind-limb muscles, elicited by stimulating the ventral roots L6–S1, caused a rise of arterial blood pressure, usually accompanied by small increases in heart rate and pulmonary ventilation: in decerebrate cats, all components of the response were much increased.

2. With tetani of different strengths, obtained by stimulating with different intensities at the same frequency, the pressor response increased with increasing tension.

3. When muscle contraction had been abolished by gallamine, or when dorsal roots L6–S1 had been sectioned, ventral root stimulation no longer caused a pressor response. The response is therefore a reflex, initiated in the exercising limb.

4. The pressor response was not affected by section of all articular nerves to knee and ankle joints, or by section of the vagi. The stimulus therefore originates in the contracting muscles alone.

5. The pressor response is potentiated by occluding the circulation through the working muscles. Reasons are discussed for concluding that the stimulus is chemical rather than mechanical, and that the 'metabolic receptors' for this exercise reflex are the free endings of group III and IV sensory nerve fibres located around the blood vessels.

INTRODUCTION

One of the main unsolved problems in exercise physiology is the nature of the stimulus giving rise to the cardiovascular and respiratory responses that muscular exercise produces. It has been known since the turn of the century that muscular activity is accompanied by an increase in arterial

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blood pressure (Bowen, 1904; Krogh & Lindhard, 1913) and it was originally postulated that the pressor response is due to a direct action of the motor cortex on the 'circulatory centres' (Krogh & Lindhard, 1913; Gillespie, 1924). This idea did not find experimental support, however, and an alternative suggestion was put forward that the pressor response is reflexly elicited by afferent fibres originating in the exercising muscle (Patterson, 1928). This hypothesis has since gained considerable strength from the work of Alam & Smirk (1937, 1938) and Asmussen, Nielsen & Wieth-Pedersen (1943). More recently, Asmussen & Nielsen (1964) and Lind, Taylor, Humphreys, Kennelly & Donald (1964) have shown that the rise in arterial blood pressure accompanying muscular contractions is potentiated when the circulation through the working muscles is occluded.

Though this work on human subjects provides suggestive, indirect evidence, direct experimental proof of the reflex nature of the pressor effect has not been obtained. It has seemed that animal experiments would have to be performed in order to establish unequivocally the reflex nature of the pressor response, and to study its characteristics in more detail. So far, however, experiments using animals have not confirmed the observations on man. In fact, only the opposite effects, i.e. depressor responses, have been described (McDowall, 1936; Euler & Liljestrand, 1947). In these experiments, the hind-limb muscles were stimulated directly in a way which would have led indiscriminately to stimulation, and subsequent blockade, of a large number of afferent fibres from the muscles, which could well have distorted the effect of any appropriate afferent input arising in the contracting muscles themselves. The possibility of artifacts such as these was avoided in the present experiments; for the muscles were activated by ventral root stimulation. As reported in a preliminary communication, muscular contractions then produce a reflex pressor response (Coote, Hilton & Perez-Gonzalez, 1969). As the small myelinated and unmyelinated afferent fibres in muscle can produce similar cardiovascular effects (Coote & Perez-Gonzalez, 1970), they are probably the sensory fibres which initiate the circulatory response to exercise in the experimental animal and in man.

METHODS

Experiments were performed on cats which were anaesthetized with I.V. chloralose (70–75 mg/kg) or decerebrated at the intercollicular level, after preliminary induction of anaesthesia with ethyl chloride and ether. Laminectomy was then performed to expose the lumbar and sacral segments L 4–S 1.

The triceps surae muscle was prepared for recording tension and a thread was tied round its tendon. The position of this marker thread in relation to a drill inserted into the lower end of the tibia was measured for an ankle flexion of 90 °; this was the muscle length in all experiments. The tendon was then detached from the calcaneum.

Arterial blood pressure was recorded from either the left carotid artery or femoral artery via a saline-filled polyethylene cannula connected to a pressure transducer (Bell and Howell Ltd., Consolidated Electrodynamics). The amplified pressure-wave form was also used to trigger a tachograph unit (Devices Ltd) to give a record of heart rate. Respiration was monitored as air-flow, using a low-volume pressure transducer (Grass Instruments Ltd, PT 5A) connected to a tracheal cannula. The output of all recording instruments was amplified and displayed on a 4-channel pen recorder (Devices Ltd).

In a few experiments the venous outflow from the triceps surae was recorded via a photoelectric drop counter (Devices Ltd), the procedures for isolating the muscle outflow being similar to those described by Hilton (1953). In these experiments heparin (1000 i.u./kg) was given I.V. at the end of all surgical procedures.

For recording, the animal was fixed in a stainless-steel frame and suspended in the prone position by means of pins, two of which were inserted bilaterally into the iliac crest, and two in the vertebrae above the level of laminectomy. A paraffin pool was made after the skin and remaining layer of vertebral muscles had been sewn to a brass ring. To expose the cord the dura was incised lengthwise. The ventral roots, L 6, L 7 and S 1 on the right side, were carefully dissected, sectioned close to the cord and placed on stainless-steel stimulating electrodes. Contraction of hind-limb muscles was elicited by electrical stimulation of these ventral roots using square-wave pulses 0·1 msec in duration at 25–50 Hz via an RF isolation unit (Grass, SIU 478A) from a stimulator (Grass, S 8). For isometric recording of contractions of the triceps surae muscle the hind limb was fixed at the knee and ankle to prevent movement and the triceps surae tendon was attached to a copper–beryllium bar on which was mounted a pair of strain-gauges. These were so arranged that tension applied to the bar caused distortion of the gauges, which formed one half of a wheatstone bridge, the output of which was displayed on the pen recorder.

RESULTS

Tetanic contractions of the hind-limb muscles, elicited by stimulation of the ventral roots L 6, L 7 and S 1 at frequencies of 25 and 50 Hz for 45–60 sec, always caused a rise of arterial blood pressure (Fig. 1).

Characteristics of the blood pressure response

Anaesthetized preparations. The time to onset of the pressor response from the beginning of the contraction period was measured in sixty-four tests carried out in four different experiments. It was estimated as the time in which the systolic blood pressure had risen by 5 mm Hg above control levels during a maximal sustained tetanus of the hind-limb muscles. The mean value was 2·5 (S.E. ± 0·19) sec. The arterial blood pressure usually reached a steady level which was maintained throughout the rest of the contraction period, as shown in Fig. 1(*c*), the maximal rise of systolic pressure being around 55 mm Hg (40 % above control). In some cases, particularly in the lower ranges of stimulus intensities, the arterial blood pressure rose continuously during the period of muscle contraction (Fig. 1*a*, *b*). Immediately after the tetanus the blood pressure fell steeply

to a level slightly above control values and then returned gradually to normal, usually within 1–1·5 min after the end of stimulation. The above records were taken from tests in which the tetani were submaximal. With tetani in response to supra-maximal stimulation (see Figs. 4, 5), the pressor response diminished as the tension began to decline, though following the latter after a variable delay. In most experiments these pressor effects were accompanied by small increases in heart rate (< 5 %) and in rate and depth of respiration. Pupillary dilatation was occasionally observed.

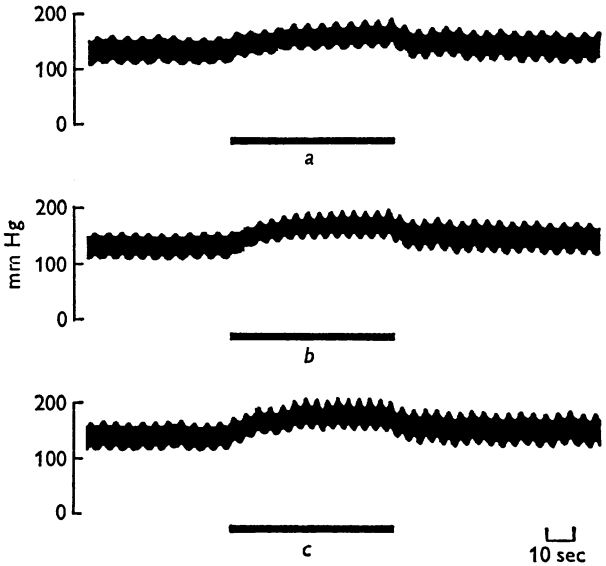

Fig. 1. Cat, chloralose. Records of arterial blood pressure showing effects of tetanic contractions of the right hind-limb muscles of different magnitudes elicited by stimulation at different strengths of the peripheral cut ends of the ventral roots L 6–S 1 at a frequency of 50 Hz for 60 sec. Ventral roots stimulated at × 2 motor threshold (*a*), × 4 (*b*), and × 5 (*c*).

In three experiments the levels of arterial blood pressure at rest were almost the same and remained steady during the course of the whole experiment. In these the question was investigated whether there is any correlation between the magnitude of the pressor response and the strength of the contraction which caused it. The tension developed by the triceps surae muscle was used as a sample of that produced in all the leg muscles, which were contracting simultaneously. To vary strength of contraction the ventral roots were stimulated at a fixed frequency of 50 Hz but with different intensities. The magnitude of the pressor response was expressed as a percentage increase above the resting level. The relationship between these responses and the peak tension developed by the muscle is illustrated

in Fig. 2. The extent to which the response increased with tension (plotted on a logarithmic scale) is indicated by the calculated regression line.

Decerebrate preparations. The pressor responses to tetanic contraction of hind-limb muscles in decerebrate cats were qualitatively very similar to those obtained in anaesthetized animals, but they were more pronounced. Maximal increases of 75 mm Hg were recorded, which is some 80 % above the resting systolic levels. Similarly, the accompanying changes in heart rate and respiration were more marked and consistent. These are illustrated in Fig. 3. Increases in heart rate ranging from 14 to 20 % were recorded in these experiments, together with marked increases in rate or

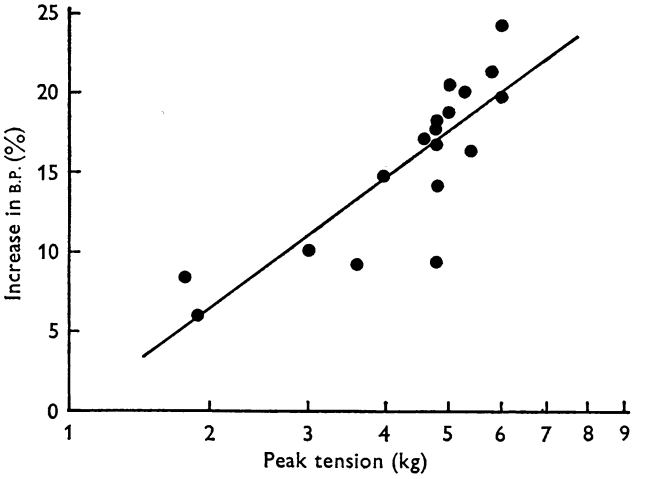

Fig. 2. Relationship between pressor responses to tetanic contractions of hind-limb muscles and tension developed by one group of these muscles (triceps surae) in three experiments. Ordinate, increases in arterial blood pressure expressed as percentages above resting level: abscissa, peak tension developed by triceps surae. Calculated regression line has been drawn.

depth of respiration, and sometimes in both. Two out of the five cats in which decerebration was carried out displayed movement of the jaw, extrusion of claws, pupillary dilatation and piloerection. A post-mortem examination showed that the level of section in these cats was rostral to the intercollicular line and even spared part of the posterior hypothalamus. Another feature of these decerebrate preparations was that on cessation of the tetanic contractions the arterial blood pressure fell steeply to a level below that before stimulation. This post-stimulation vasodepressor effect was often accompanied by a bradycardia.

In three of these cats, blocking the action of the vagus with atropine sulphate (0·5 mg/kg, I.V.) did not alter the pressor, heart rate or respira-

tory responses during contractions, but post-stimulation bradycardia no longer occurred.

Neurogenic origin of the response

These responses are caused by muscular contractions; for when contractions are abolished by gallamine triethiodide (Flaxedil, 3 mg/kg I.V.) ventral root stimulation produces no change of arterial blood pressure, heart rate or respiration.

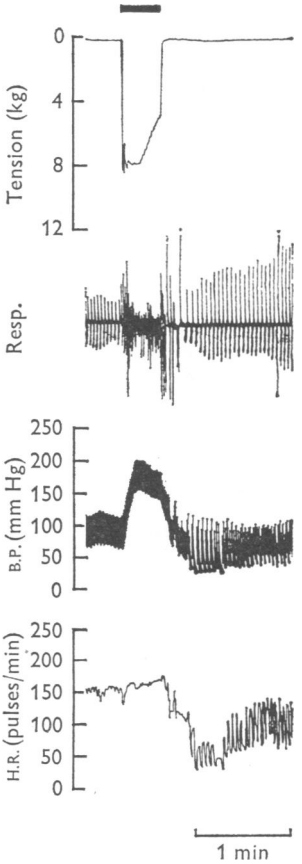

Fig. 3. Decerebrate cat. Records (from above downwards) of tension developed by triceps surae, respiration (Resp.), arterial blood pressure (B.P.) and heart rate (H.R.). At signal, tetanic contraction of the right hind-limb muscles elicited by stimulating peripheral cut ends of ventral roots L6–S1, at 0·3 V and 50 Hz. Both common carotid arteries tied.

The response is, in fact, reflex in origin, the stimulus arising in the exercising limb. When the sensory input to the spinal cord from the contracting hind-limb muscles was interrupted by section of the dorsal roots L6, L7 and S1, one by one, on the stimulated side, the size of the pressor

response progressively decreased: it was abolished when all three roots had been cut (Fig. 4). In other experiments the dorsal roots were crushed close to the dorsal root ganglion: this was equally effective in abolishing the responses.

It was unlikely that this reflex originated in joint receptors, as had been suggested for the respiratory response by Comroe & Schmidt (1943), since, in the present experiments, the limb was fixed in a way that precluded

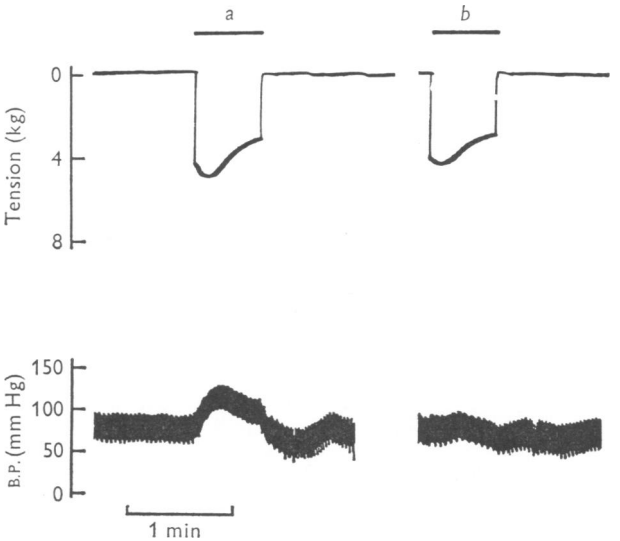

Fig. 4. Cat, chloralose. Records of arterial blood pressure and tension developed by triceps surae. At signals, tetanic contractions of the right hind-limb muscles elicited by stimulating peripheral cut ends of ventral roots L6–S1 at 0·4 V and 50 Hz, before (*a*) and after (*b*) section of dorsal roots L6–S1.

joint movement. Moreover, this possibility was examined specifically in three experiments in which the articular nerves supplying the knee, and the peroneal, tibial and sural nerves supplying ankle and foot, were sectioned close to the joints. Sectioning these nerves had no effect on the responses subsequently elicited, so a reflex from joint receptors can be excluded. The possible participation of lung inflation afferents was excluded by sectioning both vagi (three experiments), which had no effect on the circulatory responses.

Effect on the responses of restricted blood flow to the contracting muscles

The results of the previous section show that the stimulus for the cardiovascular and respiratory responses arises within the contracting

muscles themselves. This stimulus could be mechanical or chemical. Evidence in favour of the latter was provided by the early experiments of Alam & Smirk (1938) on man, who found that occlusion of the circulation through the contracting muscles potentiated the response. Both Alam & Smirk (1938) and Lind, McNicol & Donald (1966) showed further that the response persisted beyond the period of muscular contraction and until the occlusion was relieved.

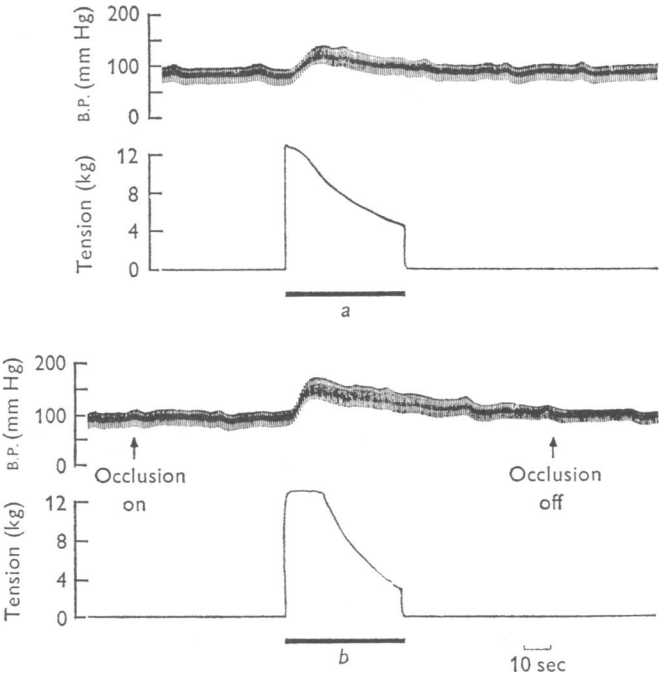

Fig. 5. Cat, chloralose. Records of arterial blood pressure and tension developed by triceps surae. At signal, tetanic contractions of hind-limb muscles elicited by stimulating peripheral cut ends of ventral roots L 6–S 1 for 45 sec at 50 Hz, before (*a*) and after (*b*) occlusion of right iliac artery (indicated by arrows).

The effect of vascular occlusion was therefore tested in the present experiments. For this purpose, a loose loop of surgical thread was placed around the iliac artery in the abdomen and passed through a mid-line incision in the abdominal wall and left protruding after the abdomen was closed. In each experiment a series of tetanic contractions of increasing strength was elicited and the maximal pressure response was determined. A tetanus slightly stronger than that yielding a maximal response was then used to elicit the control pressor response. The iliac artery was occluded and a similar supramaximal contraction was elicited 1 min after

the beginning of occlusion. Fig. 5 shows a typical result of such a test. The response to contraction developing a peak tension of 12·8 kg with normal circulation is shown above (*a*), and that to a contraction developing 13·0 kg tension during arterial occlusion is shown below (*b*). The pressor response was greater in *b*, where the mean arterial blood pressure rose from 90 to 150 mm Hg (65 %), than in *a*, where the rise was from 90 to 125 mm Hg (40 %). In addition, the rate of rise of pressure was increased during arterial occlusion, the values being 4 mm Hg/sec for *a* and 8 mm Hg/sec for *b*.

There was, in this and most such experiments, a slightly increased tension during occlusion, though the parameters of ventral root stimulation were the same. This was the reason for using control contractions which were already supramaximal for the pressor response produced with unrestricted circulation. The slight increase in tension developed by the muscle with impaired circulation cannot therefore have been responsible for the potentiation of the pressor reflex response. Further evidence supporting this occlusion was obtained in an experiment in which submaximal tetani were used. In this experiment a tetanus developing 1 kg tension in triceps surae was just about threshold for the pressor responses, producing only a 5 % increase in mean pressure with unrestricted circulation. Partial occlusion of the circulation to the limb for as little as 25 sec during the tetanus produced an increase in mean arterial blood pressure of 25 %. The blood flow through triceps surae was monitored at the same time to confirm the effectiveness of the occlusion.

In most experiments occlusion was maintained after muscle contraction had ceased. The arterial blood pressure then did start to fall at the end of contraction, but it fell more slowly than in the tests carried out with free circulation, a further fall to the control level occurring when the occlusion was released (also shown in Fig. 5).

Relationship between local hyperaemic response and strength of muscle contraction

It is well known that, when skeletal muscles contract, a pronounced, local functional hyperaemia occurs. In view of the possibility that the chemical cause of this phenomenon is the same as that initiating the reflex pressor response, the relationship was examined between varying strengths of contraction of triceps surae, as had been achieved above by ventral root stimulation at 50 Hz with different intensities, and the extent of the resulting functional hyperaemia in this muscle.

In plotting the results shown in the graphs of Fig. 6 (three experiments), the changes in conductance were first calculated by dividing blood flow (ml./min) by mean arterial pressure (mm Hg) at different times during the

tests. The resting levels were almost the same in each experiment and remained steady during the whole experiment: the increase in conductance, expressed as a percentage increase above the resting level in each experiment, has been plotted against peak tension of triceps surae in graph (*a*). If there is a real, functional relationship between resistance

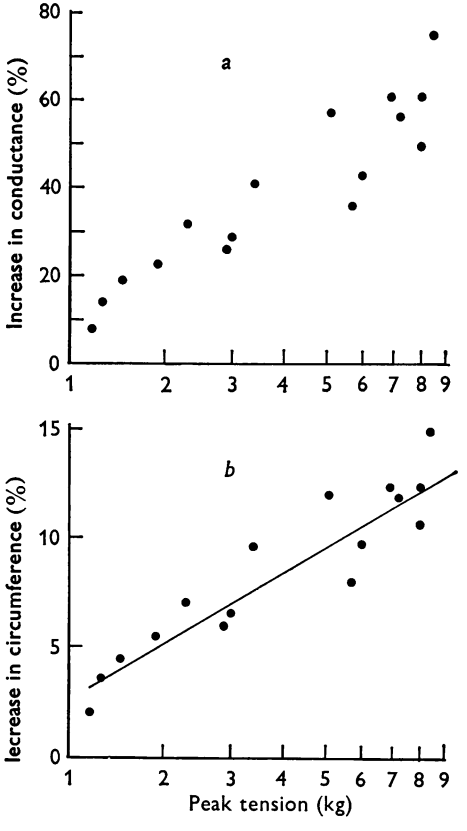

Fig. 6. Relationship between the local hyperaemic response and strength of muscle contraction in triceps surae (three experiments). *a*, ordinate, increase in conductance (flow/pressure) in triceps surae, expressed as percentage above resting level: abscissa, peak tension developed by triceps surae. *b*, ordinate, increase in resistance vessel circumference ($\sqrt[4]{(\text{conductance})}$) in triceps surae, expressed as percentage above resting level: abscissa as in *a*: calculated regression line has been drawn.

vessel size and the concentration of a chemical product of contraction, some measure should be sought corresponding to fibre length in the walls of resistance vessels. As an approach to this ideal, the conductance figures have been used to calculate values related to 'resistance vessel circumference'. This was achieved by taking the fourth root of the calculated con-

ductance, both resting and during tests, and expressing each increase as a percentage above the resting level in each experiment. These increases have then been plotted against peak tension of triceps surae (graph *b*). As in the case of the pressor responses to contraction (Fig. 2), the extent to which 'vessel circumference' increased with tension (plotted on a logarithmic scale) is indicated by a calculated regression line.

DISCUSSION

The present investigation has shown that muscle contraction in the cat gives rise to a reflex rise in arterial blood pressure. This is similar to the reflex pressor response to sustained hand-grip in man described by Lind *et al.* (1964) and analysed by Lind *et al.* (1966). Indeed, the suggestion that the pressor response to muscular exercise in man is dependent on a reflex from the exercising muscles is quite an old one (Patterson, 1928; Alam & Smirk, 1937). One possible reason contributing to the fact that it has not gained much favour among physiologists is the lack so far of a suitable model from animal experiments. The early attempts of McDowall (1936) and Euler & Liljestrand (1947) to provide one seemed, on the contrary, to show opposite effects; for they found falls of arterial blood pressure to accompany contraction of hind-limb muscles in anaesthetized animals. These apparently contradictory findings can now be explained by the different methods used to bring about muscular contraction. In these early experiments direct electrical stimulation of the hind-limb muscles was used, a procedure which would have stimulated many afferent fibres and could have blocked transmission in them subsequently. Moreover, the low-threshold afferent fibres are those from mechanoreceptor nerve endings which are known to cause, when stimulated all together in this way, a reflex fall of blood pressure. This, therefore, would be the primary effect of the stimulation used. Further, any afferent discharge originating in the contracting muscles subsequently would be distorted in quite an unpredictable way. In the present experiments the muscles were made to contract by stimulating the appropriate ventral roots, thus leaving the afferent supply from the muscles intact and avoiding any such artifacts. Under these conditions a pressor response was invariably obtained, accompanied by tachycardia and an increase in rate and depth of respiration. The anaesthetic may have had some depressant effect on this response; for it was much larger when the experiment was repeated in decerebrate preparations.

Lind *et al.* (1966) felt sure that the response in man was reflex; for when they occluded the circulation in the exercising arm, thus preventing any possible chemical mediator from leaving the limb in the venous blood, the

response was, if anything, enhanced. More recently, Lind, McNicol, Bruce, Macdonald & Donald (1968) came very close to demonstrating the existence of the reflex mechanism in humans when they showed that the pressor response could not be evoked in a patient with syringomyelia when he performed hand grips on the affected side. Lind *et al.* (1966) had emphasized that the pressor responses were not associated with pain; for discomfort did not arise from sustained hand-grip contractions until about half-way through a contraction held to fatigue. Similarly, Lewis (1942) and Dorpat (1952) stated that the earliest onset of pain during sustained contractions occurred after 20–25 sec. In the present experiments the arterial blood pressure began to rise 2·5 sec after the beginning of contraction, and frequently reached peak value after 15 sec. Extrapolating from the work in man it is reasonable to conclude that the responses took place in the absence of an afferent input that would have given rise to pain.

In the present investigation, the reflex nature and origin of the response could be proved; for, if muscle contraction was abolished by gallamine, or the appropriate dorsal roots had been sectioned, ventral root stimulation had no effect on blood pressure. On the other hand, after cutting all articular nerves the responses were unimpaired, so they must have originated in the contracting muscles themselves. A further possibility that had to be considered is that the primary reflex response to muscular contraction is respiratory, and that increased ventilation led secondarily to the rise of blood pressure and heart rate through the enhanced activity of lung inflation receptors. This was tested by cutting both vagi, after which the cardiovascular responses to muscle contraction were unchanged.

The next question to be considered therefore was that of the nature of the receptors within the muscles that were initiating the reflex cardiovascular and respiratory responses to exercise. From the work in man it had seemed possible that the receptors were responding to some material produced during contraction, since circulatory occlusion usually enhanced the pressor response to muscle activity (Alam & Smirk, 1938; Staunton, Taylor & Donald, 1964). In our experiments too, occluding the arterial inflow to the hind limb was found to increase the magnitude and rate of rise of the pressor response to contraction. As is well known, the blood flow through the triceps muscle is reduced during tetanic contractions, due to mechanical compression of the intramuscular vessels, and this reduction of flow is naturally greater with increasing strengths of contraction. Similar results were obtained by Humphreys & Lind (1963) in human subjects. It is apparent that artificial occlusion of the circulation only exaggerates that which commonly occurs normally during a tetanic contraction.

In experiments without occlusion it was shown that the magnitude of

the pressor response was proportional to the tension developed by the triceps surae muscle during isometric contractions. Tension was recorded in this muscle as a sample of the activity in the rest of the hind-limb musculature. There will have been differences in absolute tension developed by the various limb muscles, but it is likely that the strength of stimulation required to elicit a maximal contraction in the triceps surae would also cause most of the hind-limb muscles to contract maximally. Therefore, it is reasonable to conclude that the pressor responses were proportional to the strength of the contractions in the whole active muscle group. A similar relationship has been found in experiments on human subjects (Gillespie, 1924; Patterson, 1928; Alam & Smirk, 1937; Lind *et al.* 1964). These findings raise the question whether the stimulus arising in the contracting muscles might not be entirely chemical. The main mechanoreceptor afferent fibres arise from muscle spindles or Golgi tendon organs; the former are unloaded during contractions in which the stimulus to the ventral roots is subthreshold for the γ-efferent fibres (as they were in the major part of our experiments), and the firing rate of the latter is not increased and may even be decreased during the relatively short periods of occlusion used in the present experiments (Perez-Gonzalez, 1969). Moreover, Coote & Perez-Gonzalez (1970) have shown that the group I and group II somatic afferent fibres do not reflexly excite any part of the sympathetic output, whereas group III and group IV afferent fibres do so. As might be expected, therefore, studies of the activity of identified afferent fibres, during contractions of a strength to elicit pressor responses, have not demonstrated firing patterns appropriate to sensory fibres producing the response in any of the known mechanoreceptor afferents, nor in non-mechanoreceptor afferent fibres conducting at rates above 17 m/sec (Perez-Gonzalez, 1969). This observation correlates well with the finding of Coote & Perez-Gonzalez (1970) that only somatic afferent fibres conducting at rates below 15 m/sec reflexly activate sympathetic efferent pathways. The possibility can therefore be excluded that mechanoreceptor endings initiate the cardiovascular reflex, either by a direct response to a mechanical stimulus or by a modification of their mechanical response by a chemical mediator, as was suggested by Dejours (1962) when considering the initiation of the reflex respiratory response to muscular contraction.

The most likely conclusion, therefore, is that the receptors concerned are connected to the group III and possibly group IV afferent fibres which have been shown to be involved in pressor reflexes, and that they are metabolic receptors stimulated by a chemical substance (or substances) released in the muscles during activity. It seems preferable to use the term 'metabolic receptors' rather than 'peripheral chemoreceptors' as the word 'chemoreceptor', has for so long been associated with the central receptors

specifically responsive to a low P_{O_2} (and to some extent to a raised P_{CO_2} and a lowered pH). Kao (1963), who showed that the reflex respiratory response in dogs could be elicited by muscle contraction brought about by ventral root stimulation, also demonstrated in cross-circulation experiments, in which the hind-limb muscles of the recipient were those giving rise to the reflex response, that rendering the donor dog hypoxic or hypercapnic did not induce any change in ventilation in the recipient animal. It is so likely that both the cardiovascular and respiratory responses are part of the same reflex, initiated by the same receptors, that these together may be properly called the exercise reflex.

There are numerous small myelinated and unmyelinated afferent fibres in muscle which have only been regarded so far as pain fibres but which could function as metabolic receptors. These fibres are connected to free nerve endings, many of which are located around blood vessels in the muscle (Lim, Liu, Guzman & Braun, 1962; Barker, Ip & Adal, 1962) and which therefore are ideally distributed to respond to accumulation within the interstitial fluid of some metabolite. Some of these small afferent fibres have already been shown to respond to non-nociceptive stimuli, such as occlusion of the artery to a muscle for only a few seconds (Iggo, 1961). The possibility that they could subserve a non-nociceptive function, such as to initiate cardiovascular and respiratory reflexes during contraction, does not exclude their participation in the pain mechanism. Indeed, according to recent theories of pain perception (Goody, 1957; Melzack & Wall, 1965) the small myelinated muscle afferents would be expected to play a non-nociceptive role even if they could signal pain under certain circumstances.

When judging the extent of the local dilatation of resistance vessels in contracting muscles in relation to the strength of contraction, it is usual to use rate of blood flow as a measure of the vascular response. In fact, however, the measure actually required is the length of the smooth muscle fibres in the vessel walls. As an approximation, in this investigation, 'conductance' was first calculated from flow and perfusion pressure (in order to take account of changes in the latter) and the fourth root of this was used as an index of change of smooth fibre length. Comparison of the graphs of Figs. 2 and 6(*b*) then show that the size of the pressor response has much the same relationship to the tension developed by the contracting muscles as has the extent of the local dilatations of resistance vessels in these muscles (as judged by the change in 'vessel circumference'). This would be consistent with the suggestion of a real relationship, i.e. that the local hyperaemic response and the reflex pressor (or exercise reflex) response are the consequence of similar processes within the muscle and are initiated by the same chemical stimulus.

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