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THE BLOOD FLOW THROUGH MUSCLE DURING SUSTAINED CONTRACTION

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It is generally agreed that the rate of the blood flow through contracted skeletal muscle has an important bearing on the chemical processes proceeding in that tissue and on the cause of fatigue. Nevertheless, the evidence regarding the effect of a strong prolonged contraction on the local blood flow is still conflicting. Recent experiments by Rein, Mertens & Schneider [1935], Kramer & Quensel [1937] and Bülbring and Burn [1939] show that the vessels in the strongly stimulated muscle of the anaesthetized dog are widely dilated after the first $\frac{1}{4}$ min. of contraction. This suggests that strongly contracted mammalian muscle receives a rich blood supply. There are, however, some reasons for thinking that strongly contracted human muscle does not do so. Dolgin & Lehmann [1930] found that arresting the circulation in the upper arm had no effect on the maximum length of time for which a strong hand-grip could be exerted and concluded that strong contraction arrested the flow in the muscle. Grant [1938] measured the flow in a section of the forearm plethysmographically and found that the increase during 1 min. strong hand-grip was trivial in comparison with that seen immediately after relaxation.

The object of this paper is to give a further proof of the ischaemic nature of human muscle during strong contraction.

It is self-evident that the blood flow through a single human muscle can never be measured directly by any of the existing methods. Our method shares this disadvantage, but it has the advantage that it gives qualitative results concerning the blood flow through a single group of human muscles.

PRINCIPLE OF METHOD USED TO SHOW INCREASE IN BLOOD FLOW THROUGH THE PLANTAR FLEXORS OF THE HUMAN FOOT

Increase in blood flow through skin exposed to air at ordinary room temperature raises skin temperature. This is the basis of methods for studying the skin circulation. The underlying principle is that the exposed skin is cooler than the blood entering it; a faster flow brings more heat from the depths of the body and the skin warms up. The principle may be put in a more general way as follows. If any relatively small part of the body is above or below general body temperature, increase in its blood supply will tend to bring its temperature nearer to body temperature. The essence of the method is the existence of an initial temperature difference between the part and its blood supply.

Grützner & Heidenhain [1878] applied the principle to the study of muscle flow in anaesthetized animals. They produced the necessary temperature difference by skinning the leg; the underlying muscle cooled by exposure.

Our method is based on the same principle, adapted for showing an increase in the blood flow through the muscles of the calf of the human leg.

Muscle temperature was measured thermoelectrically, to the nearest 0·02° C., 3–6 cm. below the skin. The temperature difference between the blood and the muscle was produced by immersing the leg, up to the knee, in water. By keeping the water at a suitable steady temperature, the temperature of the resting muscle could be brought to any desired steady level, above or below body temperature.

The main difficulty was that during activity, variations in muscle temperature were caused by "heat production" as well as by blood flow. Careful controls were needed to avoid confusion. To see if a contraction increased the blood flow its effect on muscle temperature was recorded:

1. When resting muscle temperature was above blood temperature, "hot" muscle:
   (*a*) With free circulation through the leg.
   (*b*) While the circulation was arrested by an inflated pneumatic cuff on the thigh.
2. When resting muscle temperature was below blood temperature, "cold" muscle:
   (*a*) With free circulation through the leg.
   (*b*) While the circulation was arrested.

We concluded that the contraction had increased the flow through the muscle if, during its performance, muscle temperature approached the temperature of the blood entering it, that is, if it fell in 1 (*a*) and rose in 2 (*a*) and if, in addition, the temperature changes attributed to hyperaemia were absent when the contractions were made while the blood supply was arrested, 1 (*b*) and 2 (*b*).

The sustained contractions used for the study of the blood flow

Owing to the relatively simple mechanics of the movement of the ankle joint the muscles of the calf of the leg were once often experimented on to find the maximum force a human muscle could exert per sq. cm. of its physiological cross-section. To find this constant it was necessary, among other things, to determine the maximum force the gastrocnemius and soleus could exert in a voluntary effort. In general the body was loaded with weights till a point came when the heels could only just be raised from the ground. The results given by Weber [1846], Koster [1868], Hermann [1898] and Reys [1915] agree fairly well that the limiting load is about 450 kg. That is, the muscles of each calf can exert on the ball of the foot a force of about 225 kg. They calculated that the pull on the Achilles tendon was about three times as much, about 675 kg. —more than half a ton.

These data have enabled us to express the strengths of the contractions we used as fractions of the maximal strength this group of muscles can exert voluntarily. This notation will have its value for comparing our results with those obtained on other muscles.

As the gastrocnemius arises from the lower end of the femur it acts both as a flexor of the knee and a plantar flexor of the ankle. The knee joint was kept fully extended in all our sustained contractions so that the gastrocnemius exerted its pull from a standard position.

The vascular changes were examined in contractions of four different strengths 0·3, 0·2, 0·1 and 0·05 maximal.

To perform the strongest contraction, the 0·3 max., the subject stood on tiptoe, with the knee straight, on the leg in the water-bath (a dustbin). The body was just steadied with the tips of the fingers. The force on the ball of the foot was about 77 kg. (average of the body weights of the two subjects).

For the 0·2, 0·1 and 0·05 max. ones the subject sat on a bicycle saddle fixed at a convenient height beside the water-bath and, keeping his knee straight, exerted a steady pressure with the ball of his foot on a stirrup

which hung in the water near the bottom of the bath. The stirrup was attached to a suitably weighted lever. The actual forces exerted by the ball of the foot in these exercises were 45, 22·5 and 12·5 kg. respectively, according to the weight on the lever.

The 0·3 and 0·2 max. contractions, and all contractions performed while the circulation was arrested, were kept up till intolerable discomfort forced the subject to give up. This meant that muscle temperature was recorded throughout, if we may use the phrase, the whole "physiological life" of these contractions—while the muscle passed from rest to physiological (but not necessarily chemical) exhaustion.

The 0·1 and 0·05 max. contractions were given up at $\frac{1}{4}$ and $\frac{1}{2}$ hr. respectively; no acute discomfort was felt.

Rhythmic contractions

Muscle temperature changes during gentle rhythmic work were also examined. The subject transferred the weight of his body from the ball of one foot to the ball of the other once every second.

Experimental

Thirty-five experiments were done, each lasted on the average 5–6 hr., making a total of over 200 hr. muscle temperature observation. Protocols of typical experiments are shown in Table I.

The authors usually took turns to act as subject. Occasionally Dr N. C. Hughes—and we are very grateful to him—acted instead.

The experiment began in the morning. The dustbin, supported over a thermostatically controlled gas flame, was filled with water and the bath stirrer was started. The temperature of the water depended on whether resting muscle temperature was to be above or below body temperature. The thermojunction was sterilized by dipping it in a test tube of boiling water. The subject stood on a chair and supported most of his weight on the leg about to receive the junction. The skin of the calf was shaved and washed with surgical spirit. The observer "scrubbed up". He threaded the sterile and carefully straightened thermojunction wires into a sterile "veterinary" $1\frac{1}{2}$ in. Record mount bore 2 hypodermic needle (external diameter 1·6 mm.), and plunged the needle through the skin on the lateral or postero-lateral surface of the subject's calf deep into the muscle. In so doing he took great care to avoid the main vessels and nerves of the leg running along the posterior border of the tibia. He

TABLE I

Exercise	Subject	Date	Junction depth cm.	Room temp. °C.	Water-bath °C.	Circulation free: Resting muscle temp. °C.	Circulation free: Change in muscle temp. during exercise °C.	Circulation free: Change in muscle temp. during first 3 min. after exercise °C.	Circulation free: Duration of exercise min.	Circulation arrested: Resting muscle temp. °C.	Circulation arrested: Change in muscle temp. during exercise °C.	Circulation arrested: Change in muscle temp. during first 2 min. after exercise °C.	Circulation arrested: Change in muscle temp. during first 3 min. after release of circulation °C.	Circulation arrested: Duration of exercise min.
Rhythmic	J.L.E.M.	2. ii. 39	4	18	41·9	39·23	−1·11	+0·29	15+	39·54	+0·43	+0·11	−2·16	2¾
	J.L.E.M.	23. ii. 39	—	19	32·9	34·60	+2·54	0·00	15+	34·75	+0·07	+0·03	+1·06	4
0·3, max. sustained contraction	J.L.E.M.	23. i. 39	3	18	41·3	39·60	+0·39	−1·03	1¾	39·64	+0·44	−0·03	−1·68	2
	H.B.	20. ii. 39	—	19	32·4	34·60	+0·17	+1·21	3	34·56	+0·28	−0·06	+1·29	4¼
0·2, do.	J.L.E.M.	30. i. 39	5	15	42·4	39·85	+0·29	−1·03	2½	39·82	+0·30	−0·02	−1·29	2¾
	H.B.	6. iii. 39	3	17	32·5	34·03	+0·18	+1·04	3¼	34·08	+0·16	+0·06	+1·16	3½
0·1, do.	N.C.H.	17. i. 39	6	20	41·8	39·75	−0·79	−0·02	17+	39·67	+0·53	0·00	−2·00	7
	J.L.E.M.	2. ii. 39	3	16	32·6	33·96	+2·21	−0·08	15+	34·05	−0·02	0·00	+0·93	6¾
0·05, do.	H.B.	1.iii. 39	4	16	42·1	40·18	−0·73	−0·04	30+	40·17	+0·25	+0·08	−1·09	10
	H.B.	24 ii. 39	4	21	32·8	34·31	+2·07	−0·19	30+	34·72	+0·15	−0·12	+0·63	15

A + sign following the duration of the exercise signifies that it was terminated for reasons other than acute discomfort.

pushed the needle home till the hilt touched the skin. He now pulled the needle out of the leg leaving the wires *in situ*. To do this successfully the wires were kept pressing into the muscle while the needle was being pulled out. When the needle was out the junction usually lay 3–6 cm. below the skin. The observer slipped the needle up the wires till it was several inches from the skin. He then bent the wires into a semicircle so that the needle could be laid flat on the skin near the puncture and fixed it to the skin with sticking plaster, so, if the wires were accidentally jerked during the experiment the strain was taken by the sticking plaster and the junction was not pulled out of the muscle. The wires were examined for kinks near the puncture, and if present, they were straightened out to ensure free movement through the skin and fascia during shortening and lengthening of the muscle.

The subject sat down on a raised chair beside the dustbin and immersed the leg with the junction in it up to the knee. The leg stayed in the water for the rest of the experiment. The observer took the temperature of the room. He recorded the muscle temperature, generally every 10 min. He checked the temperature of the water, which remained all day within a few tenths of the desired figure.

(*a*) In a "hot" muscle experiment, as Table I shows, the water was kept at about 42° C. Muscle temperature rose asymptotically from about 37° to about 39·5° in about 1½ hr., and then stayed almost constant, about 2·5° below the temperature of the water. It stabilized below the temperature of the water because the muscle was kept cool by the resting blood flow; arresting the circulation was soon followed by a slow steady rise in muscle temperature.

(*b*) In a "cold" muscle experiment the water was kept at about 32·5°. Muscle temperature stabilized very slowly, in 2–3 hr., at about 34·5°, about 2° above the temperature of the water. (Arresting the circulation stopped the warming effect of the resting flow and caused a slow fall in muscle temperature.)

As soon as the temperature of the muscle had stabilized the subject did one of the exercises described earlier. In doing the exercise the subject hardly ever felt any pain due to the wires in his leg. During the exercise, and for several minutes afterwards, the observer recorded muscle temperature at ¼, ½ or 1 min. intervals. Then came a second long period of rest, with a light lunch, while muscle temperature settled down again. Then the circulation in the leg was arrested and, 2 min. later, the same exercise was repeated. 2 min. after the exercise the circulation was released and about an hour later the experiment finished. On some days

the experiment lasted longer, another of the exercises was done, first with the circulation free and later while it was arrested. After the experiment the length of the wires withdrawn from the leg was measured. Occasionally the insulation was cracked, if so the wires were revarnished before the next experiment (see below).

Method used for arresting the circulation. We are grateful to Dr G. W. Pickering for the specifications of the 6½ in. pneumatic thigh cuff we used. As the exercises caused contraction of the thigh muscles which might have protected the artery, to some extent, from the pressure of the cuff, we used a cuff pressure of 230 mm. Hg; it was thrown in from a reservoir. In some control experiments the leg was made bloodless by bandaging it from the toes to above the knee with a wide rubber bandage; the cuff on the thigh was then inflated and the bandage taken off. The skin was deadly pale, and, as the strongest of our exercises had no effect on the colour, we concluded that the arrest could be relied upon to be effective in all our experiments.

The discomfort caused by the cuff was usually quite negligible compared with that which came lower in the leg towards the end of the "physiological life" of a contraction.

Thermoelectric method. Most observers have used the hypodermic needle type of thermojunction for human deep temperatures. Foged [1930] took the temperature of the venous blood with a flexible wire thermojunction which he threaded through a hypodermic needle inserted into a vein in the ante-cubital fossa. We chose the flexible wire rather than the hypodermic needle because we thought it would be less likely to break, or cause pain or injure the contracting muscle. Grant & Pearson [1938] independently adopted the same method.

In our early experiments we were sometimes troubled because the observed temperature changed very suddenly at the beginning and end of contraction. This was traced to "junction shift". During contraction the muscle thickened from side to side and the anatomical relationships of the skin and muscle changed. If the wires were gripped more firmly by the skin and fascia than by the muscle the junction was drawn a little towards the surface of the muscle. As there was a temperature gradient of about 2° C. between the muscle surrounding the junction and the surface of the leg, the movement of the junction brought it in contact with muscle at a slightly lower temperature and this caused an apparent change in muscle temperature. An equal and opposite temperature change occurred on relaxation as the junction slipped back to its original position. To fix the junction firmly in the muscle the last ¼ in. of the wires was roughened so as to catch in the muscle, and they were made as smooth as possible where they passed through the skin.

Hill [1928] used thermoelectric apparatus with a sensitivity of about 10^{-5}° C. and found that if the junction was not insulated from contact with the tissue stray electrolytic E.M.F.'s caused errors. Grützner & Heidenhain [1878] stress the importance of perfect insulation, though their apparatus was only accurate to about 10^{-2}° C. Our apparatus was only accurate to about 0·02° C., but we thought it easier to insulate the junction than to see whether lack of insulation caused error.

The copper-constantan thermocouple was made as follows. The constantan (Eureka) wire was 0·122 mm. in diameter and 3 m. long. Its resistance was 120 Ω, all that was necessary in the external circuit for critical damping. It was enamelled and double-cotton covered. The enamel insulated the wire from the water. Each of the two copper wires was 2 m. long, 0·274 mm. in diameter, enamelled and double-cotton covered. The cotton was stripped off all the wires for about a foot from their ends. The junction to go into the muscle was made as follows. One of the copper wires was laid alongside the constantan wire with its end level with the end of the constantan. The ends were put in a clamp fixed at the top

of a stand. A weight was hung on them about 6 in. under the clamp. This kept them parallel and in contact; they were then stuck together with several coats of Bakelite varnish. Downing [1935] has described the use of this varnish. The wires were then unclamped and the ends, which had been protected from the varnish by the clamp, were separated. The constantan was wound round the copper in a spiral of about 8 turns for $\frac{1}{4}$ in. The ends were then cut off level, hard soldered, and varnished. The spiral near the junction provided the rough surface for the muscle to grip, and the part of the wires traversing the skin and fascia was smooth and free from twists.

The constant-temperature junction was made by hard soldering the other end of the constantan wire to an end of the other copper wire, and varnishing. It was fixed near the bulb of a thermometer placed in paraffin in a vacuum flask in a gas-heated incubator kept at about 37° C. (an electrically heated incubator was tried but discarded because of stray E.M.F.'s in the thermocouple circuit). The thermometer was graduated in tenths of a degree, hundredths could be read by eye with a lens and device for avoiding parallax.

The galvanometer was a Cambridge Scientific Instrument Co. d'Arsonval. Its sensitivity (when critically damped with an external resistance of 120 Ω) was $7{\cdot}6 \times 10^{-6}$ V. per mm. at 1 m. The internal resistance was 8·5 Ω and period 2·4 sec. The working distance used was 1 m.

The simple circuit consisted essentially of the galvanometer, an all-copper double-pole throw-over switch, a 120 Ω copper resistance and the thermocouple. By means of the switch the galvanometer could be connected to the resistance for short-circuiting, or, to the couple for readings.

The apparatus was carefully standardized by taking several readings at known junction temperature differences. The relative accuracy of the temperatures recorded was about 0·02° C. 1° C. junction temperature difference caused a deflexion of about 50 mm. The recording time was about 3 sec., very little more than the period of the galvanometer.

Bazett & McGlone [1927] have shown that deep temperatures taken with a thermo-junction are generally a little too low, as a little of the heat of the tissue round the junction is conducted away along the leads. Analogous errors may have been present in our readings. As our conclusions are based on relative and not absolute temperature changes this possible source of error is irrelevant.

Results

In describing the results we shall often speak of experiments on "hot" and "cold" muscle, but it must be understood that muscle temperature was never outside physiological limits. For example, in vigorous exercise rectal temperature rises above what we shall call "hot" and it drops below "cold" in a bare leg rested for an hour or so exposed to air at ordinary room temperature.

In any given experiment we did not know for certain whether the junction was in the gastrocnemius or the soleus. J. L. E. M. judged from the direction in which he inserted the needle that the junction usually lay in H. B.'s soleus. H. B. thought he usually inserted the junction into J. L. E. M.'s gastrocnemius. Probably the temperature changes were examined many times in each muscle. Yet the changes caused by a given strength of contraction were always the same in these two subjects. We therefore think that a given strength of contraction caused similar temperature and vascular changes in both muscles.

Fig. 1, drawn from data obtained in the typical experiments shown in Table I, shows the changes in muscle temperature during the actual performance of the exercises. The top half shows the results for "hot"

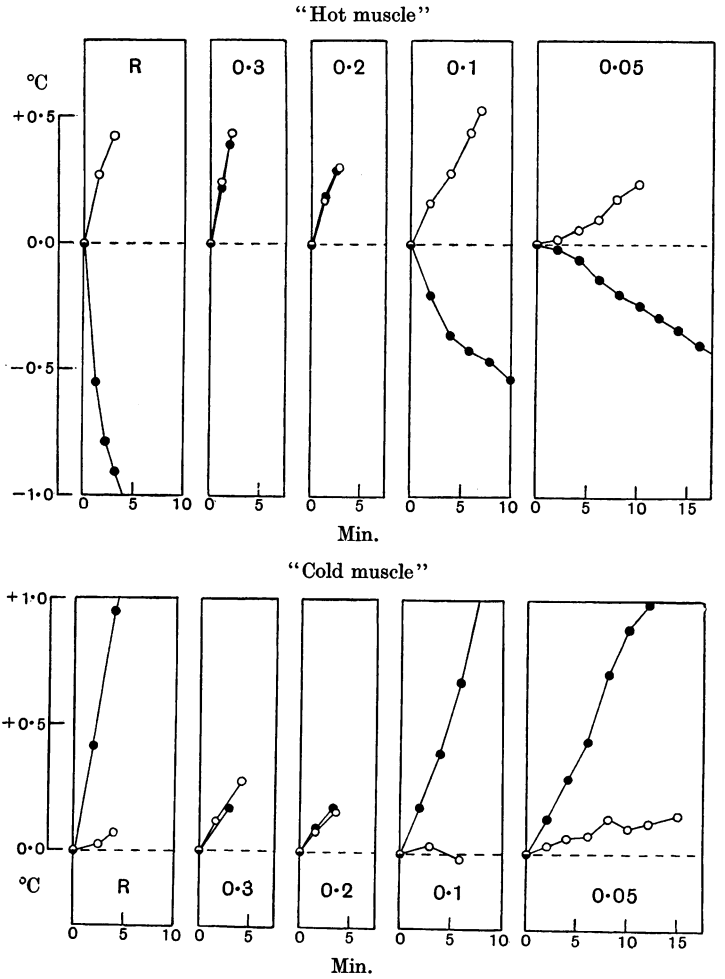

Fig. 1. Change in muscle temperature during the performance of rhythmic work, *R*, and 0·3, 0·2, 0·1 and 0·05 max. sustained contraction. • Exercise done with the circulation free. o Exercise done while the circulation was arrested.

muscle, resting temperature 39·5–40° C.; below are the results for "cold" muscle, resting temperature 34–34·5° C. The lines joining black dots show the temperature changes with the circulation free, those joining the circles

while it was arrested. In all cases the exercise began at time "0". The last point on each curve was taken just at the end of the exercise, when the subject said the discomfort was intolerable. With the circulation free, rhythmic work and weak static work (0·1 and 0·05 max.) could be kept up for so long that the end-point is not shown on the diagrams. Before the beginning of the exercise the temperature of the muscle had not changed by more than 0·05° C. during the previous 20 min. In any given exercise on "hot" or "cold" muscle the "circulation free" and "circulation arrested" curves are comparable, as the experiments were done on the same subject on the same day and with the junction in the same place in the calf.

Rhythmic work may be considered first. The vascular changes during this type of exercise are described here partly to illustrate how the method works, as rhythmic work is known to increase muscle flow, and partly to show the contrast between the effects of rhythmic and strong static work. Fig. 1 shows the effect of rhythmic work on "hot" muscle with the circulation free. Immediately after the work began the muscle started to cool very quickly. This could hardly have been due to an endothermic chemical change. It was more probably due to a marked hyperaemia. The relatively cold blood rushing through the hot muscle cooled it. This was confirmed by the other control experiments. When the exercise was done with "hot" muscle while the circulation was arrested the muscle did not cool, therefore the blood flow was responsible for the cooling. When the exercise was done with "cold" muscle, and the circulation free, the muscle warmed up very quickly. This is exactly what would be expected if there had been a marked hyperaemia. The muscle would be warmed by the rush of relatively warm blood. This is checked by the fact that the rapid rise in temperature did not occur when the exercise was done on "cold" muscle while the circulation was arrested.

Taking this evidence as a whole it amounts to proof that the rhythmic work was accompanied by increase in muscle flow, as would be expected on general grounds. Barcroft & Kato [1915] have shown that short repetitive bursts of tetanic stimulation increased the flow through the dog's gastrocnemius.

Some further points may be added. The curves show that while the circulation was arrested the work could not be kept up for as long as 5 min. With the circulation free it was kept up for 15 min. quite easily and stopped before any distress was felt. Evidently the circulation plays a very important functional role in the performance of rhythmic work, as has already been shown by Lewis, Pickering & Rothschild [1931]. As

will be seen later the circulation through muscle is of no functional importance in strong static work.

The rise in muscle temperature during exercise with the circulation arrested is interesting though it is unnecessary, for the present purpose, to discuss its cause in detail, and we merely give the probable explanation. The principal factors concerned are (*a*) "heat production" due to the rapid metabolism accompanying the activity, (*b*) the removal, by the arrest, of the thermal influence of the resting blood flow. Arresting the blood flow through "hot" muscle removes the cooling effect of the resting flow so the muscle temperature gradually rises towards that of the water in the bath. Arresting the circulation in "cold" muscle stops a warm resting flow and the muscle begins to cool towards the water temperature. That is, in a "hot" muscle experiment the rise in temperature during exercise with the circulation arrested is due to "heat production" + heat entering the limb from the water. In a "cold" muscle experiment the rise in temperature is due to "heat production" − heat loss from the limb to the water. In a "hot" muscle experiment the "heat production" is exaggerated, in a "cold" one it is reduced. The same applies to the temperature changes in the other exercises performed during arrest, and it will not be necessary to draw any further attention to this point.

We may now turn to the results obtained with the 0·3 and 0·2 max. sustained contractions, results which are entirely different from those of rhythmic work. The two points shown in Fig. 1 are: (*a*) during the exercise muscle temperature rose at practically the same rate in each "hot" or "cold" experiment whether the circulation was free or arrested—there was no evidence of hyperaemia. If the flow had stopped altogether during contraction the results would presumably have been those seen in Fig. 1; we cannot be sure that the flow was brought to an absolute standstill, but it is certain that it did not rise much above the resting rate, which, in the experiments on cold muscle, must have been extremely small, say 1 c.c./100 c.c. muscle [Grant & Pearson, 1938]. (*b*) All these exercises were kept up till the subject could bear them no longer. The "physiological life" of these contractions was the same whether the femoral vessels were patent or occluded, the blood flow through the muscle, if any, was of no functional significance.

On the other hand, Fig. 1 shows that the results for the weaker 0·1 and 0·05 max. contractions resembled those of rhythmic work. With the circulation free a very distinct hyperaemia occurred during the exercises; the "physiological life" of these contractions was shortened by arrest, the hyperaemia had a functional value.

To sum up, 0·3 and 0·2 max. contractions of the flexors of the foot are not accompanied by hyperaemia, the flow is probably arrested in the muscle; 0·1 and 0·05 max. contractions and rhythmic work are accompanied by marked hyperaemia.

The vascular changes during the recovery period may be inferred from the muscle temperature changes during the first 3 min. after exercise, given in Table I. Very marked hyperaemia set in a few seconds after the 0·3 and 0·2 max. contractions relaxed. On the other hand, the hyperaemia accompanying the 0·1 and 0·05 max. contractions and the rhythmic work did not become accentuated after the exercise.

Table I shows the temperature changes during the first 3 min. following release of the circulation. Marked reactive hyperaemia always occurred.

Discussion

The experiments just described establish the fact that a single group of human muscles, the plantar flexors of the foot, are almost or quite ischaemic during strong contraction (0·3 and 0·2 max.) and are markedly hyperaemic during weak contraction (0·1 and 0·05 max.). The facts are probably explained as follows. Anrep & v. Saalfeld [1935] and Grant [1938] have recently added to the evidence in favour of the liberation of vaso-dilator substances in active muscle. We attribute the hyperaemia during weak contraction mainly to the action of these metabolites. There seems to be only one possible explanation for the suppression of the hyperaemia during strong contraction, namely, the compression of the potentially dilated vessels by the taut muscle fibres, and this is borne out by the immediate appearance of the hyperaemia a few seconds after the muscle relaxes. Conclusive evidence already exists to show that contracted muscle can impede the blood flow mechanically, both in man and in the dog [Lindhard, 1920 *a*, *b*; Anrep, 1935; Kramer & Quensel, 1937; Grant, 1938]. The critical strength of contraction of the plantar flexors of the foot above which the hyperaemia was latent till relaxation was about 0·15 max., equivalent to the exertion of a force of about 35 kg. on the ball of the foot, with a corresponding strain of about 100 kg. on the Achilles tendon, conditions which would be fulfilled in an adult standing on tiptoe on both feet.

Our results will now be discussed in relation to other work. Dolgin & Lehmann [1930] found that weak hand-grips could be exerted for longer when the circulation in the arm was free than when it was arrested, but that the performance of strong grips was unaffected by the arrest. They concluded that the circulation in the active muscle was maintained

and was of functional value during the weak grips, but that during the strong grips the muscles became ischaemic. Our experiments support their conclusions. Asmussen & Hansen [1938] recently extended earlier work by Lindhard [1920 *a*, *b*] and found that the rate of pulmonary oxygen intake increased both during and after sustained contractions so strong that they could only be exerted for 2 or 3 min. They concluded that the flow through the active muscles increased both during contraction and again after relaxation when the mechanical opposition to the flow had vanished. At first sight these results seem to conflict with the ischaemic nature of strong contraction indicated by our experiments. It seems likely, however, that in Asmussen & Hansen's experiments the pulmonary oxygen intake would be the composite expression of events proceeding in a very large number of muscles in the legs and adjacent parts of the trunk and that the strength of contraction would differ considerably from one muscle to another. If that were the case the most powerfully contracted muscles might be ischaemic and the increased oxygen intake might be associated with the greatly increased flow through those muscles which were not contracting so vigorously. Similarly, the slight increase in blood flow found by Grant [1938] in a section of the forearm during the exertion of a strong hand-grip may have been the expression of a marked hyperaemia in some muscles and an ischaemia in others.

Rein *et al.* [1935], Kramer & Quensel [1937] and Bülbring & Burn [1939] found that during prolonged strong nervous stimulation of the anaesthetized dog's gastrocnemius muscle the vessels became widely dilated. In the plantar flexors of the human foot this finding would be expected during weak but not during strong contraction. Two alternative explanations seem possible. There may be a genuine difference in the behaviour of the muscle flow during strong contraction in human and in dog's muscle. This seems rather unlikely. Alternatively the contractions studied in the animal experiments may actually have been weak and not strong ones. In the dog experiments a contraction elicited by maximal nervous stimulation has been assumed to be a strong one. The assumption is a natural one, but does not seem entirely self-evident in the light of a critical examination of the actual pull exerted by the maximally stimulated muscle. As far as we are aware the maximum motor tetanus of the decerebrate dog's gastrocnemius is unknown. Eccles & Sherrington [1930] found an average value of 12·5 kg. for cats averaging 3·5 kg. in weight, which suggests a value of approximately 30–40 kg. for 10 kg. dogs. Kramer & Quensel [1937] do not mention the strength of the

contractions studied in their experiments. Rein *et al.* [1935], if we interpret their experiments correctly, tied the Achilles tendon to a 1 kg. weight, so that the tension developed during contraction could not have exceeded that amount. Bülbring & Burn [1939] recorded isometric tensions of about 18·5 kg., but these soon fell to a steady level of about 5 kg. Anrep & v. Saalfeld [1935] recorded, on the same muscle, an isometric tension of 2·7 kg. during a contraction lasting 5 sec. These figures suggest that the contractions elicited in the above experiments on the anaesthetized dog may not have been strong ones. The idea gains probability for another reason. The highest recorded steady tension in the above experiments was 5 kg. As the force exerted on the pad of the dog's foot is about one-third of that applied to the Achilles tendon, 5 kg. on the tendon would correspond to 1·7 kg. on the pad, this would not be sufficient to support one-quarter the weight of a 10 kg. dog. It seems possible that some factor common to all the experiments on the anaesthetized dog may have prevented strong stimulation from eliciting strong contraction; if it elicited weak contraction the accompanying hyperaemia would correspond to that also found during weak contraction of the flexors of the human foot.

Summary

1. Recent work shows that the needs of strongly contracting muscle are met by a poor vascular response in man but a liberal one in anaesthetized animals.

2. The effect of sustained contraction on the blood flow through a single group of human muscles, the plantar flexors of the foot, has been examined qualitatively by a new method.

3. 0·05 and 0·1 maximal contractions of this group are accompanied by marked hyperaemia which gradually subsides during the recovery period.

4. The length of time for which contractions of this strength can be maintained is very much shortened by previous arrest of the circulation in the leg, therefore the hyperaemia normally accompanying them is of functional value.

5. 0·2 and 0·3 maximal contractions are not accompanied by hyperaemia; the flow is probably arrested in the muscle. Marked hyperaemia sets in a few seconds after relaxation.

6. The length of time for which contractions of this strength can be maintained is unaffected by previous arrest of the circulation, therefore the blood flow through the active muscle, if any, is of negligible functional value.

7. The suppression of the hyperaemia during the stronger contractions is believed to be due to compression of the potentially dilated vessels between the taut muscle fibres, this becomes a dominant factor when the strain on the Achilles tendon exceeds about 100 kg.

8. These conclusions are believed to be consistent with the previous results obtained on man. The reason for the discrepancy between the experiments on human and animal muscle is discussed.

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