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THE DURATION OF SUSTAINED CONTRACTIONS OF THE HUMAN FOREARM AT DIFFERENT MUSCLE TEMPERATURES

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Heating of the whole body by immersion in water for short periods (Vernon, 1924) or of a limb by application of hot fomentations (Hall, Schamp, Brown & Davis, 1944; Hall, Munoz & Fitch, 1947) has been shown to reduce the strength and duration of voluntary rhythmic and sustained contractions. More recently, Nukada & Müller (1955) and Nukada (1955) showed that when a limb was immersed for a short period in water ranging from 10 to 40° C (rhythmic contractions) and from 20 to 40° C (sustained contractions) the duration of contractions to fatigue steadily increased from the higher to the lower water temperatures. These experiments on human subjects have led all these observers to the conclusion that the influence of temperature on the performance of muscular contraction is due to alterations in the proportion of blood flowing through the muscle and the skin of the exercised limb.

The possibility that the temperature of the muscle might be associated with the duration or strength of contraction has been dismissed by these workers, but the evidence of Ellis & Beckett (1954) and Hadju (1951) casts some doubt on the validity of such a conclusion. Working with isolated muscle preparations, they showed that both strength and duration of contractions, in the absence of a blood supply, depend on muscle temperatures.

Lind & Samueloff (1957) measured in man the duration of successive sustained contractions when the interval between the contractions was varied and when the arm was kept in water at 18 or 34° C throughout the experiment. They found that contractions were always longer in water at 18° C when the interval between contractions was 20 or 40 min.

The present experiments were designed to discover (*a*) whether there was a further deterioration in muscular performance when the forearm was heated in water above 34° C, and (*b*) if further improvement occurred in the strength and duration of contractions when the water was cooler than 18° C. Blood flow, muscle temperatures and action potentials were measured on the exercised

limb in an attempt to explain the changes of muscular performance with temperature. Some of the results have already been briefly reported (Clarke, Hellon & Lind, 1957*a*, *b*).

METHODS

The sustained contractions were made on a simple hand-grip dynamometer illustrated in Fig. 1. The finger grip was connected through a universal joint to a mild steel bar 1·5 cm thick. Two strain gauges (each 500 Ω) were cemented to this bar and formed two arms of a conventional Wheatstone bridge, imbalance of which produced deflexion on a spot galvanometer. The strain gauges were fixed to the upper part of the bar to keep them out of the water. The dynamometer was attached to a stand mounted on castors, to ensure that only flexor muscles were involved in producing the required tension. The arm was held so that an angle of 90° was maintained between upper arm and forearm.

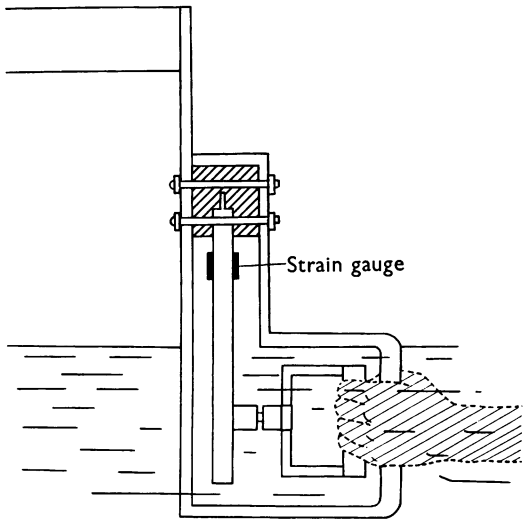

Fig. 1. Diagrammatic representation of the hand-grip strain-gauge dynamometer.

Four healthy young men trained in the use of the dynamometer acted as subjects. At the start of each experiment the subject immersed his forearm in the water-bath and immediately made two brief (<3 sec) maximal contractions: 1/3 of the mean of these contractions became the target tension for the subsequent sustained contractions. The arm remained in the bath throughout each experiment, and after 30 min immersion the first of five successive contractions to fatigue was made. Twenty minutes rest was allowed between contractions so that the later contractions would not be affected by the early period of recovery associated with post-exercise hyperaemia, in which the effects of temperature are known to be absent or obscured (Lind & Samueloff, 1957). Each subject monitored his own performance on the galvanometer. No subject performed more than one experiment in any 24 hr period. The end point of a contraction was taken to be the moment when the required tension could no longer be maintained. Although discomfort was experienced during the latter part of some contractions this did not affect the end point, which was sudden and well defined. Several trial experiments were necessary before the subjects could persevere to this end point, but once they had become accustomed to continue the contractions despite the discomfort, the duration of contractions in water at a given temperature was

consistently repeatable. The coefficient of variation for experiments repeated in water at the same temperatures was 3·90%.

The temperature of the water during each experiment was maintained constant to ±0·5° C and care was taken to provide vigorous stirring of the water over the forearm to ensure maximum heat exchange. Water-bath temperatures of 2, 10, 14, 18, 26, 34 and 42° C were used, in random order.

Blood flow in the forearm was measured with a mercury-in-rubber strain gauge plethysmograph (Whitney, 1953) placed 6 cm above the mid position along the forearm to record from the part of the arm which shows the greatest change in blood flow after exercise (Clarke & Hellon, 1957). A cuff round the wrist was inflated to 200 mm Hg 1 min before measuring the blood flow. Records of flow were taken at the rate of 4/min during the 2 min immediately before each contraction. Five seconds after the end of the contraction recording was restarted and blood flows were measured for a further 5 min. Although the flow had not always returned to the resting level by this time, it was thought desirable to release the wrist cuff to readmit blood to the hand, since only 20 min was allowed between each contraction. In cases where blood flow did not return to the previous resting level within 5 min, the values minus the mean initial level were plotted on semi-logarithmic paper against time in minutes and the resulting straight line extrapolated to zero (i.e. the mean initial level). The area under this curve was taken to be the hyperaemic response in ml./100 ml. tissue in that region of the forearm. In some experiments the recording of blood flow was continued during the contractions.

Muscle temperature was measured approximately midway between the skin and the centre of the forearm, using a thermocouple of 40 s.w.g. copper-constantan wire. The thermocouple, in a 22 gauge hypodermic needle, was inserted obliquely for about 4 cm in a distal direction into the brachioradialis muscle at the elbow. The needle was then withdrawn, leaving the thermocouple *in situ* at a vertical depth from the skin of about 2 cm. The oblique insertion prevented kinking of the wire or discomfort during the contractions. Readings were taken from the thermocouple every minute except during the contraction and for 5 min after the contraction, when readings were taken every 15 sec. When subcutaneous temperatures were recorded the thermocouple was inserted parallel to the skin by a similar technique, to a vertical depth of 1–2 mm. Great care was taken to avoid placing the subcutaneous thermocouple in the muscle or close to a blood vessel. Checks were made of the siting of these thermocouples by X-ray photography. Also, the temperature gradient vertically to a depth of 4 cm (i.e. to about halfway through the arm) through the brachio-radialis muscle just below the elbow was measured after the forearm had been immersed for 30 min in water at 2, 10, 18 and 26° C. Control experiments were carried out at each stage of the present series of experiments to ensure that neither the arterial and venous cuffs used in the measurement of blood flow nor the presence of the thermocouples affected in any way the duration of the sustained contractions.

Action potentials were recorded by means of silver suction electrodes placed over the superficial flexor muscles of the forearm. The potentials were amplified, rectified and integrated electronically, and displayed on two Dekatron scaling units connected by a two-way switch to the input. Calibration showed that the integrator counts were identical with each other and were proportional to the input voltage over the range used. The time constant of the amplifier was 0·2 sec; this ensured that low frequencies which can occur with the onset of muscular fatigue would not be excluded.

The arm was withdrawn from the water to perform contractions from which action potentials were recorded because of the difficulty of insulating the electrodes under water. During contractions the arm was lightly wrapped in cotton-wool to minimize heat exchanges. Results were obtained from two subjects when the muscle temperatures were 35, 27 and 20° C, the integrated count being measured every 10 sec during the contraction using the integrating units alternately through the two-way switch.

Precautions were taken to avoid general body heating or cooling. Oral temperatures taken every 10 min throughout each experiment did not vary greatly ($\not>$ 0·4° C). Unless otherwise stated, the term 'muscle temperature' in this paper refers to the temperature measured half way between the skin and the centre of the arm.

RESULTS

Water temperature and duration of sustained contractions

Fig. 2. shows the duration of the first of the five successive sustained contractions made at each temperature of the water. Only the first of the five contractions is shown, for the sake of simplicity; the curve for the first contraction was similar in shape to that for any of the five contractions (see

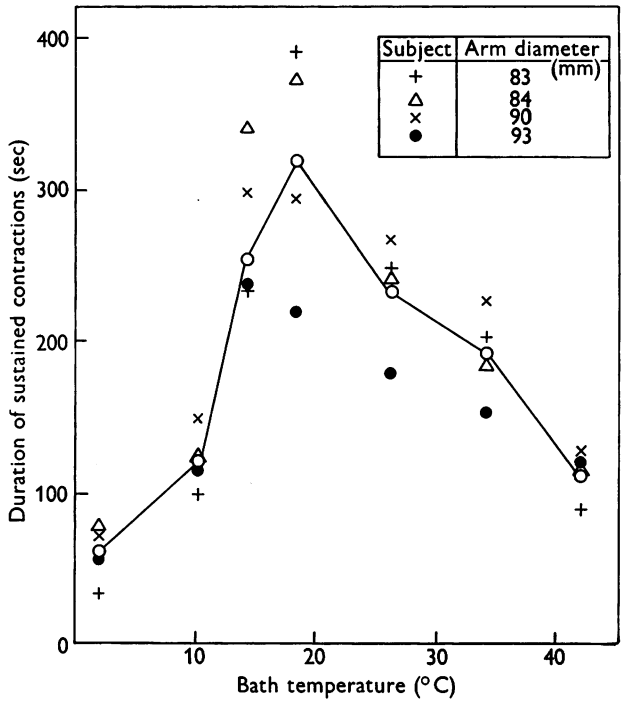

Fig. 2. Showing the individual (symbols) and mean (○—○) durations of the first of five successive sustained contractions in water at seven temperatures. The diameter of the forearm of each subject, taken 6 cm above the mid position of the forearm, is shown inset.

Fig. 3), and showed the same peak. The curve showing the mean duration of contractions revealed that the optimum water temperature for sustained contractions was 18° C. Higher or lower water temperatures resulted in a marked reduction in the duration of the contractions.

The individual results showed that while all the subjects displayed similar performances, there were differences. It is clear from Fig. 2 that for the two subjects with the thinnest forearms their first sustained contractions were longest in water at a temperature of 18° C, while for the other two subjects the longest sustained contractions occurred when their arms were immersed in water at 14° C. It seemed likely from this that there might be a critical

muscle temperature for the maximum duration of a sustained contraction and that subjects with thinner arms reached this critical muscle temperature after their forearms had been immersed in water at 18° C for 30 min. Subjects with thicker arms, however, would require to immerse their forearms for the same length of time in cooler water to achieve a similar muscle temperature—in this case in water at 14° C.

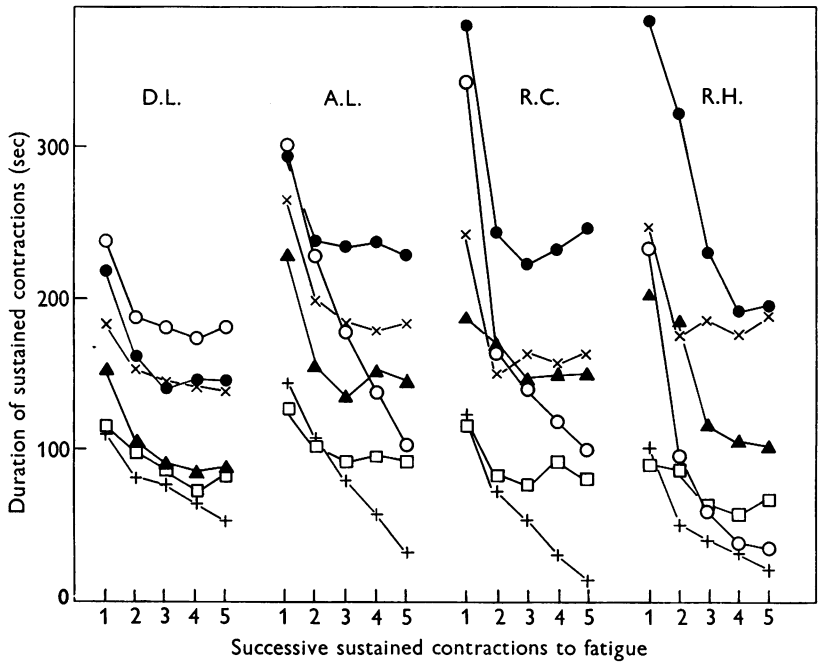

Fig. 3. The duration, for each subject, of the five successive sustained contractions in water at each of the six temperatures. The symbols represent observations obtained when the water was 10° C (+), 14° C (○), 18° C (●), 26° C (×), 34° C (▲) and 42° C (□). Results in water at 2° C are not shown, since only one contraction was completed at this temperature.

Fig. 3 shows the duration of the five successive sustained contractions at each water temperature for each of the four subjects. The pattern of response was similar for all subjects. When the water temperature was 18° C or above, the duration of the sustained contractions fell from the initial value to a steady level for the last two or three contractions. The duration both of the first and of the later sustained contractions shown in these curves appeared to be a function of the water temperature.

At water temperatures lower than 18° C there were slight individual differences which again appeared to be associated with the forearm diameter. The initial contractions of subjects R.C. and R.H. were shorter in water at 14 than at 18° C. In addition, the duration of successive sustained contrac-

tions for these subjects fell steadily throughout the experiment in water at 14 and 10° C. Subject A.L., who had a thicker forearm than these two subjects, showed the same steady fall in the duration of successive sustained contractions in water at these temperatures even though the duration of the initial contraction in water at 14° C was slightly longer than that in water at 18° C. Subject D.L. had the thickest forearm of the four subjects and showed his best performance for all five contractions when his arm was immersed in water at 14° C, but the duration of his sustained contractions fell steadily in water at 10° C.

In water at 2° C only the first of the five contractions was completed, since all the subjects came near to fainting about 5 min after the end of the contraction. This appeared to be caused by the pain due to the arterial occlusion of the hand in such cold water.

Muscle temperatures and duration of sustained contractions

Fig. 4 shows the mean muscle temperature for two subjects at each temperature of the water. During the first 30 min the muscle temperature fell or rose smoothly, depending on the temperature of the water. After the first sustained contraction the muscle temperature rose sharply in water at all temperatures, except in water at 42° C where it fell steeply. These changes of temperature were presumably due to the post-exercise hyperaemia, the incoming blood heating the muscle except in water at 42° C, where the temperature of the incoming blood was lower than that of the muscle. This change of muscle temperature due to the hyperaemia reached a peak about 5 min after the end of the contraction and thereafter the muscle temperature fell (or in water at 42° C, rose) owing to heat exchange with the water. This cyclic procedure occurred after each contraction.

It is evident from Fig. 4 that when the temperature of the water was 18° C or higher, all five contractions in any one experiment were made when the muscle temperature was about the same as that at which the first contraction occurred. The rise or fall of muscle temperature due to the hyperaemia was balanced by the subsequent cooling or heating of the muscle in the interval between contractions. This was not so when the arm was immersed in water at 14 or 10° C, where the rise in muscle temperature due to the hyperaemia was outweighed by the convective cooling from the water, so that successive sustained contractions were made with a steadily falling muscle temperature. The over-all pattern of muscle temperature at which each contraction was made was very similar to the pattern (shown in Fig. 3) of the duration of contractions in water at the six temperatures. The duration of the last three contractions fell progressively in water at 14 or 10° C and it was only in these water-baths that muscle temperature continued to fall. These observations also suggest that there is a critical range of muscle temperature for the

performance of sustained contractions. The changes of muscle temperature during the contractions themselves were variable from individual to individual but customarily rose only slightly (not more than 1° C).

Fig. 5 shows the temperature gradients obtained by inserting the thermocouple, vertically to the skin, through the brachio-radialis muscle after 30 min immersion in water at 2, 10, 14, 18 and 26° C. The temperature

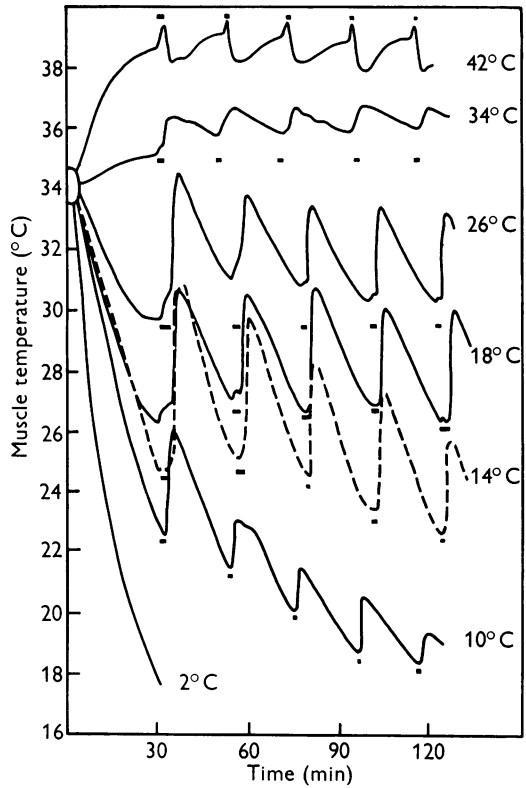

Fig. 4. Muscle temperatures, measured half way between the skin and the centre of the forearm in the seven water temperatures. The contractions are represented by the solid blocks associated with each curve. Each curve is the mean of results obtained from two subjects.

gradient from the subcutaneous area towards the middle of the forearm (i.e. from 0·5 to 3·5 cm) is approximately linear. Comparison of these results with those shown in Fig. 4 confirms that the vertical depth of the obliquely inserted thermocouples used during contractions was about 2 cm from the skin.

Fig. 6 shows the duration of the sustained contractions plotted against muscle temperature. The curve depicting the muscle temperature at which the first contraction is made is separate from the other curves at all points, since the duration of the first sustained contraction was always greater than sub-

sequent contractions (see Fig. 3). Similarly, the curve showing the muscle temperature at which the second contraction was made tends to be separated from subsequent contractions. The optimal muscle temperature for all sustained contractions was between 25 and 29° C, measured at a vertical depth of 2 cm from the skin. The last three contractions were all fitted by a single curve. At muscle temperatures of 27° C and above the duration of the last

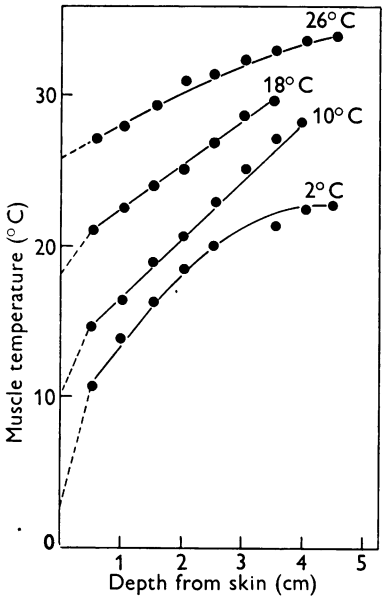

Fig. 5

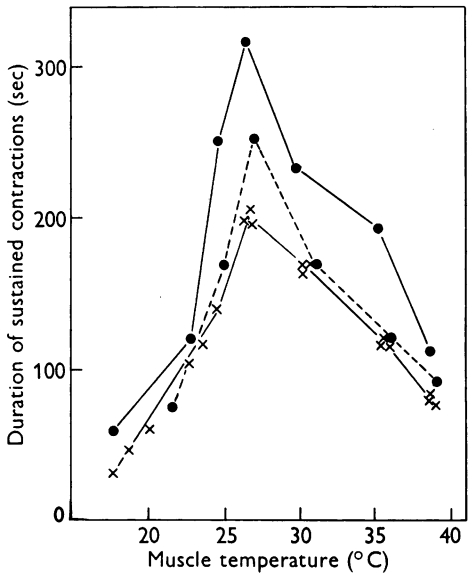

Fig. 6

Fig. 5. Temperature gradients measured vertically from the skin through the forearm after it had been immersed in water at 2, 10, 18 and 26° C for 30 min.

Fig. 6. The average duration of the five sustained contractions at recorded muscle temperatures. The upper curve represents the first contraction, the intermediate curve the second contraction, while the third, fourth and fifth contractions are all represented by the lower curve.

three contractions remained constant, as did muscle temperature. With muscle temperatures below 27° C (observed in water at 14, 10 and 2° C) successive sustained contractions were made with steadily falling muscle temperature; the duration of these contractions also fell and the curve is therefore drawn through points scattered over a wide range. These results again support the conclusion that muscle temperature is associated with duration of sustained contractions.

Muscle temperature and short maximal contractions

A possible hypothesis for the duration of contractions falling at muscle temperatures lower than 27° C is that there is some interference either with the neuromuscular transmission or in the ability of the muscle itself to con-

tract. In order to test this hypothesis experiments were carried out on the ability of the muscle to produce maximum tensions before and after immersion of the forearm for 30 min in water at various temperatures. Each of the four subjects made three short (< 5 sec) maximum contractions in 1 min at the start of the experiment. After the arm had been immersed in water for 30 min a similar series of three maximum contractions was made. The results are shown in Fig. 7. It is clear that the subjects were able to exert the same or

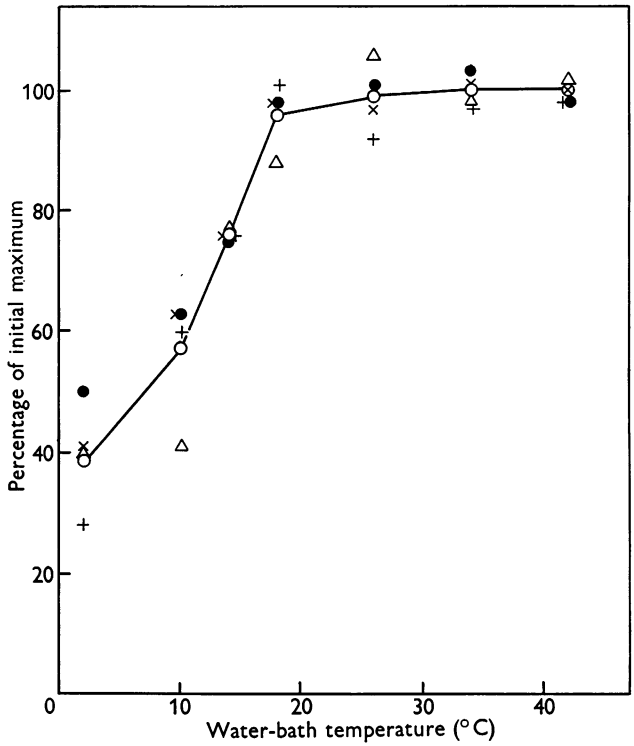

Fig. 7. The individual (symbols as in Fig. 2) and average (○—○) maximal tensions, expressed as a percentage of initial maximum tension, recorded after the forearm had been immersed in water at each of the seven temperatures for 30 min.

nearly the same tension after 30 min immersion in water at 18° C or above, but that in water cooler than this the maximum tension exerted fell considerably to a value of some 40% in water at 2° C.

Relevance of subcutaneous temperatures to sustained contractions

From the foregoing experiments it seems clear that the muscle temperature is associated with the duration of sustained contractions. This belief was rejected by Nukada & Müller (1955), and it remains possible that the results obtained here might be explained by their hypothesis that the partition of

blood between subcutaneous areas and the muscle, or even that the temperature of the subcutaneous area can influence the duration of contractions.

It is possible to affect the subcutaneous and muscle temperatures differentially by immersion of the forearm in hot and then in cold water, or vice versa; in this way, the subcutaneous temperature (and presumably the skin blood flow) can be altered much faster than the temperature of the muscles. Table 1 shows the results obtained by such experiments. The first two subjects immersed their arms in cold water until the muscle temperature was below the optimal muscle temperature for sustained contractions. The arm

TABLE 1. Showing the duration of sustained contractions obtained after the muscle and subcutaneous temperatures had been dissociated from their normal relationships by sudden changes of temperature of the water in which the forearm was immersed. The subcutaneous and muscle temperatures were measured at the start of each contraction and the resting forearm blood flow in water at each temperature. Also shown are the predictions of the duration of each contraction, assuming that muscle temperature alone or subcutaneous temperature alone affected this

			Temperature at start of contraction		Duration of sustained contraction (sec)		
					Predicted		
Subject	Water temp. (° C)	Forearm blood flow (ml./100 ml. tissue/min)	Subcutaneous (° C)	Muscle (° C)	Subcutaneous	Muscle	Observed
A.L.	12–42	2·5– 7·5	37·6	25·5	184	285	290
D.L.	10–45	2·5–13·0	39·8	25·1	122	237	247
R.C.	45–11	39·0–16·0	20·0	36·6	356	190	188
R.H.	45–10	19·0–10·5	23·0	35·5	323	186	184

was quickly transferred to hot water and the contraction was made when the muscle temperature was about 25° C, at which time the subcutaneous temperature was much higher. Estimates were made of the length of time the arm should maintain the contraction assuming that it was affected by (1) muscle temperature only, or (2) subcutaneous temperature alone (the assumption may be made that subcutaneous temperature affects skin blood flow). These estimates were based on the results of previous experiments where the duration of sustained contractions and the muscle and subcutaneous temperatures were known. The final column of the table shows the actual duration of the contraction, which can be seen to be similar to that predicted assuming that the muscle temperature only was associated with the duration. Prediction of the duration based on subcutaneous temperatures was grossly inaccurate. Conversely, the other two subjects first placed their arms in hot water and when the muscle temperature had been raised the arm was transferred to a cold bath. The contraction was made when the muscle temperature was much higher than the optimal temperature, with a subcutaneous temperature similar to that obtained in water at 18° C. Once again the actual duration of the contractions appeared to be dependent only on muscle tem-

perature. Blood flows were taken for several minutes before leaving the first water-bath and again before the contraction was made in the second water-bath. It is clear that there was a considerable difference in blood flow through the forearm in the two water-baths for any given subject, and this probably represents variation in skin circulation.

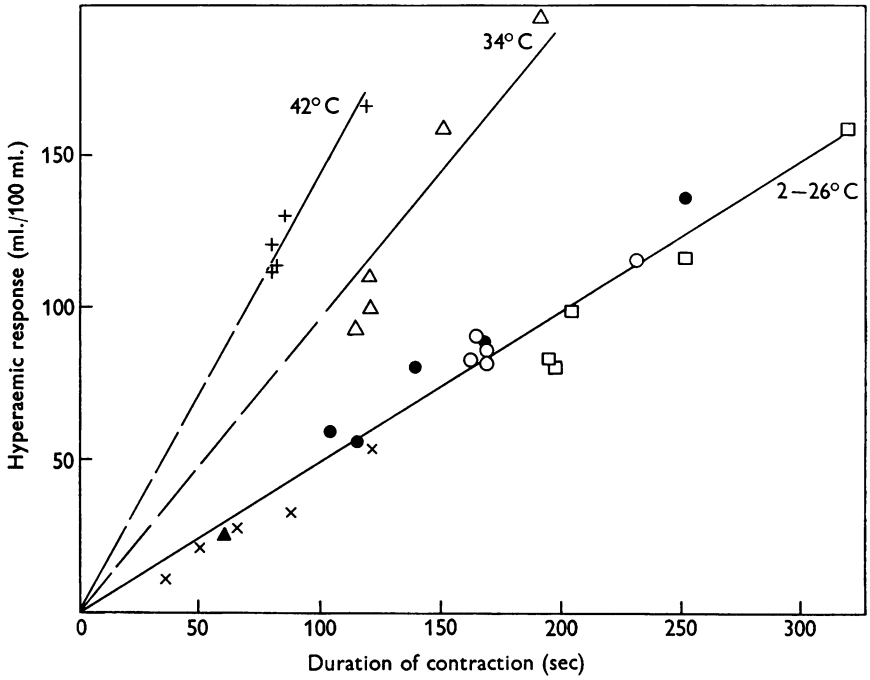

Fig. 8. The mean hyperaemic responses for four subjects after sustained contractions in water at seven different temperatures: water temperatures were (° C) 2 ▲, 10 ×, 14 ●, 18 □, 26 ○, 34 △ and 42 +.

Blood-flow measurements

The mean post-exercise hyperaemic responses for the four subjects at different water temperatures are shown in Fig. 8. The results were similar for each subject with exceptions which are noted below. There appeared to be an approximately linear relationship between the duration of sustained contraction and the hyperaemic response at each temperature of the bath. All the results at water temperatures from 2 to 26° C have been reasonably well fitted by a single line. Those obtained from water at 34° C lie along a separate and steeper line, while at 42° C this difference was even more marked. These findings indicate that at bath temperatures above 26° C the hyperaemic response for a given duration of contraction increases with temperature. One subject failed to show this increase at 34° C, and one other subject produced

similar hyperaemic responses in water at 34 and 42° C which were higher than at lower bath temperatures.

Blood-flow records were obtained on only three of the four subjects during the progress of contractions. The changes in blood flow in excess of the preceding resting level are plotted in Fig. 9. All the subjects showed a slight

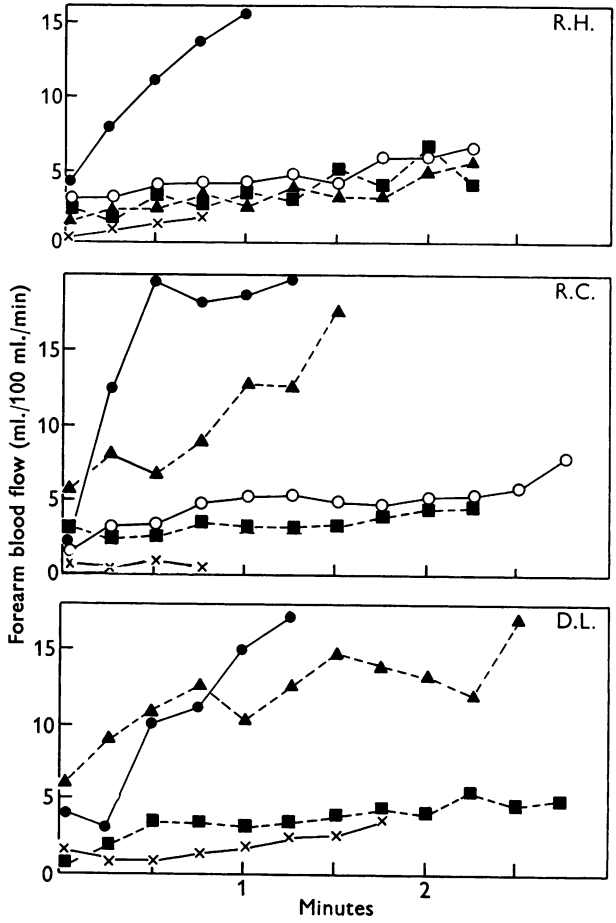

Fig. 9. Forearm blood flow measured during the performance of sustained contractions in three subjects when their forearms were immersed in water at different temperatures (records are missing for subject D.L. in water at 26° C). Water temperatures were (° C) 14 ×, 18 ■, 26 ○, 34 ▲ and 42 ●.

increase in flow on starting the contraction, but no pronounced change as the contraction proceeded in water at temperatures from 14 to 26° C. Two of the subjects showed an increase in blood flow during the contraction in water at 34° C, and all three showed an increase at 42° C. Although not so clear-cut,

these results obtained in the course of the contractions appeared to be affected by temperature in much the same way as were the post-exercise hyperaemias (Fig. 8).

Electromyographic recordings

If the reduction of both maximal tension and duration of sustained contractions at muscle temperatures lower than 27° C is due to the failure of some of the more superficial fibres to contract owing to cooling, this ought to be readily demonstrable by electromyography. A reduction in the number of

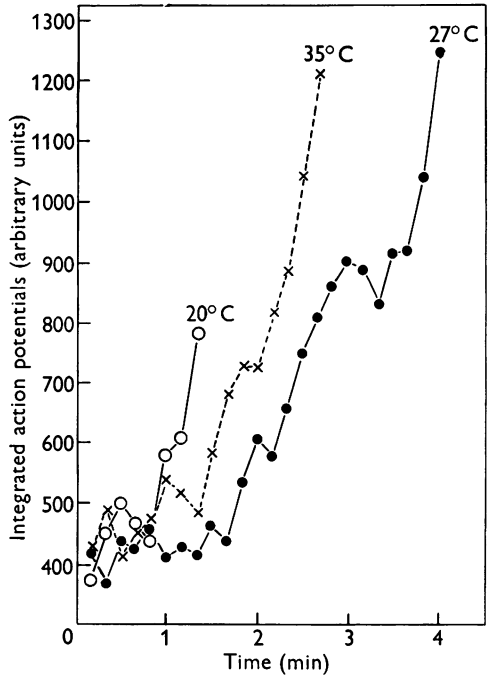

Fig. 10. Typical integrated muscle action potentials recorded throughout sustained contractions at three muscle temperatures.

active fibres ought then to provide a smaller integrated action potential count at the termination of the contraction than would be found at muscle temperatures of 27° C and above. Fig. 10 shows typical results obtained from one subject, with muscle temperatures of 35, 27 and 20° C. The integrated action potentials when the muscle temperature was 27° C rose slightly during the first half of the contraction, after which they increased rapidly to a maximum of about three times the initial value at the termination of the contraction. This pattern is similar to that described by Edwards & Lippold (1956) in sustained contractions of leg muscles.

When the muscle temperature was 35° C the action potential curve was similar to that found at 27° C except that the increase of electrical activity

began earlier. But when the muscle temperature was 20° C the integrated count increased soon after the start of the contraction and rose at the end of the contraction to a peak of activity only twice that of the initial value, only half way between the difference of initial and final activity with muscle temperatures of 27 and 35° C.

DISCUSSION

The results from these experiments show that after the arm has been immersed in water for 30 min the optimal water temperature for maintaining sustained contractions is 18° C. This corresponds to a muscle temperature of 25–29° C, some 6–10° C lower than that found in the normal, resting, clothed forearm (Barcroft & Edholm, 1946). With muscle temperatures higher or lower than this critical range, the duration of the sustained contractions is shorter. Before discussing possible reasons for this effect of temperature on the duration of sustained contractions, some other features of the results require consideration.

Although the muscle temperatures shown in Fig. 4 were taken midway between the skin and the centre of the forearm, they ought not to be considered as average forearm muscle temperatures. Fig. 5 shows that a large vertical temperature gradient exists through the arm, and the temperature of the deeper tissues will vary according to the thickness of the arm at any given point along the forearm. It may well be that a gradient also exists along the longitudinal axis of the arm. In terms of function of muscle fibres it cannot be assumed that the activity of cooler parts of the muscle balances that of the warmer parts, a point which is discussed below.

In calculating the extent of the post-exercise hyperaemia, it has been assumed that little or no increase in skin blood flow occurs during or after the exercise and that the hyperaemic response is confined to the vascular bed of the muscles. Evidence that this is so with limbs at, or below, normal temperature has been provided by Grant & Pearson (1938), Cooper, Randall & Hertzman (1951) and Coles & Cooper (1957); there is no comparable evidence on the hyperaemic response in water at 42° C, but the very slight increase in muscle temperature during the contraction and the fall in temperature during the post-exercise hyperaemia make it improbable that the skin circulation is increased by the conduction of heat from the underlying muscles.

The blood flow through the forearm was not decreased during the course of a contraction; on the contrary, in water at 34 and 42° C there was generally a slow increase of blood flow. This agrees with Grant's (1938) findings that there is a slight increase in blood flow during a 'strong hand grasp'. Barcroft & Millen (1939) investigated blood flow during sustained contractions of the calf muscles and concluded that muscle blood flow was suppressed at a tension between 10 and 20% of the maximum. This is a considerably smaller percentage than the present tension of 33% of the maximum, where a hyperaemia existed. Differences in muscle size and leverage may account for some of this

discrepancy, but probably the greatest part of it is due to different methods of establishing maximum tension.

Our results do not support the hypothesis postulated by Nukada & Müller (1955) who suggested that heating the arm diverted blood from the muscle to the skin, thus placing the muscle at a disadvantage in a subsequent contraction. Table 1 shows that when the arm is transferred from cold to hot water and vice versa the blood flow through the arm is rapidly affected. These changes almost certainly occur largely in the skin vessels. The duration of sustained contractions, observed when the muscle and subcutaneous temperatures (and presumably, blood flows) were thus dissociated from their normal relationship, strongly suggests that it is the muscle temperature alone which is associated with the duration of the sustained contraction and that the temperature of the subcutaneous tissue or of the skin or the blood flow through the skin vessels plays little or no part in determining the duration of sustained contractions.

Merton (1954*a*) has shown that the muscular fatigue in man, produced by a sustained contraction, is not associated with the failure of the neuromuscular junction. He found that action potentials over the muscle did not diminish during fatigue and he concluded that the fatigue occurred solely in the muscle. Naess & Storm-Mathisen (1955), who performed similar experiments, agreed with Merton that fatigue was entirely peripheral, but they found a reduction of the action potentials over the muscle and concluded that in some subjects at least the neuromuscular junction may be the limiting factor in fatigue. In the present experiments, where only local temperatures in the forearm were altered, the effects of different muscle temperatures on the duration of sustained contractions must also be peripheral. Presumably muscular fatigue may be induced by (1) accumulation of one or more metabolites in the muscle substance, (2) failure to renew at an adequate rate the materials necessary for contraction, (3) failure of the neuromuscular junction, or by a combination of these possibilities.

It is generally assumed that although the metabolic factors cause the hyperaemia following muscular exercise, the causative agent is, as yet, unknown (cf. Hilton, 1953). Local anoxia is unlikely to be the hyperaemic agent (Love, 1955). Clearly the metabolites must accumulate during the contraction, to be removed during the subsequent period of hyperaemia. The work of Dornhorst & Whelan (1953) and of Patterson & Shepherd (1954) suggests that the substance or substances concerned is only removed slowly, at a rate which is unaffected by blood flow when the latter is above a certain low level. For the purposes of this discussion the assumption is made that the metabolite accumulating during a contraction to cause the ensuing hyperaemia is the same as, or closely associated with, the substance which accumulates and is responsible for the failure of the muscles at the end of the contraction. It would appear then

from the results in Figs. 8 and 9 that the rate of accumulation of metabolites is increased with bath temperature where this is above 18° C, so that the duration of the contraction becomes shorter and the subsequent hyperaemic response becomes greater for a given duration of contraction. The effect of temperature on the hyperaemia is not linear, and this is shown more clearly in Fig. 11 where the hyperaemic responses at muscle temperatures of 27° C and above are plotted after contractions of 90 sec duration. The hyperaemic response increases rapidly above a muscle temperature of about 30° C.

It is clear from Figs. 2 and 7 that the fall in duration of sustained contractions is not associated with reduction in the ability of the muscles to exert maximum tension when the temperature is 27° C or more. On this evidence,

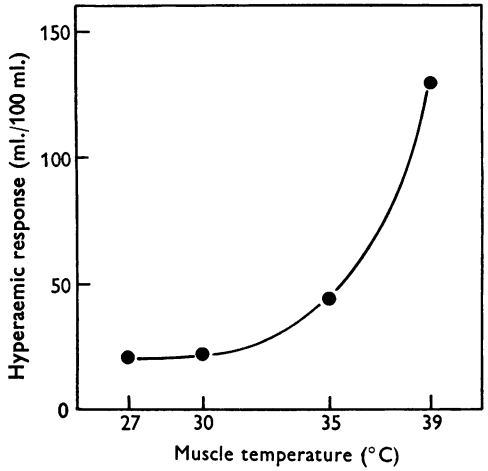

Fig. 11. The post-exercise hyperaemia measured at muscle temperatures from 27 to 39° C after contractions of 90 sec duration. Data taken from Fig. 8.

together with the increase of hyperaemic responses with increasing temperature of the water it may be postulated that the reduction in the duration of contractions with increasing bath temperature above 18° C is due to a more rapid accumulation of metabolites as the temperature increases. This agrees with the findings of Abramson, Kahn, Tuck, Turman, Rejal & Fleischer (1957) who found a higher rate of metabolism, with rising temperature, in human muscles.

There is no reason to believe that there is any other basic reason for fatigue at lower temperatures. If the same metabolic agent is responsible for fatigue at lower muscle temperatures then it would be expected that as the muscle temperature is reduced below 27° C, the duration of the sustained contractions would not be lowered. But since the duration of contractions falls abruptly with muscle temperatures lower than 27° C, we must postulate the existence of some other factor responsible for this fall in duration.

The fall in duration of sustained contractions with water temperatures below 18° C is paralleled by the fall in maximum tension which the muscle can exert (Figs. 2 and 7). Metabolic factors are unlikely to play any part in the differences observed in maximum contractions. This suggested that with cooling there might be a failure of the transmission of nerve impulses, or of the neuromuscular junction or even of the muscle substance itself.

The effect of low temperature on nervous transmission is complex (e.g. Whitteridge, 1948; Brooks, Koizumi & Malcolm, 1955; Douglas & Malcolm, 1955), but the balance of evidence suggests that the rate of conduction of impulses is slowed and the size of the action potential is reduced at temperatures below about 20° C. Nerve block appears to occur at temperatures of 10° C and below. The muscle temperatures shown in Fig. 3 are only representative temperatures for the whole muscle and Fig. 4 shows that there is a considerable temperature gradient through the muscle at cooler water temperatures, the areas nearer the skin falling several degrees lower than the representative temperatures. It is clear that a considerable proportion of the muscle was at a temperature lower than 20° C when in water at 14, 10 and 2° C. Greater or lesser degrees of modification of the nervous transmission would be present in such conditions, which might account for the fall in duration of sustained contractions.

The integrated potentials recorded showed that at muscle temperatures of 27 and 35° C the electrical activity at the end of the contraction was similar, but with muscle temperature of 20° C the activity at the end of the contraction was much lower. This supports the belief that some fibres are inactive in cooler parts of the muscle at such temperatures.

The production of acetylcholine in ganglia is affected both by the rate of stimulation and temperature (Brown, 1954; Kostial & Vouk, 1956). Cooling below 20° C causes a large reduction in the production of acetylcholine. If a similar reduction occurred at the motor end-plate, a reduction in the duration of sustained contractions would be expected.

Cooling of nerves also results in asynchrony of impulses, since the rate of conduction along cooled nerves varies according to the size of the fibres. While Merton (1954*b*) has shown that asynchrony of motor impulses leads to a reduction in the size of a muscle twitch, he has also shown that rapid tetanus of 'desynchronised volleys' does not differ, except initially, from one made up of synchronous volleys. Thus asynchrony of nerve volleys due to cooling is unlikely to explain the fall in duration of contraction.

There is no reason to suppose that cooling affects the ability of the muscle itself to contract. The evidence of Weber & Portzhel (1954) is that mammalian muscle retains its ability to contract to full tension, or nearly so, when it is cooled to temperatures well below those described here.

The most reasonable interpretation of our results is that muscular fatigue

is caused basically by a limiting accumulation of metabolites. The rate of production of metabolites is reduced with lower temperatures, so that fatigue is induced more slowly as the temperature falls. This causes a marked increase of the duration of 'sustained contractions to fatigue' from muscle temperatures of 39–27° C. At muscle temperatures below 25° C we postulate that an increasing number of muscle fibres, from the outer surface inwards, become inoperative owing to interference with nervous or neuromuscular transmission. Thus the tension required to be maintained by any subject in such conditions would have to be made with fewer active units, so that fatigue would occur more quickly. Also, the maximum tension which the muscles could exert would be reduced with cooling of the muscle. Such a concept readily explains the results obtained.

SUMMARY

1. The duration of submaximal contractions held to the point of fatigue was measured for four subjects after they had immersed their forearms for 30 min in water at temperatures between 2 and 42° C.

2. Contractions were longest after the arm had been immersed in water at 18° C, above and below which temperature of the water the duration was shorter. Immersion of the forearm in water at 18° C for 30 min resulted in a muscle temperature of about 27° C measured half way between the skin and the centre of the forearm.

3. The maximum tension that could be exerted after immersion of the forearm for 30 min was the same as the initial value in water at 18° C or more; in water at temperatures below 18° C the tension that could be exerted fell sharply with falling bath (and muscle) temperature, to a value of some 40% (of the maximum) in water at 2° C.

4. The post-exercise hyperaemic response was greater for a given duration of contraction in water at 34 and 42° C than at lower temperatures. The rate of blood flow through the muscle during contractions also increased by a greater amount when the water temperature was higher.

5. Integrated muscle action potentials showed that at the termination of the contraction there was no difference in electrical activity over the muscle when its temperature was 27 or 35° C; when it was reduced to 20° C there was considerably less electrical activity at the end of the contraction.

6. The implications of these results have been discussed. It is considered likely that as muscle temperature increases above 27° C the rate of metabolism increases and results in the earlier accumulation of metabolites so as to cause fatigue; at muscle temperatures below 27° C a proportion of the more superficial muscle fibres do not contract, as a result of interference in nervous or neuromuscular transmission due to cooling.

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