THE INFLUENCE OF EXERCISE ON THE INORGANIC PHOSPHATES OF THE BLOOD AND URINE.

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It seems very probable from the work of Embden and his co-workers that a hexosephosphate, "Lactacidogen" (1), plays an important part in muscle contraction as the precursor of lactic acid. It became interesting therefore to determine how far the inorganic phosphate of the blood was affected by the changes taking place in muscle.

The changes in lactic acid content following exercise have been studied directly and indirectly by several workers (2). Hitherto the inorganic phosphate has been noted only in a few observations for Embden and Grafe (3) by Teigle and in a single experiment by Haldane, Wigglesworth and Woodrow (4). In both of these a rise was noted after exercise. It was thought desirable that these isolated observations should be confirmed and extended.

Methods. In most of the experiments the exercise took the form of the subject running vigorously up and down the laboratory stairs, 40 ft. in height, until he was exhausted. The exercise usually lasted four or five minutes and involved five or six ascents. All experiments were done in the morning. Samples of blood were taken at various times during the experiment and the inorganic phosphate content of the whole blood estimated by Briggs' method (5).

As Barr and Himwick (2) had found a difference in the lactic acid content of arterial and venous blood, several determinations of inorganic phosphate were done in Exp. 2 on venous and finger blood. The latter was taken as being equivalent to arterial blood (3, p. 108). Blood was drawn simultaneously from the median basilic vein and from the finger at a stage of the experiment when the phosphate content of the blood was falling rapidly. The values obtained were within 2 p.c., the limits of experimental error. It was therefore concluded that the analysis of finger blood was as reliable a way as any of determining the phosphate content of the blood as a whole, and this source only was used in the rest of the experiments.

Blood was drawn into a crucible containing 1·2 mg. of sodium oxalate. A little over 1 c.c. was collected and two estimations were done, using 0·5 c.c. of blood for each. This slight modification of Briggs' method worked very well and enabled us, when necessary, to take a large number of samples at short intervals. Over 200 estimations were done in duplicate with an average error for a single observation of about 1 p.c. from the mean. In the later experiments samples of urine were taken at frequent intervals during the experiments and analysed for inorganic phosphate, ammonia, chlorides, and in some cases for other constituents. The phosphate was estimated by Briggs' method(5). The ammonia by a vacuum distillation method described by Stanford(6). The chlorides by Volhard's method. Urea was estimated by Marshall's method(7) and sulphates gravimetrically. A copious diuresis—500–1500 c.c. per hour—was produced by drinking large quantities of water. In this way it was possible to obtain samples at such short intervals as ten minutes, and by analysis to follow the rate of excretion of various constituents. It was demonstrated by Wigglesworth and Woodrow(8), and is our experience also, that this rate in the case of inorganic phosphate is quite independent of the extent of the water rate, provided the latter is above a certain minimal value (about 200 c.c. per hour). We found the same to be true of ammonia, but only approximately true for the other constituents investigated. It will be seen, however, that the variations in the rate of excretion due to the water rate were always insignificant compared to the variations under investigation. The behaviour of phosphates during water diuresis will be dealt with more fully in a subsequent paper.

Inorganic phosphate of blood.

The typical behaviour of the blood phosphate curve in these experiments may be seen in Fig. 1. A small rise took place during and immediately following the exercise reaching a maximum, probably 3–4 minutes after the running had ceased. This was followed immediately by a rapid fall well below normal which attained its maximum value in about three quarters of an hour. A slow recovery to normal followed taking two to three hours for completion.

The typical behaviour of the rate of phosphate excretion may also be seen in Fig. 1. A sudden great increase, *after* the period of exercise is followed by a certain degree of suppression.

We publish in full the protocol of this experiment No. 8 selected from a group of fifteen experiments on ten different subjects. None of the

subjects had been taking regular vigorous exercise for some weeks before the experiment and they may be called "untrained." The results of

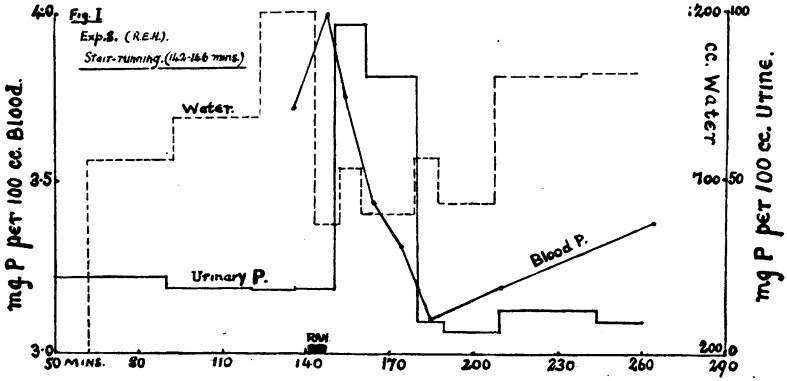

Fig. 1.

these experiments are summarised in Table I. The "short exercise" was the vigorous stair running already described. A sample of blood was taken from one to five minutes after the cessation of exercise and the increase of the inorganic phosphate content of this sample above the "normal" value is expressed as a percentage in column "Rise per cent.,"

Exp. 8. "Stair running" R.E.H.

Inorganic phosphate in blood. Mg. P per 100 c.c.

Time	*a*	*b*	Mean
135 mins.	3·72	3·72	3·72
142 „	Stair running for four minutes		
147 „	4·10	3·94	4·02
155 „	3·74	3·75	3·75
165 „	3·46	3·42	3·44
175 „	3·36	3·23	3·30
185 „	3·08	—	3·08
210 „	3·28	3·10	3·19
267 „	3·37	3·39	3·38

Inorganic phosphate in urine

Time of collection	Vol. c.c.	Mg. P per 100 c.c.	Rate P per hour mg.	Rate H_2O per hour c.c.
55 mins.	175	10	21·0	210
90 „	270	2·73	21·1	772
120 „	450	2·18	19·6	900
135 „	293	1·64	19·3	1172
150 „	148	3·22	19·3	592
160 „	122	13·23	96·0	732
180 „	204	13·45	82·3	612
190 „	131	1·24	9·7	786
209 ,	314	1·01	6·7	660
242 „	564	1·02	10·5	1025
262 „	345	·74	7·7	1035

Table I. The "normal" value was obtained by taking the mean of values determined on two samples of blood drawn shortly before the exercise, usually at about twenty minute intervals. In the earlier experiments these two values sometimes varied considerably, the second normally being nearly always lower than the first. The changes due to exercise were always larger than these variations, and in later experiments the difficulty was overcome by the subject's sitting still for at

TABLE I. Short exercise by untrained subjects.

Exp.	Subject	Rise per cent.	Fall per cent.	Remarks
1.	J.B.S.H.	16	34·5	
2.	R.E.H.	5	24	
3.	G.A.R.	9	29	
4.	R.E.H.	7	10	Two successive runs with 2 hours interval
		2	19	
5.	J.T.E.	11	22	
6.	D.B.	8	22	
7.	A.C.	12·5	18	
8.	R.E.H.	8·1	17·2	
9.	J.B.S.H.	22·7	22	
10.	J.J.	11·3	30·8	
Average for men... ...		10·2	22·6	
11.	M.M.	10	26	Exps. on three women
12.	B.E.H.	11	9	Exercise not very vigorous
13.	B.E.H.	4	13	Exercise more vigorous
14.	C.E.L.	17	2	„ very „
15.	C.E.L.	6	2	„ „ „
Average for men and women		10	18·8	

TABLE II.

Exp.	Subject	Exercise	Excess P in urine mg.	PO_4 per cent.	NH_3 per cent.	Cl per cent.	Change in *p*H
7.	A.C.	Short	43	604	—	—	—
8.	R.E.H.	„	34	437	—	—	—
9.	J.B.S.H.	„	45	360	221	26·8	—
10.	J.J.	„	40	330	216	26·6	—
17.	R.E.H.	Ran 15′	24·4	340	—	—	—
18.	R.E.H.	„ 20′	47	628	—	—	—
23.	G.B.	„ 20′	23·4	307	—	—	—
		Trained					
27.	H.K.B.O.	Short	25·6	277	155	54	7·3–6·2
28.	E.H.F.	„	39·5	300	113	7·6	6·8–5·1
30.	R.H.B.	„	26	455	90	56	6·4–5·0

A few determinations on sulphates and urea showed no very marked changes from normal.

least an hour before the commencement of the experiment. This is a precaution it would be well to take in all further work on blood phosphates. The percentage fall is calculated in a similar way from the

normal value and the lowest value obtained in any experiment. There is no guarantee that samples of blood were obtained exactly at the maxima and minima of the phosphate concentration curve. The later sampling was planned, however, from knowledge of the general form of the phosphate curve derived from earlier experiments, and it is unlikely that any large errors have been introduced.

The figures for all the urine determinations are summarised in Table II, and those for one experiment given in full in Table III. In order to make the results more comparable the amount of each constituent excreted during the half hour immediately following the exercise has been calculated and expressed as a percentage of the amount excreted during a normal half hour. Occasionally samples could not be obtained exactly at the time desired. For instance no sample of urine could be obtained from H.K.B.O. for 7 minutes after running. The urine secreted during these 7 minutes was therefore mixed in the bladder with urine secreted during the 14 minutes before and during his exercise. In estimating the amount excreted during these 7 minutes it has been assumed that the rate of excretion was constant over the whole period. This is certainly an untrue assumption and the error thus introduced tends to diminish the figure representing the change in rate of excretion, but in most cases probably by no very great amount.

As a result of the increased rate of phosphate excretion a certain amount of phosphate has been lost by the body during the three quarters of an hour subsequent to the exercise over and above the amount normally excreted. Because of the bearing this has upon the fall in blood phosphate the amount so lost has been calculated and expressed in milligrams of phosphorus in Table II ("Excess P in urine").

Before discussing possible causes for the rise in blood phosphate we must consider eight experiments done on more prolonged exercise. The results are summarised in Table IV. The exercise took the form of running on the flat for 15 minutes or longer. In Exp. 22 the subject was a member of a crew rowing a full course on the Cam. In Exps. 20 and 21 samples were taken every seven minutes during half an hour's running. The results seem to indicate a rise during the first 10 or 15 minutes after which the value oscillates irregularly about the higher level for the rest of the run, and then falls rapidly on ceasing the exercise. The rises were, on the average, considerably higher than for the short exercise, giving an average of 25 p.c. against 10 p.c., and taking 10 or 15 minutes to reach equilibrium.

Now the workers on lactic acid in the blood have observed that it is

TABLE III. Exp. 27. H.K.B.O. Ran stairs 9 times 109–116 mins.

Time of collection mins.	Vol.	*p*H	Water rate	% P Mg/100 c.c.	Rate P Mg/hr.	Rate NH_3 MgN/hr.	Rate Cl Mille Mols. per hr.	Rate urea grams per hr.	Rate sulphate Mg per hr.
31	202	7·2	391	2·7	10·6	27·5	21·7	1·66	—
57	524	7·4	1210	0·733	8·9	28·3	26·0	1·09	94·6
81	518	7·2	1270	0·840	10·6	31·3	29·2	1·21	96·6
102	225	6·9	643	3·28	21·1	25·5	12·9	0·77	58·2
112	160	6·2	960	9·68	93·0	50·0	7·7·	1·05	100·8
125	221	6·3	1020	2·96	30·2	40·0	14·7	1·03	110·9
140	313	6·8	1252	1·15	13·8	44·4	20·0	1·18	117·1
158	398	7·2	1324	0·833	11·0	27·4	25·8	1·07	100·2
177	430	7·0	1357	0·857	11·6	25·9	30·5	0·92	122·8
200	495	6·9	1290	1·075	13·9	—	31·8	1·06	—
210	222	6·8	1330	1·36	18·1	28·5	31·9	1·08	—

TABLE IV.

Exp.	Subject	State	Exercise	Rise per cent.	Fall per cent.
16.	J.B.S.H.	Untrained	Ran 13′	23	17
17.	R.E.H.	,,	,, 15′	29·6	16·5
18.	R.E.H.	,,	,, 20′	35	13·8
19.	G.A.R.	,,	,, 8′	17	18
20.	R.E.H.	,,	,, 50′	15	6
21.	R.E.H.	,,	,, 35′	13	10
22.	G.B.	Trained	,, 15″	27	0·3
23.	G.B.	Rower	,, 20″	39	7
			Mean	24·8	11·1

TABLE V. Short exercise by trained subjects.

Exp.	Subject	Rise per cent.	Fall per cent.	Remarks
24.	G.B.	5	13·5	A rowing man
25.	R.E.H.	7	15	Partly trained
26.	W.E.T.	Lost	12	Rugby "Blue"
27.	H.K.B.O.	41·5	0	Running "Blue"
28.	E.H.F.	10·5	14	Sprinter
29.	W.G.O.	5·3	8	Rowing man
30.	R.H.B.	12·3	2·8	Running
	Mean	11·2	9·3	

after short vigorous exercise that most is liberated (Haldane and Quastel, and Hill, Long and Lupton). The lag of 10 to 15 minutes observed in the production of phosphate would seem to indicate that phosphate ions, if set free within the muscle cell, diffuse out comparatively slowly, taking 10 or 15 minutes to reach equilibrium. Moreover, the increase of phosphate, we observed, is not nearly molecularly equivalent to the increased lactic acid observed by Hill, Long and Lupton in similar forms of exercise(2). Our average figure for short exercise is an increase of 0·3 mg. P per 100 c.c. Hill, Long and Lupton observed, in several experiments on "standing running" for 5 to 10 minutes, an increase on the average of about 80 mg. of lactic acid per 100 c.c.

and the molecular proportions are one ion of phosphate to about one hundred lactic ions. This great discrepancy cannot be entirely due to the slower diffusion of the phosphate as even when equilibrium is obtained the rise is never more than 1·5 mg. of phosphorus per 100 c.c. A probable explanation is furnished by the work of Embden and Lawaczeck(9) who state that after contraction of an isolated frog's muscle, a large part of the inorganic phosphate set free was recombined with glycogen to form more lactacidogen. A similar process assumed in intact human muscles would account for the comparatively small liberation of phosphoric acid observed.

We thought that the rise in blood phosphate might be due to the acidosis following the exercise(4). In order to test this as far as possible several experiments were done on the effect of a short period of CO_2 acidosis. In two experiments on J.B.S.H. and R.E.H., lasting 20 and 30 minutes respectively, a rise of 10 p.c. was observed in the blood phosphate. In two shorter experiments on the same subjects no significant increase was observed. It is obvious that the rise of phosphate produced by CO_2 acidosis is a much slower process than that produced by exercise, which appears immediately and seems to reach equilibrium in about a quarter of an hour. 6–8 per cent. CO_2 was breathed in these cases.

It remains possible that the intense acidosis produced locally in the muscles may play a considerable part in the raising of blood phosphate, but the probability is that the major part is produced by the breakdown of lactacidogen.

The subsequent sudden fall below normal is more difficult to explain. The most natural place to look for an explanation is in the direction of the sudden increased phosphate excretion observed immediately after exercise. In Exp. 9, as much as 45 milligrams of phosphorus in excess of the normal excretion was lost by the body within three quarters of an hour after running, and at first sight it seems probable that this might account for the sudden drop in blood phosphate. There are, however, one or two considerations that make it unlikely that this is the sole cause.

In the first place the amount excreted is hardly sufficient to account for the drop observed. In Exp. 9 for instance, even if we assume that all the excreted phosphate comes from the blood and none from the lymph and tissues, the 45 mg. excreted would not account for the drop observed, a drop in this experiment from a maximum of 4·6 mg. P per 100 c.c. blood to a minimum of 2·9 mg. That is to say, the blood lost 1·7 mg. P per 100 c.c. and assuming that J.B.S.H. has only 4 litres of blood—

a very moderate estimate as he weighs 100 kgrs.—then he has lost from his blood alone 68 mg. of phosphorus. Of this 68 mg. only 45 mg. or about two-thirds appeared in the urine. In this case then it is quite impossible that the kidneys are responsible for the whole of the fall in blood phosphate. In many other cases it becomes possible only by assuming that all the phosphate excreted comes from the blood which does not in turn draw any from the lymph and tissues. That this is very improbable is shown especially by experiments on CO_2 acidosis. In an experiment in which J.B.S.H. remained in the chamber for an hour, it was not until over an hour after the subject had left the chamber that his blood phosphate began to fall. In no case of CO_2 acidosis did it ever fall below normal. Yet during this period the body was losing large quantities of phosphate through the kidneys, producing only a comparatively slow reduction in blood phosphate (Fig. 2). This goes on

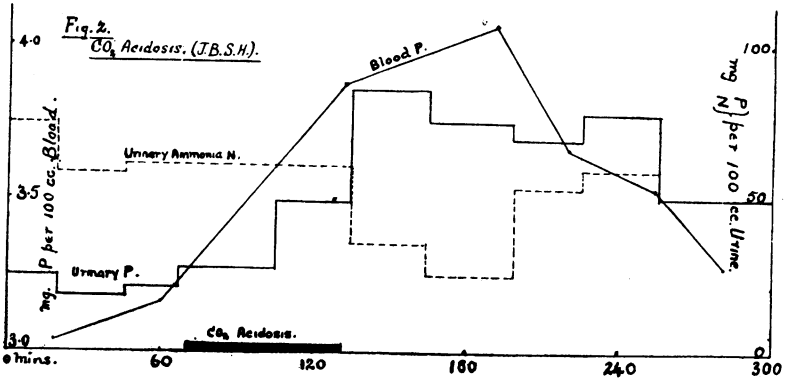

Fig. 2.

for some hours after the acidosis has come to an end, showing that there is some reserve the blood can call upon to replenish that lost through the kidneys.

Since then the changes in the blood after exercise cannot be entirely due to the kidney, what other causes may there be? A possible explanation appears when we consider the rapid re-synthesis of lactacidogen from inorganic phosphate and glycogen noted by Embden in many of his experiments. So marked is this phenomenon that it was only when he had seriously depleted the glycogen stores of his animals by starvation and strychnine poisoning that he was able to demonstrate any great lowering of lactacidogen in the muscles after exercise. If a similar process of recovery takes place in man for some time after a sudden

short burst of vigorous exercise the sudden call for inorganic phosphate may well play a considerable part in lowering the blood phosphate.

Further support to the view that the muscles are directly concerned with these changes is afforded by the fact that there is a marked difference in the phosphate curves given by trained and untrained men. The results of seven experiments on seven different subjects are summarised in Table V. All had been taking regular vigorous exercise of various forms to within a few days of the experiment. It will be seen that in no case was the fall below 15 p.c. and the average was only 9·3 p.c. contrasted with the average fall of 22·6 p.c. for the seven untrained men. The rise on the other hand is substantially the same for both groups, viz., 11·2 p.c. for trained as against 10·2 p.c. for untrained. A point that possibly has some significance is that the rower or the Rugby player gave falls of 13·5 p.c. and 12 p.c. respectively, while the four runners of the University Athletic Club, whose leg muscles were presumably even better trained, gave an average fall of only 6·2 p.c. In attempting to explain the cause of this difference one should note that on the average the trained man excreted less phosphate than the untrained (see Table II), but for reasons specified above it is unlikely that this is responsible for more than a small part of the difference observed. It may be said also that in the trained man the equilibrium is upset less for the same amount of exercise than in the untrained. There was, however, no apparent connection between states of exhaustion and fall in phosphate. In Exp. 10 for instance I.J. was comparatively fresh after the exericse yet gave a fall of over 30 p.c. In Exp. 30 R.H.B. was as exhausted and distressed as any of the untrained subjects yet only gave a fall of 2·8 p.c.

It is then difficult to avoid the conclusion that the untrained muscle absorbs more phosphate from the blood after exercise than the trained muscle. In this connection some experiments by Embden and his co-workers are of interest. Experiments carried out by them on the red and white muscles in rabbits, and on the muscles of summer and winter frogs, all show an increased lactacidogen content in the more active muscle. Moreover, the transition from the low lactacidogen content of the winter state to the high lactacidogen content of the summer state can be brought about very rapidly by bringing frogs from a cold to a warm temperature for a few days (11). It is possible therefore that during the progressively increasing activity of a muscle, brought about by training, the lactacidogen content of the muscle is increased; and if some increase takes place immediately after each burst of exercise by

an untrained muscle, then this would account for the greater demand for phosphate by such a muscle. A well-trained muscle presumably already contains a maximum amount of lactacidogen. But it is difficult to account for the anomalous results given by the three women subjects (Table I). One especially, C.E.L., although she was very exhausted never gave any sign of a fall below normal. The exercise was of the same nature as in the other experiments and was as vigorously carried out. We can only suppose that in this case the re-synthesis mechanism is either absent or works so much slower than normally that it produces no appreciable fall in blood phosphate.

On the whole, however, it seems probable that re-synthesis of lactacidogen plays a conspicuous part in causing the sudden fall below normal observed in the majority of experiments, and that the resulting absorption of phosphate seems to be more extensive in the untrained muscle than the trained.

Phosphate excretion.

Embden and Grafe(3) noted an increase in phosphate excretion after prolonged strenuous exercise by well-trained men, and attributed the negative results obtained by former workers to insufficient training and work. As they considered the phosphate output of a "working" day in contrast to a "resting" day it seems probable that the phenomenon they observed is distinct from the one noted by us. Hartmann(11) has also studied the effect of exercise lasting an hour or two hours on the phosphate excretion, collecting the urine in two-hourly samples. He notes a slight inconstant rise in the sample immediately following the exercise, which is followed by a compensating fall in the subsequent sample. It is no doubt the relatively infrequent collection of samples by these workers that masked the great but fleeting increase of phosphate excretion that takes place for a few minutes after short vigorous exercise; for it is frequently followed by a compensating suppression after about an hour. The probable cause of this phenomenon may now be considered.

It is probable that the fleeting rise of the blood phosphate above normal caused a certain amount of increased excretion(8), but it is unlikely that this was the only cause. In the first place the rate of phosphate excretion showed no proportionality to the rise of blood phosphate. In one experiment (No. 7), a comparatively small rise of 12·5 p.c. in the blood phosphate was followed by an increase of over 500 p.c. in the phosphate excretion within the next half hour. More conclusive evidence that another factor is at work is given by Exp. 8,

of which the full protocol is given. It will be seen that during the secretion of the sample collected at 180 mins. the blood phosphate fell from about 3·50–3·15 mg. P p.c., and was all the time below the normal level 3·72. Yet the phosphate excretion during this period was at the rate of 82 mg. per hour—over four times the normal rate. Similar results have been obtained in other experiments. It may be suggested that this "lag" in the phosphate excretion is not real and is due only to contamination by concentrated urine left behind in the kidney, ureters and incompletely emptied bladder. It is very improbable that this introduces much error, because when, during a normal resting period, the concentration has changed rapidly owing to changes in water rate no corresponding change has been observed in the apparent rate of excretion.

A further cause presents itself on consideration of the work on the reduction in alkali reserve after exercise(2) from which it is found that after short vigorous exercise lactic acid was liberated in the blood to an extent sufficient to neutralise a quarter of the alkali reserve, and this did not entirely disappear for nearly an hour. During this period, therefore, there exists a condition of acidosis, practically entirely compensated, similar to that sometimes met with in diabetes. It is well known that in such cases the rate of phosphate and ammonia excretion goes up. Haldane, Hill and Luck, in their experiments on ammonium and calcium chloride ingestion observed the same effect in a compensated acidosis produced experimentally(12). If the rise of phosphate excretion was in any way due to this cause a similar rise in ammonia excretion would be expected, and, therefore, in several experiments the ammonia excretion was determined. It will be seen in Table II that in the case of two untrained subjects (Exps. 9 and 10) the ammonia excretion was more than doubled just after exercise. Among the trained subjects there was in one case (Exp. 27) an increase of 50 p.c. but in the two others no very significant change. It may be noted here that Dautrebande and Davies(13), in an experiment on exercise, noticed an increase in ammonia nitrogen in the urine collected after the exercise.

In Exps. 27, 28 and 30, *p*H determinations were done on the urine by Miss Watchorn, using Michaelis' method, and in each case a marked fall in *p*H was observed after exercise (Table II). These observations serve to support the idea that exercise produces a fleeting but profound reduction in the alkali reserve to which the kidneys react vigorously and rapidly by excreting phosphate at a low *p*H and in some cases by excreting more ammonia. The rapidity with which this fre-

quently takes place over short periods is remarkable. In 20 minutes A.C. (Exp. 7) excreted 33·1 mg. of phosphorus, a rate over eight times his normal.

In this connection should be mentioned two experiments on urine secreted during and after prolonged CO_2 acidosis by J.B.S.H. and R.E.H. In both cases the phosphate excretion went up to three or four times its normal rate. The ammonia, however, showed a slight tendency to drop and certainly no indication of a rise. This might be expected as the acidosis was not of a type that involves a great excess of anions which have to be removed (as when the blood is flooded with lactic ions). The increased phosphate excretion follows closely the blood phosphate curve and is probably in this case due to this factor alone (cf. Wigglesworth and Woodrow(9)). The results are not in agreement with those found by Davies, Haldane and Kennaway(14), who state that the ammonia excretion in two experiments on H.W.D. went up after CO_2 acidosis.

In view of these marked changes observed in the behaviour of the kidney after exercise it became interesting to investigate other constituents in the urine. Determinations were done in several experiments of chlorides, sulphates, and urea. Two experiments on urea gave 102 p.c. and 105 p.c. respectively of the normal rate. This leaves out of account the fall in rate observed in all cases *during* the actal running, which has been noted by many observers and put down to the effect of vasoconstriction(15). Only one complete experiment was done on sulphates, and this gave a value of 116 p.c. but no extreme changes in rate were noted in another incomplete experiment. On the other hand in the five experiments in which the chlorides were estimated they showed a profound suppression (Table II). At most they were only a little over half the normal value and in one case (28) only 7·6 p.c. of the normal. This cannot be due to the general suppression of kidney function due to exercise(15) as during the period from which these figures were calculated there was greatly increased phosphate and ammonia excretion and normal urea and sulphate excretion. It is improbable also that it is due to such causes as sweating(16) as the period of exercise is so short; or to some shift of chloride ions into the cells due to the acidosis, reducing the plasma chloride because although Dautrebande and Davies(13) confirmed the increase of chlorides in the cells they found no diminution of the plasma chlorides after short severe exercise. Embden and Grafe noted a fall in chloride excretion accompanying the increased phosphate excretion following prolonged exercise(3).

Figures by Dautrebande and Davies(13) also show the same effect and it has been noted by others that the excretion of phosphate and chloride show an inverse relation[1]. This alone seems hardly sufficient to explain the results obtained after exercise as during the CO_2 acidosis on R.E.H. there was no significant change in the chloride excretion, although the phosphate excretion was more than doubled. We feel the cause of the phenomenon must be left undecided until further work has been done.

Summary.

1. It is shown that after short vigorous unaccustomed exercise the inorganic phosphate in blood first rises a little above, then falls considerably below, the normal value in men.

2. In men in athletic training and taking exercise regularly this fall below normal is much less marked.

3. There is a sudden fleeting but very marked increase of inorganic phosphate excretion for about three quarters of an hour after short vigorous exercise, followed in some cases by suppression.

4. There is a less constant but occasionally very marked increase of ammonia excretion.

5. There is a constant very marked suppression of chloride excretion during the same period.

6. It is thought probable that the changes observed in blood phosphate are due mainly to changes in the lactacidogen content of the muscles. The changes observed in the urine seem to be due mainly to the fleeting acidosis caused by the lactic acid formed.

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