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Part VI. Distribution of Lactic Acid Between Plasma and Red Cells During Work and Recovery

by

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Abstract

Lactic acid concentrations in whole blood, plasma and red cells were determined on samples drawn from the femoral veins of men during 2 to 3-minute runs to exhaustion on the treadmill. Plasma lactate concentration increased rapidly during the runs, but the rise in whole blood was slowed by the delayed diffusion of lactate into the red cells during the first 1 to 2 minutes of work. In one man plasma lactate in femoral vein blood began to decline within 30 seconds after a 3 minute exhausting run, while lactate continued to diffuse into the red cells for 10 minutes following the run. In another man plasma and cell lactates continued to rise for 2 to 20 minutes respectively following a 2-minute run to exhaustion. This delayed diffusion of lactate into the red cells contributes to the continued increase in whole blood lactate following strenuous work.

The delay in the distribution of lactate in an exhausting run and recovery must involve not only circulatory distribution and diffusion into the less active tissues of the body, as previously shown (part V of this report), but also diffusion of lactate from the plasma into the red cells. Others (1, 4, 5) have investigated the diffusion of lactate into the red cells in vitro, but no thorough study of this process has been made in the intact man. Our purpose in making the present study was to approach the problem directly in the intact organism by determining plasma, whole blood and red cell lactate concentrations in blood flowing directly from the working muscles (femoral vein) of men during exhausting runs and recoveries.

METHOD

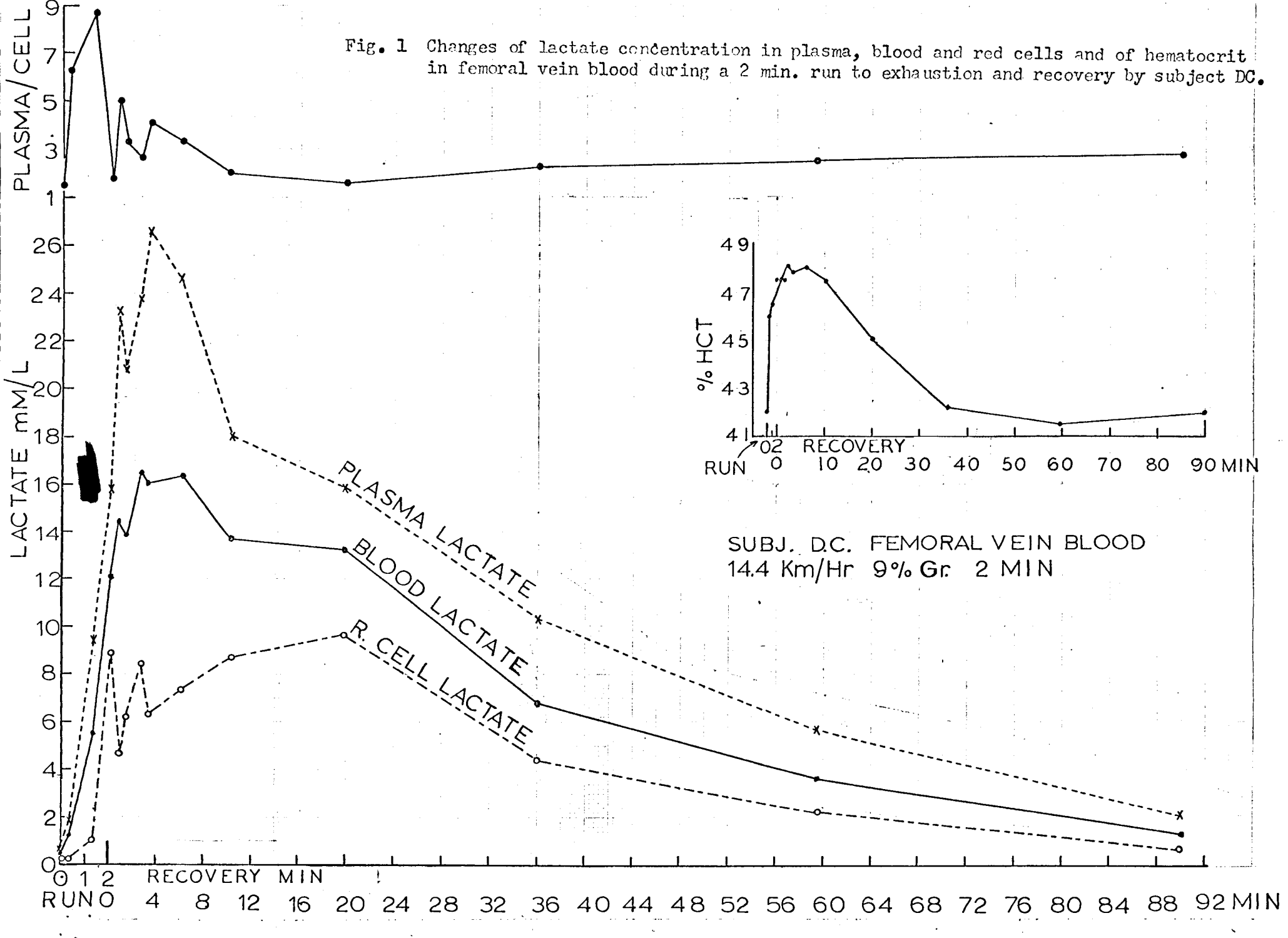
The sampling and analytical methods employed in these experiments were the same as those employed in the preceeding study (part V of this report). Blood was sampled from the femoral vein during exhausting treadmill work and recovery and immediately cooled in 3 ml. plastic centrifuge tubes in melting ice to prevent diffusion of lactate into or out of the cells (4). An aliquot was removed for determination of whole blood lactate and hematocrit. The remaining blood was then centrifuged for 5 minutes at 5000 rpm in a refrigerated centrifuge (1-3°C). Separated plasma was then precipitated in perchloric acid solution for the determination of plasma lactate. Hematocrits were determined by centrifuging blood samples in capillary tubes (2).

Red cell lactate (Lc) was calculated from the determined values of whole blood lactate (L_{W_b}), plasma lactate (Lp), and hemotacrit (Vc) by the formula of Johnson et al. (2): $Lc = \frac{L_{wb} - Lp\ (1-Vc)}{Vc}$

RESULTS

The results of experiments on subject DC and RT are given in Figs. 1 and

Fig. 1 Changes of lactate concentration in plasma, blood and red cells and of hematocrit in femoral vein blood during a 2 min. run to exhaustion and recovery by subject DC.



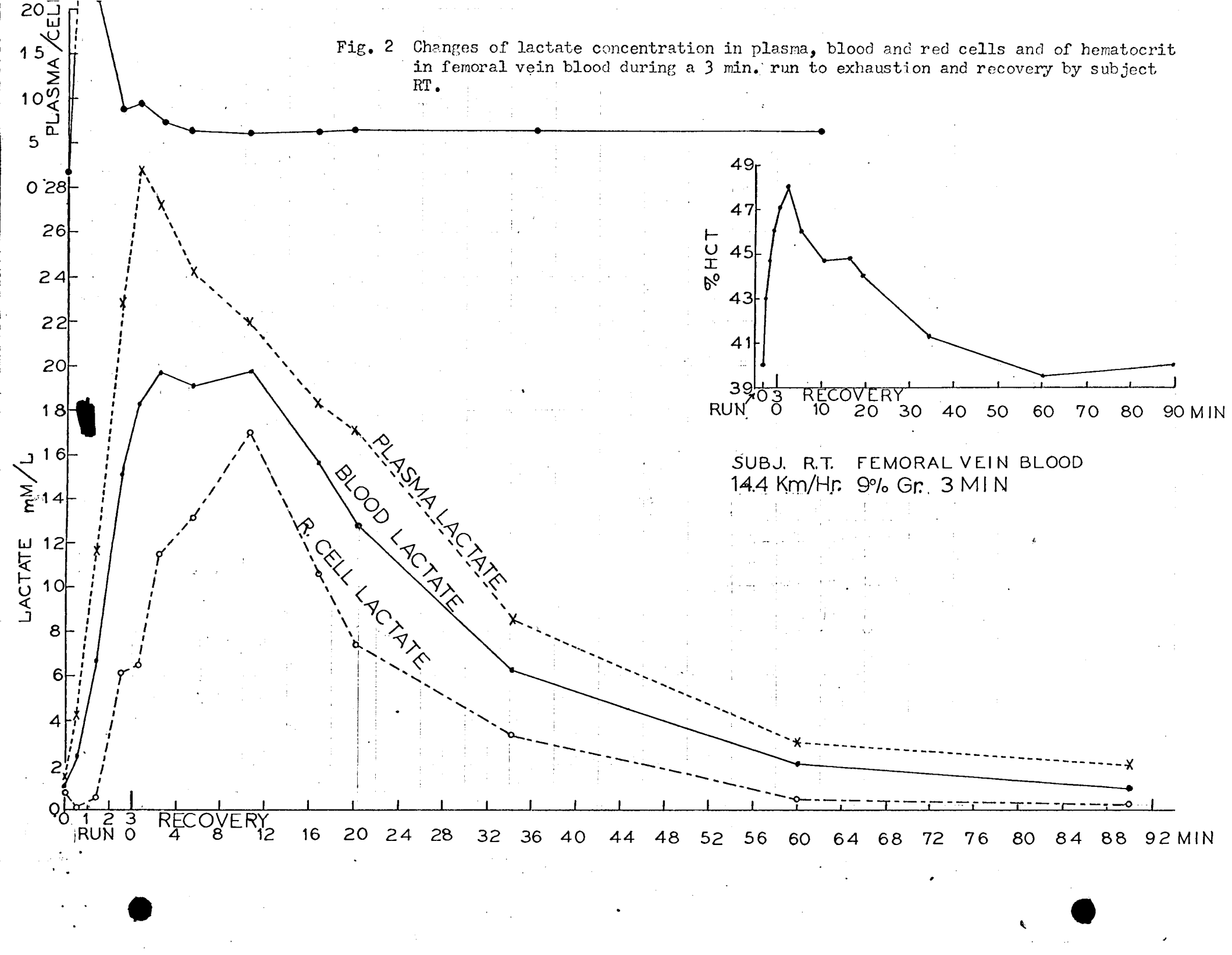


Fig. 2 Changes of lactate concentration in plasma, blood and red cells and of hematocrit in femoral vein blood during a 3 min. run to exhaustion and recovery by subject RT.

2. In femoral vein blood of the men at rest before work the plasma lactate concentrations were higher then the lactate concentrations in the red cells; the plasma to cell lactate ratios (P/C) were 1.5 to 1 and 1.8 to 1 respectively.

In subjects DC, who ran to exhaustion in 2 minutes, plasma and whole blood lactates rose only to 16 and 12 mM/l respectively during work and continued to rise during the first 3½ minutes of recovery. The plasma lactate declined rapidly in the next four minutes while whole blood lactate did not begin to decline until after the 6th minute. Cell lactate did not rise until the 2nd minute of work after which it rose rapidly to 8.4 mM/l at the end of work and continued to rise slowly through the 20th minute of recovery after which it began to decline. Concentrations in plasma and cells slowly converged toward the intermediate concentration in whole blood as recovery progressed. During the run P/C increased greatly until lactate began to move into the cells during the 2nd minute of work after which it decreased. It returned to near the pre-work level in the first 10 minutes of recovery and did not change through the remainder of the recovery period.

In subject RT, who continued to run for 3 minutes before reaching exhaustion, femoral vein plasma and whole blood lactates rose 26.5 mM/l and 17.5 mM/l respectively during work. Plasma lactate reached a peak within the first half minute of recovery and then declined at a steady rate, whereas whole blood rose an additional 2 mM/l during the first 2½ minutes of recovery and remained at this level through 10 minutes of recovery when it began to decline in a course similar to, but at a lower level than, that of the plasma lactate concentration. As in the case of DC no rise in cell lactate was observed in RT until the 2nd minute of work after which it rose rapidly to 6.4 mM/l.

In recovery it continued to rise to 17.0 mM/l in the 11th minute and then declined. In work P/C rose much the same as it did in DC but to much higher levels and failed to return to the resting level in recovery

and remained elevated throughout recovery even after a constant ratio was reached at 6 minutes.

It should be pointed out that the red cell lactate values are calculated from three determined values: whole blood lactate, plasma lactate, and hemotacrit and that small errors in the determination of any or all of these values may exaggerate variations in the calculated cell values.

DISCUSSION

These data show a difference in lactate concentration between plasma and cells in rest and work as previously reported by others (1, 3) and which may be attributed primarily to the difference in water content of plasma and cells (4). The continued rise of whole blood lactate in the blood of the femoral vein during recovery is due in part to the continued diffusion of lactate into the red cells from the plasma.

In the first 1½ minutes of work there was little or no increase in the lactate concentration in the red cells of the femoral vein blood even though plasma lactate was increasing rapidly. We have no explanation for this, but it suggests that some initial conditions involving concentration gradient, pH, pCO_2, etc. (1, 4) may be required before lactate begins to diffuse into the cell. This finding differs somewhat from previous work by Johnson et al. (5) whose data showed that diffusion of lactate into the red cells began at once in experiments carried out at 36°C in which plasma high in lactate was mixed with cells low in lactate thus imposing an immediate high concentration gradient. Under these conditions 10 minutes were required for complete equilibrium of plasma and cells (P/C=2.5 to 1) and about 2 minutes were required for half equilibrium (P/C=5 to 1). Our experiments differ from theirs in that in vitro the concentration gradient is increased gradually as lactate from the working muscles diffuses into the plasma increasing its lactate concentration. Johnson et al. (5) also

postulates that the continued increase in whole blood lactate in the first few minutes of recovery following exercise is not due to the continued production of lactate after the exercise is over, but due to "a slowly increasing cell lactate in plasma that has a lactate level held essentially constant for a few minutes by a relatively large muscle mass from which lactate is passing into the extracellular fluid." In Fig. 2 subject RT showed a drop in the plasma lactate in femoral vein blood beginning within 30 seconds in recovery following a 3-minute run. This and other experiments (see part V of this report) do not support the above hypothesis. This rapid drop in plasma lactate indicates that there is no continued production of lactate by the muscles after the exercise is stopped and that diffusion from the extracellular fluid of the muscle into the plasma is not greatly delayed. However, diffusion into the red cells is delayed and it is this delay which brings about the continued elevation of the whole blood lactate in the femoral vein following exercise. Subject DC (Fig. 1) did not show this sudden drop in plasma lactate at the end of work. He was able to run only 2 minutes before reaching exhaustion, which, for him, was not sufficient time for circulatory adjustment to occur, and this resulted in greater accumulation of lactate in the leg muscles. The removal of this accumulated lactate was responsible for the continued rise in plasma lactate in early recovery.

Previous studies in this laboratory (see part V of this report) have shown that during periods of high blood lactate concentrations lactate diffuses into inactive tissues and that in early recovery the return of this stored lactate to the circulation is mainly responsible for the continued rise and prolonged elevation of lactate in arm vein blood following an exhausting run in which most of the lactate has been produced in the leg muscles. The data just presented in this paper demonstrates that the delayed diffusion of lactate from the plasma into the red cells also contributes to the continued increase in whole blood lactate following work.

References

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