THE PHYSIOLOGICAL RESPONSES TO WORK PERFORMED UNDER NORMAL CONDITIONS AND IN SEVERE HEAT, BY BANTU MINERS WITH SPECIAL REFERENCE TO BLOOD LACTATE AND PYRUVATE

A Thesis

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CHAPTER 1

INTRODUCTION.

At the end of the nineteenth century it was known that lactic acid was a constant constituent of the blood of dogs, rabbits and even man. It was also known that lactic acid was produced during muscular contraction, but there was much controversy over the question of the chemical processes that took place in the muscle and the origin of the acid produced.

In isolated muscle preparations Fletcher and Hopkins[1] found that under anaerobic activity, lactic acid was formed, which disappeared on administration of oxygen. On the basis of this discovery they postulated a theory that in contracting muscle a close correlation exists between lactate production and oxygen supply.

The discovery that muscle glycogen was actually the precurser of lactic acid led Meyerhof to develop his theory of muscular contraction. He stated that lactic acid was closely connected with the contractile process and furthermore that a "lactic acid cycle" was present in muscle. According to Meyerhof the extra oxygen consumption after activity accounts for the removal of approximately one-fifth of the lactic acid, while the remainder is reconverted into glycogen[2].

Guided by principles established while working on isolated muscle, Hill et al.[3] attempted to apply these same principles to the complete normal animal. They concluded that in mild and moderate exercise a "steady state" was found, where the oxygen intake and oxygen supply to working muscles balanced oxygen utilization with little or no lactate accumulation. In contrast to this type of exercise it was found that in strenuous exercise

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1. W.M. Fletcher and F.G. Hopkins. "Lactic Acid in Amphibian Muscle". The Journal of Physiology 35:247, 1907.
2. O. Meyerhof, 1920. Arch. f.d. Physiol. 182:284. Cited by W.F.H.M. Mommaerts, "Muscular Contraction" P.11, Interscience Publishers, Inc. New York, 1950.
3. A.V. Hill and H. Lupton. "Muscular Exercise, Lactic Acid and the supply of oxygen. "Quarterly Journal of Medicine, 16:135 - 171, 1923.

the oxygen supply to the active muscles fell short of the oxygen need (requirement) with the result that a large proportion of the lactic acid formed was not reconverted to glycogen and therefore it accumulated in the muscles.

The Meyerhof theory of muscle contraction became untenable with the discovery of Lundsgaard [4], that muscles poisoned with monoiodoacetate will contract in the absence of oxygen, without forming lactic acid. The relationship between lactate and "oxygen debt" postulated by Hill et al. proved to be incorrect, owing to various other investigations which have appeared to show either a different relationship [5] or no relationship at all [6, 7, 8]. It therefore became obvious that blood lactate alone was inadequate to explain the degree of anaerobic metabolism taking place in muscles owing to oxygen deficiency.

The increase in knowledge about the anaerobic and aerobic phases of glycolysis and especially about the lactic dehydrogenase system in recent years, emphasized the importance of considering not only lactate but especially pyruvate in conditions of oxygen lack. From the equation of the lactic acid dehydrogenase equilibrium: Pyruvate + $DPNH_2 \rightleftarrows$ Lactate + DPN it is clear that changes in pyruvate will affect lactate levels as much as does oxygen lack.

In the light of these discoveries Huckabee [8] re-examined the relationship between oxygen debt and blood lactate and pyruvate, and postulated a theory that the rate of "excess lactate" production, a simple function of changes in both pyruvate and lactate,

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4. E. Lundsgaard: "Untersuchungen über Muskelkontraktionen ohne Milschsäurebildung". Biochem. Z., 217:162, 1930.

5. R. Margaria et al. "The possible Mechanism of Contraction and paying the oxygen debt, and the role of lactic acid in muscular contraction". American Journal of Physiology, 106:689, 1933.

6. W.H. Owles. "Alterations in the Lactic acid content of the blood as a result of light Exercise". Journal of Physiology, LXIX: 214 - 237, 1930.

7. O. Jervell. "Investigations of the concentration of lactic acid in blood and urine under physiologic and pathologic conditions". Acta Med. Scand., Suppl . XXIV, 1, 1928.

8. W.E. Huckabee. "Relationships of Pyruvate and lactate during anaerobic Metabolism. II. Exercise and Formation of Oxygen Debt". Journal of Clinical Investigation, 37:255 - 263, 1958.

is highly predictive of the body oxygen debt. "Excess lactate" formation or the quantity of anaerobic metabolism during exercise depends on the discrepancy between oxygen demand in the tissues and rates of supply, and this, on final analysis, depends on the blood circulation.

Heat, as well as active movements, influence the circulatory system. Both affect in various degrees and directions the pumping action of the heart, the distribution of the cardiac output to the various parts of the body, hemodynamic relations, the blood volume and its regional distribution, and vasomotor activity.

In most heat studies reported, the time exposure was of long duration while the activity consisted of a work rate, submaximal, and well below crest level. From the numerous reports a fairly concise picture can now be presented, showing the physiological responses to submaximal work in heat.

A careful survey of the literature revealed that information regarding the effects on the human body of hard and very hard work, when combined with severe heat exposure, is either limited or does not exist. It is, therefore, the principle aim of this study to investigate the dual stress of maximal work load and severe humid heat on the human body and especially the effect of alterations in the circulatory dynamics on muscle metabolism.

STATEMENT OF PROBLEM:

The present study was chiefly concerned with two main problems.

Firstly, in citing the literature, dealing with the lactate pyruvate relationship in blood during and after exercise, it was obvious that only a limited amount of information is available. Apart from the fact that different types of exercise were employed, the investigations were conducted in various ways making comparisons of the results nearly impossible. Asmussen (9) investigated moderate

9. E. Asmussen, "Pyruvate and Lactate Content of the Blood During and After Muscular Work". Acta Physiologica Scandinavica 20: 125, 1950.

work on a bicycle ergometer sampling blood during and after work, while Tepperman and Tepperman [10] applied moderate and heavy work loads on a bicycle ergometer, sampling blood only after cessation of exercise. In the latter case it appeared from data obtained that the work load was only moderate and the period of exercise too short.

Friedemann et al. [11] and Johnson and Edwards [12] studied the effects of work, performed under partly anaerobic conditions while blood was sampled only after the work had stopped. The "strenuous" work loads applied by Goldsmith [13] must have been only moderate because the results were very low compared with those of other investigators.

Owing to the great many variables presented by previous reports and especially the conflicting results of different work loads on the blood lactate and pyruvate, it was felt that a study should be made of the effects of progressive work loads on the blood lactate and pyruvate, as well as the pulse rate, cardiac output and oxygen consumption. Only one type of exercise should be studied, in this case cycling on a stationary bicycle ergometer, and the work loads chosen in such a fashion that it covers the range from very light work to the testee's maximal capacity.

Secondly, it is a well known fact that work imposes a stress on the circulation and if the work is done in a hot environment a second stress is superimposed upon the first. Under such conditions the circulation is unduly taxed with a dual purpose of supplying the active muscles with oxygen and secondly the transfer of heat from the

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10. Teppermann and Teppermann, "On the Blood Lactic response to measured exercise in hypoxic human subjects". Journal of Clinical Investigations, 27:176, 1948.

11. Friedemann et al. "The level of Pyruvic and lactic acids, and the lactic-pyruvate ration, in the vlood of human subjects. The effect of food, high altitude light muscular activity, and anoxia at high altitudes". Journal of Biological Chemistry, 157:673, 1945.

12. R.E. Johnson and H.T. Edwards. "Lactate and Pyruvate in Blood and Urine after Exercise". J. Biol. Chem. 118:247, 1937.

13. G.A. Goldsmith, "The blood lactate-pyruvate relationship in various Physiologic and Pathologic States". Amer. J. of Med. Sci. 215: 192, 1948.

core of the body to the periphery, the so called conductance [14].

Reports by Lee [15] and many others to the effect that a standard task performed in a hot environment is associated with a higher pulse than in a cool one, suggest a different threshold value for lactate and possibly pyruvate, in different environments, for the same individual. Careful gleaning of the literature revealed that the blood lactate and pyruvate response to exercise, performed in a hot environment, has never been studied before.

It was deemed necessary therefore to repeat a similar study as the one described in (1) supra, but with the one exception, that it should be carried out in severe humid heat.

The specific aims of this study are the following :-

(1) The determination of lactate and pyruvate in the blood, during and after exercise on a bicycle ergometer, under normal conditions and in severe humid heat, (Wet Bulb temperature 92°F. Dry Bulb Temperature 96°F.)

(2) To see whether any relationship exists between the changes in blood lactate and blood pyruvate.

(3) To find out if any relationship exist between the changes in blood lactate or pyruvate and heart rate or cardiac output.

(4) To plot the increase in lactate and pyruvate in relation to work rate and make comparisons between the two evironments tested.

Limitations:

It is felt that the value of this study was to some extent reduced by certain limitations.

The subjects were tested in the post-absorptive state which could possibly have influenced the lactate and pyruvate values.

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14. J.D. Hardy and G.F. Soderstrom. "Heat loss from the Nude Body and Peripheral Blood flow at temperatures of 22°C. to 35°C. The Journal of Nutrition 16:493 - 510, 1938.

15. Lee, D.H.J. and G. P.B. Boissard, "The Effect of Exercise in Hot Atmospheres upon the Pulse Rate". University of Queensland Papers, Vol.1 Number 6, 1941.

It has been shown by Friedemann et al. (11) that food has an effect on the blood lactate and especially the blood pyruvate levels.

On the other hand Friedemann et al. brought forward evidence that weaker performances were obtained with fasting subjects than with the same persons in the post absorbtive state.

Due to the psychological background and strange superstitions of the raw African mine labourers used in this study, the collection of blood from a deep artery by means of an indwelling needle was practically impossible. Peripheral blood from the finger tip was therefore used and these samples may or may not have reflected the true lactate and pyruvate content of arterialized blood (16 - 18).

Because of the time consuming procedure of gas analysis and certain technical difficulties encountered with cardiac output measurements, this measurement was restricted to two individuals only. Furthermore, the use of the climatic room for this study was restricted to a short period only and made it impossible to study a large and random number of people.

The activities of the subjects outside the laboratory were not controlled so that some of their pre-experimental activities could have affected the results.

Significance of Study:

An attempt was made in this study to investigate certain aspects of physiology that was all but ignored by other workers in the same field. Pyruvate changes in the blood during work or exercise has attracted very little attention up to the present time. It is hoped, therefore, that this study will make a small contribution

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11. Friedemann et. al. (1945) Loc. cit.

16. Barr, D.P. and H.E. Himwich. "Studies in the physiology of muscular exercise. 11. Comparison of arterial and venous blood following vigorous exercise". The Journal of Biological Chemistry, 55: 525, 1923.

17. Himwich H.E., R.O. Loebel, and D.P. Barr, "Studies of the effect of exercise in diabetes. I. Changes in acid-base equilibrium and their relation to the accumulation of lactic acid and acetone". The Journal of Biological Chemistry, 59: 265, 1924.

18. Eggleton, M.G. and C.L. Evans. "The lactic acid content of blood after muscular contraction under experimental conditions". Journal of Physiology, 70: 269, 1930.

towards a better understanding of the behaviour of this metabolite in the blood during work, and also point out its relationship with lactic acid.

The heat studies can be considered as unique in several ways. With one exception [19] no person has ever attempted to study the effects of heat on a human being performing maximal work. The data from these studies will certainly be informative, while the blood lactate and pyruvate data will cast more light on the problem of the efficiency of the circulation to supply oxygen to working muscles, under severe heat stress. The possible effects of acute heat exposure on performance was clearly brought out in this study.

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19. Kronfeld et al. "Strenuous Exercise in A Hot Environment". Journal of Applied Physiology, 13(3):425-429, 1958.

CHAPTER II

REVIEW OF RELATED LITERATURE.

The investigation of Fletcher and Hopkins [1] brought methodological progress and their technique in fixation of the muscle preparation was possible for the quantitive and reproducible results that could be presented. They found that resting aerated muscle contains at most 0.015 per cent lactic acid while in resting muscle under anaerobic conditions lactic acid is formed at a slow speed to a constant end value of 0.40 per cent of the wet muscle. Furthermore they made the interesting discovery that under anaerobic activity lactic acid was formed, which disappeared on administration of oxygen.

Guided by principles established by working on isolated muscles, Hill and Lupton [2] attempted to apply these same principles to muscular exercise in man. By experimentation they determined the "oxygen debt" for certain exercises, and therefrom calculated the possible amounts of lactic acid present in the blood. They maintained that for certain exercises the lactid acid reaches a "steady state" value, similar to the concept of oxygen consumption, meaning that lactate production equals lactate removal. In contrast to these findings it has been shown by Owles [3] that in very mild exercise no increase in lactate was observed, while an oxygen debt was formed of 1 liter in both subjects tested. In a thorough study made on this problem by Margaria et al. [4] it was shown that no extra lactic acid appears in the blood during and after exercise involving oxygen debts of less than about 4 liters. Furthermore they found no simple

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1. W.M. Fletcher and F.G. Hopkins, "Lactic acid in Amphibian Muscle". The Journal of Physiology 35: 247, 1907.
2. A.V. Hill and H. Lupton. "Muscular Exercise, lactic acid and the supply of oxygen." Quarterly Journal of Medicine, 16: 135 - 171, 1923.
3. W.H. Owles. "Alterations in the Lactic Acid content of the Blood as a result of light Exercise ....." Journal of Physiology, LXIX: 214 - 237, 1930.
4. R. Margaria et al. "The possible Mechanism of Contraction and paying the oxygen debt, and the role of Lactic acid in muscular contraction." American J. Physiology, 106 : 689, 1933.

correlation between the oxygen uptake after exercise which falls rapidly, and the slow reduction in blood lactate. These authors have shown that the oxygen debt curve plotted in relation to time can be fractionated in two components, the "alactacid" and "lactacid" components. The "alactacid" mechanism of contracting and paying the oxygen debt took place at a much faster rate than the "lactacid" mechanism which is responsible for about 1/3 of the total oxygen debt.

It has been found that with the onset of exercise the blood lactate increased to a maximum, the magnitude of its maximum depending on the intensity of the work load [5]. Hill [2] expressed the view that once the maximum concentration had been attained, it remained constant throughout the duration of the exercise. In contrast to Hill's view of a "steady state" lactate, Bang [6] confirmed the findings of Jervell [5] that blood lactate concentration was dependent on the intensity of the work load and further pointed out that the lactate decreases even though the activity was continued in a steady state.

This latter finding of Bang was confirmed by a report of Flock, Ingle and Bollman [7] in experiments on rats. They found a transitory production of lactic acid in the muscles at the beginning of exercise, which was not severe enough to cause persistent formation of lactic acid. These findings were disputed by Eskildsen [8] who maintains that small steady muscle pyruvate and blood lactate levels in excess of the resting values probably represent merely a new balance between rates of formation and removal.

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5. O. Jervell. "Investigation of the concentration of Lactic Acid in Blood and Urine". Acta Medica Scandinavica. Suppl. 22-25, 1927-28.

6. O. Bang. "The Lactate content of the Blood during and after muscular exercise in Man". Skand. Arch. Physiol. 74.Suppl. 10,51. 1936.

7. E.V. Flock et al. "Formation of lactic acid, an Instant Process in Working Muscle". Journal of Biological Chemistry 129:99, 1939.

8. P. Eskildsen, Acta Med. Scand. 127:171, 1947 as cited by R.G. Bannister, "Muscular Effort". British Medical Bulletin 12:222, 1956.

It has been reported by Hill, Long and Lupton [9] and Long [10] that even during very mild work an increase of lactic acid over the resting value is found. They investigated the effect of a 3.3 to 4.5 m.p.h. walk in their studies and found a 6.1 - 37.7 mg.% increase over the resting values. In contrast to their findings Owles [3] stated that the intensity at which lactic acid increases over the resting value corresponds to an oxygen consumption of 1.8 liter per minute. That no increase of lactate occurred during walking, was confirmed by a study reported by Friedemann et al.[11] who investigated the effect of a slow as well as a faster walk of 4.0 m.p.h. Other authors reported slightly different values from the value quoted by Owles.

mild exercise

It was found that the blood lactate level decreases immediately with cessation of exercise [4, 12], and experimental proof was put forward by Margaria et al.[4] that the recovery rate was such that the Logarithm of the excess lactate is a linear function of time. The removal proceeds at a comparatively slow rate, fifty per cent of the concentration of lactate in the blood being removed in 15 minutes. With very severe exercise of short duration it has been found that the lactate tends to rise even after cessation of the activity, the maximal value being obtained from 3 to 7 minutes thereafter [6, 13, 14]. This rise in the lactate content of blood after exercise was considered to be a lag in the diffusion of lactate

Cessation of exercise

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9. Hill, Long and Lupton, Proc. Roy. Soc. B. 96:438 1924, 97:84, 155, 1924-25.

10. Long, Ibid 99:167, 1926.

11. T.E. Friedemann et al "Pyruvic Acid. III. The level of pyruvic and lactic acids, and the lactic-pyruvic ratio in the blood of human subjects. The effect of food, light muscular activity and anoxia at high altitude." Journal of Biological Chemistry, 157:673, 1945.

12. E. Asmussen. "Pyruvate and Lactate Content of the Blood during and after Muscular Work." Acta Physiologica Scand., 20:125-136, 1950.

13. R.E. Johnson and H.T. Edwards. "Lactate and Pyruvate in Blood and Urine after Exercise". J. Biol. Chem. 118:427, 1937.

14. Friedemann and Barborka. The significance of the Ratio of Lactic to Pyruvic acid in the Blood after exercise". Journal of Biological Chemistry, 141: 993-994, 1941.

from the muscles into the blood [6], while other [4, 15] maintained it to be an actual post-exercise production of lactic acid.

Owles [3] has reported that training brings about a decrease of lactate concentration at a certain working intensity, which has been confirmed by Bang [6] and Edwards et al. [18]. Furthermore he pointed out that if an individual is unaccustomed to a given task he would carry it out with a higher lactate response than another type of work with the same metabolic cost but for which he was trained. It follows thus that one of the many factors which may determine the lactate response of an individual to a given task or exercise is the individual's exercise tolerance or physical ability. Experimental proof to this effect has been reported by several workers [16, 17], who found higher blood lactate responses to a submaximal exercise for untrained persons than those considered well-trained.

Training

The "threshold value" beyond which increases in blood lactate appear is also dependent on the physical fitness of the individual, and it has been reported [19, 20] that for unfit subjects, a lower critical intensity of exercise is required, to exceed this value.

melksuur-drempel

It has been stated by Robinson and Harmon [21] that a well-trained person can reach higher values as compared with an

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15. E.V. Newman. "Distribution of Lactic Acid between Blood and Muscle of Rats". American Journal of Physiology. 122:359, 1938.

16. D.B. Dill et al. "Industrial Fatigue". Journal of Industrial Hygiene. 18: 417-431, 1936.

17. R.E. Johnson and L. Brouha. "Pulse rate, blood lactate, and duration of effort in relation to ability to perform strenuous exercise. Rev. canad. Biol. 1:171, 1942. Cited by Astrand 1952.

18. Edwards et al. Cited by Astrand, 1952.

19. L.C. Cook and R.H. Hurst. "Blood lactic acid in man during rest". J. Physiology, 79:443, 1933.

20. Newman, Dill, Edwards and Webster. "The rate of Lactic removal in Exercise." Amer. J. Physiol. 118:457, 1937.

21. S. Robinson & P.M. Harmon. "The lactic acid mechanism and certain properties of the blood in relation to training". American J. Physiology. 132 : 757, 1941.

untrained. This finding of Robinson and Harmon was confirmed by observations made by Knehr, Dill and Newfeld [22].

It is not surprising, owing to the relationship between lactic acid and physical ability, that certain workers [17, 23, 24], made use of experiments on the lactate response to exercise as a test of physical fitness. For a fixed task Johnson and Brouha [17] found the lowest lactate response to running experiments in a group of well-trained oarsmen while the unfit-group had the highest lactates. Crescitelli and Taylor [23] found that, as a group, the less fit individuals gave significantly greater blood lactate values throughout the entire period of the lactate response to the exercise than did fit individuals. They also found that the total excess urine lactate in response to the exercise period tested was significantly related to the fitness of the group, as the less fit individuals showed larger quantities of this metabolite. Taylor [24] tested 32 subjects and found that heart rate and blood lactate are the most reliable submaximal measures of fitness.

Ryffel [25] was the first to carry out a direct quantitative determination of lactic acid in urine after severe exertion of short duration. One of the most exhaustive studies made on the excretion of lactic acid in the urine following exercise was made by Liljestrand and Wilson [26]. They found, after exercise of sufficient intensity, quantities ranging from 140 to 1,370 mg. over the resting level. Jervell [5], Johnson and Edwards [13] found that the peak

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22. C.A. Knehr et al, "Training and its Effect on Man at Rest and at Work". American J. Physiology. 136:148, 1942.

23. F. Crescitelli and C. Taylor. "The Lactate response to exercise and its relationship to Physical Fitness". Amer. J. Physiology 141:630, 1944.

24. C. Taylor, "Some properties of maximal and sub-maximal exercise with reference to Physiological variation and measurement of exercise Tolerance." Amer. J. Physiology, 142:200, 1944.

25. J.H. Ryffel. "Experiment on Lactic acid formation in Man". Journal of Physiology 29:XXIX, 1909/1910.

26. Liljestrand and D.W. Wilson. "The excretion of Lactic acid in the urine after muscular exercise". Journal of Biological Chemistry. 65:773, 1925.

lactate excretion occurs at 10 - 20 minutes after cessation of exercise and that the excretion is complete in forty to fifty minutes. Jervell estimated that the hourly excretion of lactate in the urine is from ten to twenty mg, but he considers it as being on the high side.

The presence of lactic acid in sweat has been demonstrated by Fishberg and Bierman [27] and values as high as 33m Eq / liter have been reported by these authors. Dill [28] made observations on the lactate content of sweat and found values of up to 20m Eq / liter when the blood lactate was only 1m Eq/liter. It was found, however, that if the necessary precautions against errors were taken the lactate content is about 10m Eq/liter [29].

lactate in sweat.

Although lactate is present in high concentrations in sweat the possible role of lactate in the functioning sweat gland has not been elucidated. Changes in the sweat gland activity during acclimatization have been studied by Weiner and van Heyningen [30] who found that in sweat of male subjects, carrying out work at high temperatures and humidity, the lactate accounts for 11 per cent of the osmotic pressure. Furthermore it was found that the lactic acid concentration may be as high as 300 mg per cent in the pre-acclimatized state but settles down to 100 mg per cent after ten to fifteen three-hourly exposures. Another interesting finding was reported by these authors in a study where the one arm of a subject was rendered ischaemic for 15-20 minutes in every thirty minutes period of sweating, over an experimental period of three hours. The lactic acid percentage was markedly increased as compared with that in the unoccluded arm, which shows a close analogy with muscle.

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27. E.H. Fishberg and W. Bierman. "Excretion of Lactic Acid in Sweat". Proc. Soc. Exp. Biol. Med. 29:1021, 1932.

28. D.B. Dill. "Life, Heat and Altitude". Cambridge, Massachusetts : Harvard University Press, 1938, p. 46, 47.

29. O. Bergheim and T. Cornbleet. American Journal of Medical Science 205 : 785, 1943.

30. J.S. Weiner and R. van Heyningen. "Lactic Acid and Sweat Gland Function". Nature, 164 : 351, 1949.

Bunting, et al,[31], pointed out that glycogen is present in cells of the sweat gland in considerable quantity, while Weiner and van Heyningen [32] in a study of the lactate content of sweat found that the concentration is not influenced by the sweat rate, the skin or rectal temperature of the subject, or the level of lactate in the blood. From the evidence put forward by Bunting et al, Woiner et al, it seems as if the lactate in sweat is a product of the gland's metabolism.

Anoxemia.

When the supply of oxygen to the interior of living cells is reduced to a state below the metabolic needs for oxygen then lactic acid is formed. If, for any reason, the oxygen content of alveolar air is drastically reduced it will result in a reduction of the oxygen tension of the gas transport system, which in its turn will affect the oxygen supply of the tissues. On those grounds the possible relationship between blood lactate and respiratory hypoxia was investigated by numerous workers. Studies were conducted in one of two ways; the experimental subjects or animals were given various low oxygen gas mixtures to breathe while the degree of anaerobic metabolism was estimated by the concentration of blood lactate, or the physiological responses of subjects to mountain climbing were investigated.

In anoxemia Macleod [33] found an increase in lactate concentration when the respiratory mixture breathed by his anaesthetized animals contained 4 to 8% oxygen. In contrast to these findings, Jervell [5] found in short experiments on men, while breathing low oxygen mixtures, no increase in lactic acid until the oxygen breathed

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31. H. Bunting et al, "Arat. Rec., 100:61, 1948. Cited by J.S. Weiner and R. van Heyningen (1949). Loc. cit.

32. J.S. Weiner and R.E. van Heyningen. "Observations on Lactate Content of Sweat". Journal of Applied Physiology, 4:734 - 744, 1952.

33. J.R. MacLeod, "The Concentration of Lactic Acid in the blood in Anoxemia and Shock". American Journal of Physiology. lV : 184, 1921.

was reduced to 7.5 per cent. Bock et al.[34] found an insignificant rise in the venous blood lactate of subjects breathing a 9 per cent oxygen mixture and the data suggests that anoxemia in various clinical conditions must reach a grave state to disturb the lactic acid concentration in the body. Myerson et al.[35] reported that when the oxygen saturation of arterial blood lies between 48 and 77 per cent, no considerable accumulation of lactic acid appears. However, their experiments on ten subjects breathing 9 per cent oxygen for 3 to 10 minutes showed a slight increase in lactic acid for 6 of the 10 subjects. Henderson and Greenberg [36] with experiments on animals found that if the inspired air contained 7 per cent or more oxygen, no increase of blood lactate was observed, but a marked hyperpnea developed. With a mixture containing less than 7% oxygen lactic acid increased but the respiration was not augmented.

Edwards [37] noted a slight rise in the resting blood lactates after a rapid ascent to 9,000 feet, but normal values were found at 20,000 feet.

Furthermore, observations made on Chilean residents at an altitude of 5.34 Km. gave values in accord with resting values at sea level. The same author found that at an altitude of 12,000 feet the blood lactate response to a maximal exertion was lower than for the same exercise at sea level, while at 20,000 feet the human body showed an inability to accumulate lactate above the resting level.

The latter finding of Edwards was disputed by Asmussen et al.[38] who found that at a decreased oxygen tension of 12% the

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34. A.V. Bock et al. "Lactic Acid in the Blood of resting Man". Journal of Clinical Investigation, 11:775, 1932.

35. A. Myerson et al. "The composition of Blood in the artery, in the internal jugular vein, and in the femoral vein during oxygen want". American J. Physiology, 98 : 373, 1931.

36. Y. Henderson and L.A. Greenberg. "Acidosis: Acid intoxication or Acarbia." American J. Physiology 107:37, 1934.

37. H.T. Edwards. "Lactic acid in rest and work at high altitude". American Journal of Physiology. 116: 367 - 375, 1936.

38. E. Asmussen et al. "Blood lactate and Oxygen debt after exhaustive work at different Oxygen tensions". Acta Physiologica Scandinavica, 15: 57 - 62, 1948.

capacity for work was lowered, but the maximum lactate concentration was practically the same or higher than in normal air.

Friedemann et al.[11] found that the pyruvic acid in the blood of resting subjects was relatively little affected by short exposures to simulated altitudes up to 15,000 feet, while the lactate pyruvate ratio showed insignificant changes in relation to altitude.

In a later study Friedemann et al.[39] found that exposure to anoxia at simulated altitudes from 15,000 to 23,000 feet resulted in elevated concentrations of lactic and pyruvic acids and a rise of the lactate-pyruvate ratio. It was also found that both lactate and pyruvate rose more with exercise in the presence of hypoxia, but the lactate-pyruvate ratio bore no relation to hypoxia during exercise[40].

Hill et al.[8] studied the influence of a variation in the inspiratory oxygen percentage on the removal of lactate from the body after exercise. The body lactate was calculated from the "oxygen debt" values and it was found that the "oxygen debt" was independent of the variations in the inspiratory oxygen percentage.

Experimental proof of Hill's hypothesis that body lactate removal is independent of the percentage oxygen inhaled, was put forward by Ström[41] who showed that the rate of lactate utilization was not significantly altered by anoxia caused by inhalation of low percentage oxygen mixtures.

Diffusion rate of lactate.

A sound knowledge of the diffusion rate of lactate and pyruvate is essential if conclusions are to be made on the total body content of these metabolites as well as the utilization rate

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39. T.E. Friedemann et al. "Work at High altitude. VI The Effect of diet and other factors on the rise of lactic and pyruvic acids and the lactate-pyruvate ratio in human subjects at simulated high altitudes". Quarterly Bulletin of North Western Univ. Med. School, 23:448-455, 1949.

40. J. Tepperman and H.M. Tepperman. "On the blood lactic acid response to measured exercise in hypoxic human subjects". Journal of Clinical Investigations, 27:176, 1948.

41. G. Ström. "The influence of Anoxia on lactate utilization in man after prolonged Muscular Work". Acta Physiologica Scandinavica, 17:440, 1949.

or rate of disappearance from the blood. It can be questioned whether determinations carried out on whole blood are representative of plasma conditions or whether the lactate ion is uniformly dispersed throughout the body water in such concentrations. The vital question whether concentration gradients exist between blood and tissues were therefore investigated by numerous laboratories.

Decker and Rosenbaum (42) have found that in the blood of humans at rest and in the post absorptive state, the lactate is evenly distributed through the water of cells and plasma. It was further found that a rise in the concentration of lactate in whole blood disturbs this ratio and a higher concentration was then found in the plasma than in the cell water. These findings are corroborated by the results of Devadetta (43) who found that addition of lactate to a suspension of blood cells in saline resulted in a distribution of the ion similar to that found in whole blood. It was further shown that washing of cells or addition of substances preventing glycolysis did not alter the distribution of lactate.

In a very thorough study, Huckabee (44) investigated the relationships of extracellular and intracellular concentrations of pyruvate and lactate ions in fresh human blood cells. He found that the concentrations of lactate and pyruvate were significantly higher in total plasma water than in total blood water. Correction factors were proposed to convert whole blood values to plasma values, being 1.22 for pyruvate and 1.08 for lactate. Calculations were also made of the concentrations of pyruvate and lactate in erythrocytes and thus the concentration gradient between extracellular and intracellular water. It was found that the average plasma pyruvate

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42. D.G. Decker and J.D. Rosenbaum, "The distribution of lactic acid in human blood". The American Journal of Physiology, 138:7, 1942.

43. S.C. Devadetta, "Distribution of lactate between the corpuscles and the plasma in blood". Quarterly Journal of Experimental Physiology, 24:295, 1934.

44. W.E. Huckabee, "Control of concentration gradients of pyruvate and lactate across cell membranes in blood". Journal of Applied Physiology 9:163, 1956.

values were 130 per cent higher than those of the cells and the lactate values were 37 per cent higher.

These findings suggest that the use of whole blood lactate values to calculate total body lactate is very much in error and that it underestimates the quantities produced in exercise. On the other hand it is still an open question whether other cells behave in the same manner as erythrocytes. Attempts were made to solve the problem by direct experimentation on the tissues of animals, but even this method was beset with difficulties making the solution of the problem very difficult. The principle difficulties were that an equilibrium state does not exist, the acid is continuously produced and removed, and its production continues while the tissue is removed for analysis.

Analysis of rat muscle and blood, simultaneously removed from resting as well as exercised animals showed no concentration gradient between the two tissues (15). Results put forward by Gesell et al (45) and Eggleton and Evans (46) in a study of the concentration gradient between plasma and muscle showed that in plasma the concentration was somewhat higher than in muscle. This is in agreement with the findings of Huckabee on erythrocytes and plasma.

According to Ghaffar (47) in experiments on frog muscle added lactate was distributed through a volume equal to the extracellular space, while Devadetta found under similar conditions that the lactate ion diffuses freely throughout the total fluid of the

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45. R. Gesell et al. "A comparison of the response of the anaesthetized dog to lowered alveolar oxygen during uniform artificial ventilation and during normally controlled ventilation." American Journal of Physiology 100; 202, 1932.

46. M.G. Eggleton and C.L. Evans. "The lactic acid content of the blood after muscular contraction under experimental conditions". Journal of Physiology, 70: 269, 1930.

47. A. Ghaffar, "Diffusion of lactic acid into and out of the voluntary muscle of the frog". Quarterly Journal of Experimental Physiology 25: 229, 1935

tissue. Alpert and Root [48] investigated the diffusion rate in the abdominally eviscerated dog and found that the lactate ion diffuse into a volume equal to about 75 percent of the body water within 40 minutes. When unanaesthetized dogs were used the diffusion rate was the same as that found with the eviscerated preparation, while the utilization rate of lactate in normal animals was about forty percent in 23 minutes.

Hartmann and Senn [49] reported an experiment where large quantities of sodium-r-lactate were injected into human subjects and the disappearances observed by blood analysis. The quantity injected raised the blood lactate to levels between 300 and 500 mg. percent. Within fifteen minutes the blood lactate content was reduced to between 30 and 65 mg % meaning that in this period more than 90 percent of the injected lactate left the blood stream. The rapid fall in the blood lactate was a result not only of diffusion of the ion, but also of excretion by the kidneys and utilization by the liver and other tissues.

Arterio-venous differences in lactate content of blood circulating through exercising muscles, as well as resting muscles were reported [50, 51], but the findings were conflicting.

Lactate removal from blood.

Although authors of numerous studies on amphibian muscle claim that this tissue has the property of utilizing lactate it has never been proved beyond any doubt that mammalian muscle has similar properties. Many reports have been put forward to show that blood supplying an inactive muscle loses some of the lactate in the process of passing through. This loss of lactate was considered only as a

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48. N.R. Alpert and W.S. Root. "Relationship between excess respiratory metabolism and utilization of intravenously infused sodiumrasemic lactate and Na(-) lactate". American Journal of Physiology 177:455, 1954.

49. A.F. Hartmann and H.J.E. Senn. "Studies in the metabolism of Na-r-lactate 1. Response of normal human subjects to the intravenous injection of Na-r-lactate. Journal of Clinical Investigation 11:327, 1932.

50. H.E. Himwich et al. "Studies of the effect of exercise in diabetes. 1. Changes in acid-base equilibrium and their relation to the accumulation of lactic acid and acetone." Journal of Biological Chemistry, 59: 265, 1924.

51. H.E. Himwich et al. "Studies in carbohydrate metabolism. 11. Glucose lactic acid cycle in diabetes." Journal of Biological Chemistry 90: 147, 1931.

diffusion phenomenum and the general conception was that if mammalian muscle can utilize lactate it is to a very limited extent.

Although it was undoubtedly established that the liver is the main site of lactate removal from the blood, certain observers put forward contrary results. According to Cherry and Crandall [52] retention of lactic acid by the liver does not occur in normal regularly fed unanaesthetized dogs. Himwich et al using anaesthetized dogs, gave support to these findings and concluded that fasting may be an appropriate stimulus for retention.

In another experiment Ivy and Crandall [53] re-investigated the problem by using unanaesthetized angiostomized animals to avoid the disturbance of carbohydrate metabolism produced by anaesthesia and surgical procedures experienced by Cherry and Crandall. The results confirmed the findings of the previous authors, i.e. a non-occurence of hepatic lactic acid retention in the non-fasting animal, but a significant retention during fasting or fat feeding. They further observed that under a wide variety of conditions, no hepatic retention of lactic acid occurred, except in the case of large amounts fed orally.

In direct contrast to these findings, Hartmann and Senn reported in two different publications, firstly the lactate removal from the blood of injected r-lactate in normal individuals [49] and thereafter the same observation made on patients suffering from acute catarrhal jaundice [54]. In the case of patients with damaged livers, the lactate removed from the blood was slightly but definitely delayed.

Alpert and Trager [55] made observations on barbiturized dogs, exercised for five minutes by tetanic stimulation, before and

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52. I.S. Cherry and L.A. Crandall. "The absence of a significant glucose lactic acid cycle (involving the liver) in normal unanaesthetized dogs". Amer. Journal of Phys. 125:41, 1939.

53. H.B. Ivy and L.A. Crandall. "Conditions in which the liver retains lactic acid". American Journal of Phys. 132:679, 1941.

54. A.F. Hartmann and M.J.E. Senn. "Studies in the metabolism of Na-r-lactate. 111. Response of human subjects with liver damage ....."Journal of Clinical Investigation, 11:345, 1932.

55. N.R. Alpert and E. Trager. "The complete dissociation of lactate removal and the excess oxygen consumption of exercise." XXth International Physiological Congress. p. 22, 1956.

after complete hepatectomy and evisceration. The lactate levels rose during exercise from 14 mg. percent to 85 mg. percent and in the normal animals the lactate returned rapidly to control values after cessation of the exercise. In the operated animals, however, the lactate remained at a high level, proving to some extent the active part played by the liver in lactate removal from the blood.

The liver (removal of L)

More direct evidence that the liver is the chief site where lactate is removed from the blood and is converted to glycogen, came from the work of Cori and Cori [56] who demonstrated the production of glycogen in the livers of rats after ingestion or injection of d-lactate.

In experiments [57] in which fasting rats were fed with lactate where the carboxyl carbon was labelled with radio-active carbon, it was found that only 32 percent of the fed lactate was incorporated into glycogen while the formed glycogen contained only a negligible amount of the radio-active carbon.

About 20 percent of the radio active carbon was lost in the expired air in the form of carbon dioxide.

In similar experiments [58] where fasting rats were fed with lactate the introduction of radio active carbon in the alpha-or beta positions of the lactate molecule, instead of the carboxylic radicle; doubled the radio activity of the carbon in the glycogen while that in the carbon dioxide of expired air was halfed. It was postulated that pyruvic acid reacts with carbon dioxide to form a dicarboxylic acid which is then decarboxylated.

Evidence that the liver is not the only organ to remove

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56. C.F. Cori and G.R. Cori. "Glycogen formation in the liver from d- and l-lactic acid". Journal of Biological Chemistry 81 : 309, 1929.

57. J.B. Conant et al. "Metabolism of lactic acid containign radio active carbon. " Journal of Biological Chemistry, 137 : 557, 1941.

58. B. Vennesland et al. "Metabolism of lactic acid containg radio active carbon in the B position. Journal of Biological Chemistry 142 : 371, 1942.

lactate from the blood came from the work of Holmes and Ashford[59] who found that brain tissue in vitro, can utilize lactate. In further studies [60] these authors tried to trace the fate of the lactate removed by brain tissue and concluded that the lactate which disappeared was not synthesized to glycogen.

Evans et al.[61] also showed that the heart could utilize lactate and from their studies it appeared that the heart utilizes lactate in preference to other types of fuel.

The fact that lactic acid is formed under anaerobic conditions and is further oxidised only under aerobic conditions was responsible for the numerous studies made on pulmonary metabolism. Very conflicting results were presented by the different workers in this field. In some cases [62, 63] it was found that the circulating blood lactate during passage through the pulmonary circuit was decreased, while others found evidence of oxidative processes in the lung [64, 65]. In contrast to these finding Evans et al.[66] found that the lung actually contributed lactate to the blood in its passage through these organs.

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59. E.G. Holmes and C.A. Ashford. "_____" Biochemical Journal 24 : 119, 1930.

60. C.A. Ashford and E.G. Holmes. "Further observations on the oxidation of lactic acid by brain tissue". Biochemical Journal 25: 2028, 1931.

61. C.L. Evans et al. "The utilization of blood sugar and lactate by the heart-lung preparation." Journal of Physiology 80: 21, 1933.

62. C.H. Beatty. "The effect of haemorrhage on lactate/pyruvate ratio and arterial-venous differences in glucose and lactate". American Journal of Physiology. 143.57, 1945.

63. D. Halmágyi et al. "Zur Frage der intrapulmonalen Milchsäuereoxydation beim Menschen". Klinischen Wochenschrift 29: 219, 1951.

64. A.M. Mitchell and A. Cournand. "The fate of circulating lactic acid in the human lung". The Journal of Clinical Investigation. 34: 471-476, 1955.

65. W.T. Goodale et al. "Coronary sinus catheterization for studying coronary blood flow and myocardial metabolism". American J. of Physiology. 152: 340, 1948.

66. C.L. Evans et al. "Utilization of blood sugar and formation of lactic acid by the lungs". Journal of Physiology 82:41, 1934.

Other factors influencing the lactate and pyruvate level of the blood.

① Exercise
② Anoxia
③ Glucose

It has been shown that exercise and anoxia increase the lactate levels of the blood, but further studies elucidated a third factor. Bueding, Stein and Wortis (67) in observations made on 23 healthy well-nourished subjects found a significant rise in the blood pyruvic acid following the ingestion of glucose. The smallest increase was 0.93 mg. percent and occurred at the end of 1 hour. Following the peak the pyruvic acid fell, reaching the fasting range at or before the 3rd hour.

A more detailed study was made by Friedemann, Haugen and Kmieciak (11) who investigated the effect of alimentation on the pyruvic acid and lactic acid levels of the blood. They found that following the ingestion of a meal, the pyruvate and lactate of the blood rose, and reached maximum values in one to two hours after which both acids rapidly declined. It was further found that following the ingestion of meat, the pyruvate level rose slowly which, according to these authors, indicates that protein ingestion may lead to an increase of keto-acids. In this regard fat was without effect.

meals

Although Jervell (5) in an early study, was unable to show an increase in the blood lactate after a carbohydrate-rich meal, the work of Goldsmith (68) confirmed the findings of Friedemann et al.

Bueding and Goldfarb (69) investigated the effect of intravenous injections of glucose on the blood lactate and pyruvate levels. They found that a single injection of glucose, was followed by a rise in both acids, which returned to the normal levels in

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67. E. Bueding, M.H. Stein and H. Wortis. "Blood Pyruvate curves following glucose ingestion in normal and thiamine deficient subjects". Journal of Biological Chemistry, 140:697, 1941.

68. G.A. Goldsmith. "The blood lactate-pyruvate relationship in various physiologic and pathologic states." American Journal Medical Sciences, 215:182, 1948.

69. E. Bueding and W.J. Goldfarb, "Blood changes following glucose, lactate and pyruvate injections in man". Journal Biological Chemistry, 147: 33, 1943.

5. O. Jervell. Loc cit.

2 hours.

Simultaneous injections of insulin after a single glucose injection had no effect on the pyruvate level, but given during a continuous infusion of glucose, it produced a further rise in the pyruvic acid level.

In an earlier study it was shown by Asmussen et al.(70) that an injection of insulin during work caused an increase in the blood lactate and R.Q. and a decrease in the blood glucose. Contrary to this, they found that an injection, under similar conditions, of adrenalin resulted in an increase in the R.Q., blood sugar as well as the blood lactate. That the injection of adrenalin causes the blood lactate to rise was confirmed by the work of Griffith et al.(71) on an anaesthetized cat. They found that the lactate output by the tissues of the leg was increased on the average by all doses of adrenalin, but the maximum output was found with the smallest dose and it was much less by all other rates of administration.

ACID-BASE EQUILIBRIUM.

In experiments on heart-lung preparations Anrep and Cannon (72) found that lactic acid in the blood rose when the alkalinity was increased, while a fall in the pH was followed by a reduction of the acid. Bock et al.(34) also found that a shift in the blood pH in the alkaline direction resulted in an increase in blood lactate. In experiments on humans, voluntary over-ventilation of 16 to 37 minutes gave increases of two to three times the resting lactate values and in some cases the pH rose to 7.6. Ingestion of large doses of bi-carbonate had no effect on the blood lactate but the pH of the blood was not raised as high as in cases of voluntary hyperventilation.

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70. E. Asmussen et al. "Hormonal influences on carbohydrate metabolism during work." American Journal of Physiology, 130:600, 1940.

71. F.R. Griffith et al. "The effect of intravenous adrenalin on blood flow, sugar retention, lactate output and respiratory metabolism of peripheral tissues in the anaesthetized cat." American Journal of Physiology, 149:64, 1947.

72. G.V. Anrep and R.K. Cannon. "The concentration of lactic acid in the blood in experimental alkalaemia and acidaemia". The Journal of Physiology. 58:244, 1924.

34. A.V. Bock et al: Loc. cit.

In experiments on dogs Gesell et al [73] found that injections of bicarbonate in amounts sufficient to raise the pH by 0.3, causes the blood lactate to increase significantly.

HEAT STUDIES.

The response of normal men to hot humid heat and their performance under heat stress has been studied by Eichna et al.[74] while the same authors [75] investigated the upper limits of environmental heat and humidity that can be tolerated by acclimatized men working in hot environments. The effects of heat stress and exercise on the renal blood flow and filtration rate was determined by Radigan and Robinson [76]. They found a greater decrease in renal blood flow in the heat which shows that the blood was shunted to the vital organs under the stress. Robinson et al.[77] investigated a possible difference in the responses of white men and negroes to work in humid heat. The difference in reactions to heat by representatives of the two races were probably due to differences in nutritional status, acclimatization and training.

Hellon et al.[78] produced positive proof that older men reacted differently in heat regulatory and circulatory responses to work in heat, compared with younger men.

Exhaustive studies have been made on the adaption of the human body to heat, the socalled acclimatization to

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73. R. Gesell et al. "The regulation of respiration. A study of the correlation of numerous factors of respiratory control following intravenous injection of sodium bicarbonate. American Journal of Physiology, 94:387, 1930.

74. L.W. Eichna et al. "Performance in relation to Environmental Temperature." Bulletin of the John Hopkins Hosp. 74:25, 1945.

75. L.W. Eichna et al. "The upper limits of environmental Heat and Humidity tolerated by Acclimatized men working in hot environments. J. Industrial Hygiene & Tox. 27 (3) 5, 9, 1945.

76. L.R. Radigan and Sid Robinson. "Effects of Environmental Heat Stress and Exercise on Renal Blood Flow and Filtration Rate." J. Applied Physiology 2 (4) : 185 - 191 , 1949.

77. S. Robinson et al. "Adaptations of White men and Negroes to Prolonged work in Humid Heat." Amer. J. of Tropical Med. 21(2) : 261 - 287, 1941.

78. R.F. Hellon et al. "The physiological Reaction of Men of two age groups to hot environments." Journal of Physiology 133 : 118, 1956.

heat [79, 80, 81, 82, 84]. The changes that were regularly observed were a progressive reduction in the increments in the skin and deep body temperature, and in the pulse rate. This was accompanied by a speeding up of the onset of sweating and an increase in the amount of sweat lost. There was a concomitant improvement in the subject's ability to perform muscular work and a noticeable reduction in his feelings of discomfort.

Eichna et al.[83] studied the changes which occured in men during acclimatization to work in dry heat, using analysis by partitional calorimetry and calculations of heat transfer. The more profuse sweating, increased evaporative cooling and decreased metabolic rate, resulted in a lowered rectal and skin temperature and an increased temperature gradient from deep tissues to surface. The increased gradient made it possible for a smaller cutaneous blood flow (70 percent) to transfer the metabolic heat to the skin.

NB

DETERMINATION OF LACTIC ACID.

For the quantitive determination of lactic acid in blood and other biological fluids many methods have been proposed and even more modifications were suggested. The older methods were not quite "specific" for lactic acid and gave too high values [5].

All methods are identical in one respect, the oxidation of lactic acid to acetaldehyde, but the choice of oxidizing reagent differed widely. The more recent methods of measuring the

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79. W.B. Bean and L.W. Eichna. "Performance in relation to environmental temperature." Fed. Proc. 2: 144, 1943.

80. Robinson et al. "Rapid acclimatization to work in hot climates". Amer. J. Physiol. 140:168, 1943.

81. D.B. Dill et al. "Changes in composition of sweat during acclimatization to heat". Amer. J. Physiol. 123: 412, 1938.

82. D.K.C. MacDonald and C.H. Wyndham. J. Applied Physiology, 3: 242, 1950.

83. L.W. Eichna et al. "Thermal regulation during acclimatization in a hot dry environment". American Journal Physiology 163:585, 1950.

84. R.F. Hellon et al. "Natural and Artificial Acclimatization to hot environment". J. Physiology. 132 : 559, 1956.

5. O. Jervell. (1927 - 28) Loc. cit.

acetaldehyde formed are either titrometric or colorimetric. The iodotrimetric method of Friedemann, Cotonio and Shaffer [85] is not quite a micromethod for estimating lactic acid, seeing that it requires 0.5 mg. of lactic acid for the usual determination in duplicate. A modification of this method described by Edwards [86] made it more applicable for blood analyses by omitting the copper calcium treatment.

The colorimetric method of Mendel and Goldscheider [87] and Miller and Muntz [88] make use of concentrated sulphuric acid as the oxidizing reagent to convert lactic acid to acetaldehyde. Mendel and Goldscheider used veratrole as the colour reagent to the colorimetric determination of acetaldehyde, while Miller and Muntz made use of the reaction between acetaldehyde and p-hydroxydiphenyl. The method most widely employed for determining lactic acid on a microscale is the colorimetric method of Barker and Summerson [89] which is based on the method of Miller and Muntz seeing that the reaction of p-hydroxydiphenyl and acetaldehyde is fundamentally far more sensitive than the veratrole reaction.

Colorimetric method for determination of LA

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85. Friedemann, T.E. ; M. Cotonio and P.A. Shaffer, "The determination of lactic acid". The Journal of Biological Chemistry, 73 : 335, 1927.

86. Edwards, H.T. "A Simplified estimation of lactate in normal human blood". The Journal of Biological Chemistry 125 : 571, 1938.

87. Mendel, B. and I. Goldscheider, Biochem. Z. 164 : 163, 1925.

88. Barker, S.B. and W.H. Summerson, "The colorimetric determination of lactic acid in biological material". The Journal of Biological Chemistry, 138 : 535, 1941.

CHAPTER 111.

METHODOLOGY.

Experimental Procedure: The seven subjects were normal male African mining recruits, except Den - being a Coloured. The physical characteristics of the subjects are presented in Table 1.

To avoid, what is commonly referred to as the "training effect" they were "conditioned" for several weeks before the testing period started. All seven subjects were tested under normal conditions of temperature while three were also tested under severe humid heat. The fourth subject who was to be tested under heat stress left just after he was fully acclimatized. The procedure of the tests were exactly the same for both environments, except that in the climatic room they worked at a wet bulb temperature of 92° Fah., a dry bulb temperature of 96° and a wind velocity of 150 feet/min.

All the tests were made in the post-absorptive state, and took place between 9 a.m. and 11.30 a.m. The subject was first weighed. After the electrodes were fastened and put in place he rested on the remodeled seat of the ergometer for at least thirty minutes. At the end of this period the resting pulse rate, rectal temperature, oxygen consumption, cardiac output and blood samples were taken. When the required resting tests were completed the subject received a "warm up" bout of 10 minutes at a work load of 2500 ft lbs/min., pedaling at 70 r.p.m. To keep the pace he had to pedal at the beat of a metronome set at 140 beats per minute. The "warm up" period was followed by five minutes rest and directly after that the subject pedalled at the determined load again at seventy revolutions per minute.

Pulse rate determinations were made every five minutes except at the very high work loads where it was taken every minute. Gas was collected from the second to the third minute and again at the tenth and eleventh minute. At the high work loads, lasting only a few minutes it was taken only at the second to third minute.

The acetylene rebreathing procedure [1a] followed immediately after the expired gas was collected. Blood samples were taken at the end of exercise usually during the last twenty seconds of exercise. At the more strenuous work loads the blood samples were taken not only at the end of exercise but also at the third, fifth and seventh minutes of recovery. The experimental procedure for subjects Ber., Jul., and Arm. was slightly different from the rest. In their case the pulse rates were taken with a stethoscope at the chest wall for two thirty second periods.

Pulse rate: The measurements were made with a portable Sanborn electrocardiograph.

Cardiac output was determined by the Grollman acetylene method [1] as modifed by Christensen [2].

Collection of expired air: Timed air collections were carried out using a rubber gas mask and a length of rubber corrugated tubing leading to a Douglas bag. Before each experiment the Douglas bags were first flushed with expired air and then cleared out. During experimentation 30 to 40 seconds were allowed for the dead space air to be flushed out before the bags were opened for collecting samples. The bags were always opened and closed at the end of an expiration [3].

Collection periods were three minutes for resting samples and one minute for working samples. Three litre gas samples were taken for analysis in rubber bags after the gas was thoroughly mixed in the Douglas bag.

1(a). A. Grollman. "The Cardiac Output of Man in Health and Disease." P. 59, Charles Thomas, Springfield, Illinois, Baltimore, Maryland, U.S.A.

1. A. Grollman. "The Determination of the Cardiac output of Man by the use of Acetylene". American Journal of Physiology 88 : 43, 1929.

2. E.H. Christensen. "Minutenvolumen und Schlagvolumen des Herzens während schwerer körperlicher Arbeit". Arbeitsphysiologie 4:470 - 502, 1931.

3. H.J. Briggs. "Physical Exertion, Fitness and Breathing". Journal of Physiology 54:292 - 312, 1920.

The volume of gas in each bag was determined by an accurately calibrated wet gas meter at constant pressure and rate of flow. The temperature was read off from a built in thermometer, and the barometric pressure noted. The volume of gas was then expressed in terms of N.T.P. after allowance was made for the gas taken for analyses.

Blood samples: In collecting blood during rest or while the subject was doing low work loads, it was necessary to keep his hand in water at 47° for a few minutes to hasten the blood flow. Thereafter his hand was dried thoroughly, a deep cut was made in his finger with an automatic spring-stiletto and after discarding the first drop of blood a sample was taken. The preliminary treatment with hot water was unnecessary during moderate and high work loads.

The blood sampling was done with a 0.5 ml. blood pipette, fitted with a rubber teat on top. A small screw clamp was fitted to the bulb of the teat and by manipulating the screw, fast and accurate adjustments could be made, while the operator had the blood in the pipette under control. With this arrangement a sample of blood could be taken in less than twenty seconds, depending on the supply of blood from the wound. Usual samples were between 0.3 and 0.5 ml and was immediately deproteinized by ice cold 20% trichloroacetic acid.

Analytical Methods: Gas; The Expired air was usually analyzed by the Haldane method while the Grollman samples containing the acetylene were analyzed on the same machine fitted with an extra bulb for absorption of the foreign gas. In carrying out the analysis, the carbon dioxide, acetylene, and oxygen are absorbed in the order named. For the carbon dioxide absorption a ten percent potassium hydroxide (KOH) solution is used. Following the absorption of carbon dioxide, the acetylene is absorbed in a reagent containing 20 percent mercuric cyanide ($Hg(CN)_2$ and 8 percent sodium hydroxide (NaOH). The oxygen is absorbed in an alkaline solution of pyrogallic acid. Duplicate analysis were made on all samples and a discrepancy of not more than 0.04 were permitted, else a third check was made.

Gas samples from subjects Ber., Jul. and Arm.were analyzed for oxygen and carbon-dioxide in the paramagnetic oxygen analyzer (Beckman, Model E2 nill-point analyzer with total range 12-22%). From the volume and composition of inspired and expired air, the rate of oxygen consumption was obtained for each period.

Blood Lactate: Lactic acid was determined by the highly specific method of Barker and Summerson (4) as modified by Hullin and Noble (5) and some minor modifications to make it more suitable for conditions, prevailing in the laboratory, at the time of the study.

Barker and Summerson described a micro method for lactic acid determinations in blood. By their procedure the blood is deproteinized with cold trichloroacetic acid, carbohydrate and interfering substances are next removed with cupric and calcium hydroxides. Lactic acid is oxidized to acetaldehyde by concentrated sulphuric acid, heated for five minutes in a boiling water bath. The colour reagent is para-hydroxydiphenyl which need 30 minutes at 30° Cent. for maximum colour development of which the excess is removed by placing in a boiling water bath for 90 seconds. The colour is read in a spectrophotometer at 560 mu.

When blood was analyzed according to this procedure it was found that duplicate samples checked poorly and reproducible results could not be achieved.

After this a modification of the original method by Hullin and Noble was tried out, which gave excellent results, which were reproduceable, but the method turned out to be too tedious and time consuming. The biggest objection against this method was the prescription of three successive copperlime treatments to remove

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4. S.B. Barker and W.H. Summerson. "The Colorimetric determination of lactic acid in biological material". Journal of Biological Chemistry, 138 : 535, 1941.

5. R.P. Hullin and R.L. Noble. "The Determination of Lactic Acid in Microgram Quantities". Biochemical Journal 55 : 289, 1953.

pyruvate or interfering substances. It was claimed by Ström [6] and later confirmed by Holmgren [7] that omitting the copper calcium treatment altogether, has no significant affect on determinations of lactic acid in blood.

In this study the copper-lime treatment was not repeated, the precipitate was not removed by centrifugation, but by vacuum filtration using sintered glass filters (No. 4). In addition the sulphuric acid was added by means of a volumetric pipette and not a burette.

Procedure: Lactic acid standard : Lithium lactate (mol.wt 95.94) was preferred because this salt is anhydrous and not hygroscopic. It was prepared as follows [8]: U.S.P. lactic acid (85 percent) was diluted with an equal volume of water and a few drops of phenol red indicator were added. Saturated 20 percent lithium hydroxide solution was added to slight excess, as is indicated by the phenol red. The solution was heated to boiling and the alkali was again added to slight alkalinity. It was then cooled, four volumes of 95% alcohol were added and after cooling, for some time, the mass of crystals was filtered off on a Buchner funnel and washed thoroughly with 95% alcohol. After being twice recrystallized from water it was dried at 100° Cent.

The stock standard: 0.2133 gm of the pure lithium lactate was dissolved in 100 ml water in a litre volumetric flask. To this was added 1 ml of concentrated sulphuric acid (analar reagent) and the volume was made up to the mark with water, and was thoroughly mixed. This solution contained 1 mg of lactic acid per 5 ml. The stock solution was further diluted to prepare the working standard.

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6. G. Ström. "The influence of anoxia on lactate utilization in man after prolonged muscular work." Acta Physiologica Scandinavica 17 : 440, 1949.

7. A. Holmgren. "Circulatory changes during muscular work in man". Scandinavian Journal of Clinical and Laboratory Investigation." 8 : Suppl. 24 pp 14 - 15 1956.

8. P.B. Hawk, B.L. Oser and W.H. Summerson. "Practical Physiological Chemistry". 13th Ed. p 75, London : J & A. Churchill, Ltd., 1954.

Procedure of analysis: 1 ml of the deproteinized blood solution was pipetted into a wide test tube containing 1 ml 20% (w/v) copper sulphate and the volume was made up to 10 ml with water. Approximately 1 gm of solid calcium hydroxide was added and was thoroughly mixed. The solution was left to stand for 30 minutes or longer before it was filtered through a No. 4 sintered glass filter.

1 Ml of the filtrate was transferred to a ground - glass stoppered Pyrex tube and 0.05 ml of 12 percent (w/v) solution $CuSo_4.5H_2O$ added, followed by 5.ml concentrated sulfuric acid with vigorous shaking. To prevent the solution from becoming too hot the lower end of the tube was immersed in an ice-water bath and the acid added very slowly. After addition, the tube was heated for 30 minutes in a water bath maintained at $60 \pm \frac{1}{2}^{\circ}$ whereafter it was cooled to $\pm 15^{\circ}$ C and the 0.1 ml, 1.5 percent p-hydroxydiphenyl reagent in alkaline solution was added. The precipitated p-hydroxydiphenyl was then thoroughly dispersed in the concentrated sulphuric acid and left in a water bath at 30° C for 20 minutes.

Once or twice during the 20 minutes interval the test tubes were agitated to redisperse the precipitated reagent, and then it was placed in vigorously boiling water for exactly 90 seconds. Thereafter it was cooled in an ice water bath, and the optical donsity of the resulting violet coloured solution was determined. For this a Beckman spectrophotometer was used at wavelength peak 560 mμ. against a set of reagent blanks, prepared by taking distilled water through the whole of the above procedures. Two different concentrations of standard solution, were analyzed in duplicate, in exactly the same way as the reagent blanks and from their value, the concentration of the unknown solution was determined. As the final dilution of the working standard should not contain more than 8 micro grammes per ml acid, it was adviseable to make up a fresh working standard every day.

Calculation: If the lactic acid concentration was plotted against the log percent transmittancy of the coloured standard solutions, a straight line was obtained, and from this calibration

curve, using the log percent transmittancy of the unknown solution, its concentration of lactic acid could be read off.

Miscellaneous information on the procedure:

1. Blood was analyzed as soon as possible after collection and was never kept for the next day.

2. The trichloroacetic acid was freshly prepared every week and was kept in the refrigerator.

3. If reproducibility and good agreement between duplicates are to be achieved, meticulous attention must be paid to detail and cleanliness of utensils.

Pyruvic acid determination: (Principles).

The blood proteins are precipitated by trichloroacetic acid and removed by centrifugation. Treatment of the clear supernatent with 2, 4 dinitrophenylhydrazine in acid solution the hydrazone of pyruvic acid is formed which with the excess phenylhydrazine is removed with toluene. The hydrazone is recovered from the toluene with aqueous sodium carbonate as the sodium salt, and this salt in strong alkaline solution is red coloured and is read on the photoelectric colorimeter.

The effect of anticoagulant and preservatives:

It is a well established fact that the pyruvate content of blood rapidly decreases on standing for a few minutes [9]. To prevent this rapid glycolysis taking place in blood certain preservatives were used e.a. iodoacetate and sodium fluoride [10].

The effects of these salts on the relation of lactate to pyruvate in blood were investigated by Bueding and Goldfarb [11].

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9. W.E. Huckabee. "Control of concentration gradients of pyruvate and lactate across cell membranes in blood". Journal of Applied Physiology. 9: 163, 1956.

10. E. Bueding and H. Wortis. "The stabilization and determination of pyruvic acid in the blood." Journal of Biological Chemistry. 133 : 585, 1940.

11. E. Bueding and W. Goldfarb. "The effect of NaF and sodiumiodoacetate on glycolysis in Human Blood". Journal of Biological Chemistry. 141 : 539, 1941.

Pyruvic acid was determined by the most commonly known method of Friedemann and Haugen (12). This method requires no blood anti-coagulant or preservatives like NaF or iodoacetate, and pyruvate determinations could very conveniently be done on the same deproteinized blood samples used for lactate estimations.

REAGENTS.

Stock Solution: Triple vacuum distilled pyruvic acid was used to make a stock solution with a final acidity of approximately 0.1 N.

Hydrazine Reagent: 100 mg were ground in a mortar with increasing small volumes of approximately 2N HCl until 100 ml have been added. The solution was filtered through a small filter paper and stored in a refrigerator.

Sodium Carbonate: A 10 percent solution was made by dissolving 10 gm in cool distilled water that has previously been thoroughly boiled. It was kept in a Pyrex bottle.

Sodium Hydroxide: A 1.5 N NaOH solution was made using thoroughly boiled water cooled down to dissolve the sodium hydroxide pellets. It was kept in a Pyrex bottle.

Calibration Curve: The standard solution was approximately diluted with 10 percent trichloroacetic acid so that 3 cc contained quantities from 0.5 micro grammes to 10 micro grammes. To establish the standard curve each different dilution was determined in triplicate. Three ml samples were taken and treated similar to the procedure followed in deproteinized blood solutions.

Procedure: Three ml of the clear supernatant solution, left over from the lactate determinations, was transferred to a 18 x 150 mm test tube. After 0.2 cc of the hydrazine reagent was added the solution stood for exactly five minutes. The formed hydrazones were extracted by adding 4 cc of toluene whereafter a rapid stream of nitrogen was passed through the mixture for two minutes. The aqueous lower layer was then removed while the liquid adhering

to the walls was dislodged by a sudden circular movement of the test tube. After removing the lower layer, 6 cc of sodium carbonate was added and the solution was again aerated with Nitrogen for two minutes. Only after the two layers of liquid were distinctly seperated was a 5 ml sample taken from the lower layer with the necessary precautions of not contaminating the sample with traces of the top layer.

Exactly 5 cc of a 1.5 N NaOH was added to 5 cc sample and was thoroughly mixed. The reading was done on a Beckman spectrophotometer at wavelength 520 mμ after 5 - 10 minutes. All blood samples were analyzed in duplicate and the mean of the two readings was used in the calculation. This was done in a similar way as that for lactate except that a different factor was used allowing for the difference in dilutions.

BICYCLE ERGOMETER.

A bicycle ergometer equipped with an electro-dynamic brake was used as the medium of exercise. A wooden chair, with arm rests, was so fixed and rigged up that it could serve as an adjustable bicycle seat. The seat was fastened to the back of the pedals and the seat height was about even with the axle of the pedal sprocket. The subject sat in the chair with his back well braced against the back support and arms resting on the arm supports thus enabling him to keep his upper body and arms in a fairly quiet and motionless position while pedalling.

The principle of the electrodynamic brake itself resides in the fact that a seperately excited direct current generator serves as load. The armature of this generator is connected to external load resistors. When pedalling the ergometer, the armature is rotated at speed and a current is generated within it.

This current dissipates electrical energy into the load resistors. By increasing the field current, the magnetic resistance to the rotation of the armature is increased and provided the speed is constant, the current generated will be proportionally greater.

More energy will, however, be needed to rotate the armature (and more electrical energy will be dissipated in the load resistors..) The field current can thus be regarded as a direct control of the energy dissipated in the load resistors.

CALIBRATION OF THE BICYCLE ERGOMETER.

NOMENCLATURE:

Applied voltage or terminal voltage	V volts
Induced emf	E_o "
Volt drop due to armature resistance	V_a "
Armature current	I_a amps
Field current	I_f "
Armature resistance and brushes	R_a ohms
Input power	W watts
Power dissipated in load	W_L "
Power loss in R_a	W_{aR} "
Power loss due to iron	W_I "
Power loss due to mechanical	W_M "
Load resistance	R_L ohms

CIRCUIT DIAGRAM:

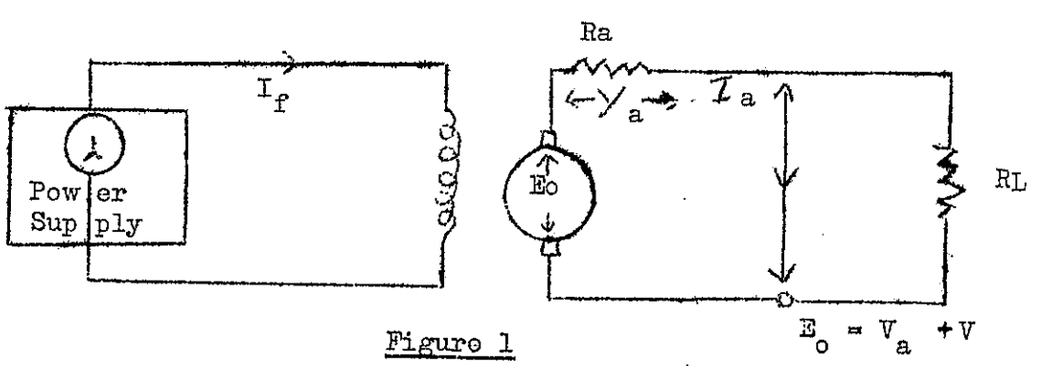

Figure 1

When the armature is rotated, an emf. E_o is induced, which is a function of I_f. By increasing I_f, E_o is increased. Energy is dissipated by connecting a load resistor R_L across the terminals. The total resistance in series consists of R_L and R_a. Therefore, by increasing I_f (from a separate power supply) one increases the load imposed on the armature of the generator. The total losses, or amount of energy dissipated consists of the following :-

a. Mechanical losses - friction.

b. Resistance losses due to resistance of armature and brushes.

c. Iron losses due to hysteresis and oddy current.

d. Resistance losses due to external load resister.

METHOD OF OBTAINING LOSSES.

1. Armature and Brush Loss - W_a

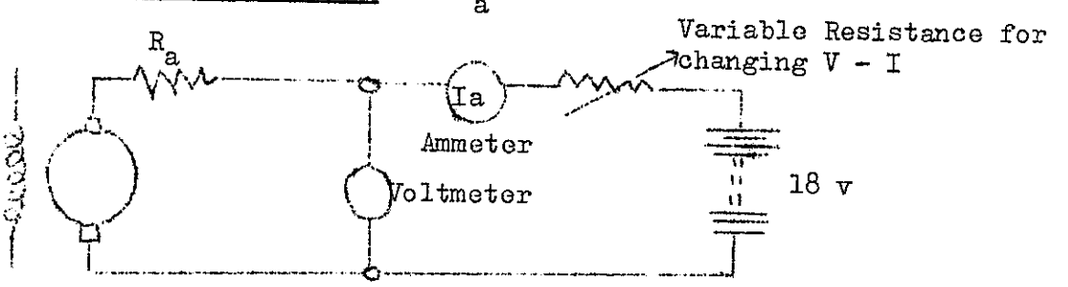

Figure 2

A number of readings for V and I_a are taken for three posistions of the armature

Arm. Resist. $R_a = V/I_a$

R_a varies with I_a, so a graph is plotted of $R_a - I_a$. (1)

2. Mechanical and Iron Losses

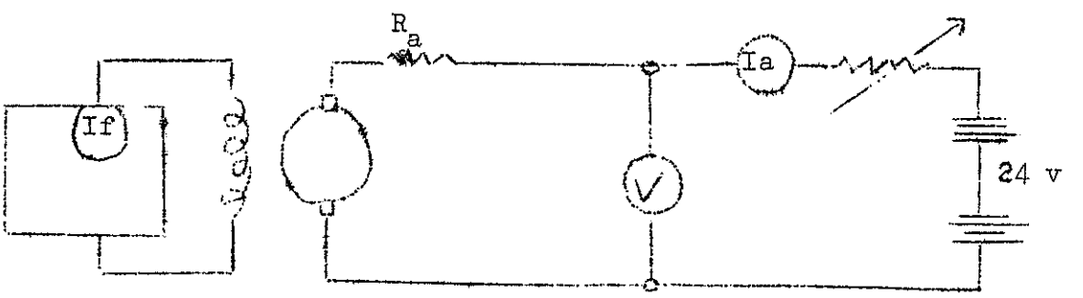

Figure 3

The machine is run as a meter at constant speed (70 r.p.m.). It is altered for various values of V, to maintain a constant speed. I_f, V and I_a are noted.

Input power $W = VI_a$.

From graph in (1) armature resistance R_a is obtained for each value of I_a.

Loss due to $R_a = I_a^2 R_a = W_{aR}$.

Therefore mechanical + iron loss

$W_M + W_I = W - W_{aR}$

$W_M + W_I = VI_a = I_a^2 R_a$ watts ⟶

Iron loss is known to have the following relation with flux intensity or field current (Figure 4).

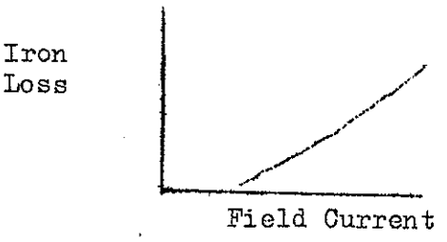

Figure 4

A graph (2) of $W_M + W_I$ is plotted against I_f and has the following shape (Figure 5).

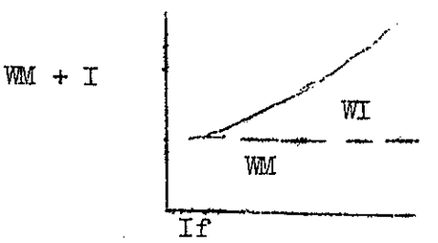

Figure 5

Since the machine was run at constant speed, the lower section, which is constant, is the mechanical loss and the upper is the iron loss.

RESISTANCE LOSS OF EXTERNAL LOAD.

Power loss = $I_a^2 R$ watts. ($R = \frac{1}{2}$ ohm). This is used in the calibrating test below.

Calibrating Test:

With the normal circuit as in Figure 1, a voltmeter is connected across the terminals. The ergometer is pedalled at constant speed. The terminal voltage is noted at each step for the whole range of I_f from 0 to 4 amps.

1. Knowing the load resistance $R_L = \frac{1}{2}$ ohm,
   Armature current = $V/R_L = I_a$.
2. From graph (1) the armature resistance is obtained, R_a, knowing I_a.
3. From graph (2) the mechanical and iron loss ($W_M + W_I$) is obtained, knowing the value of I_f at each step.
4. Armature losses = $I_a^2 R_a$.
5. External load loss = $I_a^2 R_L$.
6. Total loss is (3) + (4) + (5).

A graph of the total load can now be plotted against field current. This then is the final calibration for a particular speed.

Note

As an indication of work rate, a recording voltmeter can be connected across the terminals.

CLIMATIC ROOM (13).

The climatic chamber is built in the form of a return flow wind tunnel with two working sections, ten feet cube, forming the test chambers, one for horizontal flow and the other for vertical upward flow. In each of the two test chambers the walls normal to the direction of air flow are built in the form of bar-grid panels so as to allow the air to flow through them, yet at the same time enabling the mean radiant temperatures to be controlled to within the specified limits.

In the two test chambers the environment can be controlled to give any combination of values of the four variables involved between the following limits :-

(a) Air temperature from 40° F to 130° F.

(b) Air velocity from 20 to 1000 feet per minute.

(c) The mean radiant temperature of each of the six enclosing surfaces can independently vary from 30°F below to 30°F above air temperature.

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13. W.L. Grant. "The design and development of a climatic chamber for the study of human reactions under different environmental conditions". Journal of the S.A. Institution of Mechanical Engineers, Dec. 1954, 133 - 206.

CHAPTER 1V

RESULTS.

Observations were made of the pulse rate, oxygen consumption, blood lactate and pyruvate at different levels of work, ranging from 2500 ft lbs/min. up to each individuals maximum work load. As repeated observations were made at the same level of work, the results are presented in terms of the mean values obtained for each individual at each work load. The data are summarized in Tables 111 (a) to V111 ~~(b)~~ and are also graphically represented in figures 1 to 27. To most of the data curves were fitted as well as the calculated 95% confidence limits.

The mean values for pulse rates obtained for seven subjects are given in Table 111(a) while the pulse rates obtained for three subjects in the heat are summarized in Table 111(b).

In Table 1V(a) are presented the oxygen consumption values obtained under normal conditions, while in Table 1V(b) values are given for humid heat.

Table V(a) presents mean values for blood lactate found under normal conditions of temperature, while Table V(b) presents the "hot" values.

In Table V1(a) and (b) are shown the blood pyruvate values found under normal conditions and in severe heat.

Values for cardiac output are represented in Table V11, and represent data for two subjects tested under both environments.

The rectal temperatures recorded for two subjects are given in ~~Table V111~~ Fig. 23. In figure 23, a comparison is made between the temperature response to work performed in heat and cold for subjects Zef. and Den. A comparison between the work rates is not justifiable, because the duration of exercise varied with the intensity of the work load. However, the duration of exercise for work intensities between 2500 ft Lbs/min. and 8000 ft lbs/min. were similar and can therefore be compared. It is obvious that rectal temperature increase with increase in work load. The striking

feature of the graph is that the rectal temperature of both subjects observed in the heat are significantly higher than for the cold. The small differences found at the higher work loads, especially in the case of subject Zef, can only be ascribed to the time of exposure to heat, being too short. It is also important to note that the "hot" curve of Den. in comparison with subject Zef's curve is significantly higher at all intensities of work load.

TABLE 1.

PHYSICAL CHARACTERISTICS OF TEST SUBJECTS:

Subject	Height (ins.)	Weight (lbs. ozs)
BER.	65	145 - 6
JUL.	63	128 - 15
ARM.	61	116 - 10
ANT.	-	136 - 15
DEN.	-	137 - 10
ZEF.	64¾	127 - 14
VIC.	64	123 - 9

TABLE 11.

RESTING VALUES FOR SEVEN SUBJECTS:

SUBJECT	LACTATE mg %		PYRUVATE mg %
	N	Mean	Mean
BER.	5	12.6	1.01
JUL.	5	9.7	1.04
ARM.	5	11.0	1.06
ANT.	17	14.4	-
DEN.	23	12. 4	-
ZEF.	11	14.4	1.80
VIC.	15	14.3	1.45

TABLE 11(b).

RESTING VALUES : PREVIOUS RESULTS.

	AUTHORS.	VALUES Lactate.	REFERENCES.
1.	Edwards (1938)	7.2 to 9.9 mg%	J. Biol. Chem. 125:571
2.	Crescitelli & Taylor (1944)	12.4 and 28.5 mg%	Amer. J. Physiol. 141:630
3.	Jervell (1928)	20 to 30 mg%	Acta Med. Scand. Suppl. 22-25
4.	Goldsmith (1948)	9.3 mg%	Amer. Journal of Med. Sciences 215:182
5.	Asmussen (1950)	10 to 15 mg%	Acta Physiol. Scand. 20-125.
6.	Bock et al (1932)	9.0 mg%	Journal of Clin. Invest. 11:775
7.	Robinson et al (1941)	18.0 mg%	Amer. J. Physiol. 21:261
8.	Wells et al (1957)	16.5 mg%	Journal of Applied Physiology 10:51
9.	Holmgren (1956)	10.1 to 10.9 mg%	Scand. J. Clin. Lab. Invest. Vol. 8; Suppl. 24 p.1-97.
10.	Present study average 7 subjects.	12.7 mg%	

TABLE 111(a).

MEAN HEART RATES ~~DYRUBG~~ During CYCLING AT DIFFERENT WORKING INTENSITIES : ROOM TEMPERATURE.

B E R .			J U L .			A R M.			A N T .				D E N .				V I C				Z E F .			
Work Rate	N	Mean	Work Rate	N	Mean	Work Rate	N	Mean	Work Rate	N	Mean	S.D.	Work Rate	N	Mean	S.D.	Work Rate	N	Mean	S.D.	Work Rate	N	Mean	S.D.
3000	2	93	3000	3	82	3000	3	116	1500	6	91.2	16.8	1500	4	80.8	6.8	5000	5	111.2	9.0	2500	5	97.2	5.6
5500	1	118	5500	3	115	5500	4	150	5000	8	153.4	13.3	5000	5	120.8	3.4	7500	7	147.7	5.6	5000	5	126.2	9.8
6700	2	138	6700	2	137	6700	4	168	7000	9	171.7	6.5	7000	9	144.3	6.2	8500	2	166.5	3.5	7500	3	170.6	10.1
7500	2	137	7500	4	148	7500	2	184	7500	7	175.9	7.2	8000	6	159.3	2.5	9000	6	166.8	8.2	9000	5	177.6	6.8
9000	4	160	9000	3	162	9000	2	178	8000	7	179.9	4.1	9000	5	169.0	.7	10000	7	167.0	7.5	10000	1	192.0	-
10000	4	155	10000	1	178				8500	8	186.1	4.5	9500	6	171.5	2.2	10500	3	178.0	5.8	10500	2	187.0	1.4
11750	4	163	11000	3	171				9000	5	189.8	1.3	10000	5	177.2	4.2	11000	1	176.0	-				
									9500	3	190.7	1.6	10500	5	178.6	3.3								
													11000	5	178.0	5.0								

TABLE 111(b).

MEAN HEART RATES DURING CYCLING AT DIFFERENT WORKING INTENSITIES : HOT ENVIRONMENT.

D E N .				V I C .				Z E F .			
Work Rate	N	Mean	S.D.	Work Rate	N	Mean	S.D.	Work Rate	N	Mean	S.D.
1800	3	103.0	11.4	1800	4	90.3	3.8	1800	4	114.0	4.2
5000	5	138.8	11.4	5000	6	130.5	6.1	5000	4	146.7	1.5
7000	6	167.2	5.9	7000	7	152.4	11.2	7000	7	171.4	5.4
8000	6	173.7	7.2	8000	6	165.0	4.9	8000	6	182.2	6.2
9000	5	182.6	5.6	9000	6	171.5	4.6	9000	7	187.0	4.4
9500	6	183.3	7.0	9500	5	178.2	4.5	9500	6	185.0	5.4
10000	5	188.0	2.7	10000	4	180.5	4.2	10000	7	185.1	5.2
10500	6	189.7	7.7	10500	5	181.2	3.3	10500	10	189.9	5.0
11000	8	192.9	4.5	11000	6	181.0	3.8	11000	3	187.3	4.0
11500				11500	4	183.0	3.2	11500	2	188.0	1.4

TABLE IV (a).

OXYGEN INTAKE DURING WORK ON THE BICYCLE ERGOMETER : ROOM TEMPERATURE.

BER.			JUL.			ARM.			ANT.				DEN.				VIC.				ZEF.			
Work Rate.	N	Mean	Work Rate	N	Mean	Work Rate	N	Mean	Work Rate	N	Mean	S.D.	Work Rate	N	Mean	S.D.	Work Rate	N	Mean	S.D.	Work Rate	N	Mean	S.D.
3000	4	1.0132	3000	4	1.0214	3000	5	1.0063	1500	6	.807	.089	1500	4	.624	.045	2500	7	.918	.074	2500	7	1.012	.091
5500	6	1.6310	5500	6	1.7150	5500	5	1.7650	5000	8	1.876	.166	5000	5	1.837	.177	5000	8	1.579	.173	5000	7	1.594	.102
6700	4	1.9686	6700	2	1.9103	6700	4	1.9315	7000	10	2.214	.137	7000	8	2.257	.156	7500	10	2.400	.184	7500	9	2.483	.204
7500	3	2.2250	7500	6	2.1591	7500	4	2.2559	7500	7	2.211	.134	8000	6	2.372	.097	8500	2	2.765	.205	9000	13	2.809	.175
9000	3	2.4960	9000	4	2.4987	9000	4	2.4006	8000	6	2.338	.090	9000	5	2.521	.039	9000	8	2.818	.256	10000	18	2.912	.182
10000	5	2.7677	10000	5	2.7942	10000	2	2.455	8500	8	2.480	.207	9500	6	2.768	.098	10000	13	2.952	.151	10500	8	3.062	.164
11750	7	3.0822	11000	5	2.7832				9000	7	2.593	.058	10000	5	2.836	.135	10500	16	2.965	.197	11000	8	3.127	.159
									9500	7	2.583	.187	10500	7	2.829	.240	11000	12	3.018	.120	11500	7	3.221	.131
													11000	7	2.840	.175	11500	11	2.983	.127				

TABLE 1V(b).

OXYGEN INTAKE DURING WORK ON THE BICYCLE ERGOMETER : HOT ENVIRONMENT.

D E N .				V I C .				Z E F .			
Work Rate	N.	Mean	S.D.	Work Rate	N	Mean	S.D.	Work Rate	N	Mean	S.D.
1800	3	.562	.169	1800	4	.668	.043	1800	4	.654	.045
5000	4	1.518	.084	5000	5	1.417	.096	5000	3	1.237	.243
7000	5	2.103	.102	7000	8	1.864	.156	7000	5	1.986	.186
8000	6	2.256	.071	8000	5	2.182	.023	8000	6	2.161	.102
9000	5	2.558	.095	9000	6	2.609	.233	9000	6	2.315	.165
9500	5	2.549	.153	9500	6	2.559	.056	9500	6	2.417	.238
10000	4	2.783	.103	10000	4	2.801	.199	10000	6	2.406	.106
10500	6	2.668	.186	10500	6	2.823	.187	10500	10	2.704	.220
11000	9	2.880	.233	11000	4	2.748	.110	11000	6	2.700	.193
11500	4	2.700	.086	11500	7	2.883	.127	11500	5	2.880	.223

TABLE V (a).

THE MEAN BLOOD LACTATE CONCENTRATION FOR SEVEN SUBJECTS : ROOM TEMPERATURE.

B E R .				J U L .				A R N .				A N T .			
Work Rate	N	Mean	S.D.	Work Rate	N	Mean	S.D.	Work Rate	N	Mean	S.D.	Work Rate	N	Mean	S.D.
3000 5500 6700	10	20.4	8.3	3000 5500	7	21.2	6.5	3000	4	18.0	3.9	1800 3000	6	17.3	5.2
7500	4	30.9	1.5	6700	3	25.9	4.8	4700	2	30.6	.85	5000	3	31.0	5.7
9000	5	66.2	7.1	7500	6	42.6	7.6	5500	4	44.9	7.9	7000	3	65.3	5.0
10000	5	74.0	7.8	9000	4	73.4	3.1	6700	6	71.9	8.4	9000	2	103.1	6.2
11600	7	101.2	5.7	10000	4	90.3	3.9	7500	3	109.0	7.7				
				11000	4	103.3	8.9	9000	2	131.3	7.4				

D E N .				V I C .				Z E F			
Work Rate	N	Mean	S.D.	Work Rate	N	Mean	S.D.	Work Rate	N	Mean	S.D.
2500	5	17.2	3.6	2500	3	11.8	3.3	2500 5000	9	21.5	2.6
5000	3	23.9	6.7	5000	5	17.3	1.9	7000	4	31.9	2.6
7000	5	45.1	11.4	6000	3	22.5	2.3	7250	6	44.9	4.7
8000	4	66.8	4.9	7000	6	29.6	2.7	7700	2	67.0	4.2
9000	4	90.1	14.6	8000	3	84.5	6.3	8000	4	76.4	4.5
10000	3	109.3	12.0	9000	5	113.0	8.1	9000	2	109.3	7.4
10500	2	107.5	3.5	10000	7	124.6	10.0	9300	2	115.5	.7
11000	4	108.3	4.0	10500	3	138.2	10.9	10000	4	123.8	2.3
				11000	3	140.5	9.9	10900	2	129.5	9.2
								11000	3	133.8	12.6

TABLE V(b).

MEAN BLOOD LACTATE VALUES : HOT ROOM (TEMPERATURE 92° F.)

D E N				V I C				Z E F			
Work Rate	N	Mean	S.D.	Work Rate	N	Mean	S.D.	Work Rate	N	Mean	S.D.
1800 2500 4000	3	17.2	3.2	2500 3500	4	12.4	2.4	2500 3600	5	11.8	2.7
5000	3	19.2	3.2	5000	4	16.3	4.5	5000	4	18.5	1.5
7000	5	43.1	10.2	5250	2	28.8	6.7	7000	6	58.7	3.4
8000	4	77.1	13.4	6500	2	48.5	9.2	8000	3	89.7	13.5
9500	2	88.0	12.7	7000	4	38.9	10.3	9000	5	105.5	3.5
10000	4	105.4	1.9	8300	2	88.5	9.2	9500	5	114.4	13.8
11000	2	115.0	4.2	9000	5	104.6	5.4	10000	7	113.2	9.5
				9500	4	113.0	3.8	10400	3	116.0	5.0
				10000	2	113.0	4.2	10500	4	126.1	7.9
				10500	3	117.9	.9	11000	3	129.0	10.8

TABLE VI(a)

MEAN BLOOD PYRUVATE VALUES WITH PROGRESSIVE WORK LOADS : ROOM TEMPERATURE.

B E R .				J U L .				A R M .				V I C .				Z E F .			
Work Rate	N	Mean	S.D.	Work Rate	N	Mean	S.D.	Work Rate	N	Mean	S.D.	Work Rate	N	Mean	S.D.	Work Rate	N	Mean	S.D.
3000 5500	5	.92	.29	3000 5500	6	1.51	.31	3000 4700	4	1.31	.35	2500	4	1.97	.38	2500	5	1.55	.40
6700	3	1.00	.29	6700	2	2.11	1.25	5500	5	1.98	.38	5000	3	1.92	.33	5000	4	2.21	.22
9000	3	2.73	.05	7500	4	2.90	.51	6700	5	2.69	.86	6000	3	2.53	.06	7000	5	3.52	.33
10000	3	3.70	.43	9000	4	3.44	.41	7500	2	2.45	.21	7000	6	3.02	.50	8000	6	4.19	.38
11600	5	4.63	.16	11000	5	4.32	.66	9000	3	3.78	.39	8000	5	4.23	.30	9000	5	4.66	.11
												9000	3	4.94	.49	10000	5	5.27	.22
												10000	5	5.09	.78	11000	4	5.80	.41
												10500	2	5.70	.47				
												11000	3	5.86	.53				

TABLE V1(b).

MEAN BLOOD PYRUVATE VALUES FOR TWO SUBJECTS : HOT ROOM (TEMPERATURE 92° F. WET BULB).

V I C .				Z E F .			
Work Rate	N	Mean	S.D.	Work Rate	N	Mean	S.D.
2500	3	1.49	.11	2500	3	2.02	.59
5000	2	1.96	.30	5000	2	2.05	.17
7000	3	3.01	.20	7000	2	3.23	.-
8000	3	3.33	.35	8000	2	3.52	.02
9000	3	4.25	.32	9000	3	3.96	.06
10000	4	4.86	.61	9500	3	4.03	.02
10500	5	4.53	.54	10000	3	4.44	.16
				10500	5	5.30	.34
				11000	2	5.06	.64

TABLE V11

CARDIAC OUTPUT FOR TWO SUBJECTS AT DIFFERENT ENVIRONMENTAL TEMPERATURES.

Z E F .			D E N .		
	COLD	HOT			
Work Rate	Litres/Min. Cardiac Output.	Cardiac Output.	Work Rate	Litres/Min. Cardiac Output.	Litres/Min. Cardiac Output.
1800	-	10.30	1800	7.00	8.0
2500	9.70	-		-	
5000	14.00	14.20	5000	15.50	15.70
7000	17.25	16.25	7000	19.00	18.75
			8000	20.50	20.20
8000	18.70	17.25	9000	21.70	21.00
9000	19.80	18.25	9500	22.20	21.20
			10000	22.50	21.40
10000	20.05	19.00	10500	22.60	21.40
10500	-	19.50	11000	22.70	21.40
11000	20.70	20.00			
11500	-	20.30			

TABLE V11 (b)

BLOOD LACTATE/PYRUVATE RATIO FOR FIVE SUBJECTS : ROOM TEMPERATURE.

REST.

		2,500	3,000	4,700	5,000	5,500	6,700	7,000	7,500	8,000	9,000	10,000	11,000
ARM.	10.3		13.7	23.3		22.6	26.7		44.5		34.7		
BER.	12.4		22.1			22.1	20.4				24.2	20.0	21.8
JUL.	9.3		14.0			14.0	12.2		14.7		21.3		23.9
VIC.	9.8	6.0	9.0		8.9	9.8		19.9		22.9	24.5	24.3	24.0
ZEF.	8.0	13.9			9.7			9.1		18.2	23.5	23.5	23.1

TABLE V111

THE WORK RATE, OXYGEN CONSUMPTION, HEART RATE AT WHICH THE "THRESHOLD VALUE" FOR LACTATE WAS REACHED FOR EACH OF THE SEVEN SUBJECTS:

Subject.	Work Rate Ft.lbs/min	Oxygen L/min.	Heart Rate Beats/min.	Capacity % of Aerobic.	Max. Aerob.Cap. L/Min.
Den	4750	1.59	120	53%	3.0
Arm	4400	1.52	137	61%	2.5
Ber.	6600	1.95	140	63%	3.1
Jul.	6300	1.82	132	65%	2.8
Ant.	4300	1.75	140	67%	2.6
Zef.	6750	2.20	155	68%	3.2
Vic.	6000	2.05	130	66%	3.1

FIG. 1. SUBJECT DEN.

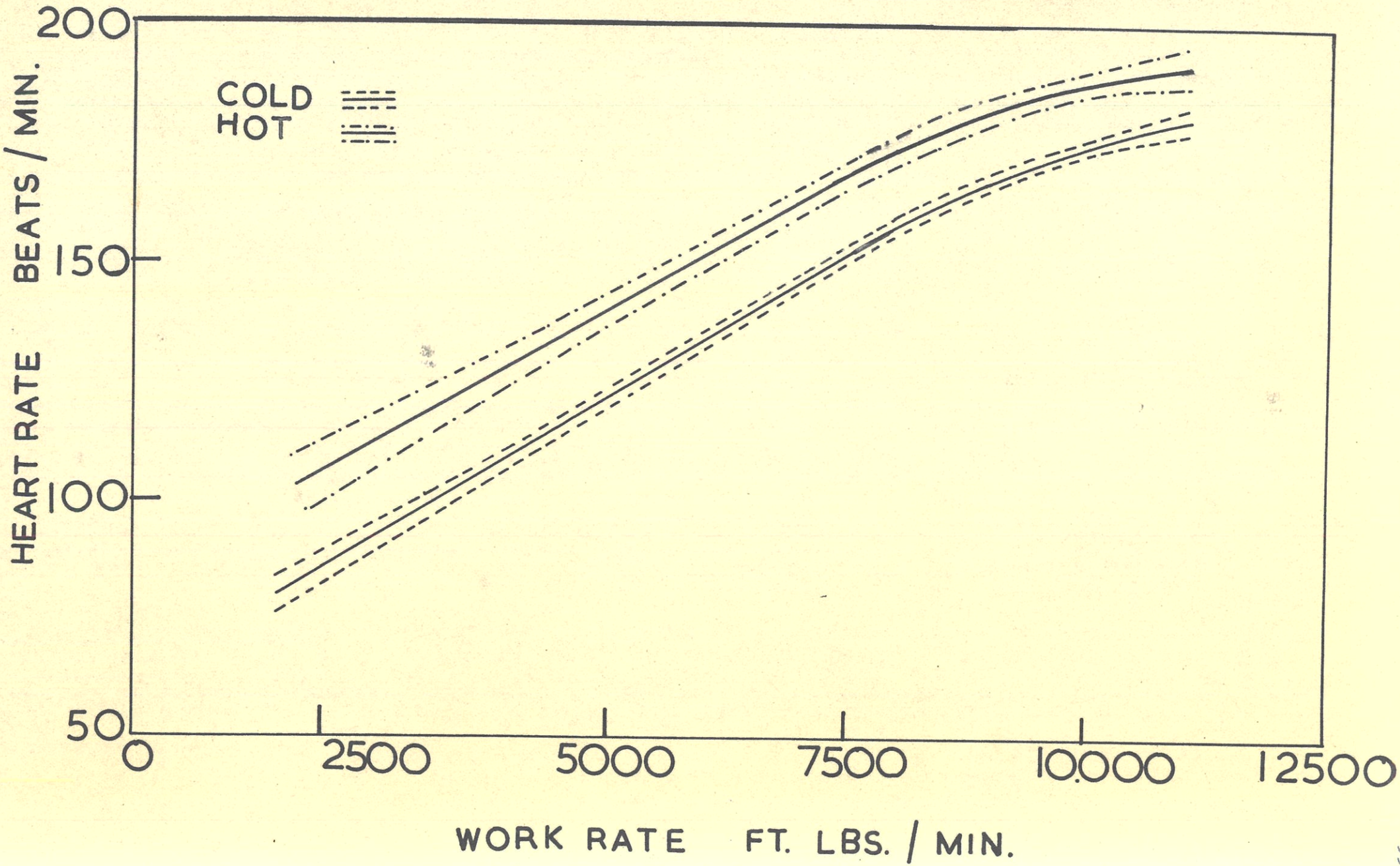


FIG. 2. SUBJECT VIC.

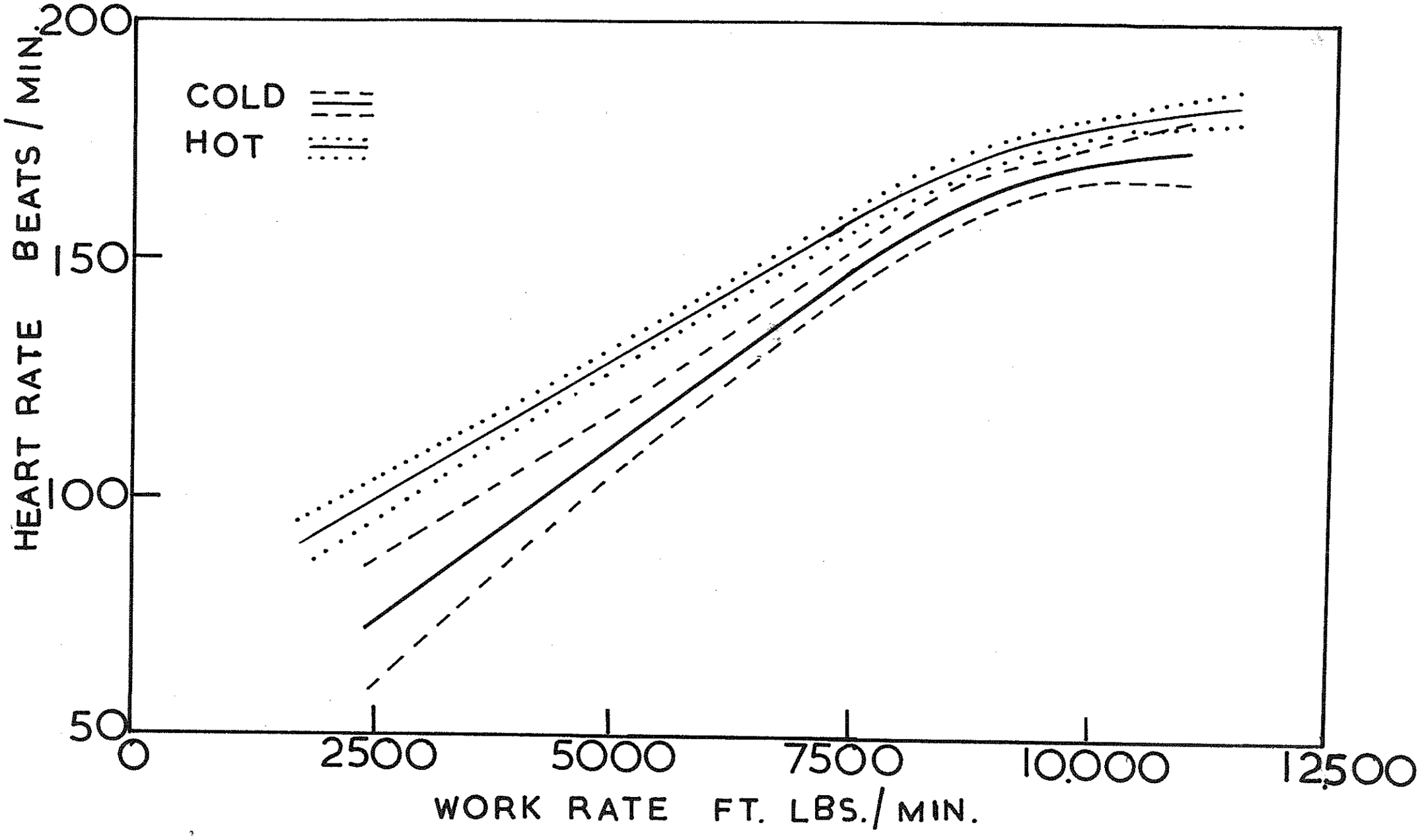


FIG 3. SUBJECT ZEF

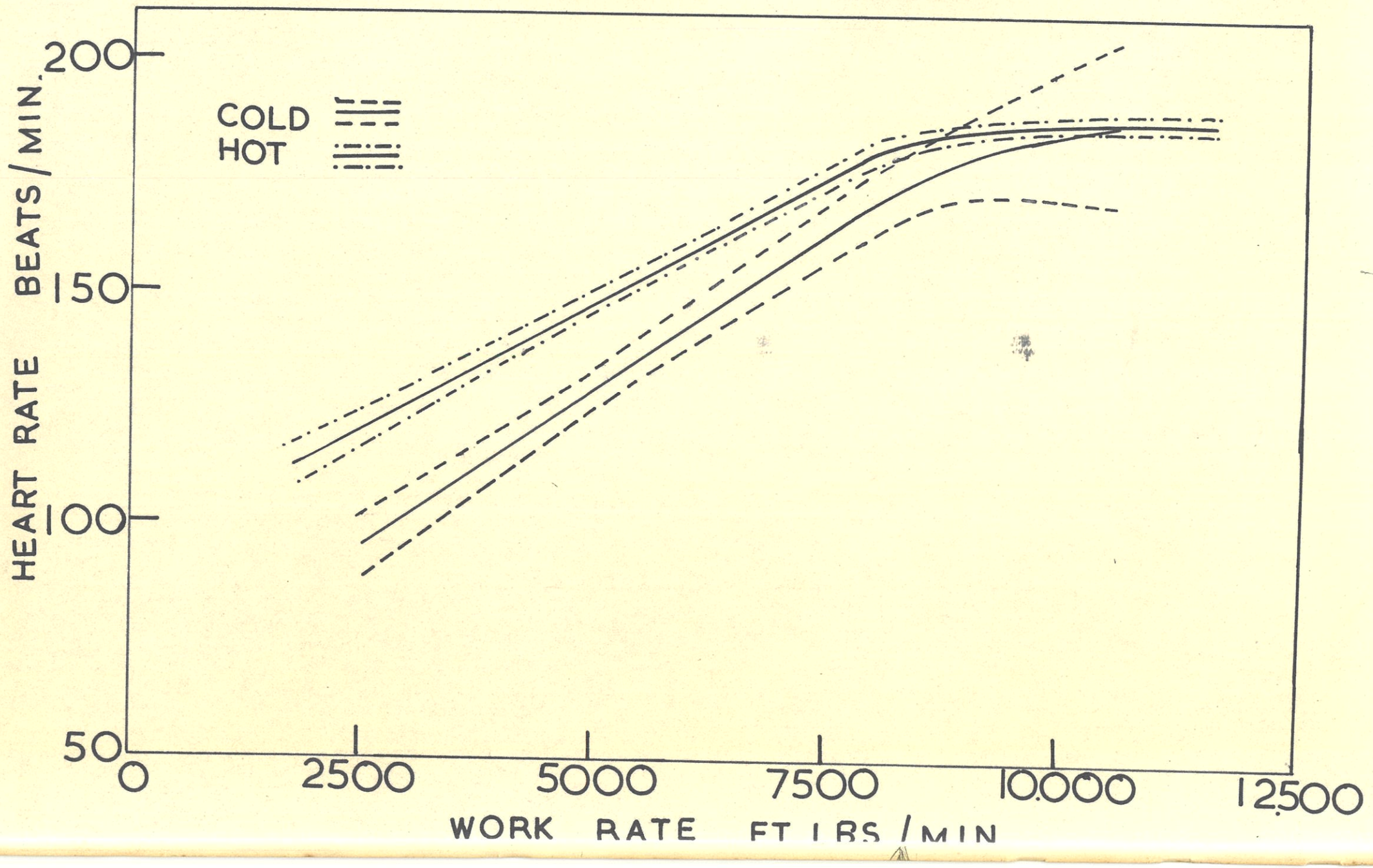


FIG. 4. SUBJECT DEN.

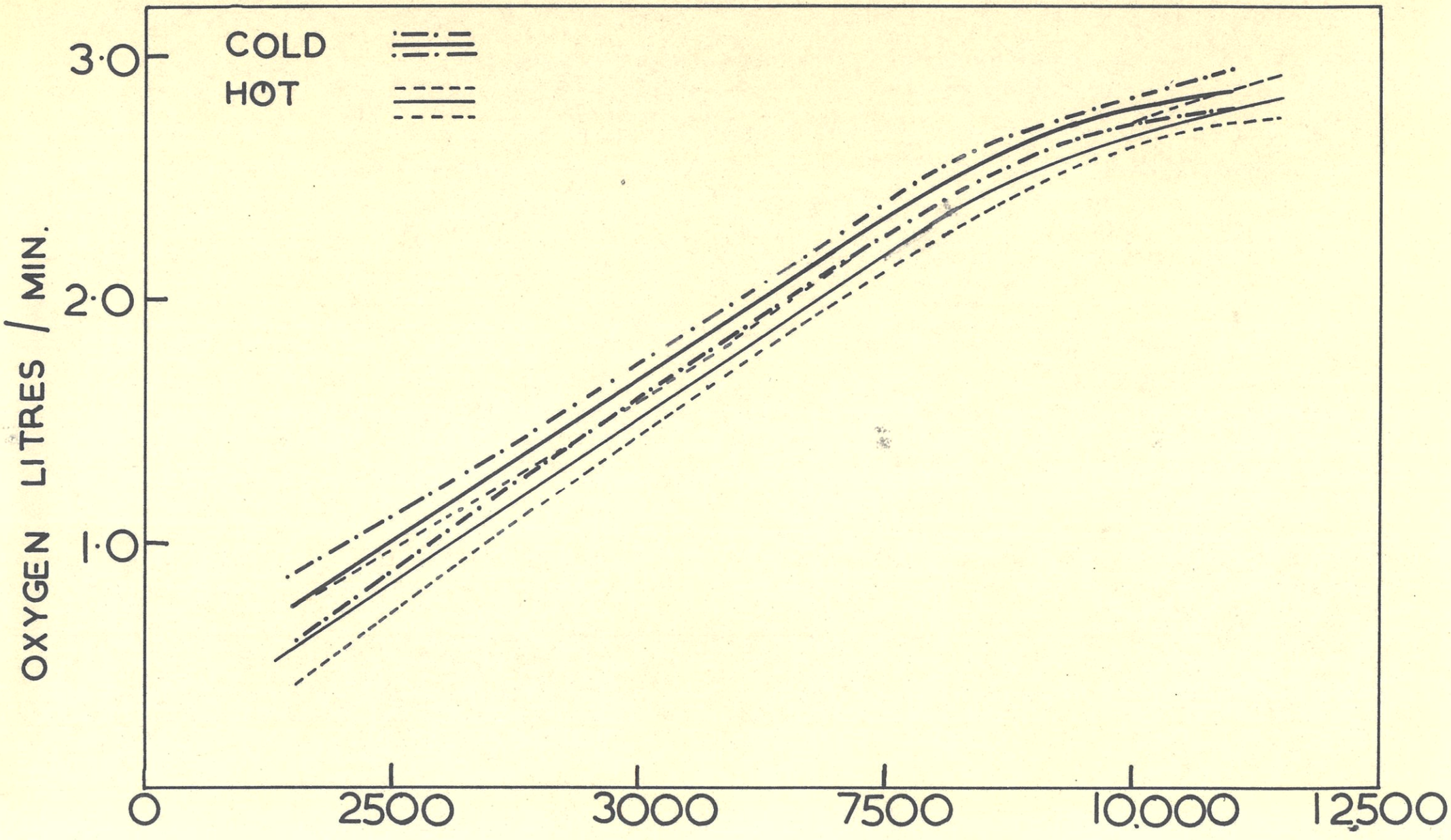
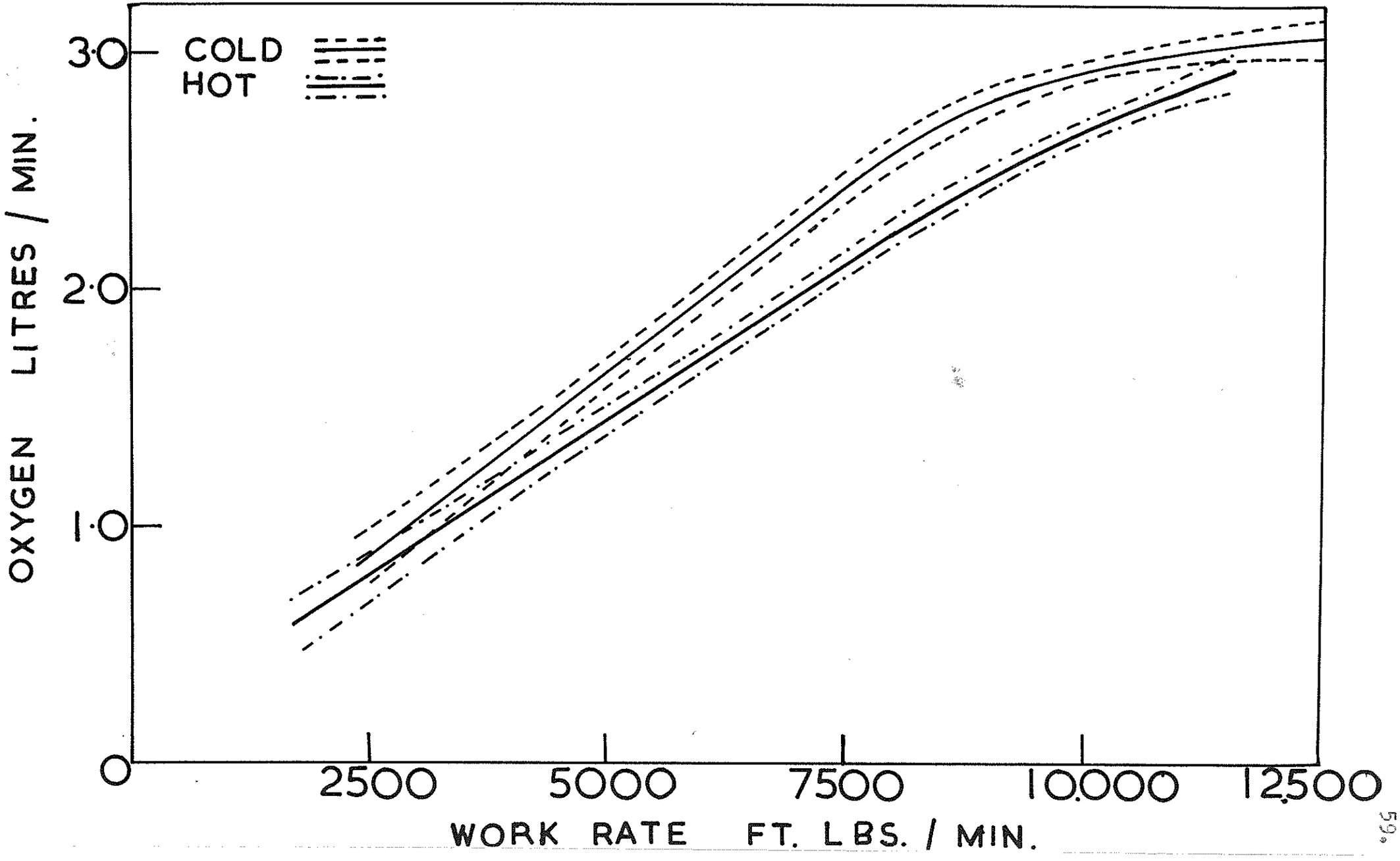


FIG. 5. SUBJE CT VIC.



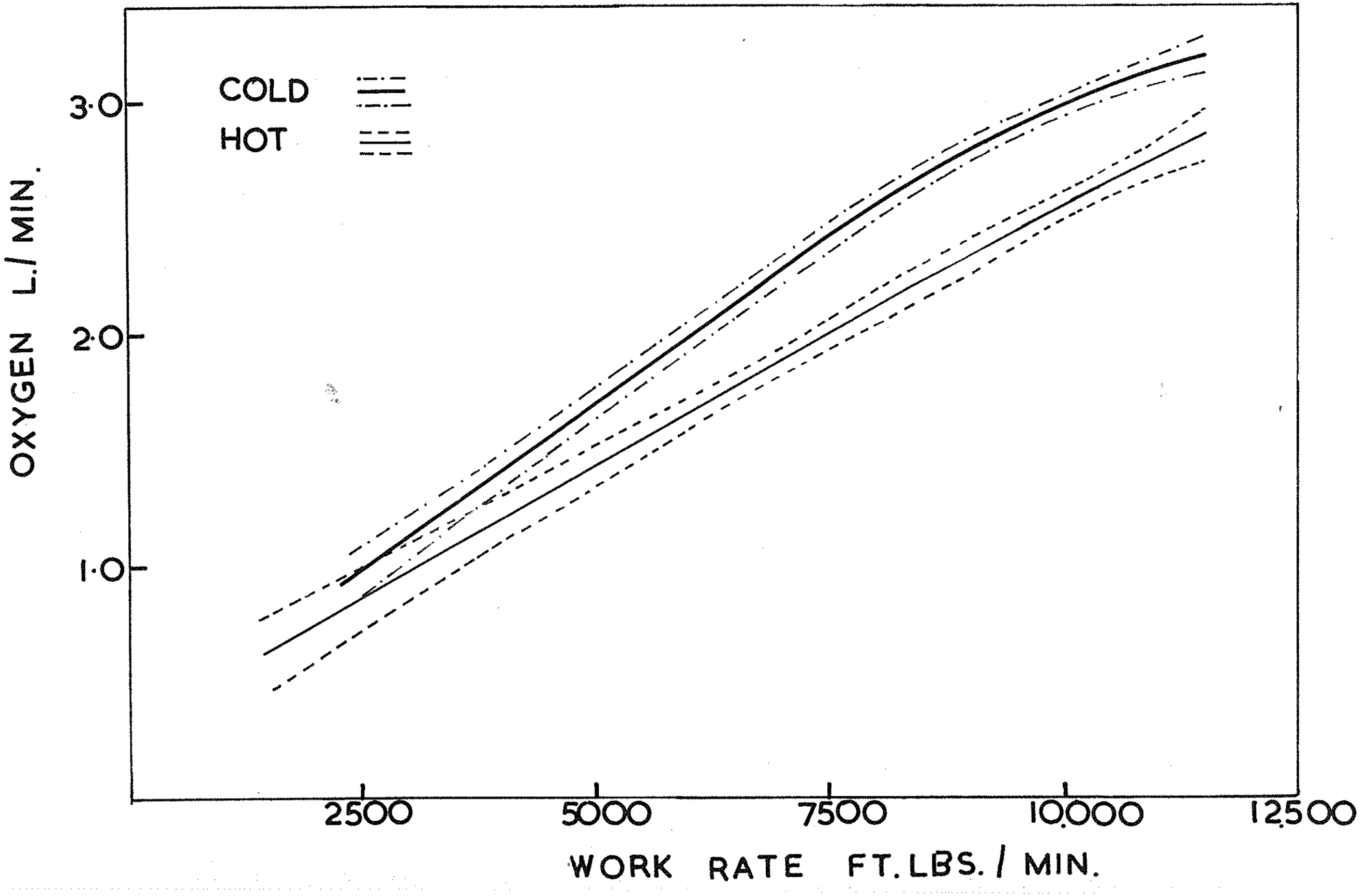

COLD
HOT
3·0
2·0
1·0
OXYGEN L./ MIN.
2500
5000
7500
10000
12500
WORK RATE FT. LBS. / MIN.

FIG. 7. SUBJECT JUL.

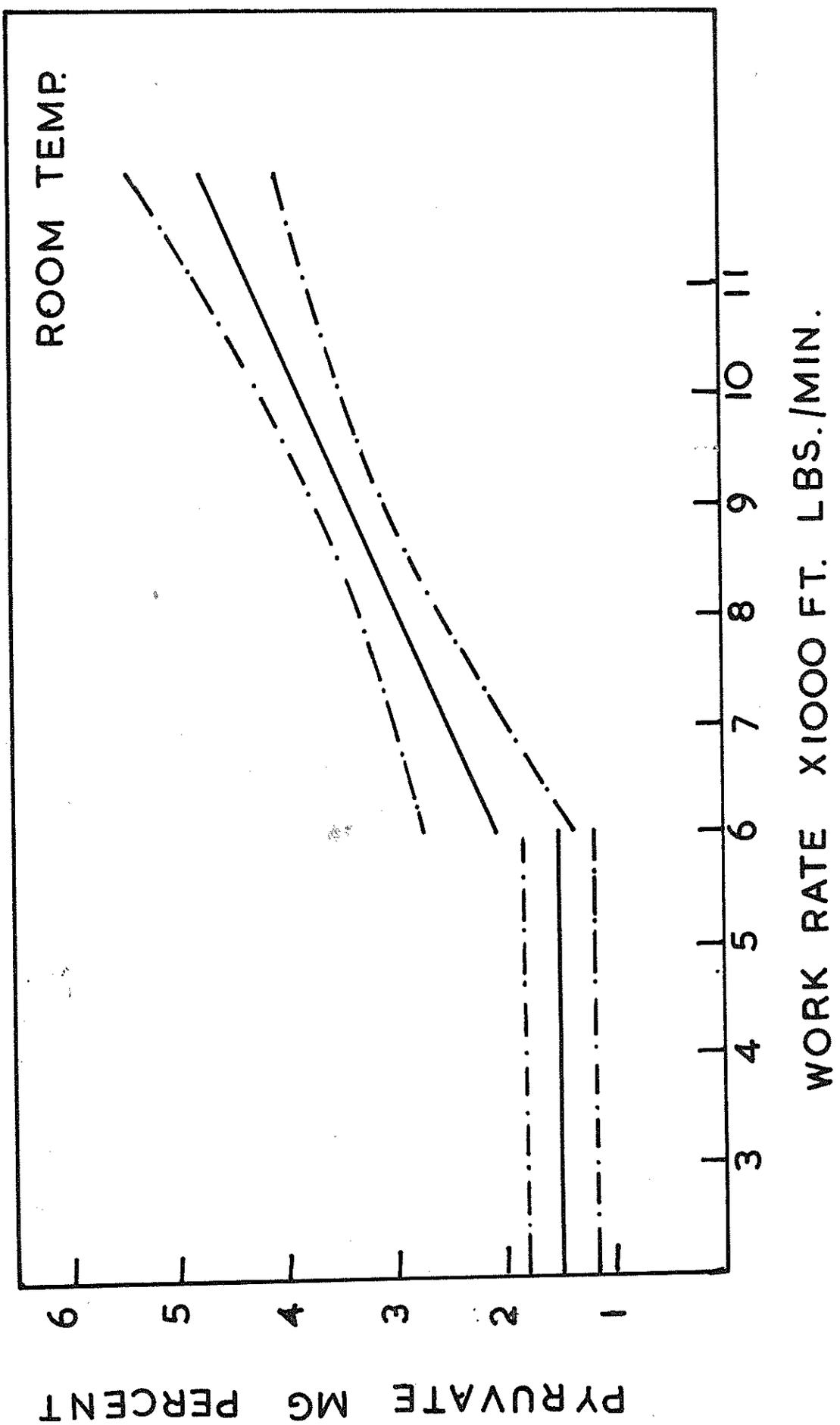


FIG. 8. SUBJECT BER.

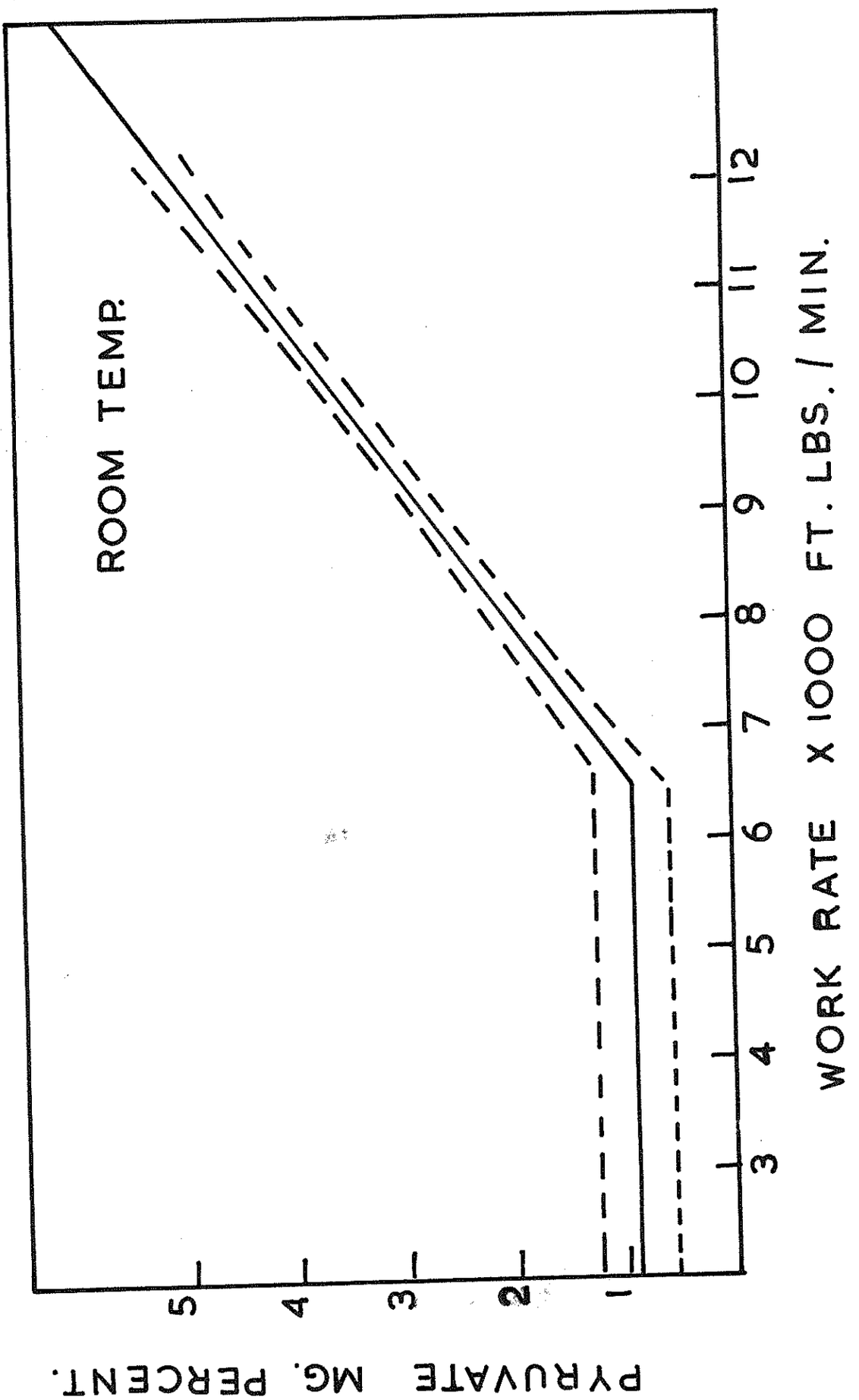


FIG. 9. SUBJECT ARM.

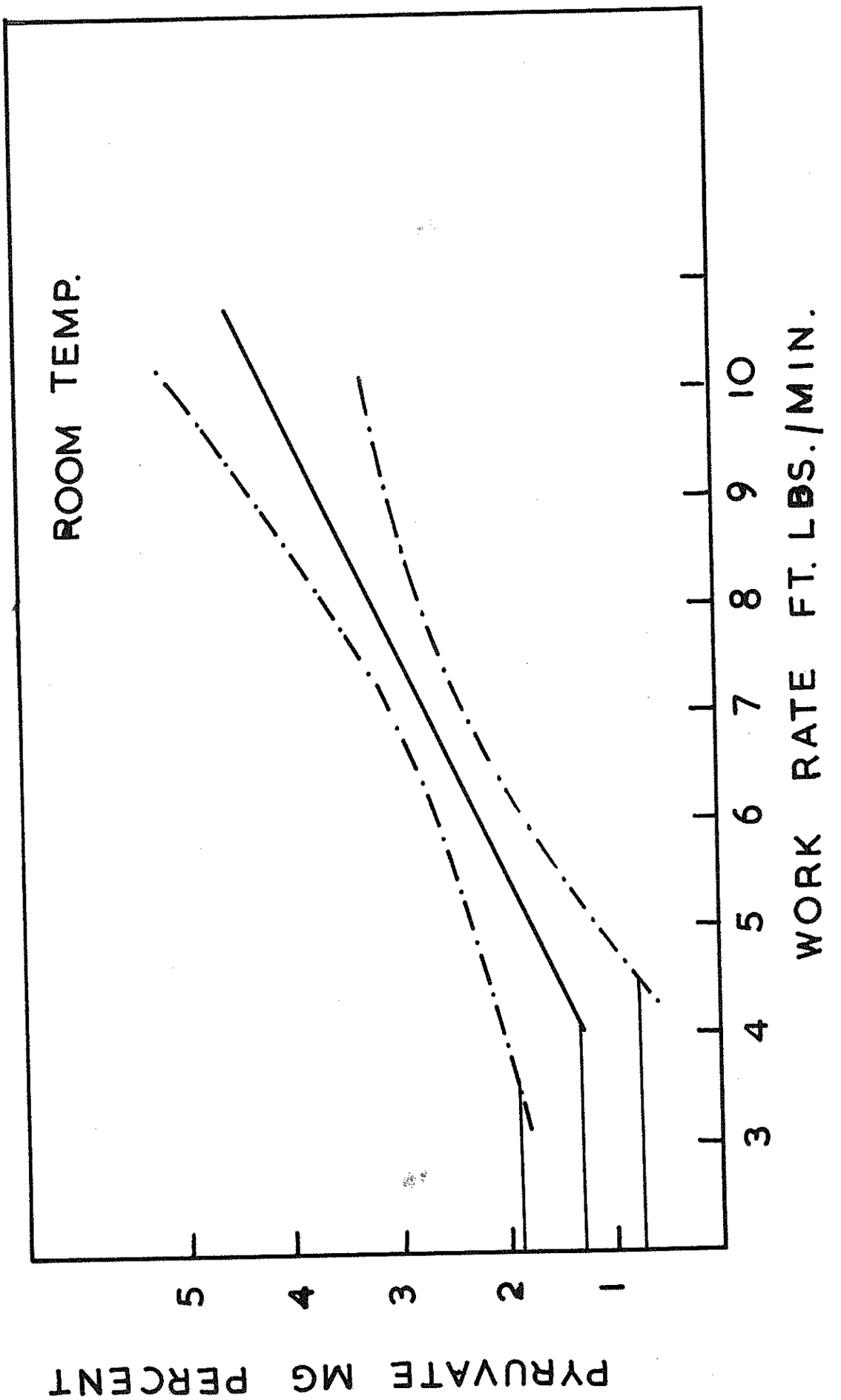


FIG. 10. SUBJECT VIC.

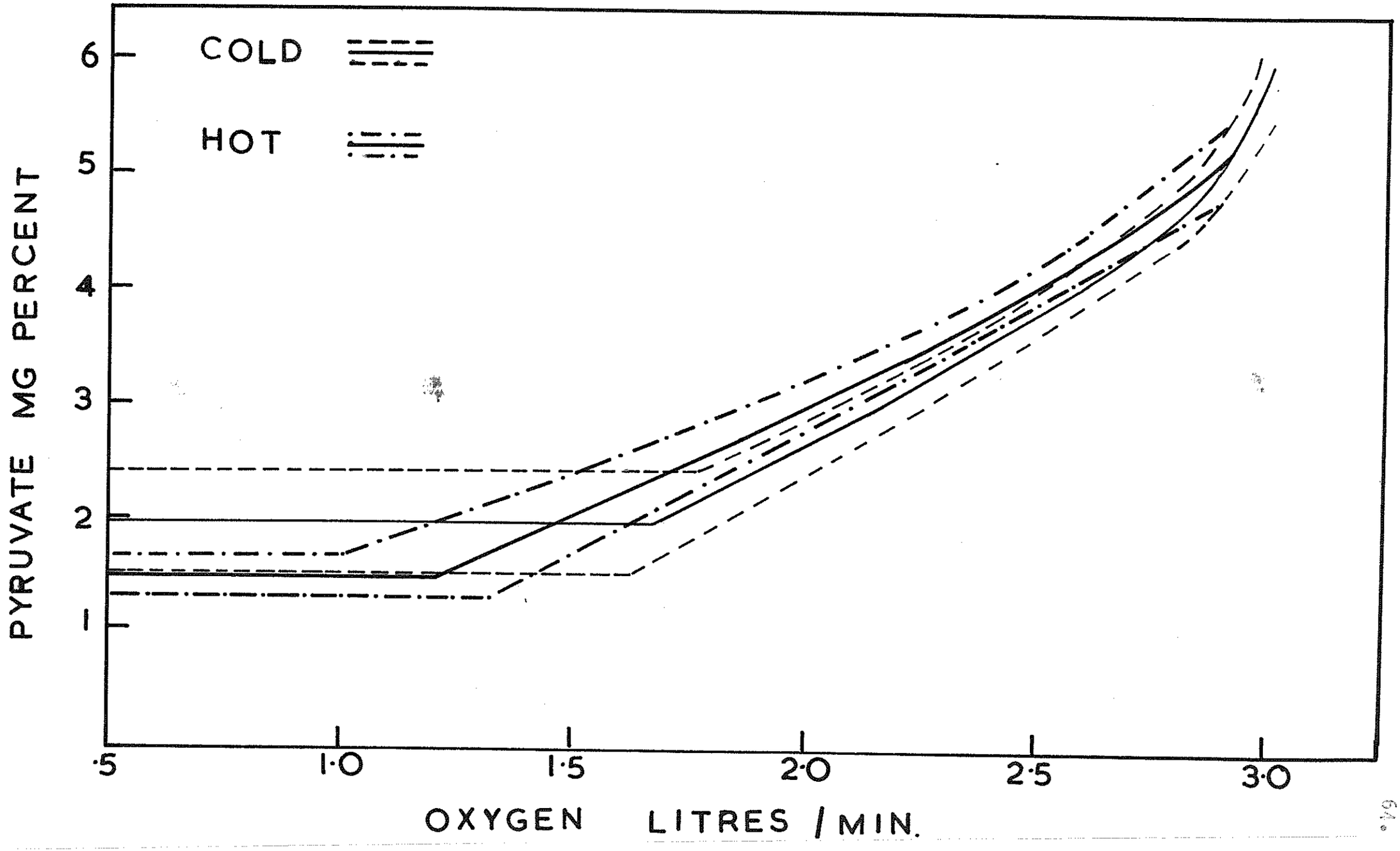


FIG. 11. SUBJECT ZEF.

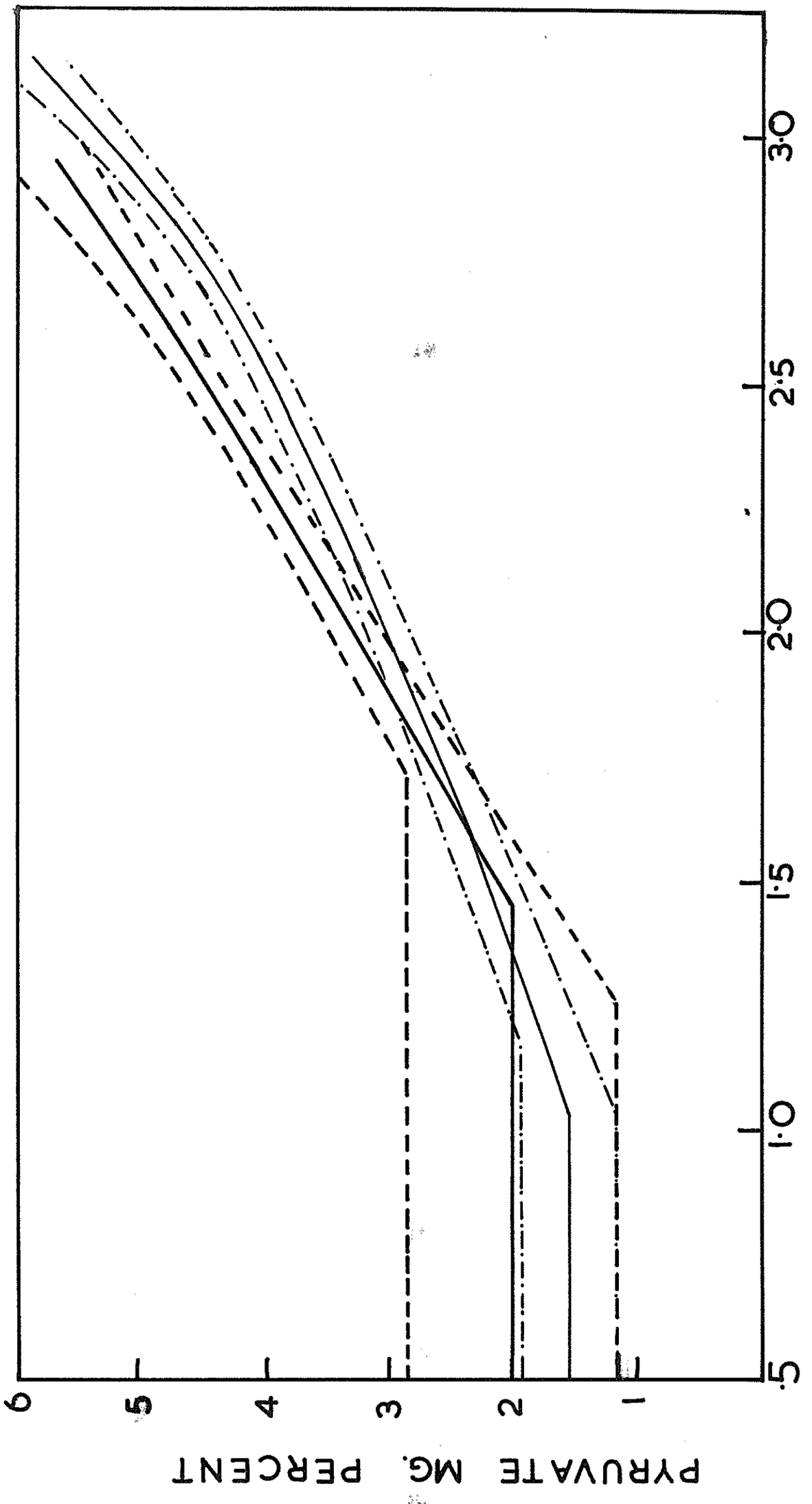


FIG. 12. SUBJECT ANT.

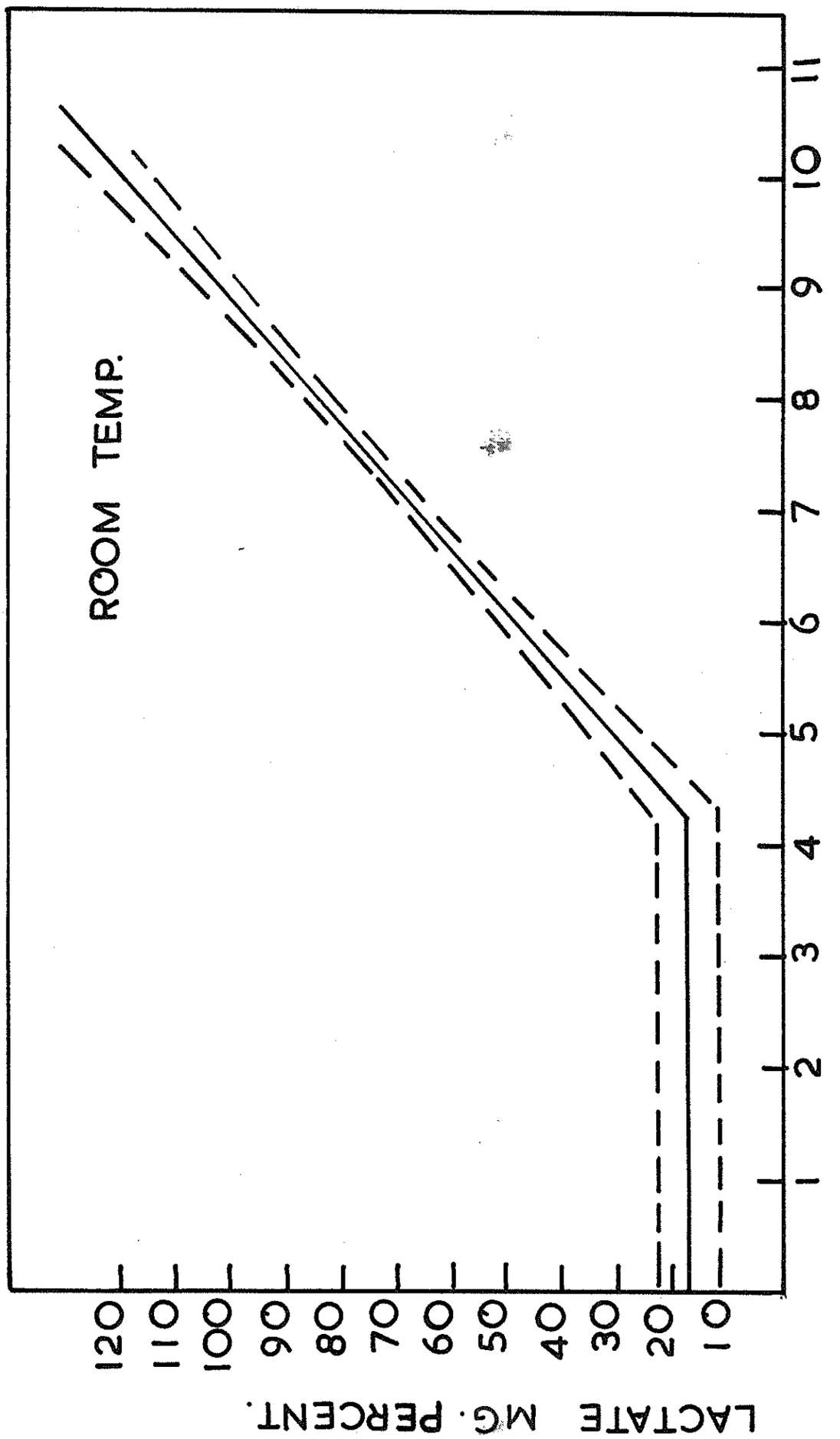


FIG. 13. SUBJECT ARM.

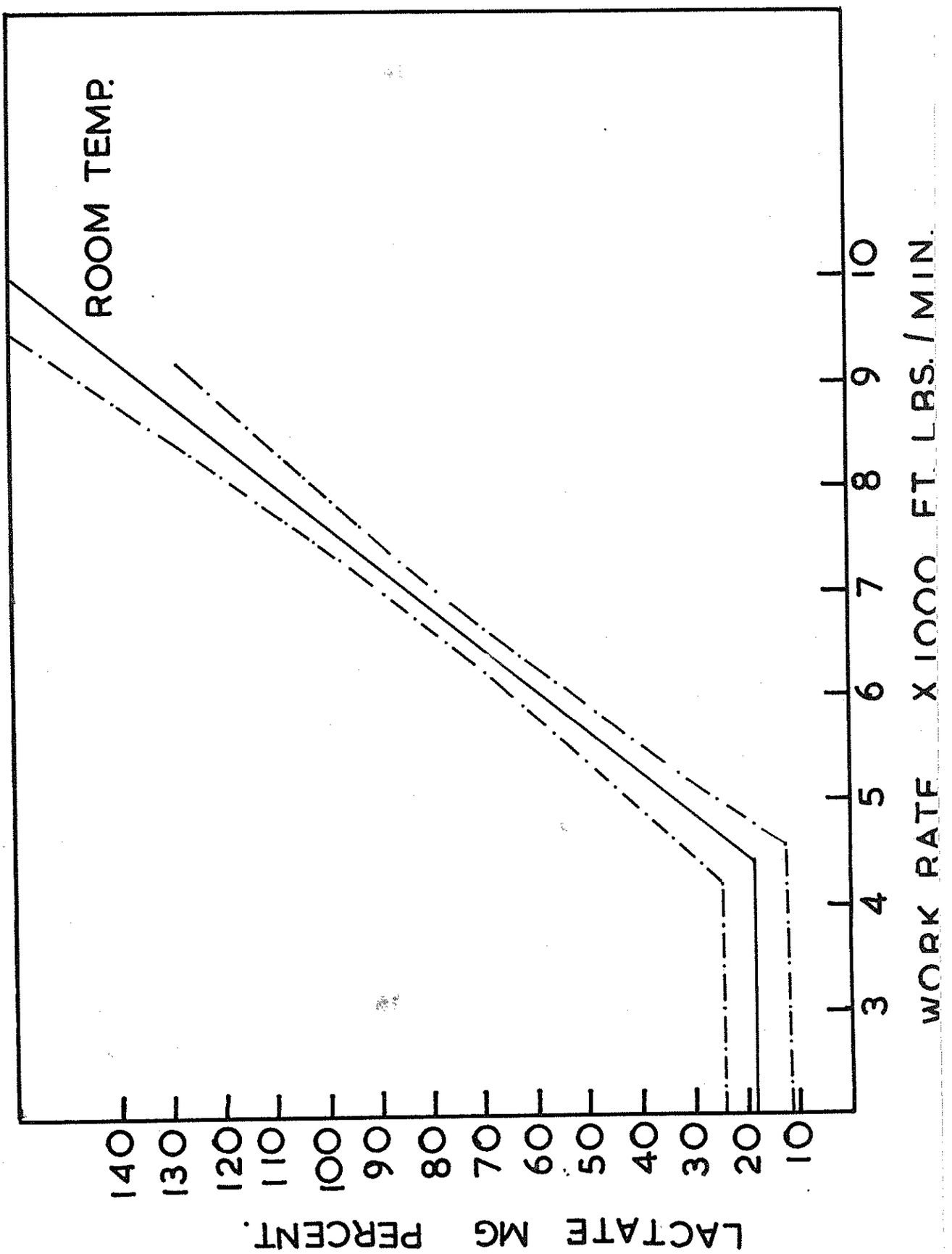


FIG. 14. SUBJECT JUL.

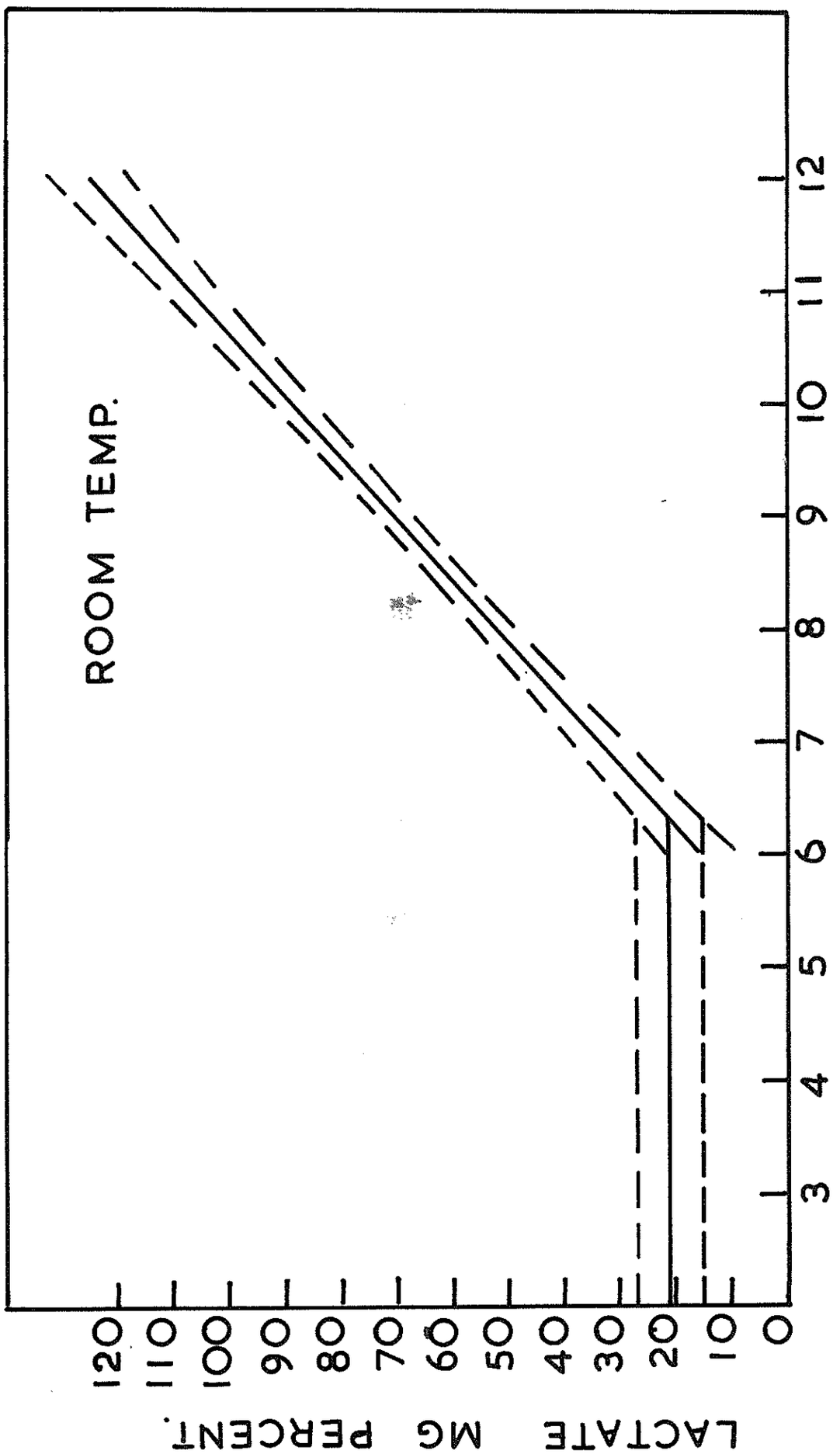


FIG. 15.

SUBJECT BER.

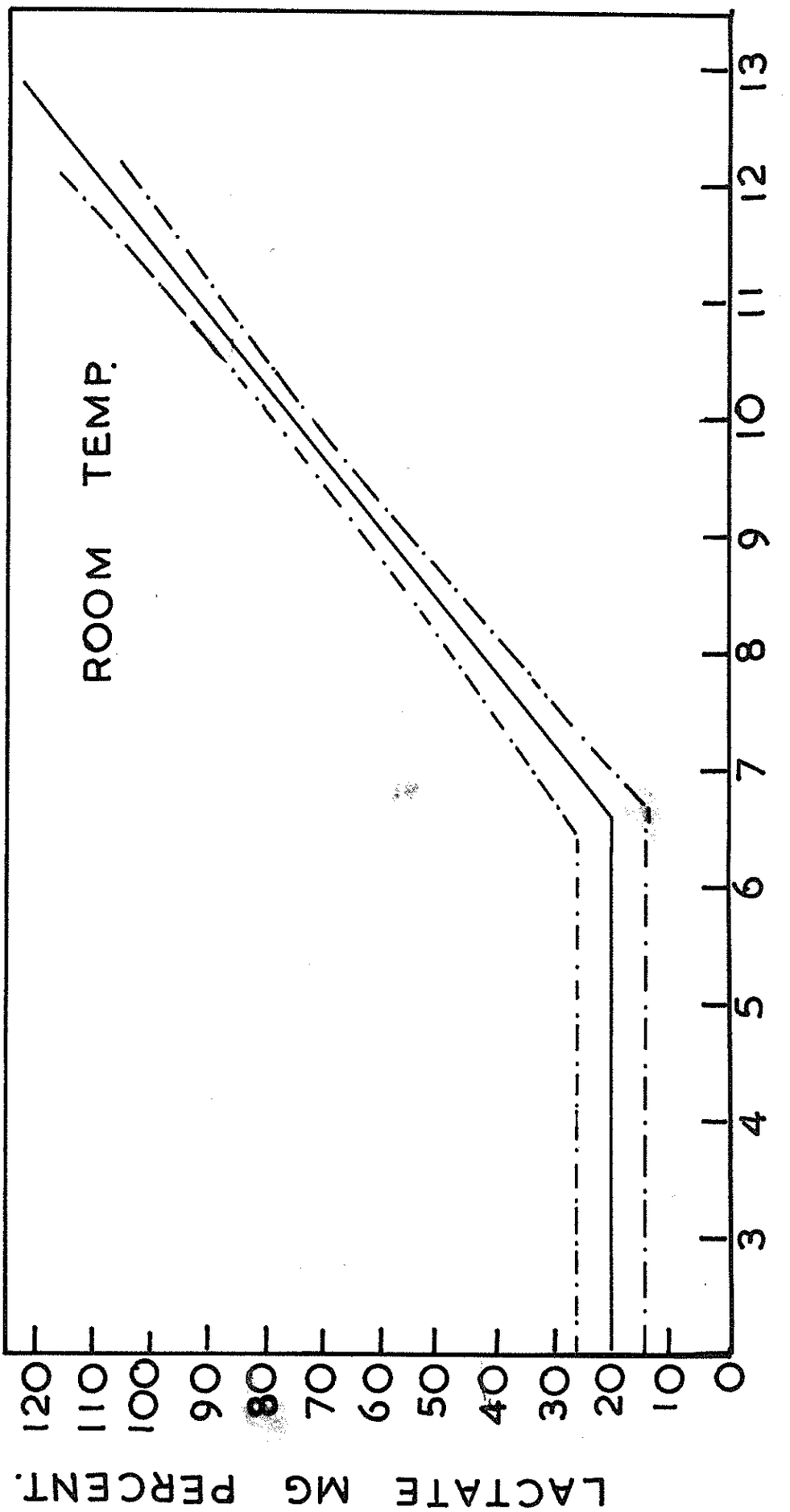


FIG. 16. SUBJECT DEN.

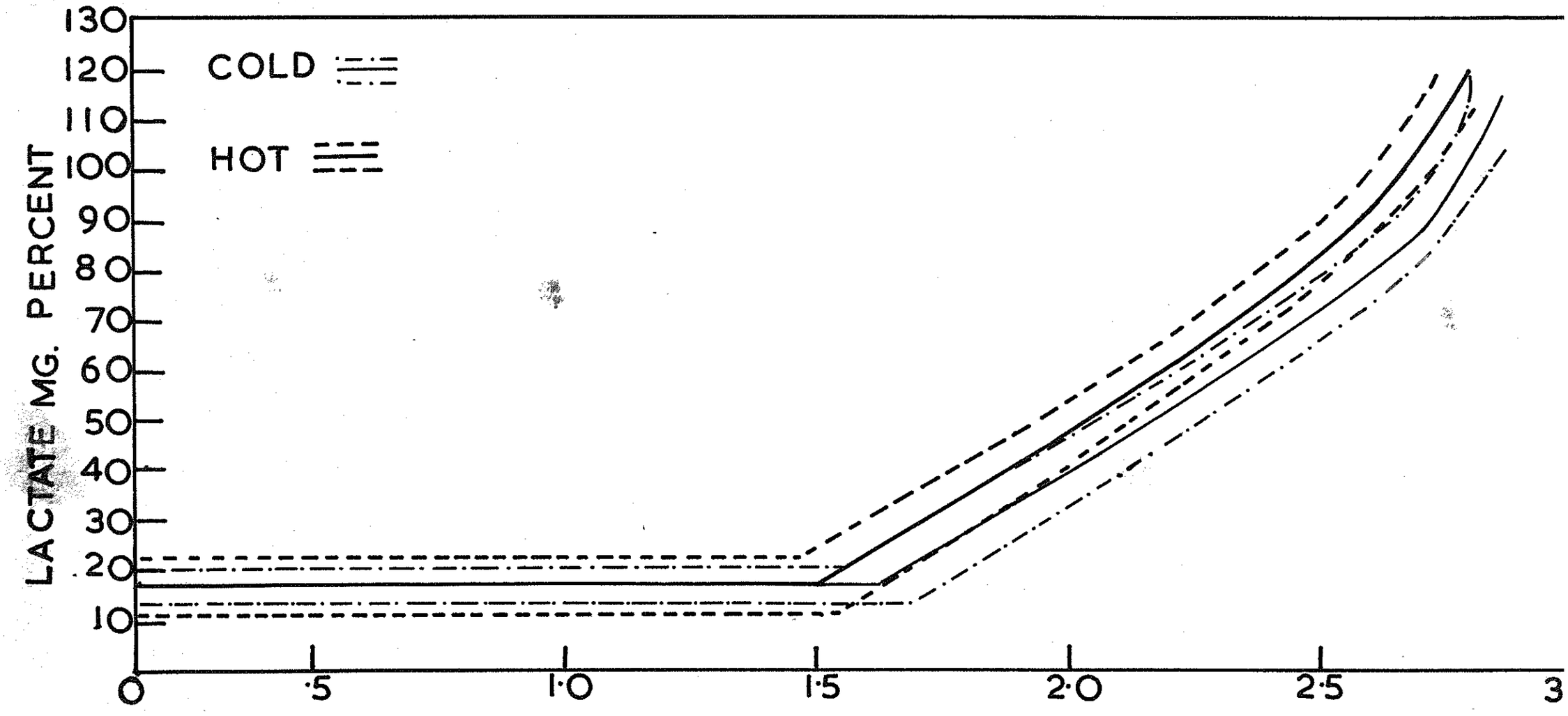


FIG. 17. SUBJECT DEN.

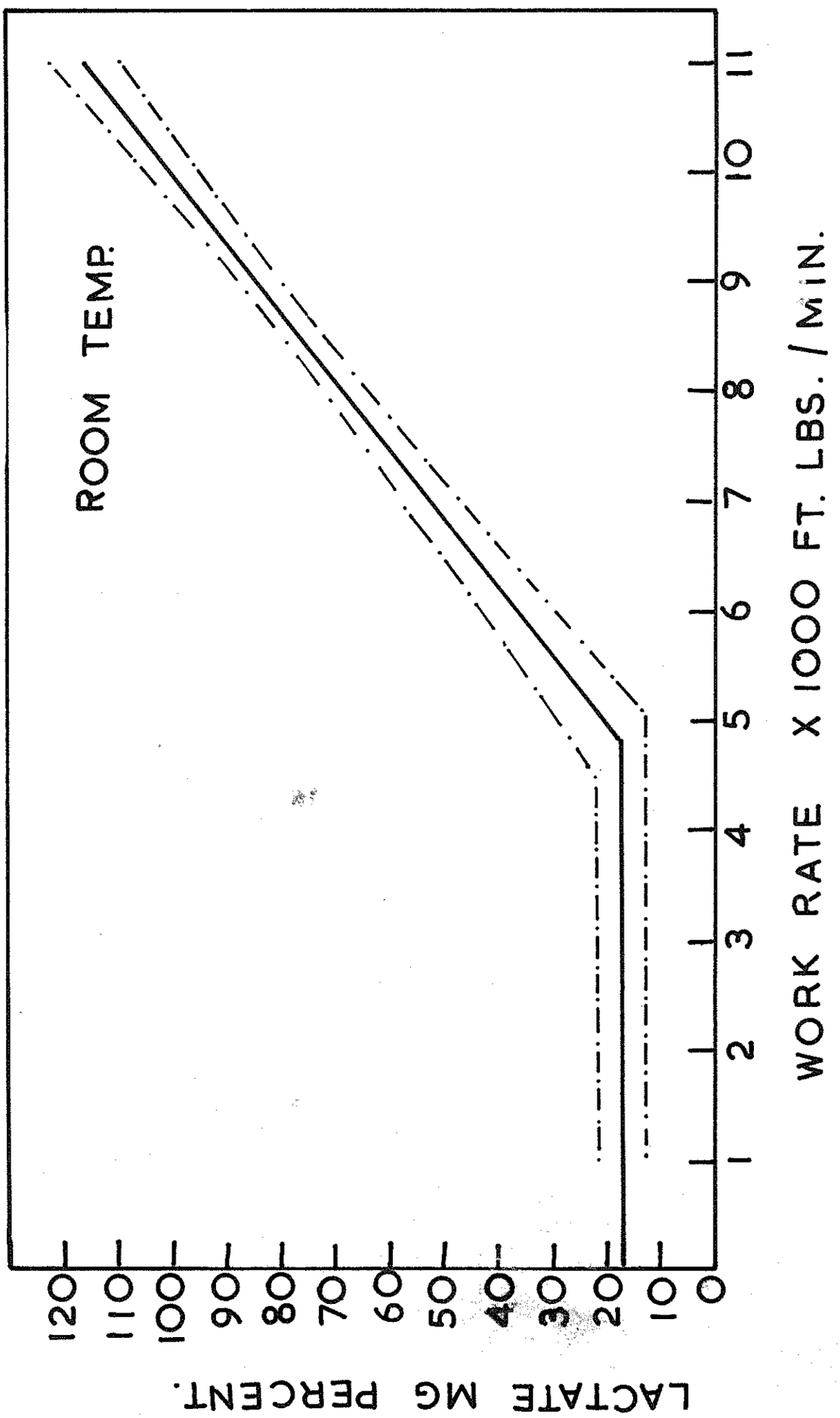


FIG. 18. SUBJECT DEN.

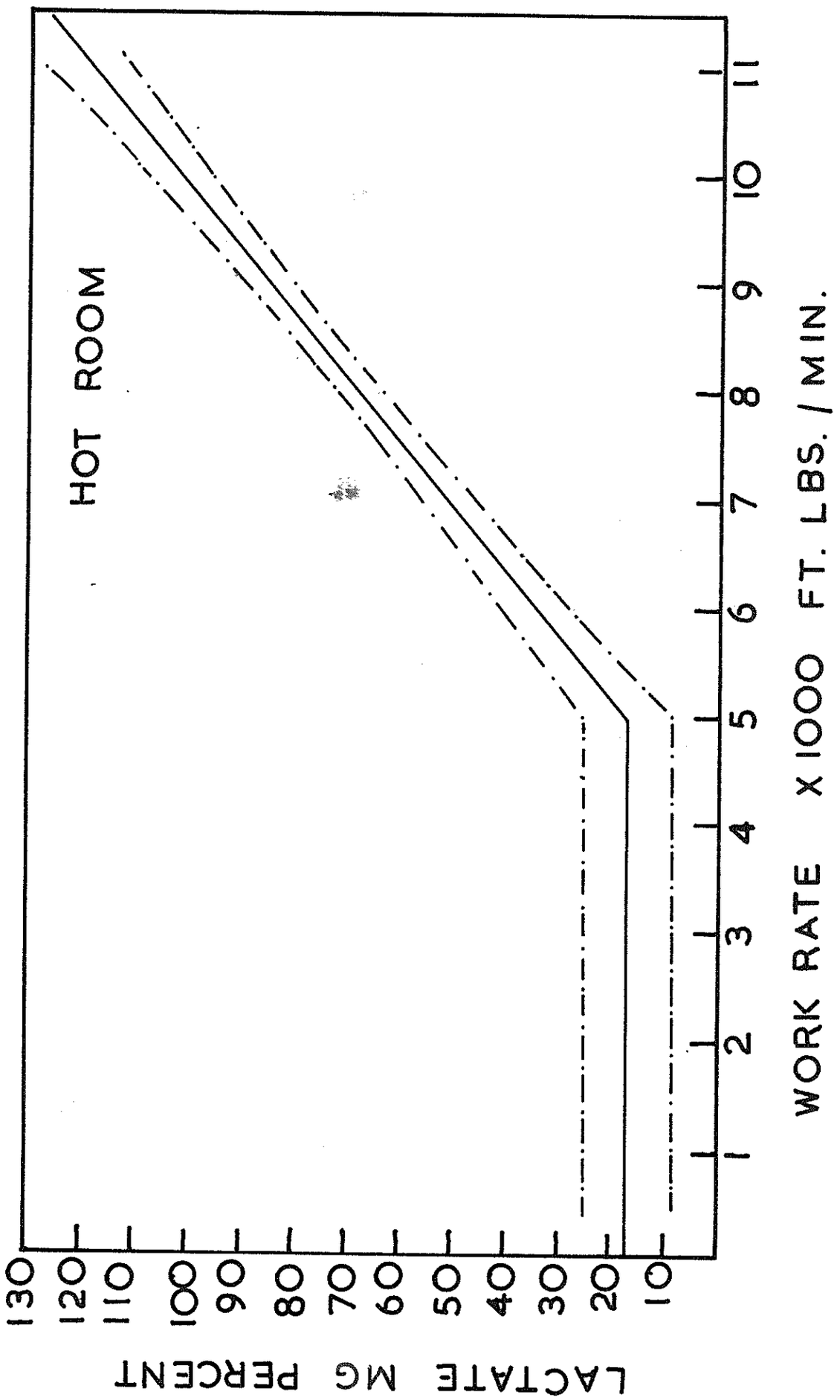


FIG. 19. SUBJECT VIC.

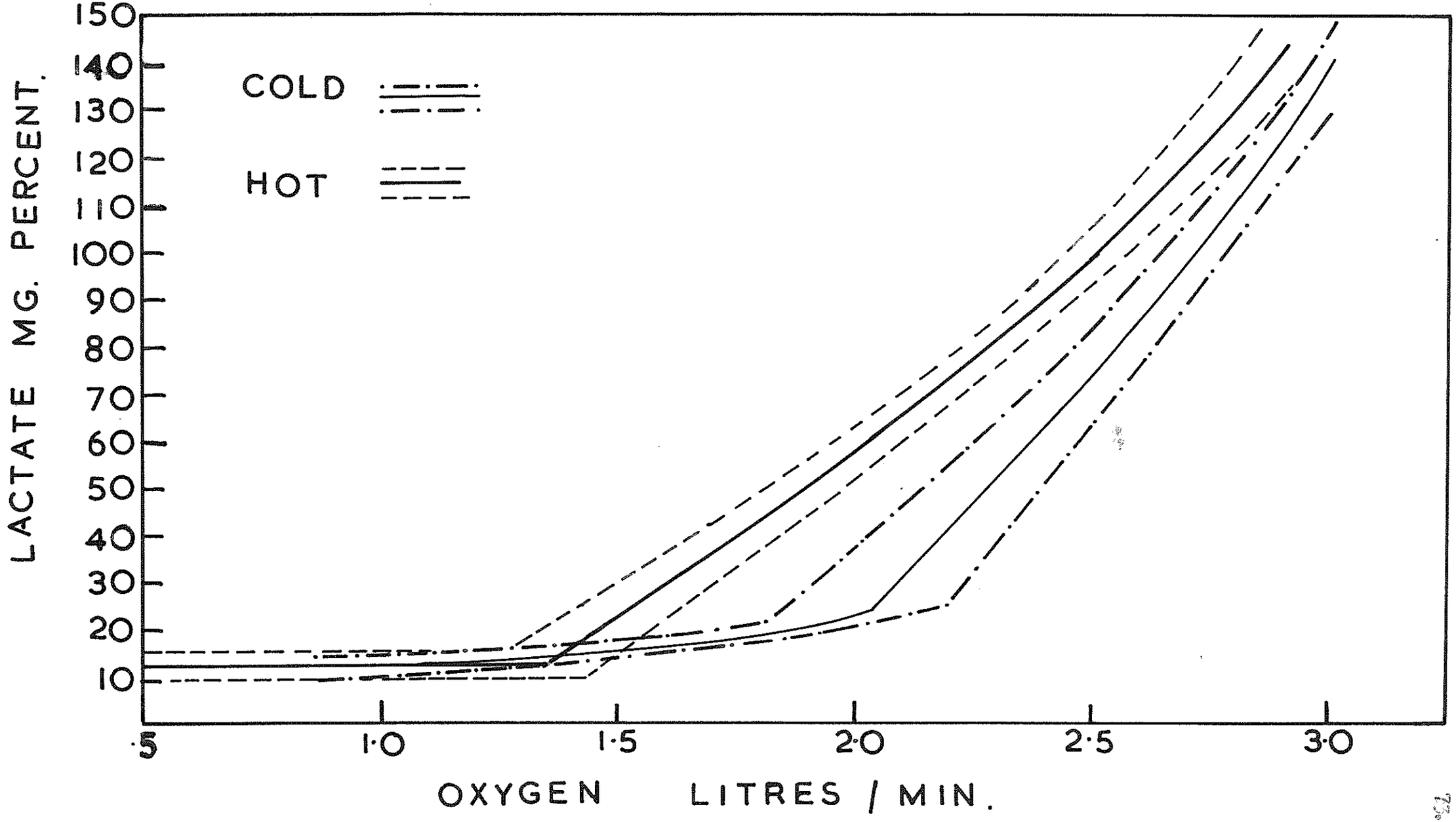


FIG. 20. SUBJECT ZEF.

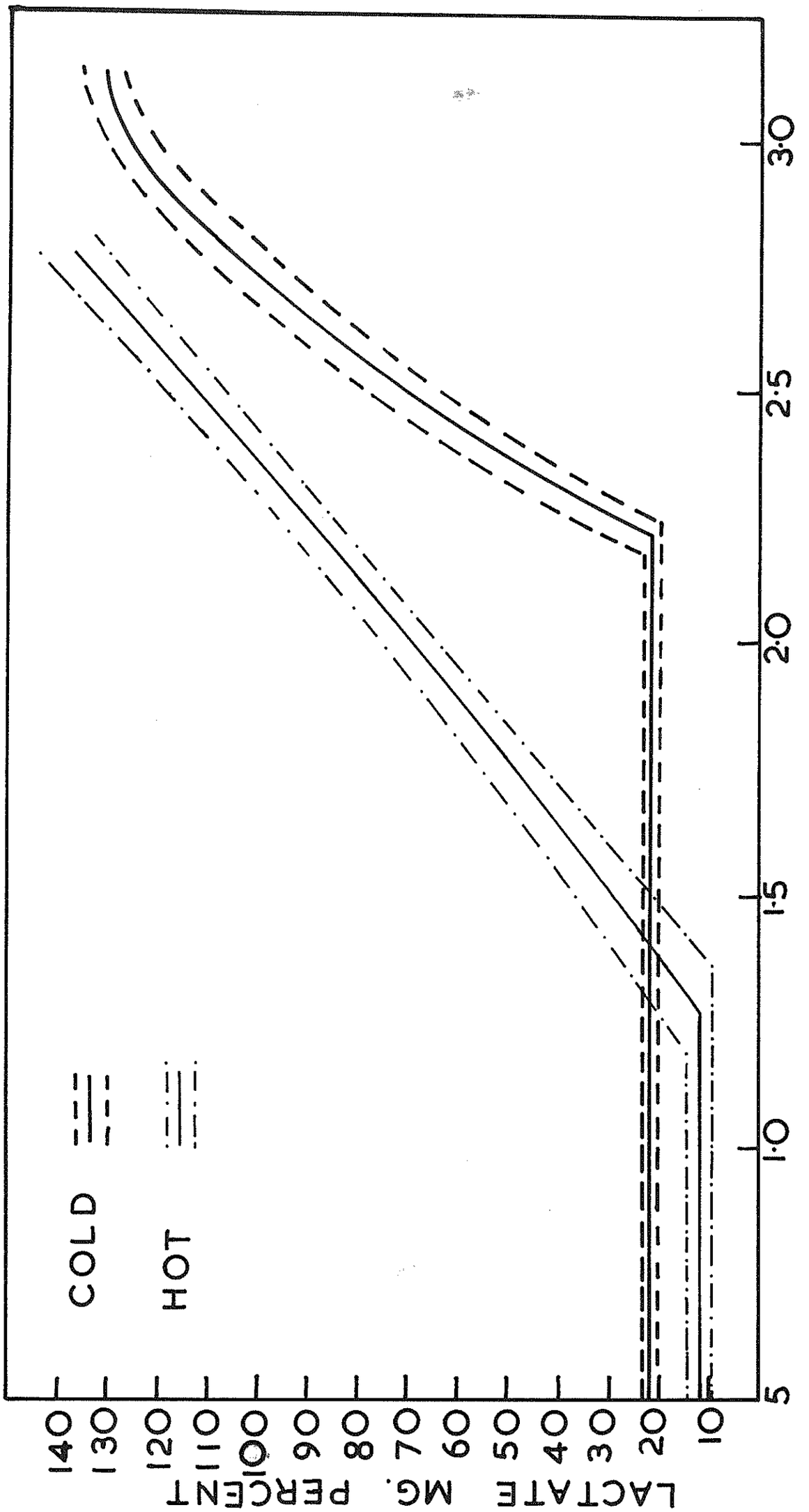


FIG. 21. SUBJECT ZEF.

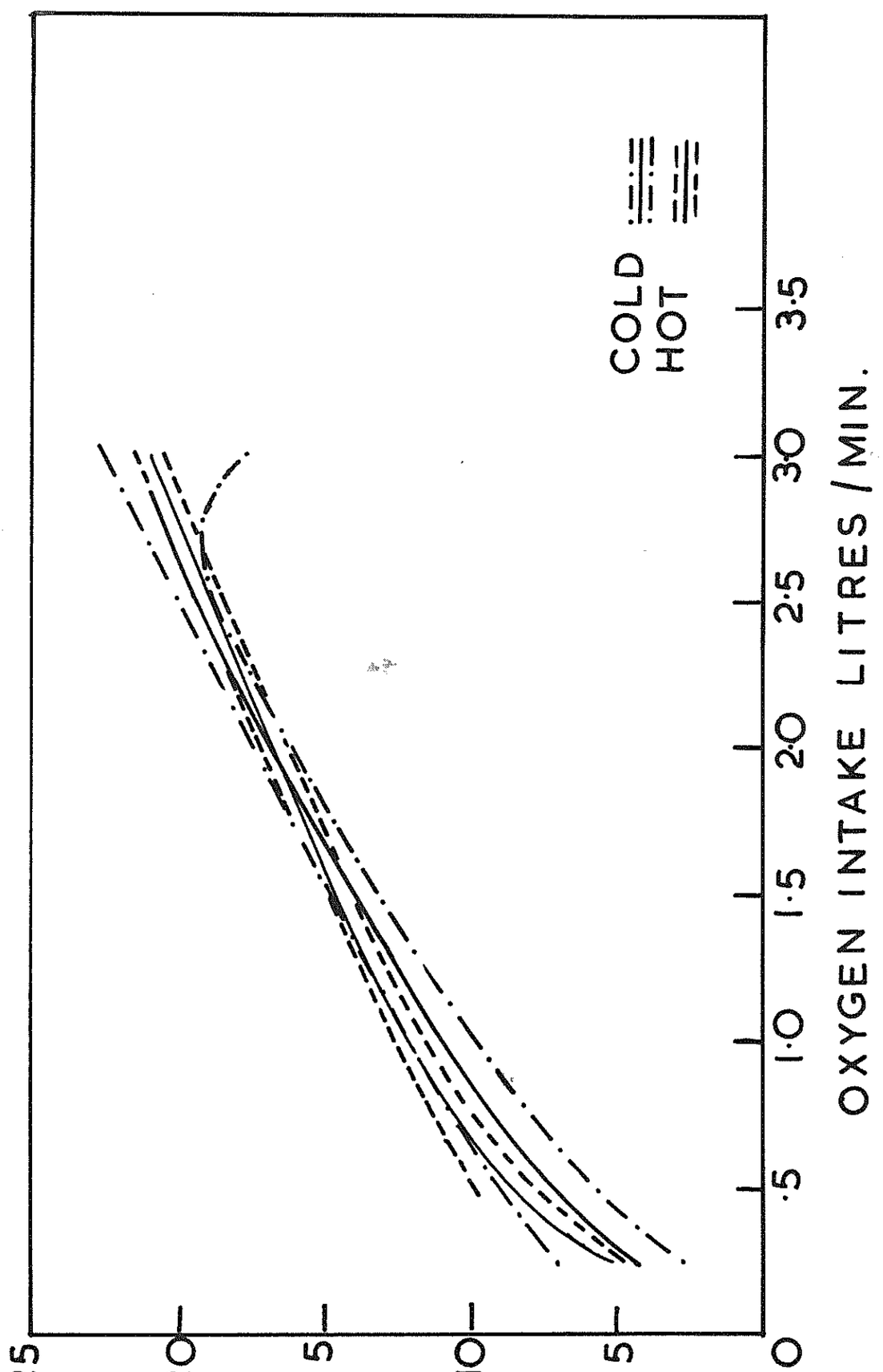


FIG. 22. SUBJECT DEN.

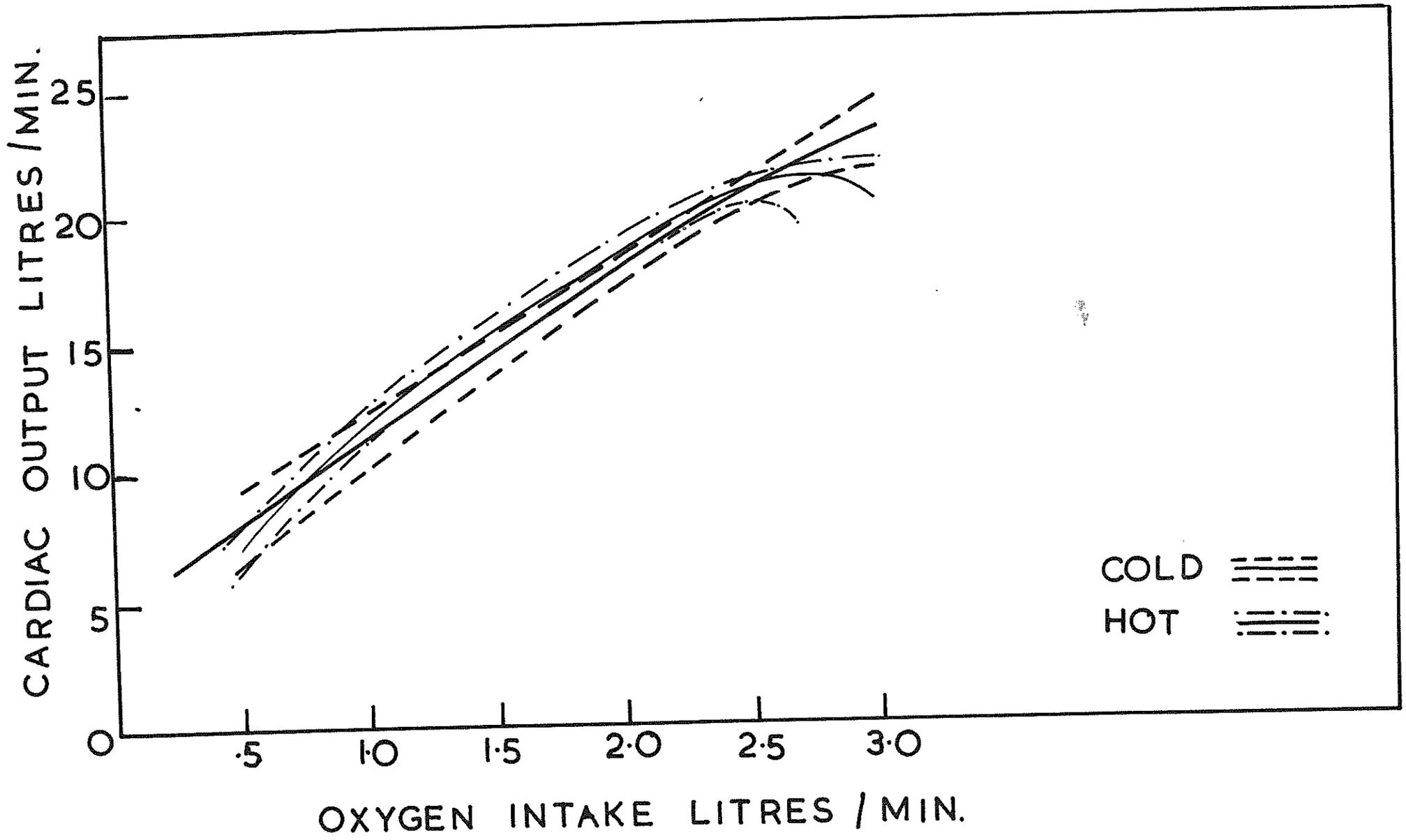


FIG. 23.

SUBJECTS. DEN.
ZEF.

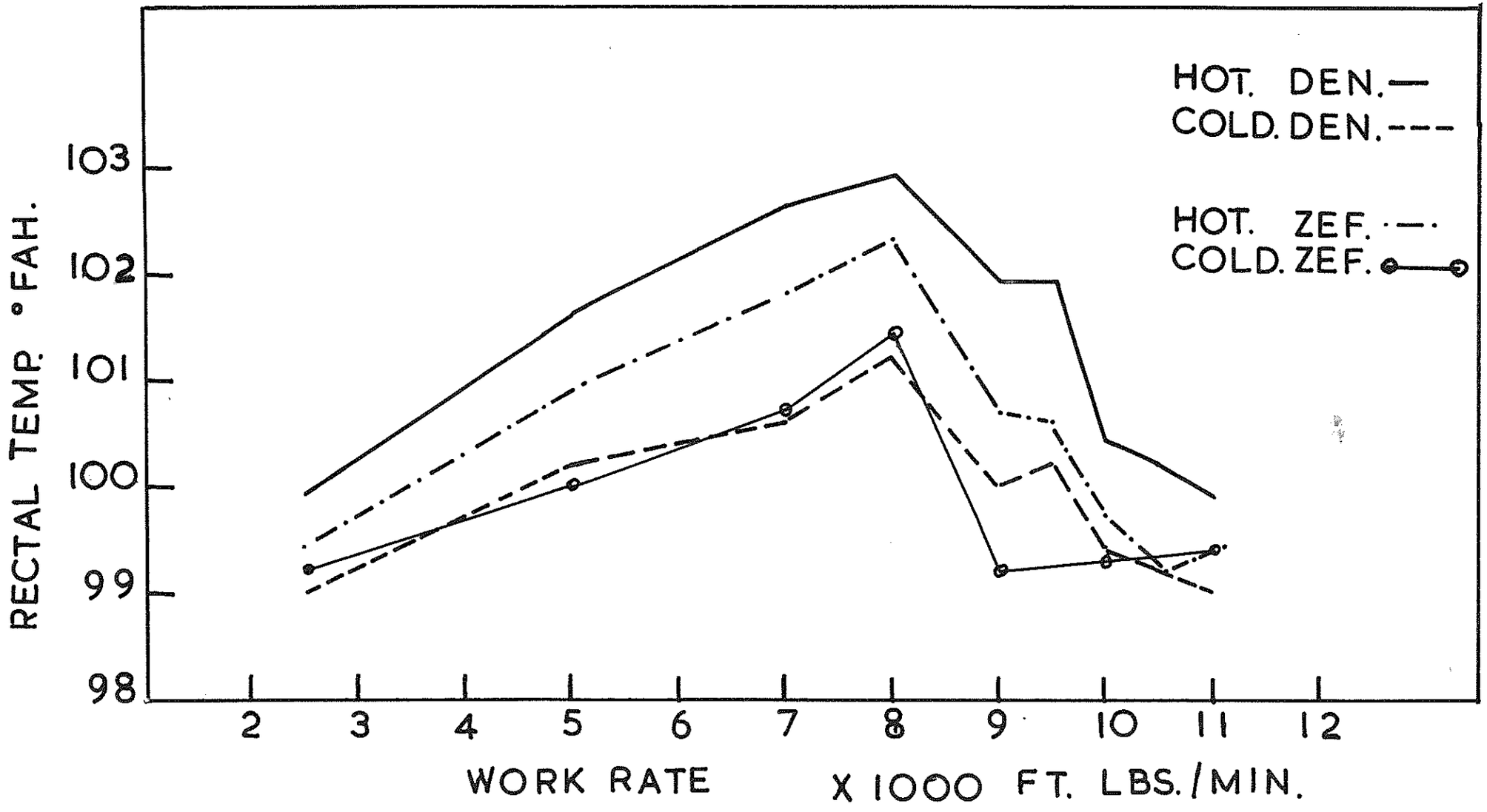


FIG 24.

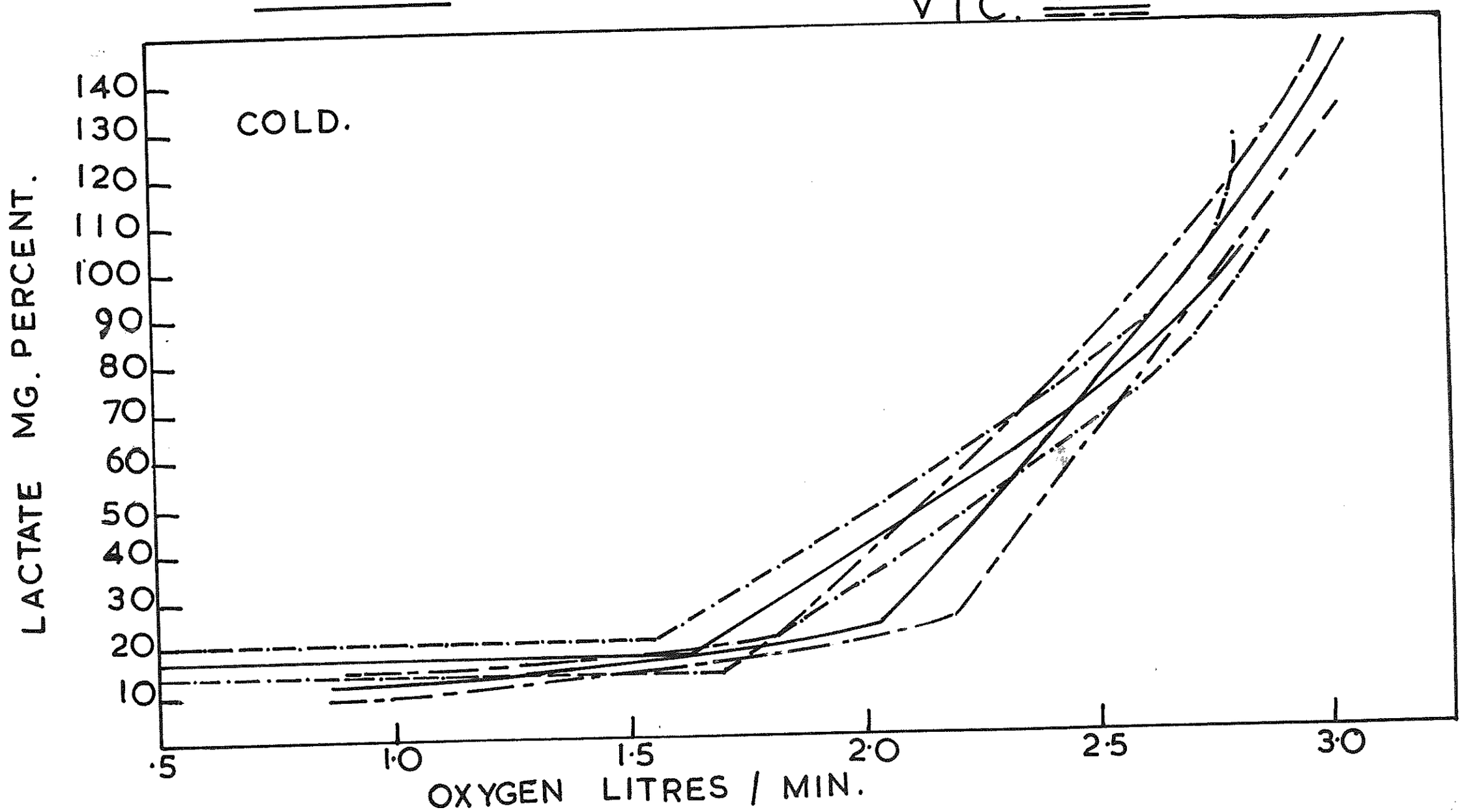

SUBJECTS. DEN.
VIC.
COLD.
LACTATE MG. PERCENT.
140
130
120
110
100
90
80
70
60
50
40
30
20
10
·5
1·0
1·5
2·0
2·5
3·0
OXYGEN LITRES / MIN.

FIG. 25.

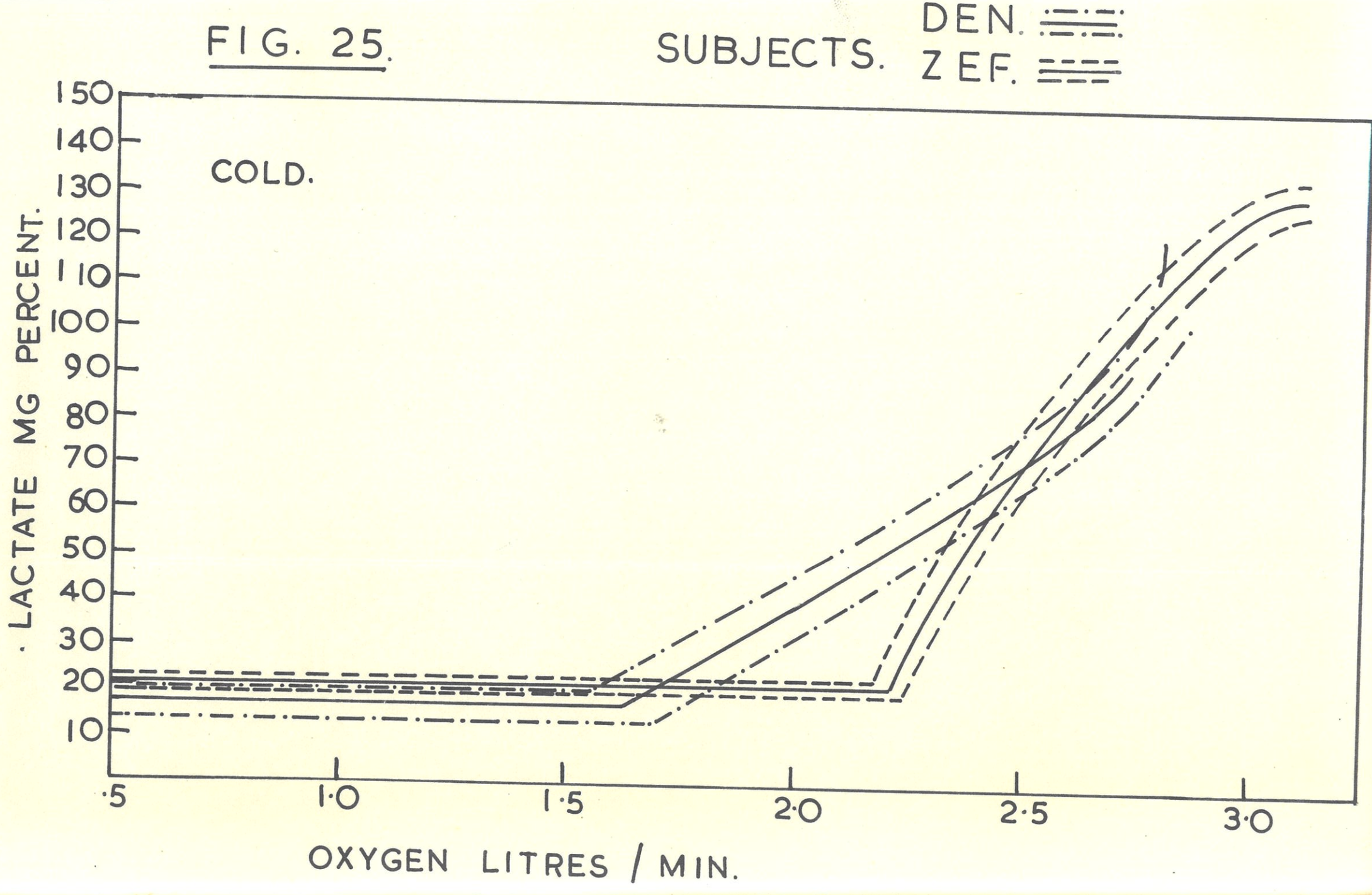

SUBJECTS.
DEN.
ZEF.
COLD.
LACTATE MG PERCENT.
150
140
130
120
110
100
90
80
70
60
50
40
30
20
10
·5
1·0
1·5
2·0
2·5
3·0
OXYGEN LITRES / MIN.

FIG. 26.

SUBJECTS. DEN. ZEF.

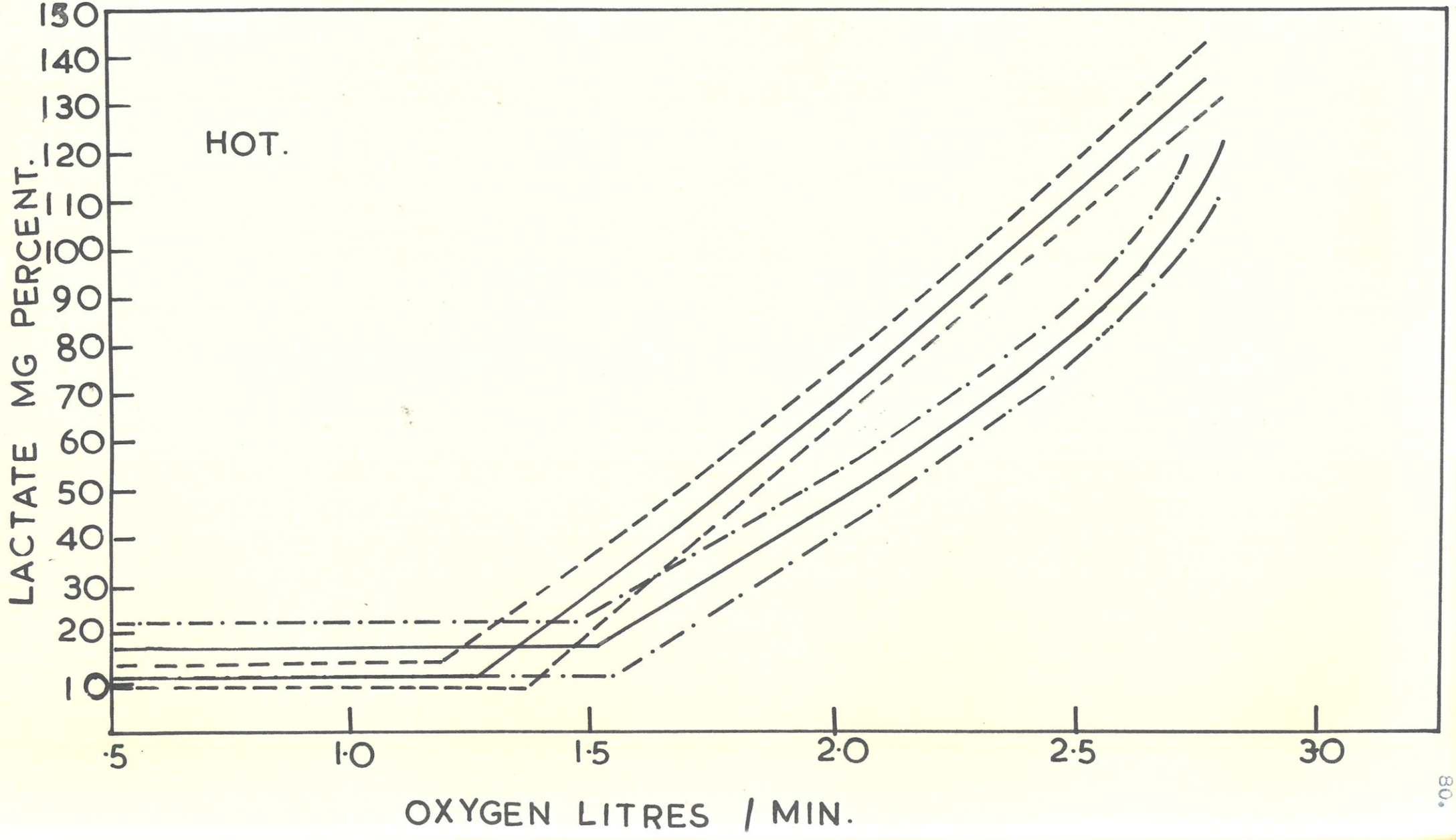
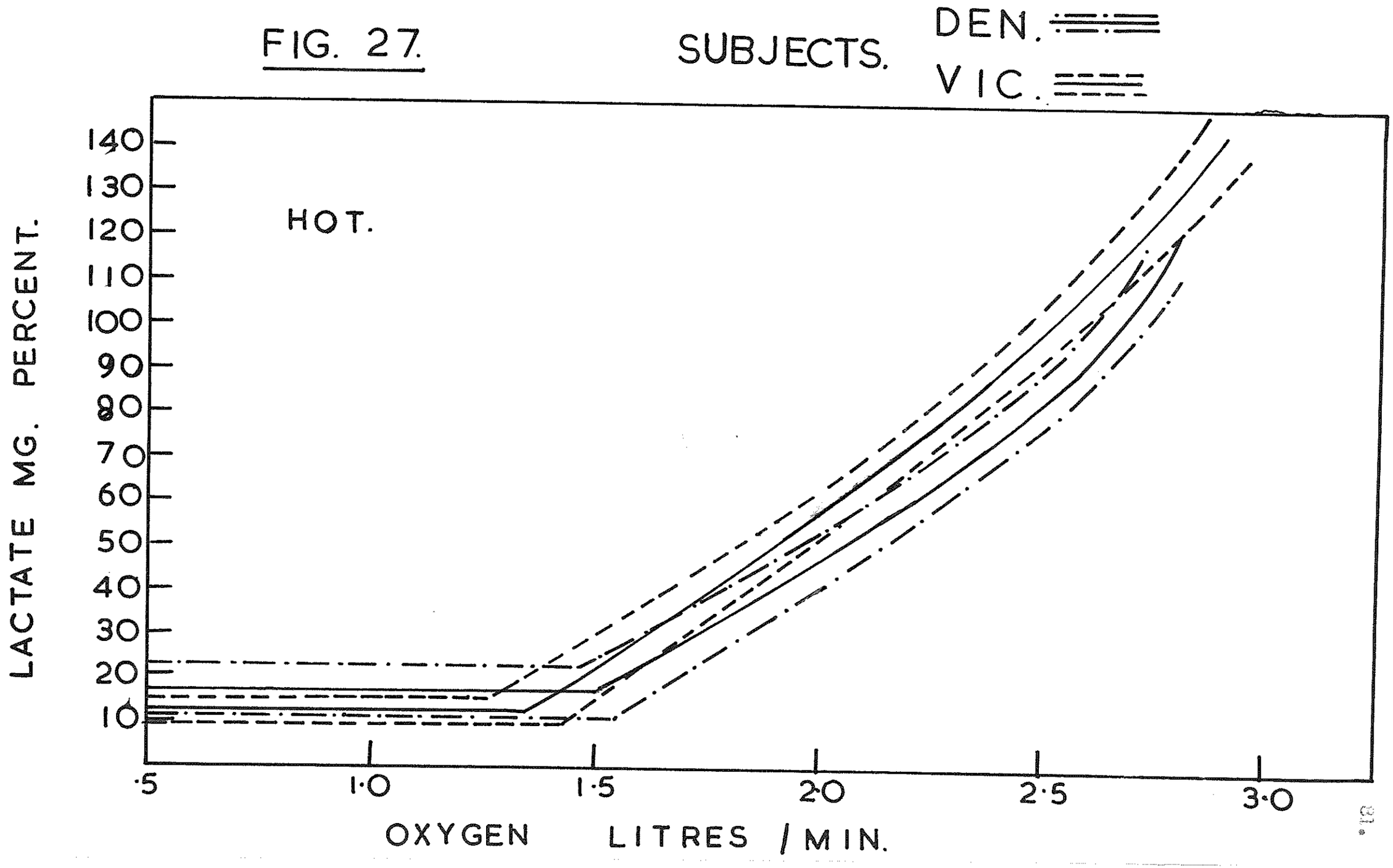


FIG. 27.


SUBJECTS.
DEN.
VIC.
HOT.
LACTATE MG. PERCENT.
140
130
120
110
100
90
80
70
60
50
40
30
20
10
·5
1·0
1·5
2·0
2·5
3·0
OXYGEN LITRES / MIN.

CHAPTER V.

DISCUSSION.

Resting Data: (Lactate and Pyruvate).

The resting data for the seven subjects studied are given in Table 11. Before any attempt is made to compare this data with published results, it is necessary to stress certain points that may have had a significant bearing on previous results. The methods of blood collection, analysis, site of sampling (e.g. arterial blood, venous or capillary blood) and subsequent treatment of samples have varied considerably in the past. Some of the older methods for lactate estimation were not specific for this acid, with the result that high resting and working values were obtained (see Jervell)[1].

It was reported that pyruvate decreased rapidly in shed blood [2], which means that unless blood was rapidly denatured (deproteinized) the blood lactate would be higher than it should be, while an under estimate of pyruvate would be given. On the other hand the use of fluoride as a preservative, accelerated the disappearance of pyruvate and increased the lactate, while iodoacetate has just the opposite reaction - increase of pyruvate and decrease of the lactate content of blood [3].

The resting samples were taken from a subject sitting quietly on the bicycle ergometer. The subjects were in the post-prandial state but the traditional use of high carbohydrate meals of native mine workers may have influenced the data to some extent [4]. However, from Table 11 (b) it is obvious that the resting values for the seven subjects are well within the normal limits.

---

1. O. Jervell. "Investigation of the concentration of lactic acid in blood and urine." Acta Medical Scandinavica. Suppl. 22 - 25, 1927 - 28.

2. Huckabee, W.E. "Control of concentration gradients of pyruvate and lactate across cell membranes in blood". Journal of Applied Physiology, 9:163, 1956.

3. Friedemann, T.E. and G.E. Haugen. "Determination of Keto-acids in blood and urine". Journal of Biological Chemistry, 147:415, 1943.

4. T.E. Friedemann, et al. "The level of pyruvic and lactic acids, and the lactic-pyruvic ratio, in the blood of human subjects. The effect of food, light muscular activity, and anoxia at high altitudes". Journal of Biological Chemistry, 157 : 673, 1945.

Exercise Data:

The results presented above show that the lactate and pyruvate concentrations in blood are relatively little affected by light muscular work. At a certain work load, specifically set and different for each individual depending on his oxygen intake, changes are found in the blood lactate and pyruvate content compared with resting figures.

For most subjects, the change in the pyruvate/lactate curves takes place at approximately the same level of work (or oxygen consumption) as does the change in the lactate/work rate curves. This agrees with data published by Asmussen (5) who found that for both his subjects the blood lactate and pyruvate increased when a certain level of oxygen intake was reached, in this case 2.5 litres/min.

From the above it is clear that the point of discontinuity of the lactate or pyruvate curve for any individual will most certainly depend on certain factors viz. state of training and his normal aerobic capacity.

Physical Fitness:

For a fixed task, unfit individuals have higher blood lactate levels than trained subjects (6) and the blood lactate response to submaximal running is significantly decreased after a training period (7). The relationship between fitness and lactate response can best be explained as an improvement of the circulatory and respiratory mechanisms. Circulatory and respiratory adjustments to a specific exercise become more efficient during training thereby decreasing the degree and duration of anaerobic metabolism (glycolysis).

---

5. Asmussen, E. "Pyruvate and lactate Content of the Blood during and after Muscular Work". Acta Physiologica Scand. 20: 125 - 135, 1950.

6. D.B. Dill et al. "Industrial Fatique". Journal of Industrial Hygiene, 18: 417 - 431, 1936.

7. Robinson S and P.M. Harmon. "The lactic acid mechanism and certain properties of the blood in relation to training". American Journal of Physiology, 132: 757, 1941.

Aerobic working capacity:

When comparisons are made between subjects at a fixed work rate, it is clear that large differences exist in the blood lactate response to exercise. If Arm, and Ant., are compared with Ber., (Maximum aerobic capacity 2.5, 3.0, 3.5 respectively) at a work rate of 6000 ft. lbs/min. it can be seen that Arm. had a blood lactate content of 84 mg% (range 80-90) while for Ant. it was 66 mg% (range 63-68) and in the case of Ber. it was only 27 mg% (range 20-34). Such comparisons reveal the fact that increases of blood lactate above resting levels will depend on the individual's maximum aerobic capacity or his physical working capacity.

Although the lactate curves (Figures 12 - 20) show great individual difference as regards the point of discontinuity, table (viii) denotes that, with the exception of one subject, all reached the threshold value for both lactate and pyruvate at a value of 61 - 68% of their aerobic capacity. In the case of Den. it was reached at 53% of his aerobic capacity. Margaria et al [8] postulated a value of 2/3 of the aerobic capacity which is in good agreement with the present data.

The intensity of work, or the level of oxygen consumption, at which blood lactate increases above ordinary resting values has been a point of great controversy in the past [9]. Crescitelli and Taylor [10] postulated a value of 2.1 litres/min. above resting value, while Taylor [11] reported figures between 1.2 - 2.0 litres/min average of 1.65. For adult males Owles [11a] found an oxygen

8. Margaria, R; H.T. Edwards and D.B. Dill. "The possible Mechanism of Contracting and paying the Oxygen Debt and the rôle of lactic acid in muscular contraction". American Journal of Physiology, 106 : 689, 1933.

9. Astrand, P.O. "Experimental Studies of Physical Working Capacity in relation to sex and age". Ejnar Munksgaard, Copenhagen. 1952. (pg 100).

10. Crescitelli, F. and C. Taylor. "The lactate response to exercise and its relationship to physical fitness". American Journal of Physiology, 141: 630, 1944.

11. Taylor, C. "Some properties of maximal and submaximal exercise with reference to physiological variation and the measurement of exercise tolerance". American Journal of Physiology, 142: 200, 1944.

11(a) Owles, W.H.. "Alterations in the lactic acid content of the blood as a result of light exercise and associated changes in the CO_2 - combining power of the blood and in the alveolar CO_2 - pressure". Journal of Physiology, 69: 214, 1930.

consumption of 1.8 litres/min. or 1.5 litres/min. above resting. Newman et al [12] gave figures as low as 1.0 litres/min. oxygen intake above resting values.

Their views seem to miss the essential point which is that this figure will to a great extent depend on the individual's aerobic capacity. This fact is brought out clearly in the present study, where individuals with varying aerobic capacity and big differences in body weight are compared. The intensity of work at which the blood lactate increases over resting values shows great individual differences; range 4300 ft. lbs/min. to 6750 ft. lbs/min. while the oxygen consumption ranged from 1.75 litres/min to 2.2 litres/min. Nevertheless it was found that for 6 of the 7 subjects studied the blood lactate increased over resting values when a level of oxygen consumption was reached equivalent to 61-68% of the aerobic capacity.

It seems, therefore, that an estimate of the level of oxygen consumption at which blood lactate increase over resting value can give a rough measure of the individual's aerobic capacity. This method is independant of body weight, and, as it implies only aerobic work, may prove useful in the investigation of the aerobic capacity of patients with clinical disorders.

In the present study only trained subjects were used, but the relationship between aerobic capacity and blood lactate may hold good for untrained subjects because, although physical training decreases the blood lactate response to submaximal workloads, it was found that physical conditioning increases the individual's maximum oxygen intake.

Pulse Rate:

Like oxygen consumption the pulse rate at which the "threshold" value for lactate was reached, also show wide individual differences. However, for five subjects the pulse rate fell within the range of 130 to 140 beats/min., while for one subject (Den.) it

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12. Newman, E.V., D.B. Dill, H.T. Edwards and F.A. Webster. "The rate of lactic acid removal in exercise". American Journal of Physiology, 118: 457, 1937.

was 120 and for another (Zef.) it was 155.

It is interesting to note that anaerobic metabolism comes into operation at or shortly after a pulse rate of 120 beats/min. is reached. The only possible explanation for this phenomenon is, that the oxygen supply to active muscles depends on the cardiac output which is the product of heart rate and stroke volume. When a pulse rate of 120 is reached the time allowed for filling the heart becomes limited and the stroke volume actually decreases so that cardiac output can only be increased by means of an even greater elevation in pulse rate, to a level of 180.

It can be assumed therefore that during work the heart can supply the working muscles with sufficient blood until a pulse rate between 120 - 140 beats/min. is reached.

At higher intensities of work the efficiency of the heart decreases owing to an actual decrease in stroke volume, so that the supply of blood to active muscles lags behind the requirements set by the metabolism of the active tissues. With further increase in intensities of work this discrepancy between supply of oxygen and requirement becomes greater with the result that the degree of anaerobic metabolism increases.

Increase of Blood Lactate:

Once the threshold value for lactate and pyruvate is reached, both acids increase rapidly with increasing work load. For all subjects this increase is linear or near-linear, although the slope for the lactate curve is much steeper in most cases, indicating a more rapid accumulation of blood lactate over pyruvate. The maximum value for blood lactate was found at a work load which the subject could keep up for only a few minutes. At the heavy work rates the highest blood lactates were found not at the end of exercise, but at the third minute of recovery, while for the pyruvate value this only occured at approximately the 13th minute of recovery. Similar observations were made by Asmussen (5)

Johnson and Edwards [13] and Friedemann and Barborka [14]. It is reasonable to assume that the diffusion rate for both acids is the same, not only because they are chemically very closely related, but also because both acids increase over their resting levels at approximately the same level of oxygen intake. The delayed appearance of the maximum blood lactate at maximal work loads must be due to a large concentration gradient existing in muscle cells, as the highest level of blood lactate is reached at a time when the oxygen intake and pulse rate have returned to pre-exercise levels.

The rate of removal of lactate can be accelarated by subjecting the individual to light exercise instead of allowing total rest after maximal exercise, so that the circulation rate is slightly increased and lactate transfer to reactive centres is speeded up.

If the diffusion rate of pyruvate is similar to that of lactate, then one explanation for the blood pyruvate reaching the maximum level so late during recovery can be ascribed to a post-exercise resynthesis from body lactate. Increase of body lactate by any means always resulted in an increase in blood pyruvate [15] which is positive proof for such a hypothesis.

Lactate/Pyruvate Ratio:

The calculated lactate/pyruvate ratios are given in Table V11(b) for five subjects. It is remarkable that for all subjects, during light exercise, the ratio is maintained with great constancy at approximately the resting figures, that is between the limits of 8 - 14. With more moderate exercise the ratio is slightly disturbed, while with more strenuous exercise the ratio is changed

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13. Johnson R.E., H.T. Edwards. "Lactate and pyruvate in blood and urine after exercise". Journal of Biological Chemistry, 118: 427, 1937.

14. Friedemann, T.E., Barborka. "The significance of the ratio of lactic to pyruvic acid in the blood after exercise". Journal of Biological Chemistry, 141: 993, 1941.

15. Bueding, E., W. Goldfarb. "Blood changes following Glucose, lactate and pyruvate injections in man". Journal Biological Chemistry 147: 33, 1943.

significantly and for all subjects at the maximal exertion a figure was found of about 20, range 21.8 to 24.0. The only exception was subject Arm., for whom a ratio was found of 34.7.

No satisfactory reason can be put forward for this peculiar discrepancy, although it is noticeable that in Arm's case the final pyruvate values are lower for the amount of lactate found compared with the other subjects. Furthermore he was the youngest member of the group tested, although his exact age is not known.

These findings are comparable with data presented by Sachs & Morton [16] on mammalian muscle preparations. It was found that during a steady state of contraction only a slight increase in lactate and pyruvate occurred, with a low ratio of lactate to pyruvate. With tetanic contraction under essentially anaerobic conditions they found a marked increase in lactate and pyruvate, but the increase was proportionately greater for lactate than pyruvate. This naturally resulted in a high ratio of lactate to pyruvate.

It is also in agreement with the result presented by Goldsmith [17] and Friedemann et al. [4] who found no significant change in the ratio during light exercise (9.3 and 14.9 respectively) while the latter author found, with severe exercise, a ratio of 28. Asmussen [5] found with moderately severe exercise lasting 20 - 30 minutes, a change in the resting value of 7 to figures between 17 to 28, but in general the ratios were about 20. He also showed that the ratio is dependent on the time of sampling and that ratios found during exercise differ significantly from those found during the recovery phase.

Friedemann and Barborka [14] suggested that the ratio is an excellent index of the oxidative processes occurring in the

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16. Sacks, J; J.H. Morton. "Lactic and pyruvic acid relations in contracting Mammalian muscle". American Journal of Physiology, 186: 221, 1956.

17. Goldsmith, G.A. "The blood lactate-pyruvate relationship in various physiologic and pathologic states". American Journal of Medical Sciences, 215: 182, 1948.

body. Furthermore it is more informative to use the ratio of lactate to pyruvate than to consider the metabolism of the individual metabolites seperately, as the latter are interrelated and a great many factors may influence their concentration in the body. The discrepancy in the ratio found for light exercise as compared with that for more severe exercise can possibly be explained along the following lines. In isolated enzyme systems, it was found that the lactic dehydrogenase system (L.D.H) favours the formation of lactic acid. If the L.D.H. system is written in its equational form:

Lactic acid $\rightleftharpoons$ Pyruvic acid + $\frac{DPNH}{DPN}$ it can be seen that lactic acid is dependent on two factors, the pyruvate concentration and the ratio of DPNH to DPN, which means the supply of oxygen. If the reaction proceeds faster to the left and therefore favour lactic acid formation, the ratio of the lactic acid to pyruvic acid would be more than unity. This probably accounts for the resting ratio of 7 - 12 found in the human body.

Under essentially anaerobic conditions, during very severe exercise when the oxygen transfer from the lungs to the working muscles is inadequate, glyceric aldehyde phosphate is oxidized to 1:3 diphosphoglyceric acid, while DPN is reduced to DPNH in the process. This reaction is coupled with the reduction of pyruvic acid to lactic acid with the simultaneous oxidation of DPHN to DPN.

In such a situation the reaction mentioned above proceeds to the left with increased velocity so that the ratio of lactate to pyruvate is increased and high values are found.

When the supply of oxygen to the working muscles is adequate, that is during light exercise where a steady state prevails, the cytochrome system and the Krebs cycle are competing with the pyruvic acid as electron acceptors for the oxidation of DPNH, as well as competing with LDH system for the pyruvic acid formed. Under these conditions a lower ratio is found which differ very little from resting values.

Maximum Values (Lactate):

The average lactate value of 118 mg% found (range 102-139 mg %) for the seven subjects after exhaustive exercise is in excellent agreement with the findings of other authors, where a similar type of exercise was used and where blood was sampled from the finger tip.

Asmussen et al.[18] reported values of 120 and 128 mg % for two athletes working on a bicycle ergometer to a state of exhaustion within 5 - 6 minutes. Astrand [9], using similar techniques, found an average figure of 110 mg %, for 28 male subjects. In contrast to the above findings Holmgren [19] found an average blood lactate of 52.8 mg % for 12 subjects working at an intensity of 2500 Kgm/min. for approximately 1 minute. A bicycle ergometer was used and blood samples were taken from the brachial artery. This low blood lactate response to exercise can only be ascribed to a short exercise period and to a pedalling rate well below the required rate of 60 r.p.m.

Friedemann & Barborka [14] reported a case where the maximal lactate value was 188 mg % with a pyruvate value of 5.2 mg %. Robinson et al.[20] found values of 116, 134 and 150 mg % for three excellent athletes after an exhaustive run. Friedemann et al.[4] using a well trained athlete, reported a maximal lactate value of 139.5 mg % and 4.98 mg % pyruvate after a run of 1 mile at a speed of 8.8 m.p.h.

Testing 6 untrained subjects on a treadmill, Wells et al.[21] found an average lactate value of 112 mg % after an

18. Asmussen, E., W. v. Döbeln and M. Nielsen. "Blood lactate and oxygen debt after exhaustive work at different oxygen tensions". Acta Physiologica scandinavica, 15: 57, 1948.

19. A. Holmgren. "Circulatory changes during maximal work in man". Scandinavian Journal of Clinical Laboratory Investigation, Vol. 8, Suppl. 24; pp 1 - 97, 1956.

20. Robinson, S., H.T. Edwards and D.B. Dill. "New records in human power". Science, 85:409, 1937.

21. Wells, J.G., B. Balke and D.D. Van Fossan. "Lactic acid accumulation during work. A suggested standardization of work classification". Journal of Applied Physiology, 10: 51 - 55, 1957.

exhaustive run. Using a treadmill Crescitelli and Taylor [10] found values as high as 320 mg % for one subject. Finger tip blood was taken and the results analysed according to the method of Barker and Summerson [22].

In the light of other findings, it is obvious that all the figures quoted by the latter authors, for all subjects tested, are at least twice as high as normal values. Their resting results of 28.5 mg% show that it must at least be halved to fall within normal limits. It is quite possible that glycolysis is responsible for the high values found, owing perhaps to a delay in the deproteinization of the sampled blood. A reasonable supply of blood from the finger tip is necessary to enable one to take a rapid sample, but this is only achieved if a deep cut is made, which is unpleasant for the person under test.

Heat Study:

Heart Rates : (Figures 1 - 3, Table 111 a & b).

Pulse rates for all subjects were significantly higher in heat than in cold, indicating that the hot environment plus work imposes a greater stress on the circulation than work performed in a cool condition.

Oxygen Consumption: (Figures 4 - 6, Tables 1V a & b).

A comparison of the oxygen consumption at different work loads in the two environments studied, shows that all the graphs have one characteristic in common. The oxygen intake increases linearly with increasing work loads, up to a certain maximum where it levels off. When the work rate was raised further, no significant increase in oxygen intake occurred. This is in agreement with data put forward by Hill [23], Astrand [9], Mitchell et al. [24] and others.

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22. Barker, S.B. and W.H. Summerson. "The colorimetric determination of lactic acid in biological material". Journal of Biological Chemistry, 138: 535, 1941.

23. Hill, A.V., H. Lupton. "Muscular Exercise, lactic acid and the supply of oxygen". Quarterly Journal of Medicine, 16: 135 - 171, 1923.

24. Mitchel, J.H., B.J. Sproule and C.B. Chapman. "The Physiological meaning of the Maximal Oxygen Intake Test". The Journal of Clinical Investigation, 37: 538 - 547, 1958.

Under normal conditions, the linear part of the oxygen curve represents a condition where mainly aerobic metabolism takes place, i.e. the metabolism of pyruvate in Krebs cycle, the so called 'main line oxidation'. As the curve approaches its asymptote the oxygen intake does not keep pace with the increase in work load, with the result that the 'branch line oxidation', anaerobic in nature, is superimposed on the aerobic phase. Part of the required energy is now supplied anaerobically, while some pyruvate functioning as hydrogen carriers, is reduced to lactate in the process.

Further comparisons reveal that for subjects Zef. and Vic. the oxygen intake in the hot room is significantly lower than for similar work loads under normal conditions. Only at the lowest and highest intensity were the oxygen intakes similar. These findings were rather unexpected, because they are direct in contrast with the data of the third subject (Den.) whose results reveals no such difference. For Den. the two sets of limits (95%) overlap along the entire range of work loads showing no difference between 'hot' and 'cold' data.

At first sight it may appear that for subjects Zef. and Vic. the work in the heat was performed with greater efficiency. However, Dill [25] pointed out that although the oxygen intake may show wide variations with different external and internal temperature for certain activities such as walking and running, for bicycle ergometry it is nearly negligible. Their data substantiate the work of Oknishi, who studied the same problem. It seems therefore that efficiency cannot account for the discrepancy in the oxygen consumption found for variations in temperature.

An alternative explanation for this finding is that under heat stress the working muscles were not adequately supplied with oxygen. The discrepancy between the 'hot' and 'cold' curves indicates

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25. Dill, D.B., H.T. Edwards, P.S. Bauer and E.J. Levenson "Physical Performance in relation to External Temperature". Arbeidsphysiologie, 3(4) : 508 - 518, 1930 - 31.

the amount of oxygen that fell short of metabolic needs for muscular work in the heat, and further shows the degree of anaerobic metabolism necessary to supplement the required energy.

If this hypothesis is true, then the blood lactates found under heat stress, should be higher than those found under normal conditions. This assumption that alterations in blood lactate are due to an oxygen deficiency is, however, subjected to certain objections put forward by Huckabee [26]. He postulated that changes in pyruvate affect lactate levels as much as does oxygen lack and concludes further, that lactate can only be used as a quantitative estimate of anaerobic metabolism, if pyruvate remains unchanged at all times. It seems therefore essential at this stage to comply with the hypothesis or conditions put forward by Huckabee, before lactate is used as an index of anaerobic metabolism.

Blood Pyruvate: (Figures 10, 11; Table VI a & B).

From the graphs it is clear that the pyruvate increased during exercise in both environments studied. For subject Vic. there is no significant difference between 'hot' and 'cold' data, while for Zef. the difference between 'hot' and 'cold' is only significant at oxygen consumptions between 1.5 and 2.0 litres/min. and this difference is small in comparison with the highly significant differences found in blood lactates and oxygen intake. It can be assumed that blood pyruvate increases with the intensity of exercise, but the magnitude of the increase is not affected by environmental temperature.

This finding is of great importance, because it immediately overcomes the main objection of Hackabee, viz. using blood lactate as a measure of oxygen supply. It can therefore be assumed that any difference between the blood lactate in hot environments compared with data obtained under normal conditions, must necessarily

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26. Huckabee, W.E. "Relationship of pyruvate and lactate during anaerobic metabolism. II. Exercise and formation of oxygen debt". Journal of Clinical Investigation 37: 255 - 263, 1958.

be due to anoxia, because the pyruvate change in blood is similar in both environments, and its affect on blood lactate will therefore be equal in both environments.

Blood Lactate: (Figures 19, 20, Table V a & b).

A comparison between the 'hot' and 'cold' data for subjects Zef. and Vic., reveals that the blood lactates in hot conditions are significantly higher, at all levels of oxygen consumption, than those found under normal conditions. This parallels the oxygen intake values found under similar conditions. Furthermore it is of interest that the increase of blood lactate over the resting values take place at different oxygen consumptions. Under normal conditions O_2 intakes were 2.2 litres/min. oxygen, for subject Zef. and 2.1 litres/min. for Vic., while in hot conditions they were 1.3 and 1.4 litres/min. respectively.

The above data, pointing to an increase in blood lactate at high environmental temperatures, lends support to the findings of Robinson et al.[27], and Dill et al.[25]. Both these authors made use of light work loads, while the time of exposure to heat was somewhat longer. Robinson studied eight subjects running on the treadmill, first at room temperature and thereafter in severe heat. For the same task under normal conditions the average lactate values were 14 mg %, while in hot conditions it rose to 22 mg %. Dill et al. on the other hand, used a bicycle ergometer, and found that in hot conditions the blood lactates were increased by as much as 4.5 to 32.4 mg % for the five subjects studied.

At room temperature, for all subjects acting as controls, the 'threshold' value for lactate was found at an oxygen consumption equivalent to 2/3 of their aerobic capacity. In heat, however, it is evident that for subjects Zef. and Vic. the threshold value for lactate was reached before the metabolic rate was 50% of

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27. Robinson, S., D.B. Dill, J.W. Wilson and M. Nielsen. "Adaptation of White men and Negroes to prolonged work in humid heat". American Journal of Physiology, 21: 261, 1941.

the aerobic capacity. It seems that in severe heat there is a decrease in the individual's aerobic apacity; although he can still reach the maximum oxygen consumption found under room temperature conditions. This is only because at these high levels of work the time of exposure is too short to have any deleterious effects on the body. The reason for this apparent decrease in aerobic capacity can only be explained as a result of circulatory insufficiency.

Christensen [28] suggested that under normal conditions an individual should not work for long periods at an oxygen consumption more than half his aerobic capacity, while under adverse conditions the level of oxygen consumption must be lower than half the aerobic capacity. Substantial evidence is provided by the present study in support of this hypothesis. It is clearly demonstrated in the case of two subjects, that certain work performed aerobically under normal conditions were carried out anaerobically under heat stress.

Unlike the curves of the other two subjects studied, comparison of Den's. 'hot' and 'cold' curves, Fig. (16) relating blood lactate to oxygen consumption, show no significant differences. His blood lactate data show good agreement with the oxygen intake data; the latter showed no significant difference due to environmental temperature.

The individual differences in regard to oxygen consumption and blood lactate, observed in this study, can only be ascribed to the efficiency of the circulation in supplying the active muscles with oxygen.

It is common knowledge that both physical training and acclimatization to heat have pronounced effects on the efficiency of the circulation. It would appear at this stage that subject Den. is more thoroughly acclimatized than the other two, because the latter show signs of an unstable circulation due to over taxation.

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28. Christensen, E.H. "Symposium on Fatigue". P. 93, Ergonomics Research Society, London, 1953.

Although the one subject (Den.) had a smaller maximal aerobic capacity than the other two, it appears that he tolerated heat stress better. It has been reported that physically fit individuals tolerated severe heat better and also acclimatized more rapidly than untrained men. Therefore, in order to draw some conclusions on the fitness of the three subjects, the lactate/oxygen curves for all three are compared in both environments, figures (24-27). In cool conditions it is obvious that the curve for Den. is significantly higher than those for the other two subjects, which are remarkably similar. In the light of experiments reported by Crescitelli and Taylor [10] and Taylor [11], who showed that unfit individuals usually have higher blood lactates than fit persons when doing similar tasks, it follows that subjects Den. appears to be the least fit of the three, when their performances are compared in the cool.

In hot conditions, however, Den. has much lower blood lactates compared with subjects Zef. and Vic. Although his lactate/oxygen curve in the heat is identical with that in cool conditions it appears that the curves of the other two subjects in hot conditions are significantly higher than their curves in cold conditions, with the result that they are also significantly higher than the 'hot' curve of Den. Using blood lactates as a criterion of physical fitness it appears that Den. is the fittest of the three subjects - he is better adapted to work in the heat.

The assumption that blood lactate is a criterion of circulatory efficiency under heat stress is only justifiable, if it can be proved that the factors eliminating lactate from the blood remained unchanged in both environments.

1. Sweat: According to Lee [29] there is no reason to believe that blood lactate is removed at a greater rate in heat than in cool conditions, although he points out the possibility

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29. Lee, D.H.K. "A Basis for the study of man's Reactions to Tropical Climates". Queensland University Papers, Vol. 1, Number 5, 1940.

that more lactate may be excreted by the sweat glands owing to increased sweat rates. More recent evidence does not bear out with his assumption and it has been shown that the lactate content of sweat is by no means correlated with blood lactate but is merely a reflection of the metabolism of the sweat gland.

2. Liver: The principle site of blood lactate removal is the liver. It has been shown that during exercise a reduction of the splanchnic blood flow takes place; while Bishop et al.(30) actually found that for cardiac patients during exercise, the hepatic flow was reduced to 65% of the normal resting value. In heat, this phenomenom may be enhanced to such an extent that water drunk during heat exposure may cause vomiting or water diarrhea, which is evidence of a blood flow to the gastro-intestinal organs too low to allow them to absorb the water. Blood lactate is removed at a greater rate during exercise than at rest (12), but in a hot environment where the liver receives less blood, the removal rate must be lower than under cool conditions.

If the liver function was impaired in heat it would have been reflected in high lactate and low pyruvate values. In this study it was found that the pyruvate did not change with environmental temperature, and this is against the suggestion that liver function is impaired. Furthermore, if a hot environment impairs liver function it must most certainly affect the blood glucose because the liver is considered as the 'glucostat' of the human or animal body. No evidence could be found in the literature that this actually occurred in heat, which substantiates the present reasoning that the efficiency of the liver in removing blood lactate remained the same under both conditions studied.(25).

3. Kidney: During exercise and thereafter a fraction of the blood lactate is excreted in the urine. It was found that during exercise the blood flow to the kidney was reduced while the filtration

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30. Bishop, J.M., K.W. Donald and O.L. Wade. "Changes in the Oxygen content of hepatic venous blood during exercise in patients with Rheumatic Heart Disease". Journal of Clinical Investigation, 34: 1114 - 1125, 1955.

rate was decreased by 45% [31]. Radigan and Robinson [32] compared kidney function and renal plasma flow during exercise under normal conditions and in severe heat. The renal plasma flow was reduced more in the hot environment while the filtration fraction was simultaneously increased. If any difference does exist between the excretion rates of lactate in the two environments it must be negligible, because it has been shown that the excretion rate of lactate in urine is low [1], especially for well-trained persons.

It has been pointed out that in hot humid environment heat dissipation rests entirely upon the circulation, the so called conductance or skin blood flow [33]. This means that in humid heat, the circulation is the main avenue whereby heat is transferred from the core of the body to the surface or skin. This can only lead one to believe that differences observed between Den. and the other two subjects in the heat, must point to a difference in magnitude of the skin blood flow. Although in several reports [34 - 35(a)] it is claimed that acclimatization brings about an increase in blood volume, and that this factor, in addition to a higher pulse rate in the heat, compensates for the increase in skin blood flow, it is usually found that in prolonged exercise in heat the peripheral resistance breaks down with the result that the circulation in general collapses. This condition is accompanied by an inability to remain in thermal balance

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31. Barclay, J.M., W.T. Cooke, R.A. Kenney and M.E. Nutt, "_____" American Journal of Physiology, 148 : 327, 1947.

32. Radigan, L.R. and S. Robinson. "Effects of Environmental Heat Stress and Exercise on Renal Blood Flow and Filtration Rate". Journal of Applied Physiology, 2:185 - 191, 1949.

33. Hardy, J.D. and G.F. Soderstrom. "Heat loss from the Nude Body and peripheral blood flow at Temperatures of 22° C. to 35° C." The Journal of Nutrition 16: 493-510, 1938.

34. Bass, D.E. et al. "Mechanisms of Acclimatization to heat in man". Medicine 34: 323 - 380, 1955.

35. Bass, D.E. and A. Henschel. "Response to Body Fluid Compartments to Heat and Cold". Physiological Reviews, 36: 128 - 144, 1956.

35(a). Bazett, H.C., F.W. Sunderman, J. Doupe and J.C. Scott. "Climatic Effects on the Blood Volume and Composition of Blood in man". The American Journal of Physiology, 129: 69- 83, 1940.

with the result that the rectal temperature is significantly elevated.

Nielsen [36] brought forward evidence that rectal temperature is related to the metabolic rate in cool conditions. As the subjects of this study were all subjected to the same work loads it can be assumed that the rectal temperature is a measure of the effectiveness of the circulation in ridding the body of excessive heat. In figure (23) a comparison is made between the rectal temperature response to work in heat and cold for subjects Zef. and Den.

Another remarkable feature of this graph is that for both subjects in cold conditions the two curves overlap along the entire range of work rates, while in hot conditions there is a significant difference between the curves; the one for Den. being higher at all work loads.

The differences between 'hot' and 'cold' rectal temperature is a measure of the combined stress of exercise and heat on the subject. For both subjects at a work rate of 2500 ft.lbs/min. the stress is clearly similar as rectal temperature in heat and cold is the same, especially for subject Zef. who showed a very small deviation from the cold value. This provide a possible explanation for his oxygen intake being the same as that in the cold, because at work rates of 10,000 and 11,000 ft.lbs/min. the difference between rectal temperature was small and at these work loads the oxygen intake was once more similar to that in cold conditions.

It is evident from data on the rectal temperature of the two subjects that subject Zef. maintained a lower deep body temperature than Den. for all rates of work. To achieve this a greater skin circulation was necessary.

The difference between Den. and the other two subjects can be explained along the following lines. For all subjects the cardiac output remained the same for both environments, at all intensities of work Figures (21, 22). This has been accomplished by a

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36. Nielsen, M. Skandinavische Archiv für Physiologie, 79 : 193, 1938.

shunt of blood from the splanchnic area to compensate to some extent for the increase in blood flow to the skin.

In the case of subject Den. the amount of blood supplying the active muscles was the same in both environments, which is indicated by similar lactate curves for both conditions. This could only be achieved if the skin circulation was somewhat reduced, which accounts for his higher rectal temperature as compared with those of subject Zef.

The other two subjects were unable to mobilize part of the skin blood flow like Den. with the result that their deep body temperatures were lower than that of Den. The supply of blood to the active muscles was insufficient with the result that a condition of relative anoxia occurred. This condition is suggested from the lower oxygen uptake and a significantly higher blood lactate value.

CHAPTER VI

Conclusions:

Under normal conditions the blood pyruvate increases with progressive work loads in a similar manner as that found for blood lactate, although the magnitude of the increase is much smaller.

During exercise the constancy of the blood lactate, pyruvate ratio will depend mostly on the blood lactate, seeing that blood lactate changes, usually alters the ratio more significantly than changes in blood pyruvate.

Under normal conditions of temperature, the blood lactate increases with exercise, above the resting value, when the oxygen consumption reaches a level of 66 to 68% of the individual's aerobic capacity. Furthermore, this increase is found at a pulse rate of 120 to 140 beats per minute.

Under heat stress the threshold value for lactate is still found at a pulse rate of 120 - 140 beats/min. but the level of oxygen intake then only represents approximately 50% of the individual's AEROBIC CAPACITY.

The apparent decrease in a persons aerobic capacity under heat stress, can only be attributed to an increase in skin blood flow.

Summary:

Seven mine recruits (six Africans and one Coloured), in the post-prandial state, were tested on a bicycle ergometer at work loads ranging from 1800 ft.lbs/min. to each individual's maximal work load.

Measurements were made of the changes in pulse rate, oxygen consumption, cardiac output, rectal temperature, blood lactate and blood pyruvate with progressive work rates.

The ratio of blood lactate to pyruvate shows no increase at the end of light exercise but is significantly changed at the end of moderate and hard work. A possible explanation is put forward to explain the change in ratio during hard work.

A close correlation is found between the increase of blood lactate over resting values, during exercise, and the individual's aerobic capacity.

For two of the three subjects studied under heat stress, the oxygen consumption at certain work rates, was found to be significantly lower in the heat than at room temperature. While the blood pyruvate showed no significant change with a change in environment, the blood lactate response to exercise increased with an increase in environmental temperature.

The threshold value for lactate under heat stress, was found at a lower level of oxygen intake than the level found at room temperature. This indicates to an apparent decrease in an individual's aerobic capacity, when subjected to heat stress. The possibility that heat stress may have impaired the rate of lactate removal by certain organs is discussed, as well as the influence of an increase in skin blood flow on the principle function of the circulation, viz. oxygen transport.

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