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Blood Pyruvic Acid in Heart Disease.

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Meakins and Long[1] were the first to study intermediates of tissue carbohydrate metabolism in heart disease. They showed a marked and proportional increase in blood lactic acid in patients with heart failure. These results were subsequently both confirmed[2] and denied[3] The importance of lactic acid as the center of carbohydrate metabolism has since been overshadowed by that of pyruvic acid, and more recently even more interest has attached to pyruvic acid because of Peters'[4] discovery that vitamin B_1 is necessary for its oxidation.

Taylor, S. Weiss, and Wilkins[5] attempted blood pyruvic acid estimations in all types of disease by measuring the blood bisulfite binding substances and showed a rise of these in failing hearts. This method of pyruvate estimation has since been shown, however, to be non-specific and inaccurate.[6, 7] This present work makes use of a specific method[8] and deals only with heart disease.

Procedure. The blood pyruvate levels of organically normal individuals and patients with decompensated and compensated heart disease were studied. The control group consisted of 5 male and 5 female carefully selected psychopaths, ranging in age from 25 to 84, non-alcoholics who were free of organic disease, had no evidence of dietary deficiency, and who were not excited or depressed. A pyruvate determination was done on each daily for 6 days and after one-half hour rest in bed. Twenty hospital patients, ages 32 to 79, with varying degrees of congestive failure, and with no fever, history of dietary deficiency, or impaired renal function other than that due to the cardiac failure, and no other organic disease, constituted the second group on whom daily blood pyruvate determinations were

1 Meakins, J., and Long, C. N. H., *J. Clin. Invest.*, 1927, **4**, 273.

2 Harris, E. W., Jones, C. N., and Aldred, C. N., *Quart. J. Med.*, 1935, **4**, 407.

3 Weiss, S., and Ellis, L. B., *Arch. Int. Med.*, 1935, **55**, 665.

4 Peters, R. A., *Lancet*, 1936, **1**, 1161.

5 Taylor, F. H. L., Weiss, S., and Wilkins, R. W., *J. Clin. Invest.*, 1937, **16**, 833.

6 Wortis, H., Bueding, E., and Wilson, W. E., PROC. SOC. EXP. BIOL. AND MED., 1940, **43**, 279.

7 Elsom, K. O., Lukens, F. D. W., Montgomery, E. H., and Jones, L., *J. Clin. Invest.*, 1940, **19**, 153.

8 Lu, G. D., *Biochem. J.*, 1939, **33**, 249.

TABLE I.*
Range and Mean Blood Pyruvate Levels of Controls.

Subject No.	24	25	26	27	28
Range**	.56-.80	.72-.84	.80-.98	.78-1.00	.80-.96
Mean	.68	.76	.92	.92	.88
Subject No.	29	30	31	33	34
Range**	.60-.88	.60-.90	.64-1.10	.60-.88	.64-1.00
Mean	.76	.79	.88	.73	.80

*Values expressed here and elsewhere as mg per 100 cc of blood.
**Range of each subject based on 6 daily determinations.

TABLE II.
Decompensated Heart Group.

Subject No.	1	2	3	4	5	6	7	8	10	12
Etiology	H	H	A	A	A	A	A	H	A	H
Severity	S	S	S	M	M	M	S	M	S	S
Peak*	3.4	1.9	2.0	2.2	1.8	2.0	2.3	1.8	1.9	2.2
Subject No.	14	15	17	18	19	20	21	22	47	49
Etiology	A	R	R	U	H	R	H	CT	A	R
Severity	S	M	M	M	S	M	S	S	M	S
Peak	2.1	1.4	1.2	1.7	2.5	1.5	1.7	1.7	1.5	1.8

Abbreviations: H, hypertensive; A, arteriosclerotic; R, rheumatic; CT, coronary thrombosis; U, unknown; S, severe; M, moderate.
*Highest blood pyruvate recorded.

made. In the third group were 12 patients, ages 20 to 62, with heart disease but without failure.

Results and Discussion. Table I reveals sharply defined limits of normality, with 1.0 as the upper limit and 0.8 as the mean.

The patients with decompensated hearts (Table II) show only one case, No. 17, whose highest blood pyruvate was not significantly elevated. The mean "peak" level for the group is 2.15, with a range of 1.2 to 3.4.* Every case but one showed changes from day to day. These variations, however, were not considered significant unless they were higher than 0.3, the maximum day to day variation of the controls. An attempt was made to correlate the daily fluctuation with the clinical condition of the same day. Making allowance for one discordant variation, 79% of the patients show daily variations in agreement with the corresponding clinical changes. Or, of a sum total of 58 daily variations in all of the cases, 43 or 74% were clinically correlative.

It should be remembered, however, that clinical estimation of the change of a heart patient's condition from day to day is often diffi-

* This roughly approximates the values that Lu[9] found in acute beriberi.

[9] Platt, B. S., and Lu, G. D., *Quart. J. Med.*, 1936, **29**, 355.

TABLE III.
Compensated Hearts.

Subject No.	35	36	37	38	39	41	43	44	53	54	55	56
Type	R	R	A	A	A	R	H	H	H	H	H	A
B. P.	0.80	0.56	0.80	0.56	0.72	0.80	0.88	0.80	0.64	0.72	1.16	0.84

Abbreviations: B. P., blood pyruvate; others as in Table II.

cult, if not at times impossible. It is quite possible, therefore, that even though the daily pyruvate level may not fit in with the clinical evaluation of that given day the former may still be the better index of the patient's true condition. That the mean peak of the severe failures was 2.13 as contrasted with 1.67 for those moderately ill, may be further evidence in this direction.

Of the 7 cases who died 3 showed a rise of pyruvate as death approached, 3 no change, and one a fall. It is interesting to note that 2 cases of edema of non-cardiac origin which were also studied showed no rise above the normal at any time. The age, sex, and type of heart disease could not be shown to bear any relationship to the pyruvate level.

For the compensated group (Table III) 12 patients with definite cardiac enlargement (10 to 110% oversized) were selected for study. The pyruvate levels were well within normal limits except for one, No. 55, who showed a value of 1.16. Of further interest were 2 ambulant patients with heart disease who appeared to be compensated except for a definite but slight edema, and they both had increased values of 1.2.

Summary. 1. There is a rise above the normal of blood pyruvic acid in heart failure. 2. This elevation approximates the degree of failure.

The author is very grateful to Dr. Emmet B. Bay for his helpful suggestions and criticism and to Dr. E. S. G. Barron for the use of his laboratory and his technical guidance.