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STUDIES IN CONGESTIVE HEART FAILURE

III. The Buffering Power of the Blood and Tissues

By COBB PILCHER, GURNEY CLARK and TINSLEY RANDOLPH HARRISON

(From the Department of Medicine, Vanderbilt University School of Medicine, Nashville, Tennessee)

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The results reported in a previous paper of this series (Harrison and Pilcher, 1930) led us to accept tentatively the hypothesis of Eppinger, Kisch and Schwarz (1927) that the buffering power of the tissues is diminished in congestive heart failure.

Since the evidence from our previous work was indirect, it seemed wise to study the matter further by more direct methods. Accordingly, the pH of the blood, determined by Cullen's (1922) method, using the micro-modification of Hawkins (1923) and the bicolor standards of Hastings and Sendroy (1924), and the carbon dioxide content of the blood, determined by analysis in the Van Slyke-Neill (1924) constant-volume apparatus were studied in patients with cardiac failure and in normal subjects, in four series of observations.

a. The pH and carbon dioxide content of the blood at rest was found to be usually within normal limits in both the "decompensated" patients and the control subjects, as shown in the left-hand parts of tables 2, 3 and 4. However, that a state of "cardiac acidosis" may exist is shown in table 1.

b. The effect of the oral administration of 15 to 17.35 grams of ammonium chloride over a period of 12 hours was found to be approximately the same on the arm venous blood (drawn without stasis) of normal and "decompensated" individuals. The data are shown in table 2. These observations were suggested by the work of J. B. S. Haldane (1924), who showed that ammonium chloride given in large amounts by mouth caused a diminution in the alkaline reserve of the blood.

c. The effect of breathing a mixture of 5 per cent carbon dioxide and 95 per cent oxygen was found to be slightly greater on the pH of the blood of three of the four "decompensated" patients than on that of any of the normal subjects. The carbon dioxide content was usually increased somewhat in both controls and patients (table 3). One patient (Ada D.) who had nephrosis with massive edema, but no evidence of heart disease, showed the greatest diminution in pH found.

TABLE 1

Spontaneous acidosis in patients with heart failure

Patient	Blood from	CO_2 content of blood	Clinical data
		volumes per cent	
A. R.	Artery	33.0	Marked dyspnea; massive edema (weight 33 pounds greater than when edema-free)
	Artery	39.3	5½ hours later; 3 hours following administration of 20 grams sodium bicarbonate. Dyspnea diminished
H. C.	Artery	31.1	August 4, 1928. Luetic aortic insufficiency; severe pulmonary edema; Cheyne-Stokes respiration; cyanosis of fingers; massive edema
	Artery	61.4	August 5, 1928. Greatly improved. Edema diminished; respirations rapid but regular. (Diuresis following "Salyrgan")
	Artery	65.2	August 7, 1928. Has broncho-pneumonia and severe anoxemia (47.5 per cent arterial oxygen saturation). Edema stationary
R. R.	Arm vein	30.8	Aortic insufficiency; terminal heart failure; blood drawn a few minutes before death; pH 7.08

d. The effect of exercise was determined in three patients with no circulatory disorder, in one patient with heart disease but neither dyspnea nor edema, and in five patients with varying degrees of "decompensation." Blood was drawn from the femoral vein of the subject before and within 30 to 60 seconds after a standardized exercise of the leg. Observations were made on both legs (with an interval between) of one control subject and of two patients with congestive failure.

TABLE 2

The effect of ammonium chloride on the carbon dioxide content of the arm venous blood

Subject	Date	Arm venous blood before NH_4Cl		NH_4Cl	Arm venous blood after NH_4Cl				Clinical data
		CO_2 content	pH		CO_2 content	Change	pH	Change	
		volumes per cent		*grams in 12 hours*	*volumes per cent*				
G. C.	February 10, 1929	53.4	7.40	17.35	38.0	−15.4	7.23	−0.17	Normal controls
T. H.	February 17, 1929	50.4	7.38	17.35	35.7	−14.7	7.27	−0.11	
C. P.	February 4, 1929	47.2	7.33	17.35	41.7	− 6.5	7.25	−0.08	
D. K.	March 10, 1929	47.5	7.39	15.00	35.8	−12.7	7.36	−0.03	Hypertensive heart disease; chronic pulmonary disease; marked Cheyne-Stokes respiration; no edema
M. A.	March 4, 1929	48.7	7.33	15.00	42.3	−6.4	7.22	−0.11	Hypertensive heart disease; paroxysmal dyspnea; slight edema of lower half of tibia. (Duration, 6 days)
A. A.	February 5, 1929	56.6	7.36	17.35	44.0	−12.6	7.11	−0.15	Hypertensive heart disease; luetic aortitis; slight edema and dyspnea
	March 12, 1929	50.0	7.30	15.00	33.2	−16.8	7.16		No edema or dyspnea
A. D.	February 18, 1929	47.6	7.36	19.00	38.4	−9.2	7.40	−0.04	Hypertensive heart disease; paroxysmal dyspnea; no edema. Liver enlarged. *Vomited 3 times*
	March 12, 1929	54.6	7.34	15.00	40.1	−14.5	7.31	−0.03	No dyspnea or edema. No vomiting
T. P.	March 12, 1929	50.1	7.30	15.00	33.9	−16.2	7.23	−0.07	Luetic aortic regurgitation; massive edema; slight dyspnea
M. T.	March 12, 1929	55.2	7.37	15.00	38.8	−16.4	7.33	−0.04	Hypertensive heart disease; no dyspnea; slight edema
F. C.	March 12, 1929	43.5	7.32	15.00	36.8	−6.7	7.20	−0.12	Hypertensive heart disease; auricular fibrillation; slight edema; slight dyspnea

TABLE 3

The effect of breathing 5 per cent carbon dioxide on the pH and carbon dioxide content of the arm venous blood

Subject	Date	Breathing air			Breathing 5 per cent CO_2					Clinical data
		pH of arm venous blood	CO_2 content of arm venous blood	Total ventilation	pH of arm venous blood	Change	CO_2 content of arm venous blood	Change	Total ventilation	
			volumes per cent	*cc. per minute*			*volumes per cent*		*cc. per minute*	
G. C.	March 26, 1929	7.33	46.0	5,800	7.32	−0.01	47.4	+1.4	11,280	Normal controls
T. H.	March 29, 1929	7.38	43.4	4,440	7.33	−0.05	44.3	+0.9	13,500	
C. P.	March 17, 1929	7.32	46.7	4,800	7.29	−0.03	48.4	+1.7	11,160	
A. A.	March 19, 1929	7.30	53.1	6,260	7.23	−0.07	52.6	−0.5	19,840	Hypertensive heart disease; no edema or dyspnea for 6 weeks previously
A. D.	March 20, 1929	7.34	46.6	6,940	7.27	−0.07	48.0	+1.4	14,200	Hypertensive heart disease; no edema or dyspnea for 4 weeks previously
T. P.	March 27, 1929	7.26	47.3	6,840	7.26		50.4	+3.1	17,680	Luetic aortic regurgitation; no dyspnea; barely perceptible edema
T. B.	March 24, 1929	7.44	47.3	3,650	7.39	−0.05	48.8	+1.5	10,540	Hypertensive heart failure; edema of feet and ankles, back of legs and thighs. Disoriented mentally
Ada D.	March 24, 1929	7.39	44.0	5,360	7.31	−0.08	46.1	+2.1	11,760	Nephrosis; general anasarca; massive edema of 5 weeks duration. No evidence of heart disease

TABLE 4

The effect of exercise on the pH and on the carbon dioxide content of the femoral venous blood

Patient	Date	Leg	pH of femoral venous blood		Change	Clinical data	
			Before exercise	After exercise			
F. R.	February 21, 1929		7.35	7.30	−0.05	Gastric ulcer. No circulatory disease	Controls
C. L.	February 28, 1929		7.40	7.36	−0.04	Lung abscess (surgically drained). No circulatory disease	
J. W.	March 2, 1929		7.43	7.40	−0.03	Mild diabetes mellitus; moderate arteriosclerosis. No other circulatory symptoms	
J. H.	January 31, 1929	R	7.28	7.24	−0.04	Mild diabetes mellitus; hypertension cardiac enlargement; no dyspnea or edema	
		L	7.26	7.22	−0.04		
A. A.	February 2, 1929		7.23	7.16	−0.07	Hypertensive heart disease; moderate edema of feet and lower halves of legs; no dyspnea at rest	
T. P.	March 11, 1929	R	7.42	7.24	−0.18	Luetic aortic regurgitation; massive edema	
		L	7.40	7.25	−0.15		
M. G.	March 2, 1929		7.33	7.31	−0.02	Hypertensive heart disease; very slight edema (first "break" in compensation); duration, 4 days	
A. D.	February 24, 1929		7.38	7.25	−0.13	Hypertensive heart disease; very slight nocturnal dyspnea until 3 days previously; slight edema; 5th "break" in compensation	
D. K.	March 10, 1929	R	7.40	7.32	−0.18	Hypertensive heart disease. General anasarca; moderate dyspnea; Cheyne-Stokes respiration	
		L	7.38	7.34	−0.04		

In five of the seven observations on "decompensated" patients, the diminution in pH was greater than any diminution found in a control subject. The average diminution in the controls was 0.04 pH and in the decompensated patients, 0.11 pH. With one exception (table 4, D. K., left leg), the degree of change in pH was proportional to the extent or duration of the edema, or both.

The changes in carbon dioxide content of the blood varied greatly in both control and "decompensated" subjects and these data are not included in table 4. Such variation is to be expected, since the carbon dioxide content of the blood during exercise varies with three different factors: the oxidation occurring in the muscles, the lactic acid "overflowing" into the blood stream and the minute ventilation.

SUMMARY AND CONCLUSION

The pH and carbon dioxide content of the blood of normal subjects and of patients with congestive heart failure has been studied before and after (*a*) administration of large doses of ammonium chloride, (*b*) breathing 5 per cent carbon dioxide and (*c*) a standardized exercise.

The findings in patients with heart failure at rest were usually within normal limits, but a state of acidosis was found in three patients with very severe symptoms.

The changes occurring after administration of ammonium chloride and after breathing carbon dioxide were usually within normal limits in patients with congestive failure.

The changes in pH after exercise were usually greater in "decompensated" patients than in control subjects and the degree of change was, with one exception, proportional to the extent and duration of the edema. This is believed to be compatible with the presence of diminished buffering power of the tissues in heart failure.

One compensated cardiac patient showed changes similar to those found in control subjects and one patient with "non-cardiac" edema showed changes similar to those in the "decompensated" patients. This suggests that edema itself may be related to changes in the tissues of patients with heart failure.

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