WATER AND ELECTROLYTE CONTENT OF CARDIAC AND SKELETAL MUSCLE IN HEART FAILURE AND MYOCARDIAL INFARCTION

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METABOLIC studies of water and electrolytes, carried out on patients during their recovery from congestive heart failure, demonstrated an uptake of potassium and sodium and a release of water by the cells.[1] From these findings, it was postulated that, during the development of cardiac failure, the cells lost potassium and sodium and gained water, presumably because of activation of osmotically inert cellular base.

The present study is a part of a series on water and electrolyte metabolism in congestive failure. Its purpose is to compare the water and electrolyte content of myocardial and skeletal muscle obtained at autopsy from patients dying of congestive failure or myocardial infarction with the composition of specimens obtained from patients dying of noncardiac causes.

The material comprised forty-seven cases. In its selection, care was taken to eliminate patients with uremia, diabetic acidosis, or other metabolic disease likely to cause alterations in the composition of myocardium or skeletal muscle independent of cardiac disease.

The patients were classified from a clinicopathologic standpoint into the following groups: I, controls, comprising sixteen patients with normal hearts at autopsy and no evidence of a metabolic disorder; II, uncomplicated left ventricular hypertrophy, found in fourteen patients who showed no evidence of congestive failure, received no cardiac glycosides during hospitalization, and died of noncardiac causes; III, congestive failure with underlying left ventricular hypertrophy, present during hospitalization and at autopsy in eight patients despite digitalization; and IV, acute myocardial infarction, responsible for death in seven patients.

METHOD

Heart muscle blocks were obtained during autopsy from four standard sites in the left ventricle: (1) anterior-apical, (2) mid-septal, (3) mid-lateral, and (4) posterior basal. In Group IV separate blocks were taken from areas of acute

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Read by title, Central Society for Clinical Research, Nov. 3, 1950, Chicago. (Abstract, J. Lab. & Clin. Med. **36**:796, 1950.)

Supported by grants from the National Institutes of Health and the Michigan Heart Association.

Received for publication Oct. 9, 1951.

TABLE I. GROUP I—CONTROL

CASE	HEART MUSCLE (PER 100 GM. OF WET TISSUE)						SKELETAL MUSCLE (PER 100 GM. OF WET TISSUE)					
	H_2O (GM.)	Na (MEQ.)	K (MEQ.)	cl (MEQ.)	Mg (MEQ.)	P (MEQ.)	H_2O (GM.)	Na (MEQ.)	K (MEQ.)	cl (MEQ.)	Mg (MEQ.)	P (MEQ.)
1	80.1	5.13	9.75	4.67	0.435	4.10	80.2	6.50	6.00	5.66	0.302	2.76
2	79.3	4.86	9.55	4.27	0.345	3.80	78.3	3.34	12.00	3.27	0.369	3.40
3	81.3	4.82	10.72	4.33	0.441	4.35	77.9	3.08	10.60	2.73	0.405	3.90
4	79.4	4.91	8.03	4.00	0.450	4.10	75.3	3.74	10.40	2.61	0.465	4.08
5	77.9	4.00	8.98	3.91	0.605	5.37	74.4	2.78	10.10	2.28	0.495	4.60
6	79.8	4.90	8.00	4.03	0.456	3.47	77.5	3.34	10.45	2.17	0.457	3.01
7	78.6	4.21	8.55	3.13	0.416	2.80	79.1	2.38	6.63	1.88	0.510	2.80
8	78.0	5.05	9.70	3.85	0.348	3.82	75.8	4.13	14.50	2.39	0.350	5.38
9	78.5	5.38	7.40	4.20	0.437	4.53	79.0	5.69	4.75	3.69	0.395	3.09
10	78.4	5.21	7.76	4.16	0.495	2.96	76.6	4.05	9.78	3.01	0.467	3.14
11	79.3	5.73	7.96	3.83	0.498	3.40						
12	78.5	5.90	6.90		0.422	4.15	74.6	5.65	8.40		0.465	4.87
13	80.2	4.82	8.19		0.435	3.67						
14	77.7	4.65	7.20									
15	78.1	3.65	7.38									
16	76.1	6.17	7.11									
Mean	78.8	4.96	8.32	4.04	0.445	3.88	77.2	4.06	9.42	2.97	0.425	3.72
Standard deviation	±1 .2	±0.60	±1.09	±0.37	±0.060	±0.65	±1.9	±1.26	±2.65	±1.00	±0.063	±0.86

myocardial infarction and from distant areas that appeared uninfarcted to gross examination. Skeletal muscle blocks were secured from the pectoralis major. Fibrous and fatty tissues were trimmed off, and the inner portions of these blocks were analyzed for water, sodium, and potassium in all samples and, in addition, for chloride, magnesium, and phosphorus in the majority of the cases.

Water content was determined gravimetrically by vacuum desiccation under constant temperature of 100° C., using the Abderhalden apparatus. Sodium, potassium, magnesium, and phosphorus were analyzed by a modification of the spectrographic method for plasma and urine, after digestion of the dried samples with nitric acid-perchloric acid mixture.[2] In those specimens analyzed for sodium and potassium only, the flame photometer was employed.[3] Chloride content was determined by the Volhard titration method after digestion of a wet sample with 5 N sodium hydroxide.

RESULTS

The values for water are reported in grams per 100 Gm. of wet tissue, those for sodium, potassium, magnesium, phosphorus, and chloride in milliequivalents per 100 Gm. of wet tissue. One milliequivalent of phosphorus represented 55.8 mg. of the element.[4] Tables I, II, III, and IV give the results in each individual case of the four respective groups. Since no consistent difference in the composition of myocardium from the four standard sites was found in the absence of infarction, the values for cardiac muscle in Tables I, II, and III represent averages of the analyses of four separate blocks. In Table IV the values for infarcted myocardium and grossly normal myocardium are recorded separately for each case. Table V summarizes the results of each group.

DISCUSSION

Group I. Controls (Table I).—Since autopsy material from accidental deaths was not available to us, the control group was selected from patients who had died suddenly from diseases such as ruptured cerebral aneurysm or who had succumbed to brief illnesses, unattended by any obvious disturbance of fluid and electrolyte balance. The sixteen hearts classified into the control group were normal to gross and microscopic examination and served as the index of the normal in fluid and electrolyte content, subject to the reservation that unpredictable chemical alterations may have occurred during the agonal state in the absence of histologic changes. In spite of possible agonal alterations in chemical composition, the results were in good agreement with those reported in the literature. The average values per 100 Gm. of wet cardiac muscle in our sixteen cases were as follows: water 78.8 ± 1.2 Gm.; sodium 4.96 ± 0.60 meq.; potassium 8.32 ± 1.09 meq.; chloride 4.04 ± 0.37 meq.; magnesium 0.445 ± 0.06 meq.; and phosphorus 3.88 ± 0.65 meq. These figures were almost identical with the mean values in five cases reported by Wilkins and Cullen,[5] who found 78.9 Gm. of water, 4.0 meq. of sodium, 7.8 meq. of potassium, 0.42 meq. of magnesium, and 3.65 meq. of phosphorus per 100 Gm. of wet tissue. The authors did not analyze for chloride. Myocardial analyses in thirteen cases of accidental death reported

by Mangum and Myers[6] gave average values of 80.8 Gm. of water, 7.1 meq. of potassium, and 3.47 meq. of phosphorus per 100 Gm. The mean potassium content of five normal hearts analyzed in Harrison's laboratory[7,8] was 7.57 meq. per 100 Gm. of wet tissue. The values obtained by Herrmann and Decherd[9] and Scott[10] in cardiac patients without congestive failure were significantly lower, perhaps because these cases did not actually represent normal controls.

The average values for magnesium and phosphorus in skeletal muscle were comparable to those in the myocardium, but water, sodium, and chloride content was lower, whereas potassium content was higher. Our findings in respect to water and potassium confirmed early work in Harrison's laboratory.[7,8] From a study of the results in individual cases in Table I, it would appear that magnesium contents of skeletal and cardiac muscle were comparable in most instances, whereas sodium, potassium, and chloride contents were neither comparable nor parallel. Skeletal muscle was subject to greater variation in water, sodium, potassium, and chloride content than the myocardium.

The foregoing differences in chemical composition were believed referable chiefly to differences in partition of water between the muscle cell and interstitial space and in small part to differences in fat content. A rough estimate of the partition of water was made by the method of Newburgh[11] on the assumption that practically all chloride was extracellular and that the concentration in the interstitial water amounted to 117 meq. per liter. Fat content of cardiac muscle was negligible and therefore disregarded, whereas that of skeletal muscle was estimated at 1 Gm. per 100 Gm. of wet tissue. Estimates of the partition of water in myocardium, made with the foregoing premises, were as follows: 32.6 Gm. of extracellular and 46.2 Gm. of intracellular water per 100 Gm. of wet tissue, whereas the corresponding estimates for distribution of water in skeletal muscle were 23.5 Gm. in the interstitial space and 53.7 Gm. within the cells. Although the validity of these figures depends upon the accuracy of the assumptions employed in the calculations, it is noteworthy that the estimated partition in these cases is in accord with derived figures obtained by others from analyses of animal muscle[12] and biopsied human skeletal muscle.[13]

The intracellular concentrations of base were estimated on the assumption that the concentrations in interstitial fluid were 143 meq. per liter for sodium, 4.0 meq. per liter for potassium, and 3 meq. per liter for magnesium. The values for cardiac muscle, expressed in milliequivalents per liter of intracellular water, were sodium 6.3, potassium 177.0, and magnesium 7.7; the corresponding figures for skeletal muscle were sodium 13.1, potassium 174.0, and magnesium 6.8. The higher intracellular concentration of sodium in skeletal muscle compensated for the lower concentrations of potassium and magnesium, so that the concentrations of total base were essentially the same in cardiac and skeletal muscle.

Since there is no satisfactory unit available to indicate the number of cells actually present per 100 Gm. of wet tissue, it is necessary to consider the cells collectively. Obviously the total water content of 100 Gm. of wet tissue or the calculated distribution of extracellular and intracellular water reflects in no way the absolute quantity of water in each individual cell or the relative quantity of water in the extracellular compartment per unit number of cells.

TABLE II. GROUP II—UNCOMPLICATED LEFT VENTRICULAR HYPERTROPHY

CASE	HEART MUSCLE (PER 100 GM. OF WET TISSUE)						SKELETAL MUSCLE (PER 100 GM. OF WET TISSUE)					
	H_2O (GM.)	Na (MEQ.)	K (MEQ.)	Cl (MEQ.)	Mg (MEQ.)	P (MEQ.)	H_2O (GM.)	Na (MEQ.)	K (MEQ.)	Cl (MEQ.)	Mg (MEQ.)	P (MEQ.)
17	77.4	5.26	9.37	4.65	0.480	3.97	75.4	3.96	10.70	4.00	0.465	4.00
18	78.0	4.91	8.30	4.13	0.500	4.43	72.3	3.44	10.40	2.54	0.618	5.51
19	77.6	4.57	7.82	3.66	0.450	3.30	74.0	3.83	9.59	2.09	0.501	3.61
20	78.8	5.30	9.26	4.80	0.438	3.48	77.6	4.35	10.61	3.78	0.394	2.75
21	79.6	4.79	8.10	3.43	0.425	3.71	75.7	3.40	12.62	2.40	0.486	4.52
22	77.1	5.18	6.50	4.75	0.475	3.33	77.3	3.60	13.90	2.20	0.613	3.92
23	75.9	5.00	8.65	4.00	0.495	4.20	75.2	4.00	12.39	3.19	0.483	4.49
24	78.8	7.61	8.53	5.22	0.445	3.09	77.1	4.56		2.82	0.649	4.76
25	79.4	4.48	7.45	3.52	0.420	3.70						
26	76.1	4.53	8.95									
27	77.0	4.57	7.55									
28	76.8	4.09	8.30									
29	75.5	4.40	8.53									
30	78.7	4.30	7.88									
Mean	77.6	4.93	8.23	4.24	0.459	3.69	75.6	3.89	11.46	2.88	0.526	4.20
Standard deviation	±1.3	±0.67	±0.75	±0.60	±0.028	±0.41	±1.9	±0.39	±1.44	±0.67	±0.084	±0.77

TABLE III. GROUP III—HYPERTROPHIED, DIGITALIZED, CONGESTIVE HEART FAILURE

CASE	HEART MUSCLE (PER 100 GM. OF WET TISSUE)						SKELETAL MUSCLE (PER 100 GM. OF WET TISSUE)					
	H_2O (GM.)	Na (MEQ.)	K (MEQ.)	Cl (MEQ.)	Mg (MEQ.)	P (MEQ.)	H_2O (GM.)	Na (MEQ.)	K (MEQ.)	Cl (MEQ.)	Mg (MEQ.)	P (MEQ.)
36	80.4	5.49	6.90	5.05	0.398	3.68	76.6	3.44	7.58	4.50	0.361	3.13
37	80.6	4.00	6.65	2.76	0.460	3.90	80.5	3.05	6.65	2.45	0.404	3.68
38	78.1	5.61	6.45	4.26	0.394	3.55	70.6	3.00	7.70	2.20	0.343	3.12
39	79.2	4.83	6.90	4.26	0.435	4.00	72.6	4.00	7.38	2.87	0.414	4.22
40	80.5	5.90	6.90	4.26	0.385	3.86						
41	78.4	4.35	7.70				73.6	4.33	9.80			
42	77.8	5.49	5.75									
43	76.0	6.90	6.98									
Mean	78.9	5.32	6.78	4.12	0.414	3.80	74.8	3.56	7.82	3.00	0.380	3.53
Standard deviation	±2.3	±0.86	±0.51	±0.56	±0.063	±0.16	±3.4	±0.52	±1.06	±0.90	±0.030	±0.45

Group II. Uncomplicated Left Ventricular Hypertrophy Without Digitalization or Congestive Failure (Table II).—The myocardial water and electrolyte content in uncomplicated left ventricular hypertrophy was almost identical with that in the control group, as shown by a comparison of the mean figures in the first two tables. Thus the average values for hypertrophied and normal myocardium, respectively, were water 77.6 ± 1.3 Gm. and 78.8 ± 1.2 Gm.; sodium 4.93 ± 0.67 meq. and 4.96 ± 0.60 meq.; potassium 8.23 ± 0.75 meq. and 8.32 ± 1.09 meq.; chloride 4.24 ± 0.60 meq. and 4.04 ± 0.37 meq.; magnesium 0.459 ± 0.028 meq. and 0.445 ± 0.060 meq.; and phosphorus 3.69 ± 0.41 meq. and 3.88 ± 0.65 meq. per 100 Gm. of wet tissue. Myers[14] correlated myocardial potassium and phosphorus with cardiac weight. His data showed a fairly constant phosphorus content over a wide range of weights and no significant difference in the potassium content of normal and moderately hypertrophied hearts, weighing up to 530 grams, but a reduction in potassium in hearts exceeding 550 grams. Since his data gave no clear indication as to whether or not patients with congestive failure of milder grade or terminal development were excluded from the series, the lower potassium content of markedly hypertrophied hearts may have been related to the greater incidence of congestive failure in this group.

Group III. Congestive Failure With Underlying Left Ventricular Hypertrophy (Table III).—Cardiac muscle from patients dying in congestive failure showed a significant decrease in potassium content to an average of 6.78 ± 0.51 meq. per 100 Gm., as compared with values of 8.32 ± 1.09 meq. in controls and 8.23 ± 0.75 meq. in uncomplicated left ventricular hypertrophy. These findings confirmed reports of other workers.[5,7,8,14] On the other hand, our data revealed no significant change in content of water, sodium, chloride, magnesium, and phosphorus in the failing cardiac muscle as compared with the normal or the hypertrophied, but compensated, myocardium.

Since all our patients with congestive failure received a cardiac glycoside during hospitalization, it was necessary to consider digitalis action as a possible causative or contributory factor in the lowering of myocardial potassium. Studies in animals[15-18] have shown that toxic doses of digitalis reduce myocardial potassium, whereas therapeutic doses cause no significant change. Inasmuch as none of the patients was clinically overdigitalized at the time of death, it was concluded that the lowering of myocardial potassium was associated with congestive failure per se, rather than with the digitalis employed in treatment. This conclusion is supported by more recent studies in another local hospital,[19] demonstrating reduction in myocardial potassium in patients who died in congestive failure without having received any cardiac glycoside.

Skeletal muscle potassium was also lower in patients with congestive failure than in controls or in the group with uncomplicated left ventricular hypertrophy. The parallelism in the fall of potassium in cardiac and skeletal muscle with congestive failure suggests that the decrease is the result, rather than the cause, of failure.

The question arose as to whether the drop in potassium content without significant change in total water reflected a decrease in concentration of potassium

in the cells or merely an increase of extracellular fluid at the expense of intracellular fluid. Accordingly, estimates of water and electrolyte partition were made in the same manner as in the control group, similar values for the extracellular concentrations of each electrolyte being assumed.* The derived values per 100 Gm. of cardiac muscle were 33.4 Gm. of extracellular water and 45.5 Gm. of intracellular water; those for 100 Gm. of skeletal muscle from patients with congestive failure were 23.6 Gm. of extracellular water and 50.7 Gm. of intracellular water. The estimated partition of water in cardiac and skeletal muscle of patients with congestive failure did not differ significantly from that in the corresponding type of muscle from the control group.

The estimated concentrations of intracellular base in decompensated myocardium were as follows: sodium 12.1 meq., potassium 146 meq., and magnesium 6.85 meq. per liter of intracellular water. Comparison of these values with the corresponding figures in the control group showed a significantly lower intracellular potassium, a very slightly lower magnesium, and a higher sodium concentration in decompensated myocardium. The total intracellular base concentration was significantly reduced below the control value of 191 meq. per liter to 164 meq. per liter.

The intracellular concentrations in skeletal muscle from patients in congestive failure, estimated in the same manner, were as follows: sodium 3.9 meq., potassium 152 meq., and magnesium 6.2 meq. per liter of intracellular water. The values for sodium and potassium were lower and those for magnesium almost the same as the concentrations in skeletal muscle from the control group. The total intracellular base concentration in skeletal muscle from patients in congestive failure was significantly reduced below the control value of 193.9 meq. per liter to 162.1 meq. per liter. It was pointed out previously that the total intracellular base concentration of skeletal muscle from the control group (193.9 meq. per liter) was almost identical with that of myocardium from the same patients (191 meq. per liter). Attention is now drawn to the fact that the total intracellular base concentration of skeletal muscle from patients with congestive failure (162.1 meq. per liter) was almost identical with that of the decompensated myocardium (164 meq. per liter), indicating comparable losses of base from skeletal and cardiac muscle during congestive failure.

The parallelism in the reduction of the concentrations of total intracellular base and potassium in skeletal and cardiac muscle during failure suggests that serial biopsies of skeletal muscle during life might serve as a useful, though indirect, index of changes in the concentration of total base and potassium in the myocardium. Furthermore, the reduction in total base and potassium of myocardial and skeletal muscle found in congestive failure confirms that previously postulated[1] on the basis of demonstrated cellular uptake during recovery from cardiac decompensation.

The significantly higher concentration of total base in intracellular than in extracellular fluid of the control group indicates either that part of the intra-

*It is recognized that errors in calculations are more likely to occur in the group with congestive failure than in the controls because of the tendency for mercurial diuretics employed in the treatment of cardiac failure to depress plasma chlorides and sodium below the assumed concentrations.

cellular electrolyte is normally bound in an osmotically inactive complex or that nondiffusable tissue protein creates sufficient osmotic imbalance to produce Donnan's effect. In congestive failure, the normal difference in the concentrations of total base in intracellular and extracellular fluid is reduced or obliterated. The reduction in concentration of total cellular base associated with congestive failure may result from either (1) activation of inert base, necessitating release of electrolyte from the cell to preserve osmotic equilibrium with the interstitial fluid, or (2) breakdown of tissue protein with consequent reduction in the Donnan effect.

Group IV. Recent Myocardial Infarction (Table IV).—Comparison of the average values obtained from the analysis of grossly infarcted myocardial blocks from Group IV with the normal averages from Group I showed a reduction in potassium and magnesium content to approximately one-half of the control values, a decrease in phosphorus to approximately 60 per cent of the control, and a marked elevation in sodium and chloride. The increase in the latter elements, expressed in milliequivalents per 100 Gm. of wet tissue, over the average control value was 3.15 for sodium and 3.29 for chloride, suggesting that these elements entered infarcted myocardium as neutral sodium chloride. Since there was no significant change in total water, the marked elevation in chloride and sodium reflected, in part, an increase in extracellular fluid at the expense of intracellular fluid and perhaps, in part, a diffusion into dead or dying cells. The postulated increase in extracellular fluid can be confirmed by histologic evidence of severe interstitial edema. Calculation of extracellular and intracellular distribution of water and electrolyte was not made because of the lack of data on the amount of chloride entering dead and dying cells.

The losses of potassium and magnesium amounted to an average of 4.36 meq. per 100 Gm. of wet tissue and therefore preponderated over the gain in sodium, with consequent decrease in total base. The reduction in potassium and magnesium could not have been explained solely by the interstitial edema, even if all chloride remained extracellular. The depletion of potassium, magnesium, and phosphorus was undoubtedly caused primarily by myocardial injury, and the content, expressed in terms of 100 Gm. of wet tissue, was further reduced by the complicating interstitial edema.

It is noteworthy that blocks taken from distant areas that did not show evidence of infarction on gross examination gave results for all five electrolytes that were intermediate between the values obtained from infarcted segments and those from normal controls. These changes were not attributable to congestive failure, since both the reduction in potassium, magnesium, and phosphorus and the elevation in sodium and chloride were greater than the deviations found in congestive failure without infarction. It is probable that acute ischemia without necrosis was responsible for the chemical changes in areas beyond the gross infarct.

The composition of skeletal muscle from patients dying of myocardial infarction corresponded more closely with that of control subjects dying of noncardiac causes than with that of patients dying of congestive failure.

TABLE IV. GROUP IV—ACUTE MYOCARDIAL INFARCTION

CASE	HEART MUSCLE (PER 100 GM. OF WET TISSUE)						SKELETAL MUSCLE (PER 100 GM. OF WET TISSUE)					
	H_2O (GM.)	Na (MEQ.)	K (MEQ.)	cl (MEQ.)	Mg (MEQ.)	P (MEQ.)	H_2O (GM.)	Na (MEQ.)	K (MEQ.)	cl (MEQ.)	Mg (MEQ.)	P (MEQ.)
A. Non-infarcted segments												
44	78.9	7.20	5.95	5.97	0.340	2.96	67.6	4.40	9.57	3.40	0.390	3.30
45	78.8	7.20	4.60	5.80	0.331	3.35	75.5	3.61	11.00	2.40	0.452	2.53
46	78.6	6.52	5.90	5.75	0.230	3.37	76.6	2.91	10.30	2.03	0.500	4.48
47	76.7	7.69	5.30									
48	78.4	4.82	6.88				73.2	2.26	8.16			
49	76.4	8.70	6.00									
50	76.5	5.69	6.77									
Mean	77.8	6.68	5.91	5.84	0.300	3.23	73.2	3.29	9.76	2.61	0.447	3.47
Standard deviation	±1.1	±1.20	±0.74	±0.10	±0.050	±0.17	±3.5	±0.79	±1.05	±0.57	±0.045	±0.80
B. Infarcted segments												
44	77.9	7.51	5.37	7.50	0.198	2.15						
45	79.6	7.95	3.93	7.10	0.294	2.65						
46	78.3	8.40	2.44	7.40	0.154	2.15						
47	76.6	9.00	3.20									
48	77.4	7.70	2.75									
49	70.6	11.59	4.85									
50	77.3	4.60	6.80									
Mean	76.7	8.11	4.19	7.33	0.215	2.32						
Standard deviation	±2.7	±1.96	±1.48	±0.16	±0.058	±0.24						

TABLE V. SUMMARY

GROUP	H_2O	Na	K	Cl	Mg	P
			Heart Muscle			
I	78.8 ± 1.2	4.96 ± 0.60	8.32 ± 1.09	4.04 ± 0.37	0.445±0.060	3.88 ± 0.65
II	77.6 ± 1.3	4.93 ± 0.67	8.23 ± 0.75	4.24 ± 0.60	0.459±0.028	3.69 ± 0.41
III	78.9 ± 2.3	5.32 ± 0.86	6.78 ± 0.51	4.12 ± 0.56	0.414±0.063	3.80 ± 0.16
IV, A	77.8 ± 1.1	6.68 ± 1.20	5.91 ± 0.74	5.84 ± 0.10	0.300±0.050	3.23 ± 0.17
IV, B	76.7 ± 2.7	8.11 ± 1.96	4.19 ± 1.48	7.33 ± 0.16	0.215±0.058	2.32 ± 0.24
			Skeletal Muscle			
I	77.2 ± 1.9	4.06 ± 1.26	9.42 ± 2.65	2.97 ± 1.00	0.425±0.063	3.72 ± 0.86
II	75.6 ± 1.9	3.89 ± 0.39	11.46 ± 1.44	2.88 ± 0.67	0.526±0.084	4.20 ± 0.77
III	74.8 ± 3.4	3.56 ± 0.52	7.82 ± 1.06	3.00 ± 0.90	0.380±0.030	3.53 ± 0.45
IV	73.2 ± 3.5	3.29 ± 0.79	9.76 ± 1.05	2.61 ± 0.57	0.447±0.045	3.47 ± 0.80

SUMMARY

The water and electrolyte content of cardiac and skeletal muscle from patients dying of congestive failure or recent myocardial infarction was compared with the composition of specimens from patients dying of noncardiac causes. Myocardial blocks from four standard sites in the left ventricle were analyzed for water, sodium, potassium, chloride, magnesium, and phosphorus in twenty-nine cases and for water, sodium, and potassium in eighteen additional cases; blocks from the pectoralis major were analyzed for all six substances in twenty-five cases and for water, sodium, and potassium in four additional cases.

The control group comprised sixteen patients with normal hearts at autopsy and no evidence of a metabolic disorder. Myocardial analyses yielded averages of 78.8 Gm. of water, 4.96 meq. of sodium, 8.32 meq. of potassium, 4.04 meq. of chloride, 0.445 meq. of magnesium, and 3.88 meq. of phosphorus per 100 Gm. of wet tissue. Estimates of the partition of water between the extracellular and intracellular compartments and of the concentrations of base within the cells were made according to the method of Newburgh. Skeletal muscle from the same patients was comparable to myocardium in total water content, but showed a relatively smaller extracellular and larger intracellular volume. The concentration of total base in the cells was essentially the same as that in myocardium, but sodium level was slightly higher and potassium and magnesium levels were slightly lower.

Uncomplicated left ventricular hypertrophy was found in fourteen patients who showed no evidence of congestive failure during hospitalization and died of

noncardiac causes. The average content of water and each electrolyte in the myocardium was almost identical with that in the normal control group.

Congestive failure, complicating left ventricular hypertrophy, was present during hospitalization and at autopsy in eight patients. Analyses of cardiac and skeletal muscle showed significant reduction in potassium below the control values, but no significant change in content of water, sodium, chloride, magnesium, or phosphorus. The estimated partition of water in both cardiac and skeletal muscle was similar to that in the controls. The estimated intracellular concentration of total base was reduced to a similar level in cardiac and skeletal muscle, largely from potassium loss. The significance of these observations is discussed.

Recent myocardial infarction was responsible for the death of seven patients. Separate analyses were made of blocks from infarcted areas and of blocks from distant areas that appeared uninfarcted to gross examination. Infarcted myocardium showed a marked but proportionate increase in sodium and chloride, reflecting a severe interstitial edema, and an even more marked reduction in potassium, magnesium, and phosphorus, reflecting primarily losses from dead and dying cells. The analyses of distant blocks that were not grossly infarcted gave results for all five electrolytes that were intermediate between the values obtained from infarcted segments and those from normal controls. The abnormalities in chemical composition of myocardium well beyond the boundaries of a recent infarct were attributed to ischemia. Analysis of skeletal muscle from these patients did not deviate significantly from the normal.

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