base concentrations. The total determined acid decreased more than the total base so that there was an increase in the undetermined acid. These same changes were observed whether the hyperthermia was carried out for 2 or 4 hours. A recovery specimen taken 2 hours after the short term experiment showed a fall in pH below the control level, a further fall in the chloride and total base concentrations, a partial recovery of the CO_2 tension and bicarbonate concentration, and a complete return of the undetermined acid to its control level.

Similar changes were observed in the pH, CO_2 tension, and bicarbonate concentration when a 0.6% salt solution was given. However, the chloride and total base concentrations increased slightly. Further, the decrease in the total measured acid was not as great as in the experiments where water was drunk, but since the total base did not decrease, the undetermined acid concentration was equally as great. A control specimen taken 2 hours after the short term experiment showed the same tendency as when water was drunk with the exception that the total base and chloride levels tended to return toward the control level.

On the whole the changes in the acid-base balance observed are those which one might expect to occur as a result of a rapid hyperventilation of short duration. The drinking of salt solution seems to prevent a loss of base and chloride from the serum and to better enable individuals to undergo this form of fever therapy.

7944 P

Further Studies on the Creatine Content of Heart Muscle.*

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Creatine in the form of phosphocreatine is thought to play a significant part in muscular activity, and the functional capacity of muscle is apparently reflected by its creatine content. It is of interest to determine in what degree the retention of nitrogenous products influences the level of creatine in heart muscle and how the creatine content is also influenced by clinical and morphological

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manifestations of cardiac incompetency. A study of the creatine content of heart muscle should therefore offer many interesting possibilities from the standpoint of both creatine metabolism and heart disease which are complementary to each other.

A preliminary report of our work dealing with this subject was presented before this Society in January, 1932, the first publication appearing in April, 1934.[1] The concentration of creatine in the left ventricular muscle of the human was found to exceed that of the right ventricle by about 30%. Attention was also called to certain correlations between creatine content, and age and heart weight.

This general study has been continued, special attention being given to the inclusion of cases with marked nitrogen retention and heart failure. It has been observed that in cases of marked creatinine retention without heart failure there is considerable increase not only in the creatine content of voluntary muscle but also in that of the left and right ventricle. In lobar pneumonia the creatine content of both the voluntary and left ventricular muscle was appreciably increased, due possibly to retention. Low figures for the right ventricle in several cases were suggestive of right heart failure. It was a singular fact that in cases of uremia with heart failure, the creatine content of the voluntary muscle was essentially normal, despite high figures for the blood creatinine, while the creatine concentration of the left and right ventricles was reduced, the reduction being greatest generally in the left ventricle. Somewhat similar observations were made in cases with cardiac decompensation with heart failure, although in some of these cases the percentage reduction was greatest in the right ventricle. That this is due to a greater degree of incompetence of the right than the left ventricle is suggested but not proven.

At the time we began our original study, Calhoun, Cullen, Clarke and Harrison[2] presented a very interesting paper in which they pointed out that a diminished potassium content of heart muscle was invariably present in failing ventricles and suggested that this change may have been a contributing cause of such failure. The observations we have made regarding creatine in heart failure would appear to parallel very closely those of the Vanderbilt investigators on potassium, and it may well be that they represent 2 phases of the same process. At the present time we are engaged in a further study

[1] Seecof, D. P., Linegar, C. R., and Myers, V. C., *Arch. Int. Med.*, 1934, **53**, 574.

[2] Calhoun, J. A., Cullen, G. E., Clarke, G., and Harrison, T. R., *J. Clin. Invest.*, 1930, **9**, 393.

with the collaboration of Mangun and Reichle[3] involving the simultaneous determination of creatine, phosphorus, and potassium, but the observations are as yet too few to warrant additional conclusions.

7945 C

Experiments on Ligation of Renal Vein.

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The effect of partial or complete obstruction of the renal vein on the secretion of urine and the histology of the kidney has been investigated repeatedly. Conflicting results reported in the literature have been reviewed by Rowntree, Fitz and Geraghty,[1] Orofino,[2] and Nicastro.[3] Little has been added to the histological description of such kidneys since Buchwald and Litten's[4] report. The outstanding feature is degeneration and atrophy of the tubules with apparently relatively normal glomeruli. Other studies have been concerned with the development of an adequate collateral circulation to maintain the functional capacity of the kidney. This is apparently much better in dogs than in either cats or rabbits, and Alesandri,[5] Rowntree, Fitz and Geraghty were able to maintain dogs in excellent condition for a time even when one kidney had been removed and the other renal vein ligated. Usually, however, even in the dog when the renal vein has been ligated a progressive atrophy of the kidney follows with decrease and finally cessation of all urinary secretion. In cats and rabbits this is the constant finding.

Orofino found that from 5 to 20 days after ligation of one renal vein in dogs, the urine from that kidney was decreased in amount, and contained lower concentrations of urea and chloride but more albumin than the urine from the normal kidney. Dicker and Demoor[6] found the volume of urine from the ligated kidney greater

[3] Myers, V. C., Mangun, G., and Reichle, H. S., unpublished observations.

[1] Rowntree, L. G., Fitz, R., and Geraghty, J. T., *Arch. Int. Med.*, 1913, **11**, 121.

[2] Orofino, A., *Ann. Ital. di Chirur.*, 1932, **11**, 924.

[3] Nicastro, G., *Il Morgagni*, 1927, **69**, 2001.

[4] Buchwald, A., and Litten, M., *Virchow's Arch.*, 1876, **66**, 145.

[5] Alesandri, quoted by Orofino.

[6] Dicker, E., and Demoor, J., *Compt. Rend. Hebdomadaires Soc. de Biol.*, 1930, **103**, 503.