Electrolyte Metabolism in Congestive Heart Failure

Yoshitsugu NOHARA and Hiroshi SAIMYOJI

The Third Medical Clinic (Director: Prof. M. MAEKAWA), Kyoto University Hospital, Sakyo-ku, Kyoto

Disturbance of water and electrolyte metabolism in congestive heart failure is a secondary phenomenon resulting from contraction insufficiency of the myocardium. The purpose of this paper is to study clinically the mechanism of this phenomenon in the cases precisely investigated at Maekawa Clinic.

I. Renal clearance in congestive heart failure

Our statistical observation in congestive heart failure shows that there is a decrease in effective renal blood flow (RBF), an effective renal plasma flow (RPF) and a glomerular filtration rate (GFR) and an increase in filtration fraction (FF), as previously reported. The more severe the grade is, according to the classification of New York Heart Association, the larger the deviation is found in each value of renal clearance. The correlation between RPF and various data of the cardiac catheterization performed within one week after renal clearance examination was studied in 43 cases. RPF has no correlation to cardiac index, but positive to arterial oxygen content and rather negative tendency to arteriovenous oxygen difference.

The above data are considered to indicate that the volume of the residual blood in lung capillary vascular bed is increased, being interpreted as a backward failure due to left ventricular or left auricular failure.

II. Participation of catecholamine in congestive heart failure

Before the treatment, the more severe congestive heart failure was, the more norepinephrine in urine was found statistically. The volume of catecholamine in urine was in positive correlation with the magnitude of venous pressure, but in reverse correlation with the magnitude of the vital capacity. Even in left ventricular failure norepinephrine excretion was fairly increased. Norepinephrine excretion was in good correlation with RPF in statistical significance, but was not so good with GFR. The data are considered to suggest that postglomerular resistance of the kidney and the increased volume of norepinephrine are closely correlated.

The extent of catecholamine participation in congestive heart failure is shown in the following cases.

Case 1: A 60-year-old man, hypertensive heart failure, grade III. Blood pressure
160/106 mm. Hg. Rest and salt restriction were not effective. Guanethidine was administered. Venous pressure, aldosterone and norepinephrine were decreased. So, T. P. P. (2, 4, 7-triamino-6-phenyl-pteridine) was added, but the effects were not satisfactory. A lanatoside C injection provided a compensation in heart failure for the first time. This experience teaches us that a postsynaptic blocking agent gives rather bad effect on congestive heart failure by releasing the catecholamine, and that, whatever the cause may be, congestive heart failure must be treated firstly by correcting contraction insufficiency of the myocardium.

Case 2: A 20-year-old female, mitral stenosis, grade II. After rest and diet treatment, lanatoside C was injected, and then trichlomethiazide was added intermittently. After that norepinephrine in urine was gradually decreased with fluctuation, and stabilized to normal value.

On the basis of the above cases, the increased catecholamine excretion in congestive heart failure seemingly represents an adaptation reaction.

III. Regulation mechanism of electrolyte in congestive heart failure

Firstly, typical cases are presented.

Case 3: A 60-year-old man, coronary heart disease, grade IV. This is a typical case compensated remarkably by digitalis under oxygen tent and salt restriction with constant K (60 mEq./day).

Case 4: A 19-year-old man, idiopathic myocardose, grade IV. Lanatoside C was not effective under salt restriction (3 g./day) and constant K. Then T. P. P. was added, resulting in remarkable naturesis and increased urine volume from the first day. T. P. P. administration was stopped after 8 days because of dehydration, and the remarkably decreased natrium excretion and increased potassium excretion were found with increased aldosterone excretion in urine. This phenomenon is considered as a "rebound phenomenon," which was also observed in 2 other cases.

Case 5: A 42-year-old man, mitral valvular disease, secondary pulmonary hypertension, grade IV-III. High aldosteronuria (30.0 µg./day) and very low value of 17-OHCS in urine were recognized (0.75 µg./day). Under rest and salt restriction (5 g./day) with constant K, heart failure was fairly improved. By lanatoside C injection, aldosterone excretion became slightly decreased (11 µg./day). Under these conditions, excessive water (about 500 ml./day) was added for 4 days, resulting in an increase of urine volume, C_osm and natrium excretion, osmotic pressure substances in urine and a little C_H2O. Namely, mainly naturesis and slight water diuresis were observed. And trichlomethiazide was added for 7 days, resulting in an increase in urine volume, C_osm, C_H2O, and natrium excretion. Namely, this is naturesis. The aldosterone excretion was low during the drug administration (3.6 µg./day), and it was increased on the 3rd day after discontinuing the drug (17~6 µg./day) with decreased Na/K ratio in urine, and on the 7th day was again normalized (6.2 µg/day).

Case 6: A 28-year-old man. Marfan's syndrome. Bacterial endocarditis, aorto-
left ventricle to right ventricle shunting syndrome, grade IV. This patient was admitted 3 times during a period of about 2 years. As shown in Fig. 1, on the 3rd admission, strong and long-term diuretic treatment was continued by spironolactone, T.P.P. and trichlormethiazide under salt restriction (1 g./day), constant K, and cardiac glycoside, resulting in artificial secondary aldosteronism for about 8 months. Lastly, aldosterone excretion fell abruptly to 5.8 µg./day and 17-OHCS excretion also was decreased to 0.30 µg./day. Then the patient lost the capacity of adaptation resulting in sudden death under hyponatremia. This case is one of refractory heart failure.

Next, the authors will discuss the summarized data. The clear correlation between the volume of aldosterone in urine and the grade of decompensation was not recognized, and the volume of aldosterone was found correlated neither to the magnitude of venous pressure nor to serum potassium and natrium level. In the refractory phase aldosterone excretion was not increased in spite of hyponatremia. This is considered to indicate that hyponatremia is normally corrected by increased aldosterone. In some cases 17-OHCS excretion was increased from low value simultaneously when the volume of aldosterone was corrected.
Fig. 2. Change in urinary aldosterone and 17-OHCS during the treatment of congestive heart failure by digitalis or thiazides administration.

Fig. 3. Change in urinary aldosterone and 17-OHCS during the treatment of congestive heart failure by spironolactone or T.P.P. (2, 4, 7-triamino-6-phenylpteridine) administration.
The excretion of aldosterone under various kinds of diuresis was as follows: Digitalis diuresis is considered as physiological as diuresis at rest. Aldosterone was decreased in all 4 cases of rest diuresis and in the majority of cases at digitalis diuresis. With thiazide administration, aldosterone was decreased, but increased after ceasing administration, showing a "rebound phenomenon" in some cases (Fig. 2). With T.P.P. administration, aldosterone was increased in cases with low aldosterone value, and decreased in cases with high aldosterone value. With spironolactone administration, aldosterone was increased in almost all cases (Fig. 3).

IV. Conclusion

Electrolyte metabolism in congestive heart failure was studied in the cases admitted to Maekawa Clinic of Kyoto University Hospital. Renal clearance, catecholamine in urine, electrolyte in serum and urine, aldosterone in urine and, in some cases, osmotic pressure and water clearance were investigated and discussed. Both diuresis at rest and digitalis administration are considered as being physiological, and that diuresis induced by various diuretics are altogether naturesis accompanied by increased urine volume. When diuretic action by drug is proved as proper, the individual is considered to be able to adapt himself to the effect, even with rebound phenomenon. But when too strong and too long-term naturesis is enforced by the drug, without considering right treatment for the etiological, structural and functional factors, the reserve of natrium necessary for the individual's life will be out of proportion to that which mineral corticosteroids can regulate. This stadium is considered to be the refractory phase of congestive heart failure.

Acknowledgment

The authors wish to thank Prof. Oshima, the chairman of this symposium, and Prof. Maekawa, the president of this meeting, for giving us the opportunity to speak at this meeting, and Prof. Maekawa for his kind guidance while carrying out this study. Acknowledgment is also made to colleagues in Maekawa Clinic of Kyoto University Hospital for their cooperation and help.

References