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Effects of hemodialysis on left ventricular performance: A Doppler echocardiographic study

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Summary

Left ventricular systolic and diastolic performance was examined using Doppler and M-mode echocardiography in 42 patients with chronic renal failure before and after hemodialysis. Twenty patients with left ventricular hypertrophy, 22 without hypertrophy and 30 normal subjects were studied. Chronic renal failure patients showed significantly larger chamber diameters of the left ventricle, left atrium and right ventricle than did normal subjects. This group also exhibited greater fractional shortening, stroke volume and cardiac output. Before hemodialysis, patients with left ventricular hypertrophy had a significantly higher cardiac output and the greater ratio of late to early diastolic peak flow velocities (A/R) than did patients without hypertrophy. After hemodialysis, there were significant reductions in blood pressure, ventricular and atrial dimensions, stroke volume and cardiac output. The velocities of early and late diastolic left ventricular filling and the deceleration rate were also significantly reduced. The heart rate, A/R, deceleration half time, and the ratio of deceleration half time to acceleration half time (DHT/AHT) were significantly increased. The greater the amount of fluid removed, the greater the changes in the above values. Patients with left ventricular hypertrophy exhibited significant reductions in fractional shortening, ejection fraction, stroke volume and cardiac output, compared to those without hypertrophy. However, patients without hypertrophy showed more significant decrease in the acceleration half time and increase in DHT/AHT than did patients with hypertrophy. These findings demonstrated normal systolic function and impaired diastolic properties in patients with chronic renal failure, who had left ventricular hypertrophy unaccompanied by dilatation.

Key words

Doppler echocardiography　Cardiac function　Left ventricular hypertrophy　Hemodialysis

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Introduction

Recent advances in pulsed Doppler technique have facilitated noninvasive measurements of intracardiac blood flow velocity to provide important information about cardiac function[1~11]. Pulsed Doppler echocardiography is clinically useful for detecting abnormalities of diastolic filling, which may be one of the earliest signs of abnormal left ventricular function. Some studies[11,12] have suggested that diastolic properties of the left ventricle are impaired when hypertrophy is present. In the present study, we measured left ventricular diameter and blood flow at the level of the mitral valve annulus, and the response to hemodialysis in patients with chronic renal failure was observed using a pulsed Doppler flowmeter in combination with two-dimensional echocardiography. The goal was to evaluate the influence of volume removal using hemodialysis on chronic renal failure patients with and without hypertrophy of the left ventricle.

Material and methods

Subjects

Forty-two patients with chronic renal failure who receiving hemodialysis for two to 15 years (mean, eight years), and 30 normal persons were subjects of the present study. There were 20 men and 22 women, 36 to 65 years of age (mean, 47 years). All subjects were in sinus rhythm with heart rates of 60 to 80 per min. The cardiothoratic ratio by chest radiography ranged from 48 to 55%. Patients were classified in two groups based on left ventricular wall thickness. The hypertrophy group consisted of 20 patients whose left ventricular wall thickness was $(LVPWT + IVST)/2 \geq 1.2$ cm. The non-hypertrophy group of 22 patients had normal left ventricular wall thickness: $(LVPWT + IVST)/2 < 1.2$ cm). The hypertrophy group consisted of 11 males and nine females, 36 to 65 years of age (mean, 48 years); the non-hypertrophy group consisted of nine males and 13 females, 36 to 62 years of age (mean, 47 years). None of the patients had pericardial disease or valvular lesions according to Doppler echocardiography. Patients with diabetes mellitus were excluded from study. The controls consisted of 30 subjects (15 males and 15 females) similar in age range with no history of hypertension and no evidence of cardiovascular disease by echocardiography.

Doppler echocardiography

Doppler echocardiograms were recorded one hour before and one hour after hemodialysis, with the subjects in the semi-left lateral position. M-mode, two-dimensional echocardiography, and pulsed Doppler studies were performed with a phased-array echo-Doppler system (Hewlett-Packard Series 77020A). Either a 2.5 or 3.5 MHz transducer was employed for the M-mode, two-dimensional echo and a pulsed Doppler system. In a pulsed mode, the sample volume length and width were 2~3 mm and it was located at depths varying from 0 to 15 cm. Transmitral flow was sampled by placing the transducer at the cardiac apex and aligning the Doppler cursor parallel to the flow by means of the four-chamber view of two-dimensional images (**Fig. 1**). Recordings were made in the pulsed mode with the sample volume positioning in the left ventricle at the level of the annulus of the mitral valve. Pulsed wave Doppler echocardiograms were recorded with the electrocardiogram on a strip-chart at a paper speed of 100 mm/s (Honeywell strip chart recorder). The flow velocity toward the transducer was displayed above the baseline of the Doppler echocardiogram as shown in **Fig. 1.** Calculations were made from the peak early diastolic velocity (R), peak velocity resulting from atrial contraction (A), acceleration half time (AHT) and deceleration half time (DHT) of early diastolic inflow, and the following ratios were calculated: A/R & DHT/AHT. The acceleration rate (ACR) and the deceleration rate (DCR) were also measured. All measurements were made based on an average of five consecutive beats.

M-mode echocardiography

M-mode echocardiography was performed using standard techniques. Measurements were

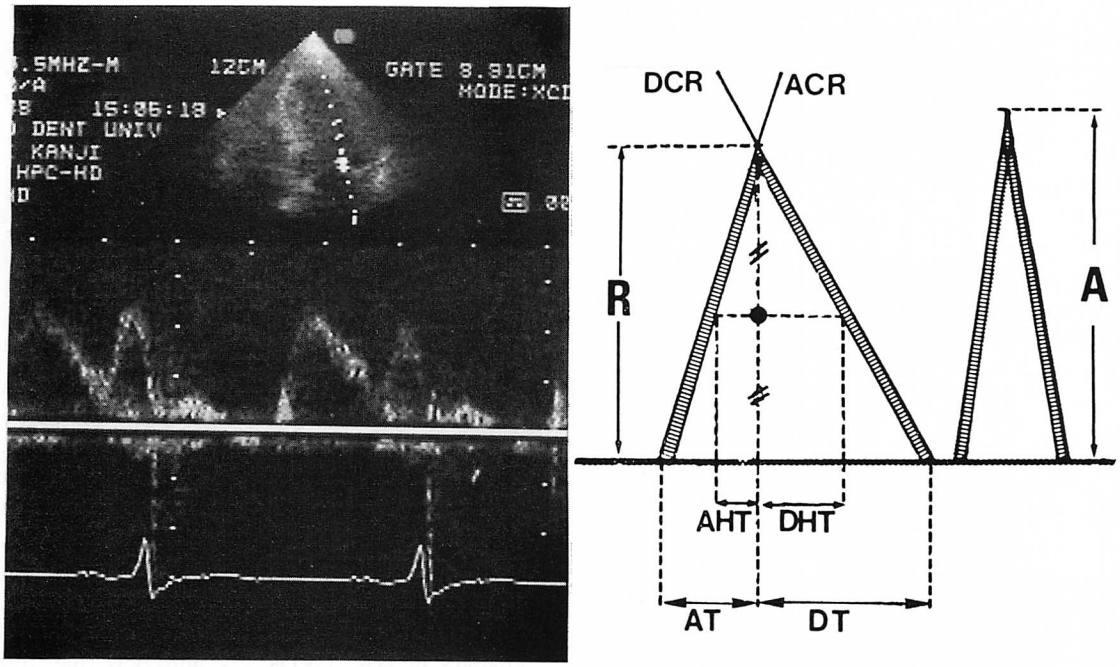

Fig. 1. Mitral inflow patterns recorded at the level of the mitral annulus using the apical four-chamber view.

R=rapid filling velocity; A=atrial contraction velocity; AHT=acceleration half time; DHT=deceleration half time; AT=acceleration time; DT=deceleration time; ACR=acceleration rate; DCR=deceleration rate.

made using leading-edge methods according to the standards suggested by the American Society of Echocardiography[13]. Left ventricular diastolic dimensions (LVDd), left ventricular systolic dimensions (LVDs), left atrial dimensions (LAD), right ventricular dimensions (RVD), fractional shortening (%FS), and left ventricular ejection fraction (EF) were determined from the M-mode echocardiogram. Stroke volume (SV) and cardiac output (CO) were calculated according to the method of Teichholz et al[14].

Statistics

All values were expressed as means±SD. Where appropriate, the Student's t test for paired or unpaired data was employed. A p value of <0.05 was considered a significant difference between the groups.

Results

Pre-hemodialysis

The decrease in body weight due to hemodialysis varied from 1.4 to 4.0 kg (mean±SD: 2.7±0.8 kg). Both the left ventricular hypertrophy and non-hypertrophy groups had significant increases in LVDd, LAD, RVD, %FS, SV, and CO, compared to the control group. Both groups had a more rapid peak velocity of atrial contraction (A) and shorter DHT, compared to the control group. Only the left ventricular hypertrophy group had a significantly larger A/R compared to the control group. In the two hemodialized groups, the hypertrophy group showed a significantly larger cardiac output, a thicker LV wall, and a higher A/R. **Table 1** summarizes the data of each group before hemodialysis.

Table 1. Hemodynamic parameters before hemodialysis

	Controls	Non-LVH group	LVH group
Age (yrs)	48±9	47±11	48±10
HR (b/min)	70±9	72±8	75±9
LVDd (cm)	4.7±0.3	5.0±0.4**	5.1±0.5**
LVDs (cm)	3.0±0.4	3.2±0.6	3.0±0.5
LAD (cm)	3.3±0.4	4.0±0.4*	3.9±0.5*
RVD (cm)	1.6±0.4	2.3±0.4***	2.1±0.4***
LVPWT (cm)	1.0±0.1	1.0±0.1	1.3±0.1***
IVST (cm)	1.0±0.1	1.0±0.1	1.3±0.1***
%FS (%)	39±6	41±5*	42±5*
EF (%)	68±6	69±8	71±8
SV (ml)	79±15	92±17*	93±22*
CO (l/min)	5.6±1.1	6.2±1.0*	7.4±1.8***
A (cm/sec)	52±15	61±11*	63±11***
R (cm/sec)	61±12	63±12	60±21
A/R	0.9±0.2	1.0±0.2	1.2±0.4**
AHT (msec)	51±9	46±6	46±13
DHT (msec)	98±16	87±15**	84±17**
DHT/AHT	1.9±0.3	1.9±0.4	1.9±0.4

mean±SD.

p values: * <0.025, ** <0.01, *** <0.005.

Abbreviations: HR=heart rate; LVDd and LVDs =left ventricular end-diastolic and end-systolic dimensions; LAD=left atrial dimension; RVD=right ventricular dimension; LVPWT & IVST=thicknesses of left ventricular posterior wall and interventricular septum; %FS=%left ventricular fractional shortening; CO=cardiac ontput. Other abbreviations: see Figs. 1 and 2.

Post-hemodialysis

Following hemodialysis, significant reductions of systemic blood pressure were observed as well as decreases in LVDd, LVDs, LAD, RVD, SV, CO, A wave, R wave and DCR. Heart rate increased. Increases in A/R, DHT, DHT/AHT were observed in both groups after hemodialysis. Comparing the values of each parameter before and after hemodialysis, heart rate increased more in the non-hypertrophy group than in the hypertrophy group. The hypertrophy group showed significantly greater reductions of %FS, EF, SV and CO compared to the non-hypertrophy group. Relative to the left ventricular inflow parameters, the non-hypertrophy group showed significantly greater changes in AHT, DHT/AHT and AT than did the hypertrophy group. Other parameters did not differ significantly between the groups. Many parameters of left ventricular inflow changed less in the left ventricular hypertrophy group than in the nonhypertrophy group. Results are summarized in **Table 2** and **Fig. 2.**

Effect of reduction in fluid volume

In determining the influence of reduction in fluid volume, each group was subdivided into two groups based on their fluid reduction. As shown in **Table 3,** in patients with left ventricular hypertrophy, greater reductions in volume resulted in greater changes in %FS, EF and DHT/AHT, than in patients with less volume removal. In the non-hypertrophy group, greater volume reduction resulted in greater changes in HR, LVDs, SV and A/R than did lesser volume reduction. Comparing the greater volume reduction groups with and without hypertrophy, %FS and EF changed more significantly in the hypertrophy group, and A/R more significantly changed in the non-hypertrophy group. In the lesser volume reduction groups with and without hypertrophy, the DHT/AHT was altered more significantly in the group without hypertrophy. These results indicate that reduction in fluid volume had a greater effect on systolic hemodynamics in the presence of left ventricular hypertrophy, compared to patients without such hypertrophy. Diastolic performance was affected to a lesser degree in patients with left ventricular hypertrophy than in those without hypertrophy.

Discussion

Effect of hemodialysis

Reduced cardiac performance has been recognized as a common clinical occurrence in patients with chronic renal failure and it has been believed to be multifactorial in etiology[15~19]. Cardiac dysfunction has been regarded as attributing to a direct effect of uremic toxins,

Table 2. Changes of parameters before and after hemodialysis

	Non-LVH group		LVH group	
	Before	After	Before	After
HR (b/min)	72±8	84±11***	75±9	83±11**
BPs (mmHg)	143±23	115±22***	156±31	134±32***
BPd (mmHg)	79±15	70±15***	82±14	75±16***
LVDd (cm)	5.0±0.4	4.5±0.5***	5.1±0.5	4.4±0.6***
LVDs (cm)	3.2±0.6	2.9±0.6***	3.0±0.5	2.7±0.6***
LAD (cm)	4.0±0.4	3.4±0.5***	3.9±0.5	3.3±0.5***
RVD (cm)	2.3±0.4	2.0±0.4***	2.1±0.4	1.8±0.3***
LVPWT (cm)	1.0±0.1	1.0±0.2	1.3±0.1	1.4±0.2*
IVST (cm)	1.0±0.1	1.0±0.2	1.3±0.1	1.4±0.2*
%FS (%)	41±5	37±8	42±5	36±8**
EF (%)	69±8	65±9	71±8	63±13***
SV (ml)	92±17	67±19***	93±22	62±21***
CO (l/min)	6.2±1.0	5.3±1.1***	7.4±1.8	5.2±1.6***
A (cm/sec)	61±11	57±11	63±11	58±11*
R (cm/sec)	63±12	48±13***	60±21	47±12***
A/R	1.0±0.2	1.3±0.4***	1.2±0.4	1.3±0.4*
AHT (msec)	46±6	42±9*	46±13	45±11
DHT (msec)	87±15	94±13*	84±17	92±22*
DHT/AHT	1.9±0.4	2.3±0.4***	1.9±0.4	2.1±0.3*
AT (msec)	80±12	65±12***	78±12	76±36
DT (msec)	158±24	162±34	145±46	165±49*
ACR (cm/sec^2)	800±232	745±181	760±200	673±158
DCR (cm/sec^2)	398±76	305±53***	421±130	287±65***

mean±SD.

p values: * <0.025, ** <0.01, *** <0.001.

Abbreviations: BPs and BPd=systolic and diastolic blood pressures. Other abbreviations: see Table 1 and Fig. 1.

hypertension, chronic volume overload, coronary artery disease, chronic acidemia, use of acetate buffer in the dialysate, hypovolemia, anemia, pericardial disease, hypoxemia, hyperkalemia, and hyperlipidemia. The major cause of death in chronic renal failure is cardiac[12,20]. The cardiac lesion consists of myocardial degeneration; i.e., hypertrophy and fibrosis. These abnormalities result in decreased cardiac function with a decrease in left ventricular compliance. Hemodialysis results in volume removal and removal of uremic solutes, either of which can affect myocardial performance. Most investigators have shown that hemodialysis improves cardiac function in patients with abnormal left ventricular function[21,22]. All the patients in the present study had normal systolic function with volume overload. Although these patients showed lower indices of systolic function after hemodialysis, the values were within normal limits. The left ventricular hypertrophy group showed greater changes in systolic hemodynamics than did the non-hypertrophy group, suggesting that the hypertrophic myocardium had an increase in inotropic function with volume removal.

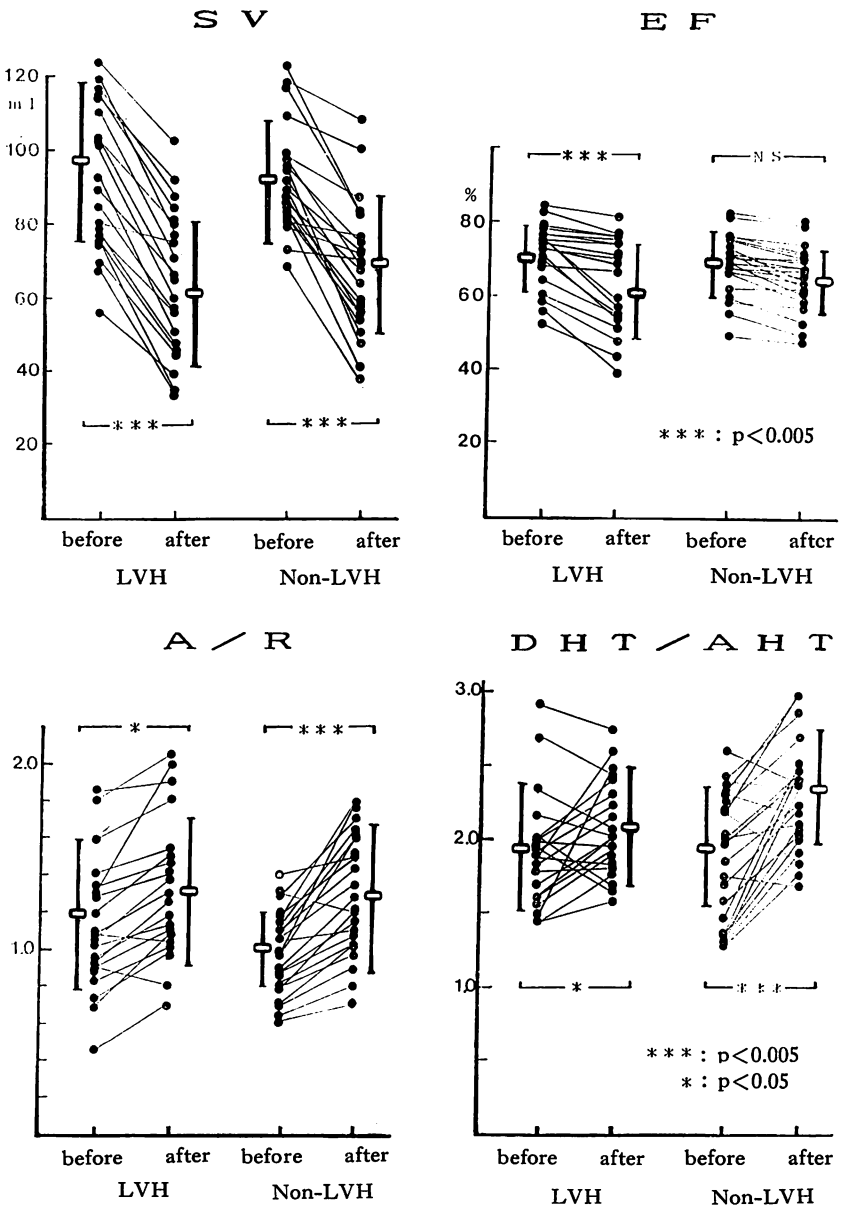

Fig. 2. The changes of SV, EF, A/R, and DHT/AHT of the left ventricular hypertrophy group and the non-hypertrophy group before and after hemodialysis.

SV=stroke volume; EF=ejection fraction; A=atrial contraction velocity; R=rapid filling velocity; AHT=acceleration half time; DHT=deceleration half time.

Table 3. Influence of volume reduction during hemodialysis

Body weight decrease (kg)	LVH group				Non-LVH group			
	3.8±0.4 (n=8)		2.1±0.4 (n=12)		3.2±0.6 (n=11)		2.0±0.4 (n=11)	
	Before	After	Before	After	Before	After	Before	After
LVDd (cm)	5.0±0.8	4.2±0.5***	5.0±0.3	4.3±0.8***	5.1±0.5	4.5±0.6***	4.9±0.3	4.4±0.4***
LVDs (cm)	2.9±0.4	2.6±0.4*	3.0±0.7	2.8±0.8*	3.3±0.7	2.9±0.7***	3.0±0.4	2.8±0.4***
%FS (%)	40±6	35±8*	39±8	37±7*	38±9	36±9*	39±5	37±6
EF (%)	70±6	63±10*	69±9	64±14*	68±10	65±10*	68±5	64±6*
SV (ml)	94±23	62±28**	93±11	62±16**	95±15	63±19**	90±8	61±11**
A (cm/sec)	65±8	63±10	63±14	58±13	60±11	56±12	62±12	58±10
R (cm/sec)	58±23	45±9*	63±24	49±16**	66±13	49±16***	58±5	45±8***
A/R	1.3±0.5	1.5±0.4***	1.1±0.4	1.3±0.5*	1.0±0.2	1.4±0.3***	1.1±0.3	1.3±0.3*
DHT/AHT	1.7±0.2	2.2±0.4**	2.0±0.5	2.0±0.4	1.8±0.4	2.2±0.4***	1.9±0.5	2.4±0.4***

mean±SD.
p values: * <0.05, ** <0.01, *** <0.005.
Abbreviations: see Table 1.

Left ventricular diastolic filling

Doppler echo cardiography is a useful means of noninvasively measuring intracardiac blood flow patterns. Several researchers have observed alterations in left ventricular filling related to aging[1~3], cardiac hypertrophy[4~6,23,24], and coronary artery disease[4,7]. In the present study the contribution of atrial contraction to ventricular filling and A/R were shown to be greater in the presence of left ventricular hypertrophy. The increase in A/R was related to reduction of left ventricular diastolic relaxation. The decrease in velocity of rapid left ventricular blood inflow was related to decreased expansion of the left ventricular wall and an increase in compensatory atrial contraction[12,13]. A primary goal of dialysis is to remove volume in a short period of time. When volume overload is decreased, the changes in diastolic parameters in patients with left ventricular hypertrophy were less than those in patients without hypertrophy. These data demonstrate that, although the hemodialized patients had normal systolic function, abnormal diastolic function was present and was probably partly compensated by accelerated flow velocities during the slow diastolic filling phase. The non-hypertrophy patients showed nearly normal responses in diastolic properties; however, those with hypertrophy had impaired compensatory function. For this reason, there were fewer changes in diastolic function. The hypertrophy group had an increased resistance to left ventricular relaxation in early diastole. Decreased left ventricular compliance was suggested by a decrease in early diastolic acceleration time and a decrease in the rate of early diastolic flow velocity deceleration. Acceleration time indicates active relaxation; deceleration time indicates passive relaxation. These values are related to diastolic function during left ventricular rapid inflow. In cases of hypertrophic cardiomyopathy and hypertensive heart disease, AHT and DHT are significantly prolonged, compared with normals. However, chronic renal failure patients had shorter AHT and DHT than did normals. This could be due to volume overload. For this reason, in chronic renal failure patients, the indices of diastolic function differ from those of other patients with left ventricular hypertrophy.

Hemodialysis in patients with LV hypertrophy

Adverse symptoms and hypotension remain as common problems during hemodialysis

procedure. The integrity of the autonomic nervous system and myocardial performance play key roles in determining how individual patient responds to volume reduction. Due to the responses of patients with left ventricular hypertrophy to volume reduction, it may be advisable to increase the frequency of hemodialysis, removing lesser volumes of fluid each time.

要　約

左室収縮・拡張動態におよぼす血液透析の影響：ドップラー心エコー図法による検討

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慢性腎不全患者42例を対象とし，左室肥大心群・非肥大心群それぞれにおける血液透析の左室収縮および拡張動態におよぼす影響についてドップラー心エコー図法にて検討した．その結果は以下のごとく要約される．

1. 慢性腎不全患者は健常群に比し，心室・心房の内径が大であり，%FS，心拍出量が有意に大きく，また心房収縮による左室流入血流速度(A 波)が大であり，左室拡張早期流入血流速度(R 波)の減速半時間 (DHT) が短かった．

2. 左室肥大心群は非肥大心群に比し，心拍出量が有意に大で，健常者群に比して A/R が有意に大であった．

3. 血液透析後，両群ともに血圧，心室・心房径，心拍出量，A 波，R 波の有意な低下を認めた、または心拍数，A/R，DHT/AHT の有意な上昇を認めた．

4. EF, FS などの収縮指標に関しては，肥大心群は非肥大心群に比べて透析後有意の減少を示したが，A/R，DHT/AHT などの拡張指標に関しては僅かな低下を認めるのみであった．

以上の結果を総括すると，血液透析による除水に対して，肥大心群は非肥大心群に比べて心内内径と収縮能の著しい減少を示したが，拡張能の減少は軽微であった．この事実は，心肥大を有する慢性透析例では強い収縮能が温存されているにもかかわらず，拡張能はすでに障害され，そのため除水に対する応答が小さいことを示唆している．

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