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Improved left ventricular contractility with cool temperature hemodialysis

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Improved left ventricular contractility with cool temperature hemodialysis. Cool temperature dialysis (CTD) has been shown to sharply decrease the frequency of intradialytic hemodialysis hypotension, but the mechanism of this hemodynamic protection is unknown. Therefore, we performed two-dimensional echocardiographic studies of left ventricular contractility in six stable hemodialysis patients before and after hemodialysis at 37°C (RTD) and 35°C (CTD). Left ventricular function was assessed by plotting the rate-corrected velocity of circumferential fiber shortening (Vcf_c) against end-systolic wall stress (σ_{es}) at four different levels of afterload. Linear regression was used to calculate Vcf_c at a common afterload of 50 g/cm^2. Changes in weight and dialysis parameters were similar following RTD and CTD. Mean arterial pressure and heart rate did not change significantly following RTD or CTD. The $Vcf_c - \sigma_{es}$ relation was shifted upward in each patient after CTD, indicating increased contractility as compared to RTD or pre-dialysis baseline. Pre-dialysis Vcf_c at an afterload of 50 g/cm^2 was similar during RTD and CTD (0.94 ± 0.24 circ/sec vs. 0.92 ± 0.22 circ/sec). Post-dialysis Vcf_c at an afterload of 50 g/cm^2 was significantly higher for CTD than for RTD (1.13 ± 0.29 circ/sec vs. 0.98 ± 0.30 circ/sec, $P = 0.0004$). Thus, cool temperature dialysis increases left ventricular contractility in hemodialysis patients, which may be a potential mechanism whereby hemodynamic tolerance to the dialysis procedure is improved.

Symptomatic hypotension remains a major problem in the management of hemodialysis patients [1, 2]. Multiple factors have been implicated including extracellular fluid volume contraction [3–6], autonomic dysfunction [7, 8], alterations in plasma osmolality [9], changes in ionized calcium [10, 11], anemia [12], membrane biocompatibility [13], cardiac dysfunction [14], and dialysate temperature [13, 15–26].

Several years ago, Maggiore et al suggested that the temperature of the blood reentering the patient affects vascular stability during hemodialysis and hemofiltration [15, 17, 18]. Sherman et al confirmed this observation by showing that episodes of symptomatic hypotension during hemodialysis occurred less frequently at lower dialysate temperatures [19, 20]. This effect was noted in stable patients as well as patients with frequent intradialytic hypotension. Lindholm et al noted that mean arterial pressure and heart rate remained more stable in patients with frequent intradialytic hypotension when cool temperature dialysate was employed [21]. In longer term studies, Marcen et al [22] and Orofino et al [23] reported that dialysis at low temperatures was associated with a decrease in symptomatic hypotension, greater weight loss during hemodialysis, and stabilization of pre-dialysis systolic blood pressure. The underlying mechanism responsible for the hemodynamic protective effect attributed to the use of cooler dialysate is unknown, although speculation that increased serum catecholamines and a subsequent increase in peripheral vascular resistance may play a role has been offered [16, 24].

The present study was undertaken to determine whether cool temperature dialysis improves left ventricular contractility independently of changes in heart rate and loading conditions. We undertook an examination of left ventricular contractility as a first step in studying the mechanism(s) responsible for hemodynamic protection with cool temperature dialysis. Accordingly, left ventricular contractility was assessed using the rate-corrected velocity of fiber shortening-end-systolic wall stress relation, which has been shown to be independent of heart rate and preload and incorporates afterload into its calculation [25, 26].

Methods

Patient population

The study included six men ranging in age from 37 to 66 (mean 55 ± 11 years). Time on hemodialysis ranged from 4 to 128 months (mean 71 ± 58). These patients were selected because they were clinically stable on chronic hemodialysis, had high quality echocardiograms, and were in sinus rhythm. The etiology of renal failure was hypertension in four subjects, glomerulonephritis in one, and diabetes mellitus in one. One patient had previous coronary artery bypass surgery and four were taking antihypertensive medications. Routine medications included phosphate binders, analgesics, antipruritics, and sodium bicarbonate. None was taking digoxin, inotropes, or beta-adrenergic blockers. The study protocol was approved by the Human Studies Subcommittee of the Dallas Veterans Affairs Medical Center, and informed consent was obtained from each subject.

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Experimental protocol

Each patient served as his own control and underwent hemodialysis on two separate occasions using different dialysate temperatures of 37°C and 35°C. The dialysis procedures were performed in random order, and both the patient and echocardiographer were unaware of which dialysis maneuver was occurring. Hemodialysis was performed mid-week or late-week. The time between maneuvers was one week in three patients, four weeks in one, five weeks in one, and 15 weeks in one. All patients remained clinically stable with no changes in medications or dry weights during the time interval between studies. During each maneuver, ultrafiltration rates were adjusted to achieve the patient's target weight.

Hemodialysis was performed using a Terumo TAF 15M first-use dialyzer (1.5 m^2) (Terumo, Somerset, New Jersey, USA) on either a Cobe Centry-3 dialysis machine (Cobe Laboratories, Lakewood, Colorado, USA) or a Drake Willock dialysis machine (Beckton, Dickinson and Co., Portland, Oregon, USA). Dialysate temperature was constantly monitored throughout the course of hemodialysis using an in-line thermometer. Dialysis flow rates were set at 500 ml/min. Blood flow rates were set at 400 ml/min and were confirmed by triplicate bubble transit times on a 50 cm racetrack. A constant ultrafiltration rate was maintained throughout the procedure. The dialysate contained 140 mmol/liter sodium, 2 or 3 mmol/liter potassium, 110 mmol/liter chloride, 30 mmol/liter bicarbonate, 11.1 mmol/liter glucose, and 1.75 mmol/liter calcium.

At the beginning of hemodialysis and after the final hemodynamic maneuver, weights were measured in triplicate using the same balance scale. Oral temperatures were recorded using a digital thermometer pre- and post-dialysis. Blood pressures during the procedure were recorded every 30 minutes using a sphygmomanometer. Blood samples were drawn prior to hemodialysis and after the post-dialysis manipulation of left ventricular afterload. Serum electrolytes, blood urea nitrogen, creatinine, and calcium were measured using a SMAC II Analyzer (Technicon Instruments Corporation, Tarrytown, New York, USA) in the hospital laboratory. Hematocrit was measured on a Coulter S + V (Coulter Electronics, Inc., Hialeah, Florida, USA). Blood ionized calcium concentration was measured using an ion-specific electrode NOVA 2 ionized calcium analyzer (NOVA Biomedial, Newton, Massachusetts, USA). Plasma norepinephrine values were measured using a radioenzymatic technique in a commercial laboratory (Smith Kline Beecham Clinical Laboratories, Dallas, Texas, USA). The normal supine norepinephrine values in this laboratory are 110 to 410 pg/ml. In three patients, plasma norepinephrine levels were measured on different dialysis days than the echocardiographic studies. Thus, no attempt was made to correlate plasma norepinephrine levels with observed changes in contractility.

Echocardiography

Echocardiographic studies were performed by the same experienced technician immediately before the initiation of hemodialysis and at the end of hemodialysis (but before the patient was disconnected from the machine). Standard two-dimensional echocardiographic views were obtained from the parasternal, apical, and subcostal windows using a Vingmed CFM 700 (Interspec, Conshohocken, Pennsylvania, USA) with a 3.0 MHz transducer. No patient had segmental wall motion abnormalities, structurally abnormal valves, or pericardial effusion. No patient had evidence of significant valvular regurgitation by Doppler color flow mapping.

Left ventricular contractility was assessed from the left ventricular stress-shortening relation, previously shown to be independent of loading conditions [25, 26]. An M-mode recording was obtained at the midventricular level (just off the tip of the anterior mitral leaflet) using two-dimensional guidance from the parasternal window. Simultaneous blood pressure was obtained using a sphygmomanometer. Immediately thereafter, a two-dimensionally directed M-mode recording was obtained at the level of the aortic leaflets. After baseline echocardiography, left ventricular afterload was altered by infusion of either sodium nitroprusside (N = 11 procedures) or phenylephrine (N 1 procedure). Nitroprusside was specifically used if the initial systolic blood pressure were greater than 125 mm Hg, whereas phenylephrine was chosen if the baseline systolic blood pressure were less than 125 mm Hg. The nitroprusside infusion was titrated to produce a stable plateau in systolic blood pressure at a value 8 to 12 mm Hg lower than the baseline reading. Repeat echocardiographic measurements were obtained at this level of blood pressure and the process was repeated until echocardiography was performed at four different levels of afterload. Phenylephrine was titrated to incrementally increase systolic blood pressure with echocardiography performed during stable plateau periods. Prior to phenylephrine infusion, atropine sulfate (0.01 mg/kg) was given to abolish reflex cardiac slowing. None of the patients experienced any adverse effects of these medications during the study.

Data analysis

M-mode recordings were analyzed in blinded fashion by an experienced echocardiographer. Measurements of left ventricular posterior wall thickness (h) and cavity dimension (D) were made at end-systole and end-diastole. Left ventricular ejection time (ET) was determined as the time from aortic valve opening to aortic valve closure and the RR interval was determined from the electrocardiogram. The rate-corrected mean velocity of circumferential fiber shortening (Vcf_c) was calculated by the following formula:

$$Vcf_c = \frac{(D_{ed} - D_{es}/D_{ed})}{ET/\sqrt{RR}} \text{ [11].}$$

Left ventricular end-systolic wall stress (σ_{es}) was calculated by the formula:

$$\sigma_{es} = (0.338)\frac{(P_{es})(D_{es})}{(h_{es})[1 + (h_{es}/D_{es})]}$$

where P_{es} is end-systolic pressure and 0.338 is a conversion factor [27]. For this experiment, peak systolic pressure was substituted for P_{es}. It has been previously shown that peak systolic pressure correlates closely with P_{es} and that the calculation of wall stress is not significantly influenced by use of peak systolic pressure [28].

Statistical analysis

Vcf_c was plotted against end-systolic stress by linear regression analysis in each patient before and after routine dialysis

Table 1. Biochemical parameters before and after hemodialysis

	Pre-dialysis		Post-dialysis	
	RTD	CTD	RTD	CTD
Sodium *mmol/liter*	141 ± 2	138 ± 3	142 ± 2	141 ± 1
Potassium *mmol/liter*	4.4 ± 0.9	4.5 ± 0.9	3.6 ± 0.6[a]	3.5 ± 0.5[a]
Chloride *mmol/liter*	104 ± 2	103 ± 1	102 ± 3	103 ± 3
Bicarbonate *mmol/liter*	22 ± 2	21 ± 3	27 ± 4[a]	26 ± 4[a]
BUN *mmol/liter*	14.6 ± 7.7	16.8 ± 10.7	7.5 ± 4.7[a]	8.7 ± 5.9[a]
Creatinine *mol/liter*	937 ± 195	919 ± 212	539 ± 65[a]	477 ± 97[a]
Calcium *mmol/liter*	2.3 ± 0.3	2.3 ± 0.3	2.5 ± 0.3	2.4 ± 0.2
Norepinephrine *pg/ml*	384 ± 171	322 ± 195	325 ± 111	542 ± 266
Ionized calcium *mmol/liter*	1.1 ± 0.1	1.1 ± 0.1	1.2 ± 0.1	1.1 ± 0.1
Hematocrit %	25 ± 2	25 ± 4	29 ± 6[a]	29 ± 5[a]

Data are presented as mean ± SD. Abbreviations are: RTD, routine dialysis (37°C); CTD, cool temperature dialysis (35°C).
[a] $P < 0.05$ compared to pre-dialysis

Table 2. Hemodynamic variables before and after hemodialysis

	Pre-dialysis		Post-dialysis	
	RTD	CTD	RTD	CTD
Weight *kg*	70.5 ± 17.8	71.2 ± 18.4	67.5 ± 17.5[a]	67.8 ± 18.0[a]
MAP *mm Hg*	109 ± 14	106 ± 12	109 ± 21	114 ± 18
Heart rate min^{-1}	85 ± 11	79 ± 13	89 ± 9	78 ± 18
Temperature °C	36.6 ± 0.3	36.1 ± 0.6	36.6 ± 0.4	35.6 ± 0.7
Vcf_c at 50 g/cm² *circ/sec*	0.94 ± 0.24	0.92 ± 0.22	0.98 ± 0.3	1.13 ± 0.29[b]

Data are presented as mean ± SD. Abbreviations are: RTD, routine dialysis (37°C); CTD, cool temperature dialysis (35°C); MAP, mean aterial pressure.
[a] $P < 0.05$ compared to pre-dialysis
[b] $P = 0.0004$ compared to all other values by ANOVA

and cool temperature dialysis. The regression equations obtained were used to determine Vcf_c at a common afterload (σ_{es} = 50 g/cm²) in order to eliminate afterload as a confounding variable in comparisons between patients. An F ratio was calculated by repeated measures analysis of variance to assess group differences in Vcf_c at 50 g/cm². A P value of <0.05 was considered statistically significant.

Comparisons of biochemical data, mean arterial pressure, and heart rate were also done by repeated measures analysis of variance with a P value <0.05 considered significant. Group data are expressed as mean values ± one standard deviation.

Results

Biochemical changes

Serum chemistries, blood ionized calcium, and hematocrit were comparable both before and after hemodialysis at 37°C and 35°C (Table 1). Both hemodialysis procedures significantly lowered serum potassium, blood urea nitrogen, and creatinine while raising bicarbonate and hematocrit. Pre-dialysis and post-dialysis weights were similar with routine dialysis and cool temperature dialysis, indicating that equivalent ultrafiltration was achieved with both procedures (Table 2). A slight decrease in oral temperature was present after cool temperature dialysis (36.1 ± 0.6°C vs. 35.6 ± 0.7°C), but this did not reach statistical significance.

Hemodynamics

Heart rate and mean arterial pressure were not significantly different pre- and post-dialysis with either dialysis maneuver (Table 2). Although cool temperature dialysis resulted in a slight increase in blood pressure over baseline (106 ± 12 mm Hg vs. 114 ± 18 mm Hg), this did not reach statistical significance. Blood pressures were stable during dialysis and no patient experienced a significant hypotensive episode (systolic blood pressure < 90 mm Hg).

Figure 1 shows rate-corrected Vcf plotted against end-systolic wall stress in a patient in this study. The relation is shifted upward after cool temperature dialysis, indicating increased left ventricular contractility. Each patient demonstrated an upward shift with cool temperature dialysis as compared to routine dialysis or pre-dialysis baseline. Figure 2 illustrates the increase in Vcf_c at an afterload of 50 g/cm² with cool temperature dialysis compared to pre-dialysis and routine dialysis. As shown in Table 2, pre-dialysis Vcf_c at 50 g/cm² was not significantly different prior to routine dialysis or cool temperature dialysis (0.94 ± 0.24 circ/sec vs. 0.92 ± 0.22 circ/sec). After routine dialysis, Vcf_c at 50 g/cm² was slightly but not significantly higher (0.98 ± 0.30 circ/sec) than the pre-dialysis value. However, cool temperature dialysis resulted in a significantly higher Vcf_c at 50 g/cm² (1.13 ± 0.29 circ/sec) when compared to pre-dialysis or routine dialysis ($F = 11.16$, $P = 0.0004$).

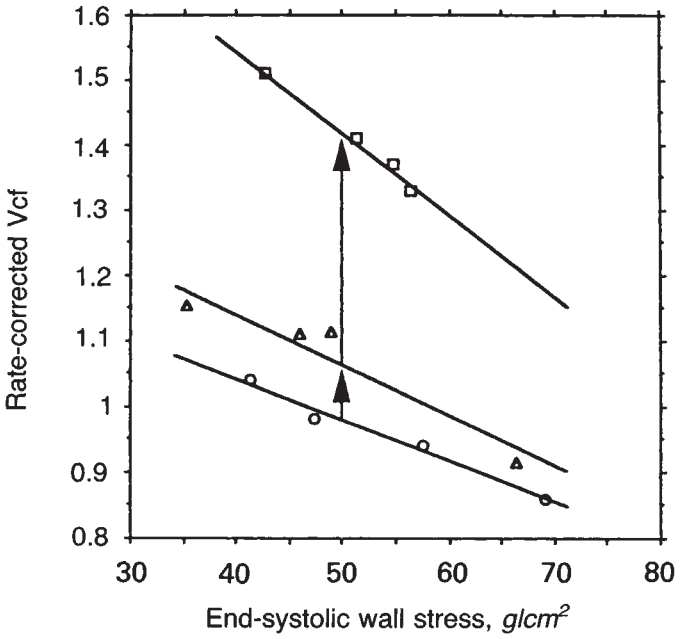

Fig. 1. *Rate-corrected Vcf plotted against end-systolic wall stress over four different levels of afterload in a patient in this study.* Circles represent measurements made at baseline (BSL), triangles represent routine dialysis at 37°C (RTD), and squares represent cool temperature dialysis at 35°C (CTD). A slight upward shift in the relation is observed from BSL to RTD. CTD results in a significant upward shift, reflecting increased left ventricular contractility.

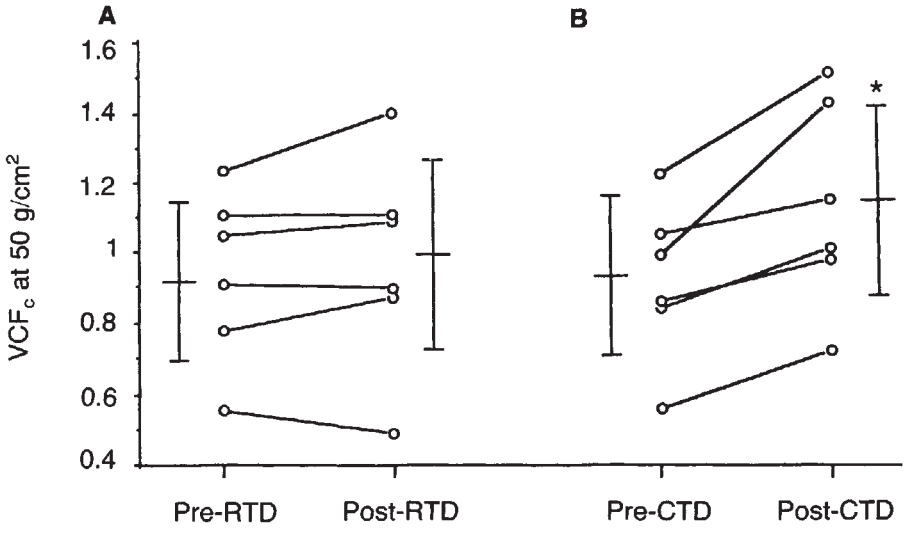

Fig. 2. *Plot showing the individual values for rate-corrected velocity of fiber shortening (Vcf_c) at a common afterload (σ_{es} = 50 g/cm^2).* Error bars depict mean ± one standard deviation. Abbreviations are: RTD, routine dialysis at 37°C; CTD, cool temperature dialysis at 35°C. P = 0.0004 vs. all others.

Discussion

Previous studies have shown that cool temperature dialysis decreases the frequency of intradialytic hypotension, improves tolerance to ultrafiltration, and improves the patient's sense of well-being [17–23, 29]. Although the advantages of cool temperature dialysis have been ascribed to increased peripheral vascular resistance [16] and catecholamine release [24], the hemodynamic effects of this maneuver have not been previously evaluated. Our data demonstrate that cool temperature dialysis improves left ventricular contractility as compared to the predialysis baseline and routine hemodialysis at 37°C.

It has been previously shown that ejection phase indices of left ventricular systolic performance are improved following hemodialysis [10, 30–32]. However, ejection phase indices are markedly dependent on heart rate and loading conditions [25, 26, 33, 34], factors which may be profoundly altered by hemodialysis. Thus, it is not clear from these earlier studies whether the observed improvement in left ventricular performance was due to increased contractility or hemodialysis-related changes in loading conditions. The rate-corrected Vcf-end-systolic wall stress relation employed in the present study is load-independent [25, 26], and has been previously used to evaluate the effects of dialysis on myocardial function [11, 35].

Although our data demonstrate improved contractility after cool temperature dialysis, the mechanism(s) by which this occurs remains uncertain. Immersion of the hand in ice water (cold pressor test) is known to cause reflex sympathetic stimulation [36]. Accordingly, it is conceivable that intravascular cooling may reflexly activate sympathetic outflow and increase myocardial contractility. Such a mechanism might also be expected to increase blood pressure and peripheral vascular resistance. There was a tendency for post-dialysis norepinephrine values to be higher after exposure to cool temperature dialysate (542 ± 266 vs. 325 ± 111 pg/ml), but this did not achieve statistical significance. It should also be noted that there was not a significant increase in heart rate during cool temperature dialysis. Previous studies using cool temperature dialysis over a longer term, have reported higher blood pressures and/or fewer episodes of hypotension [15–24]. In this acute study, mean arterial pressure also tended to be higher after cool temperature dialysis (114 ± 18 mm Hg vs. 109 ± 12 mm Hg), but this did not reach statistical significance, perhaps due to the small number of patients studied. Peripheral vascular resistance was not measured in this study.

Both routine hemodialysis and cool temperature dialysis resulted in similar changes in serum potassium, blood urea nitrogen, creatinine, bicarbonate, and hematocrit (Table 1). Blood ionized calcium, which has been shown to correlate directly with left ventricular function [10, 11], was similar post-dialysis with both routine dialysis and cool temperature dialysis. However, significant improvement in contractility was only present in the latter group. Thus, a change in blood ionized calcium was not a variable by which cool temperature dialysis increased left ventricular contractility in this study. Similarly, changes in potassium concentration were also equivalent during both procedures.

Limitations

The number of patients enrolled in the study was small. Thus, we were not able to determine whether the increases in contractility with cool temperature dialysis are quantitatively different in patients with normal as compared to depressed left ventricular function, nor could we determine whether the response to cool temperature dialysis is modified by left ventricular hypertrophy.

We used peak systolic pressure rather than end-systolic pressure to calculate end-systolic wall stress. End-systolic pressure can be calculated from a calibrated carotid pulse tracing [25, 26], and is theoretically more appropriate for determining end-systolic stress. However, peak systolic pressure correlates closely with end-systolic pressure, is easier to obtain, and results in accurate calculation of end-systolic wall stress [28]. Finally, calculation of circumferential wall stress from short-axis two-dimensional echocardiograms may be more accurate than meridional M-mode measurements of wall stress, particularly in patients with dilated cardiomyopathy or aortic stenosis [37]. However, all of the patients in this study had normal left ventricular chamber dimensions and none had aortic stenosis.

Conclusions

This study demonstrates that cool temperature dialysis is associated with increased left ventricular contractility as assessed by a load-independent method. Further investigation is necessary to determine the precise mechanism by which this occurs.

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