

LEFT VENTRICULAR FUNCTION DURING HAEMOFILTRATION AND HAEMODIALYSIS: A COMPARATIVE STUDY

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Summary

The relationship between systemic haemodynamics and left ventricular performance during HF and HD was evaluated by M-mode echocardiography. In patients with normal cardiac function and similar rates of fluid removal, special attention was paid to the effects of solute transport and buffer. Convective transport, independent of the replacement buffer, produces an increase in systemic vascular resistance with no changes in ventricular function. In contrast, a diffusive transport induces different effects depending on the dialysate buffer: acetate decreases vascular resistance and bicarbonate significantly improves myocardial contractility shifting the cardiac function curve to the left.

Introduction

Symptomatic hypotension occurs often during conventional haemodialysis (HD) limiting the extent of ultrafiltration [1]. Characteristically, haemofiltration (HF) and isolated ultrafiltration (UF) lead to improved tolerance with easier fluid removal [2,3]. This clinical difference may be related to effects on systemic haemodynamics, myocardial performance or both. Although peripheral haemodynamics during HF and HD have been widely investigated, comparative cardiac function studies are rare. HD and HF change electrolyte balance, acid-base status, plasma volume, uraemic toxin concentrations as well as systemic haemodynamics. all of which are factors capable of modifying cardiac performance. Nevertheless, their acute effects on ventricular contractility have not been systematically studied. The present work provides further information on the consequences of solute transport and the role of replacement buffer on vascular haemodynamics and left ventricular function non-invasively by M-mode echocardiography.

Material and methods

Five groups of 10 stable end-stage renal failure patients on regular HD for more than six months were studied. HF, HD and sequential UF diffusion (DF) and

buffers: Acetate (Ac), Lactate (La) and Bicarbonate (Bi) were used (Table I). Patients with fluid overload, clinical signs of heart failure, cardiac enlargement on X-ray, uncontrolled hypertension or taking digitalis preparations were excluded from the study. On the basis of a previous echocardiogram, the existence of ventricular contraction abnormalities, subclinical pericardial effusions or contractility parameters below normal were also criteria for exclusion.

TABLE I

Procedure	Buffer (mEq/L)	Mean weight loss (kg)	Membrane
HF-Ac	Acetate: (37)	1.89 ± 0.73	0.6m ²
HF-La	Lactate: (40)	1.79 ± 0.92	0.6m ²
HD-Ac	Acetate: (37)	1.80 ± 0.81	1.1m ²
HD-Bi	Bicarbonate: (37)	1.83 ± 0.69	1.1m ²
UF/DF	--/Acetate: (37)	1.76 ± 0.74/- -	1.1m ²

Immediately pre- and post-procedure, controlled M-mode echocardiograms were performed including a simultaneous carotid pulse tracing for calculation of systolic ejection time. Ventricular dimensions were obtained at the level of the chordae tendinae. End-diastolic diameter at the peak of the R wave of the ECG and end-systolic diameter at the point of maximal septal contraction were measured in five consecutive cycles and the mean was calculated. Mean arterial pressure (MAP) was obtained by adding one-third of the pulse pressure to two-thirds of the diastolic pressure. From the above data, ventricular volumes [4], stroke volume and cardiac index (CI) were derived. Whole ventricular function was estimated by the ejection fraction (EF) and myocardial contractility by the velocity of circumferential fibre shortening (VCF) [5] and the exponential rate of fibre shortening (ERFS) [6]. The systemic vascular resistance (SVR) was calculated by dividing the MAP by the CI and expressed as arbitrary units.

The reproducibility of the echocardiographic data was assessed in repeated calculations of the stroke volume in nine normal subjects, giving a mean coefficient of variation of 5.1 per cent. Linear regression analysis demonstrated a correlation coefficient of 0.92 for stroke volume measurements by two independent observers. Statistical significance was calculated by Student's t-test for paired data.

Results

Weight loss was comparable in the five groups of patients (Table I). With both forms of HF there were no changes in MAP (-3%, -4%), but CI fell (-16%, -18%; $p < 0.02$). A significant rise in SVR (+24%, +21%; $p < 0.005$) was seen, irrespective of Ac or La being present in the replacement fluid (Figure 1). During HD-Ac, MAP decreased (-20%; $p < 0.005$) as SVR dropped (-22%; $p < 0.02$)

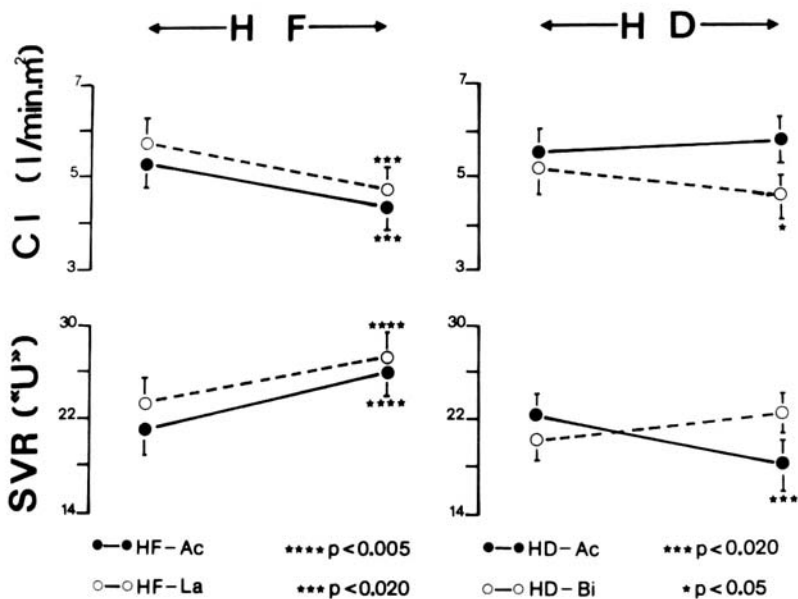


Figure 1. Systemic haemodynamic results for HF and HD

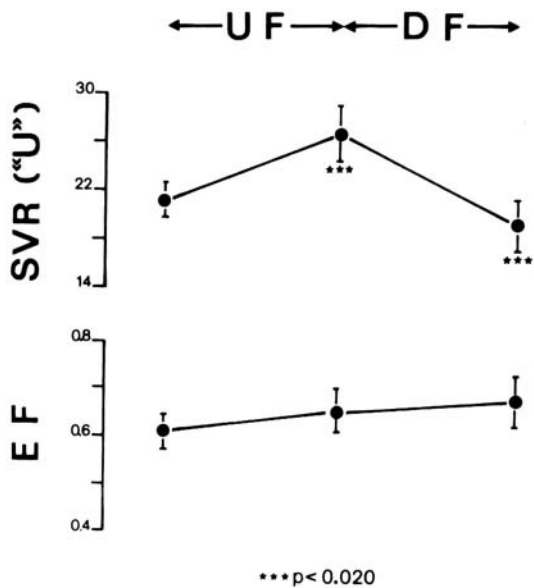


Figure 2. Main results for sequential UF/DF

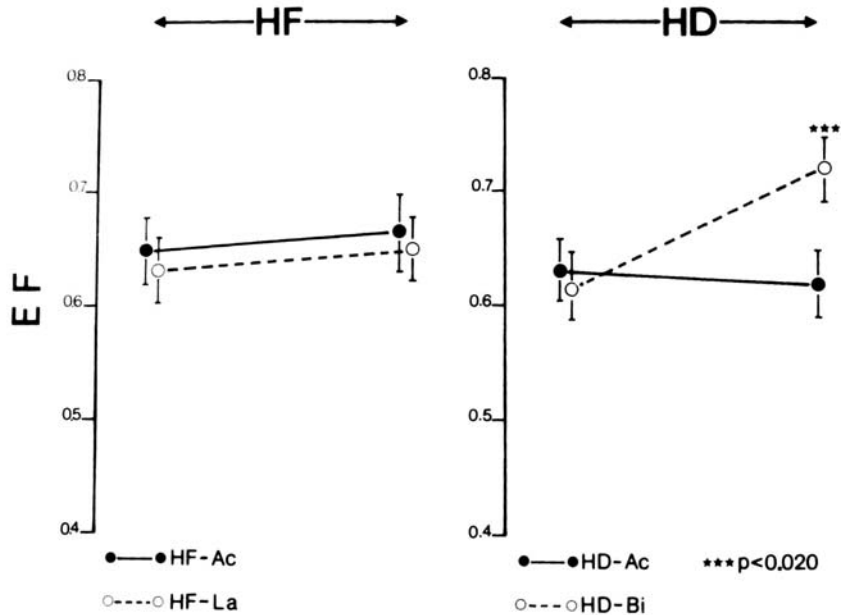


Figure 3. Ventricular function during HF and HD

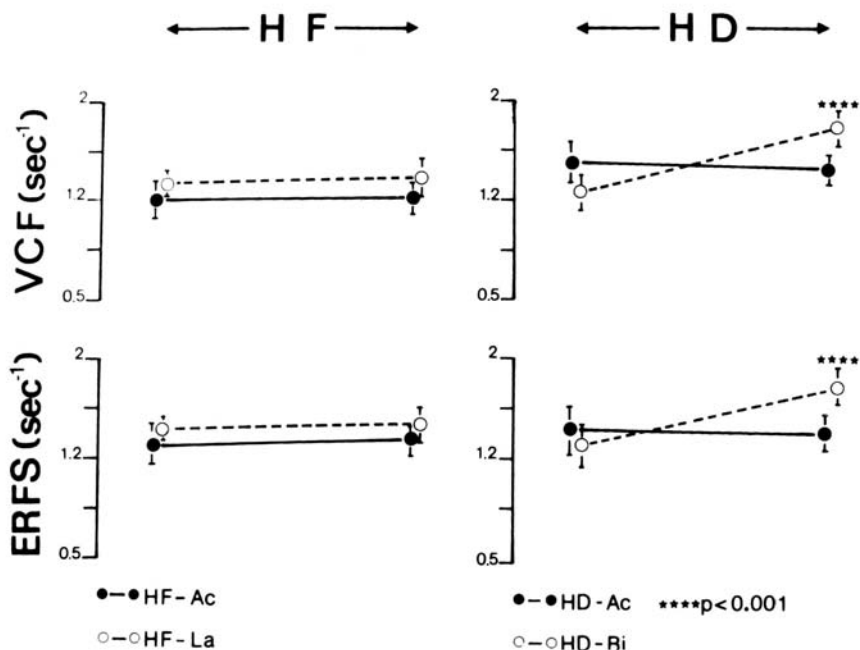


Figure 4. Myocardial contractility during HF and HD

though CI was maintained (+2%). HD-Bi showed no changes in SVR (+4%) and a slight decrease in CI (-9%; $p < 0.05$) (Figure 1). UF induced a fall in CI (-17%; $p < 0.02$) and an increase in SVR (+24%; $p < 0.02$), whereas DF reduced SVR (-31%; $p < 0.02$) (Figure 2).

No significant changes in cardiac performance were detected with HF, HD-Ac, UF or DF. HD-Bi produced a significant improvement in whole ventricular function (EF: +17%; $p < 0.02$) (Figure 3), which was even more remarkable when contractility parameters were considered (VCF: +29%; ERFS: +25%; $p < 0.001$) (Figure 4).

Discussion

HF with either acetate or lactate buffers and UF induced identical cardiovascular influences. These procedures were associated with an increase in SVR capable of counterbalancing the observed decrease in CI [7]. On the other hand, HD produced rather different haemodynamic changes in relation to the dialysate buffer. After HD-Ac the fall in SVR, maintenance of CI and arterial hypotension are in contrast to the striking vascular stability and CI reduction found after HD-Bi. Excluding isolated DF, a comparable stroke volume decrease was obtained at the end of all procedures; this is in agreement with the equivalent ultrafiltration rate and weight loss in the five groups of patients. In this setting the CI depends on heart rate changes rather than on ventricular volume modifications induced by the procedure itself [8].

The fact that UF and isolated DF yielded equal haemodynamic reactions as HF and HD respectively, suggests that the physical modality of solute transport plays a major role. Recent studies referred to the vasodilatory effect of acetate as the main factor involved in vascular instability [9]. However, according to our results, this does not seem to apply when solute removal is conducted by convective transport such as during HF-Ac.

Since the acute effects of HF and HD on cardiac performance remains unclear, we looked at whether the two modalities of solute transport induced distinct myocardial contractility influences. Furthermore, as acetate has been known to produce negative inotropic reactions, this agent may be cardiodepressor when added to substitution fluid or dialysate [10]. These points prompted us to study patients with intrinsic normal functional parameters on clinical and echocardiographical grounds. In this selected patient population we could not find significant changes in ventricular function with HF, HD-Ac, UF and DF, indicating that solute transport does not contribute a particular influence. Moreover, our observations are at variance with the stated acetate-induced contractile depression and suggest that acetate has no deleterious effects, at least in patients whose cardiac reserve is normal.

It has been reported that acute reductions of afterload, such as those achieved during fluid removal, can enhance cardiac function if venous return is kept constant [11]. But in HF and HD both afterload and preload fall concomitantly [12]. Thus, venous return is also proportionally reduced and this explains the lack of change in myocardial function. However, HD-Bi showed a marked improvement in cardiac performance and contractility despite similar degree of

fluid withdrawal and uraemic metabolite removal. Although the extent to which the removal of acute 'toxins' improves ventricular function is uncertain, factors linked to HD-Bi such as more rapid correction of acidosis, better arterial oxygenation and maintenance of SVR may be responsible for the observed enhancement in contractility [13].

Previous studies have demonstrated a good correlation between angiographic and echocardiographic measurements of the left ventricular volumes [14]. Additionally, the sensitivity and reproducibility of M-mode echocardiography for serial evaluation of the contractile state has been recently emphasised [15]. We recommend this non-invasive and non-hazardous technique for the examination of patients with cardiovascular HD side-effects in order to assess whether myocardial dysfunction is a contributing factor. In patients with this condition a trial of bicarbonate-containing dialysate rather than HF seems worthwhile.

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