

THE INFLUENCE OF FLUID REMOVAL DURING HEMODIALYSIS ON HEMODYNAMIC PARAMETERS

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BACKGROUND

Fluid loading and removal are inherent aspects of hemodialysis but little is known concerning the effect of these movements on intradialytic hemodynamics. A non-invasive regional novel impedance cardiography device was used to assess these in chronic hemodialysis patients in order to improve quality of care, specifically reduction intradialytic events, particularly hypotension, guiding by appropriate interventions and ultrafiltration prescriptions¹.

RESULTS

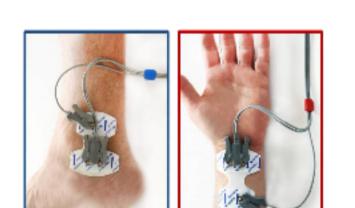
Intradialytic hypotension (IDH) occurred in 10(42%) of 24 patients with mean SBP decreasing from 136 ± 21 to 107 ± 19 (P<0.01) and mean DBP 69 ± 12 to 57 ± 9 (P<0.05) from pretreatment. In the 14 non IDH patients SBP decreased from 138 ± 26 to 130 ± 20 (P=0.3730) and mean DBP from 68 ± 10 to 64 ± 7 (P=0.3443). IDH patients could be divided into two subgroups according to their hemodynamics:

- 1). VASODILATATION (n=5, 80% being diabetic). TPRI decreased during treatment by $29\pm4\%$ from $2,411\pm164$ to $1,712\pm162$ [dyn*sec/cm5*m2] (P<0.001 but CPI did not change significantly. SBP decreased by 30 ± 11 from pretreatment of 148 ± 19 mmHg (P<0.01). Mean ultrafiltration volume was $2,115\pm550$ ml. Midodrine administered pre and during dialysis eliminated the IDH⁷.
- 2) CARDIAC POWER REDUCTION (n=5, 40% diabetic). CI decreased by 11% from 3.3 ± 0.2 to 2.9 ± 0.2 , CPI⁸ decreased by $25\pm5\%$ from 0.61 ± 0.09 to 0.45 ± 0.06 [w/m2] (P<0.01), TPRI did not change significantly and SBP decreased by 27 ± 7 from pretreatment of 124 ± 18 (P<0.01). Mean ultrafiltration volume was $2,420\pm530$ ml.

In the 14 non-IDH patients Insignificant decreases in TRPI and CPI occurred. The effect of intradialytic drug removal was observed in 2 patients: 1) **Hydralazine** - TPRI increased by 69% from 2,159 to 3,469, SBP from 104 to 143, CI decreased by 22% from 2.7 to 2.1, HR was not changed and SI decreased from 43 to 33. 2) **Nadolol** - CI increased by 38% from 2.6 to 3.6, HR increased from 83 to 98, SI from 31 to 37 and TPRI decreased from 2,887 to 1,905.

METHODS

Hemodynamic profiles of 24 hemodialysis patients chronic were investigated using the device (NICaS, NI Medical, Petach Tikva, Israel). Following the placing of electrodes wrist and on contralateral ankle, Stroke volume index (SI), Cardiac output index (CI), Total peripheral resistance index (TPRI) and Cardiac power index (CPI) were calculated^{2,3,4,5,6}. Measurements were made pre-,mid-,just before the end and 10 min post treatment and were 4-5 times in each repeated individual.



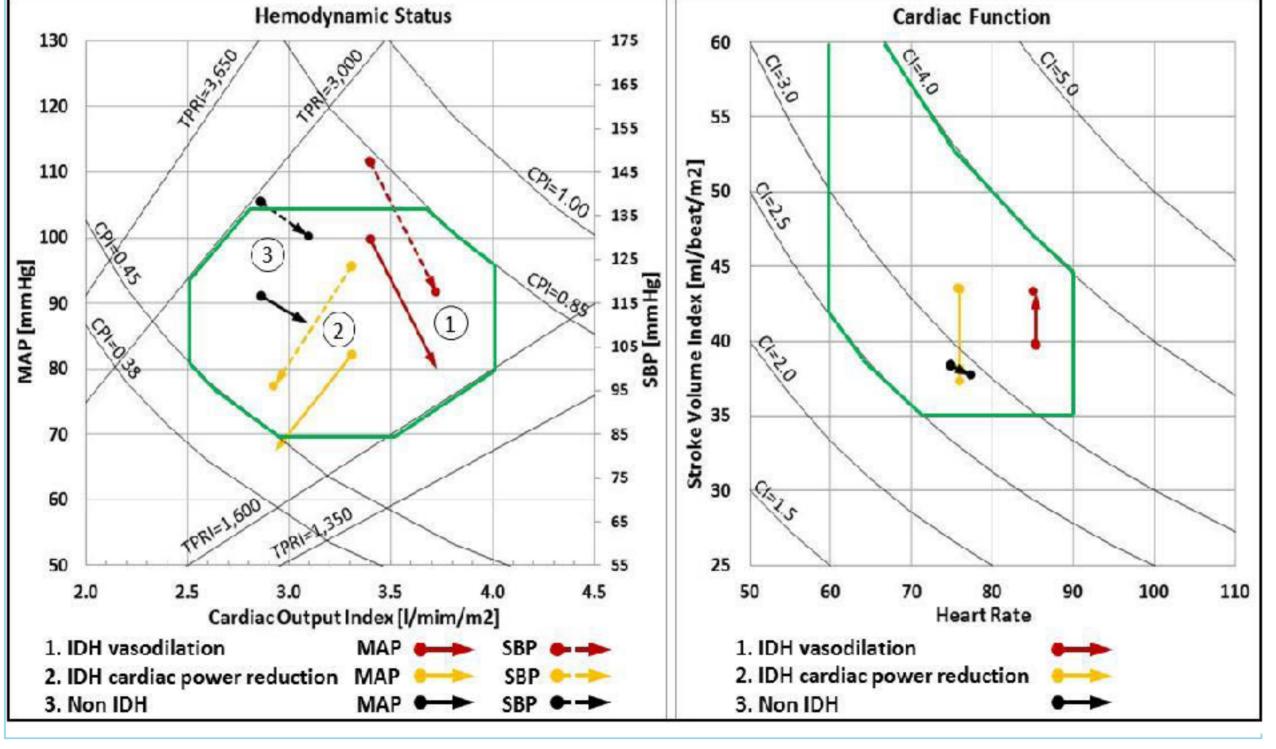


NICaS sensors NICaS monitor

All subjects, IDH and non IDH groups demographics and pretreatment hemodynamics

Demographics All Subjects			Demographics All IDH			Demographics Non IDH			
Parameter Mean STD +/-		Parameter	Mean	STD +/-	Parameter	Mean	STD +/-		
N	24 (100%)		N	10 (42%)		N	14 (58%)		
Male	13 (54%)		Male	6 (50%)		Male	7 (50%)		
Asian	9 (38%)		Asian	4 (40%)		Asian	5 (36%)		
Hispanic	6 (25%)		Hispanic	2 (20%)		Hispanic	4 (28%)		
Black	4 (16%)		Black	2 (20%)		Black	2 (14%)		
White	5 (21%)		White	2 (20%)		White	3 (21%)		
Age	67	8	Age	56	8	Age	66	5	
Pre Weight [kg]	71.5	15	Pre Weight [kg]	74.6	16.9	Pre Weight [kg]	69.4	12.7	
Post Weight [kg]	69.5	14	Post Weight [kg]	72.3	16.8	Post Weight [kg]	67.5	12.3	
Pre BMI [kg/m2]	26.2	5.0	Pre BMI [kg/m2]	28.9	5.2	Pre BMI	26.0	1.7	
TBW [% of w]	51.1%	7.8%	TBW [% of w]	48.5%	7.1%	TBW [% of w]	53.8%	4.4%	
Diabetes	14 (58%)		Diabetes	6 (50%)		Diabetes	8 (57%)		
Heart Failure	6 (25%)		Heart Failure	1 (10%)		Heart Failure	5 (36%)		
Pre BP [mmHg]	137/68	23/11	Pre BP [mmHg]	136/69	21/12	Pre BP [mmHg]	138/68	26/10	
Pre HR	77	8	Pre HR	80	7	Pre HR	75	9	
Pre SI [ml/m2]	10	6	Pre SI [ml/m2]	42	4	Pre SI [ml/m2]	39	8	
Pre CI [I/min/m2]	3.1	0.5	Pre CI [I/min/m2]	3.3	0.3	Pre CI [I/min/m2]	2.9	0.5	
Pre CPI [w/m2]	0.62	0.15	Pre CPI [w/m2]	0.68	0.16	Pre CPI [w/m2]	0.57	0.12	
Pre TPRI	2,511	651	Pre TPRI	2,219	253	Pre TPRI	2.720	770	

Hemodynamics mean trends of the 3 groups



IDH due to Vasodilation and IDH due to Cardiac power reduction demographics and pretreatment hemodynamics

	graphics odilation		Demographics IDH cardiac power reduction			
Parameter	Mean	STD +/-	Parameter	Mean	STD +/-	
N	5 (17%)		N	5 (21%)		
Male	3 (60%)	845	Male	3 (60%)		
Asian	4 (80%)		Asian	0 (0%)		
Hispanic	0.00		Hispanic	2 (40%)		
Black	1 (20%)		Black	1 (20%)		
White	-		White	2 (10%)		
Age	64	9	Age	67	8	
Pre Weight [kg]	65.5	5.1	Pre Weight [kg]	83.7	20.3	
Post Weight [kg]	63.4	5.2	Post Weight [kg]	81.2	20.2	
Pre BMI [kg/m2]	25.5	4.5	Pre BMI [kg/m2]	32.3	3.3	
Pre TBW [% of w]	52.0%	8.2%	Pre TBW [% of w]	44.6%	4.6%	
Diabetes	4 (80%)		Diabetes	2 (40%)		
Heart Failure	in <u>u</u> s	3.5	Heart Failure	1 (20%)		
Pre BP [mmHg]	148/77	19/13	Pre BP [mmHg]	124/62	18/4	
Pre HR	85	7	Pre HR	76	3	
Pre SI [ml/m2]	40	1	Pre SI [ml/m2]	44	4	
Pre CI [I/min/m2]	3.4	0.3	Pre CI [I/min/m2]	2.9	0.5	
Pre CPI [w/m2]	0.76	0.18	Pre CPI [w/m2'	0.61	0.09	
Pre TPRI	re TPRI 2,411 164		Pre TPRI	2,027	158	

Statistical analysis of hemodynamic parameters trends during dialysis treatment

	IN.	SBP	MAP	31	нк	Ci	CPI	IPRI
All patients	24	Pre: 137±23 Lowest: 121±23 Diff.: -16±14 P < 0.01	Pre: 91±14 Lowest: 81±11 Diff.: -10± 9 P < 0.01	Pre: 40± 8 Lowest: 39± 8 Diff.: -2±13% P < 0.374	Pre: 77± 9 Lowest: 79±11 Diff.: 2± 9% P = 0.312	Pre: 3.1±0.5 Lowest: 3.2±0.7 Diff.: 5±19% P = 0.273	Pre: 0.62±0.12 Lowest: 0.54±0.09 Diff.: -11±12% P < 0.01	Pre: 2511±776 Lowest:2197±743 Diff.: -12±22% P < 0.01
Subgroup	N	SBP	MAP	SI	HR	CI	СРІ	TPRI
IDH vasodilation	5	Pre: 148±19 Lowest: 118±18 Diff.: -30±11 P < 0.01	Pre: 100±14 Lowest: 80±13 Diff.: -20±6 P < 0.01	Pre: 40± 1 Max: 43± 2 Diff.: 9± 5% P < 0.01	Pre: 85± 7 Lowest: 85±8 Diff.: 0± 8% P = 0.998	Pre: 3.4±0.3 Lowest: 3.7±0.6 Diff.: 10±14% P = 0.204	Pre: 0.76±0.18 Lowest: 0.67±0.22 Diff.: -12±13% P = 0.076	Pre: 2411±16 Lowest:1712±163 Diff.: -29±4% P < 0.01
IDH cardiac power reduction	5	Pre: 124± 9 Lowest: 96±15 Diff.: -27± 7 P < 0.01	Pre: 82± 4 Lowest: 68± 7 Diff.: -15± 3 P < 0.01	Pre: 44± 4 Lowest: 37± 5 Diff.: -14± 3% P < 0.01	Pre: 76± 3 Lowest: 76± 7 Diff.: 0± 5% P = 0.9023	Pre: 3.3±0.2 Lowest: 2.9±0.2 Diff.: -11± 4% P < 0.01	Pre: 0.61±0.09 Lowest: 0.45±0.06 Diff.: -25±5% P < 0.01	Pre: 2027±15 Lowest:1763±22 Diff.: -13± 7% P < 0.01
Non IDH	14	Pre: 138±26 Lowest: 130±20 Diff.: - 8±10 P < 0.05	Pre: 91±14 Lowest: 87±11 Diff.: - 4± 6 P < 0.05	Pre: 39± 8 Lowest: 38± 8 Diff.: - 1±13% P = 0.632	Pre: 75± 9 Lowest: 77±11 Diff.: 3± 6% P = 0.253	Pre: 2.9±0.5 Lowest: 3.1±0.7 Diff.: 9±21% P = 0.156	Pre: 0.57±0.12 Lowest: 0.53±0.09 Diff.: - 6± 9% P < 0.05	Pre: 2720±770 Lowest:2525±742 Diff.: - 5±26% P = 0.2938

CONCLUSIONS

Reported here for the first time in dialysis patients, the use of noninvasive regional impedance cardiography technology in order to provide insight into hemodynamic parameters which can be used to guide appropriate management.

Surprisingly, vasodilatation was the main mechanism in 50% of the patients who developed IDH. Autonomic dysfunction common in diabetic patients as the cause of vasodilation requires further investigation^{9,10}. Midodrine seems to be the most effective intervention in these patients.

Other 50% of the patients developed IDH due to a decrease in cardiac output which can be probably attributed to preload changes but cardiac diastolic dysfunction could have contributed as indicated by Cardiac Power decrement.

We believe that this new approach will increase understanding of cardiovascular events during dialysis and give physiological basis for pharmacological and other prophylactic intervention during IDH.

It will also provide insight into the effects of dialyzable cardiac drugs. The methodology could be useful in comparative population studies.

REFERENCES

- 1. Ritz E, et al. Cardiac changes in uremia and their possible relation to cardiovascular instability on dialysis. Contrib Nephrol 1990;78:221–229
- 2. A. Handt, M. Farber, J. Szwed: Intradialytic measurement of cardiac output by

thermodilution and impedance cardiography. Clinical Nephrology 1977; 7(2):61-64

- 3. G Cotter, A. Schachner, L. Sasson, H. Dekel and Y. Moshkovitz: Impedance cardiography revisited. *Physiological Measurement* 27 (2006) 817-827, July 2006
- 4. Paredes O.L. et al: Impedance cardiography for cardiac output estimation reliability of wrist-to-ankle electrode configuration. *Circulation Journal* 2006; 70: 1164-1168
- 5. Cotter G. et al: Accurate, non-invasive, continuous monitoring of cardiac output by Whole Body Electrical Bio-impedance. Chest 2004; 125: 1431-1440
- 6. Cotter G. et al: The role of cardiac power and systemic vascular resistance in the pathophysiology and diagnosis of patients with acute congestive heart failure. European Journal of Heart Failure; 5 (2003) 4433-4451
- 7. A. House et al: Midodrine appears to be safe and effective for dialysis-induced hypotension: a systematic review. Nephrology Dialysis Transplant 2004; 19: 2553-2558
- 8. Mendoza D et al: Cardiac power output predicts mortality across a broad spectrum of patients with acute cardiac disease. *American Heart Journal* 2007; 153: 366 370
- 9. Converse RL Jr. et al. Paradoxical withdrawal of reflex vasoconstriction as a cause of hemodialysis-induced hypotension. *J Clin Invest* 1992;90:1657–1665
- 10. Tatsuya S. et al. Hemodialysis-associated hypotension as an independent risk factor for two-year mortality in hemodialysis patients. *Kidney Intern* 66:1212–1220







