

---

## Clinical Determination of Dry Body Weight

Bernard Charra, Guillaume Jean, Jean-Marc Hurot, Jean-Claude Terrat, Thierry Vanel, Cyril VoVan, Foued Maazoun, Charles Chazot

Centre de rein artificiel, Tassin, France

---

**W**hile nephrologists wait for the ideal, non invasive, inexpensive, precise, and reproducible tool to evaluate extracellular volume (ECV), they need to exert their clinical acumen in the quest of that holy grail, dry weight (DW).

Estimation of DW using a clinical approach based on blood pressure (BP) and ECV is feasible and reliable as shown by successful experiences in various dialysis modes over more than three decades. But a need still exists to resolve difficulties associated with accurate assessment of BP (methods and circumstances of measurement, and the confounding effects of antihypertensive drugs) and ECV (evaluation of weight changes unrelated to ECV, lack of specificity and sensitivity of clinical symptoms, lag time, confusion in terminology). An essential point in clinical assessment of DW is that a normal BP is at the same time the target and the crucial index of DW achievement. For this reason, a trial-and-error “probe” process has to be used at intervals to make sure that the dry weight target point is correctly estimated.

The various “non clinical” methods proposed for dry weight assessment increase the complexity and the cost of hemodialysis. They are, in the present state of things, more clinical research than practice tools. They do not replace clinical judgment.

(*Hemodial Int.*, Vol. 5, 42–50, 2001)

### Key words

Dry weight, blood pressure, extracellular volume, sodium

### Introduction

Determining and maintaining dry body weight is often reported to be the thorniest clinical problem in maintenance hemodialysis [1,2]. The opinion that “better methods of determining dry weight are required” [3] is shared by many nephrologists. Expecting the ideal, non invasive tool, which would give a “one-glance” assessment of the extracellular volume (ECV) is, by our experience, an unrealistic hope. Finding and maintaining dry weight (DW) requires clinical skills and judgment without which nephrologists cannot achieve DW and thereby control blood pressure (BP) in their dialysis patients.

---

### Correspondence to:

Bernard Charra, MD, Centre de rein artificiel, 42 Avenue du 8-Mai-1945, Tassin 69160 France.  
email: bcharra@aol.com

In chronic renal failure (CRF), a progressive increase in ECV affects both interstitial space and plasma volume. As shown by Guyton [4] in uremic dogs, a sodium load results—through transient increase of cardiac output and total peripheral resistances—in an increased BP, which turns on the natriuresis. The increased natriuresis brings ECV back to almost normal within a few weeks. This process explains how CRF leads to hypertension (HT) without obvious saline overload, and especially without edema. It is of note that when implementing a strict DW policy, residual diuresis and natriuresis shut off almost completely within the first few weeks of long dialysis.

Owing to the intermittent nature of hemodialysis (HD), the patient oscillates between a “wet”, saline-overloaded state just before the session and a “dry” state just after. During the few hours of ultrafiltration (UF) during an HD session, the plasma volume is reduced to a low point. Refilling [5] from the interstitial space lags some hours behind [6], so that, just at the end of the session, the patient is hypovolemic. A transient orthostatic hypotension is not abnormal at that time; it disappears within a few hours when refilling is complete. Owing to the delay in refilling, any method that bases ECV estimation on plasma volume at the end of the session [for example, vena cava diameter, atrial natriuretic peptide (ANP), cyclic guanosine monophosphate (cGMP)] systematically overestimates the ultrafiltration and underestimates the actual ECV value.

The new trend toward more frequent dialysis will reduce problems associated with the rapid removal of ECV, because the amount of ECV to be removed is so much less [7,8].

### What is dry weight?

Intermittent dialysis substitutes for the native pressure–natriuresis system [9], whereby, from the analysis of BP and ECV, the physician (or person in charge of the dialysis) evaluates the sodium and water extraction needed to return ECV to normal.

Many definitions of DW have been suggested since Thomson *et al.* [10] initially proposed the term.

Several definitions of DW are based on the presence [11] or absence of symptoms of hypovolemia or hypervolemia [12]. But a wide weight and ECV gap exists between these two symptomatic extremes. Therefore, DW is better defined as the body weight achieved at the end of dialysis at which the patient remains constantly normotensive until the next dialysis without antihypertensive medication [13].

### How is ECV assessed?

Three main sources of information are used in ECV evaluation. First, conditions leading to ECV excess (usually excessive salt intake) or depletion (usually excessive loss) and symptoms (dyspnea, headache or dizziness, fatigue, cramps) need to be identified by detailed history. Second, current clinical signs are, of course, essential. The BP (with postural changes) is critical to a DW evaluation. It gives a direction and an estimate of the ECV change that weight change confirms and measures more accurately. One must also look for edema and evaluate the neck veins with the patient lying flat [or measure central venous pressure (CVP) if the patient's blood access is a deep vein catheter]. Third, an x-ray evaluates the heart size (cardiothoracic ratio) and lung density. Hematocrit, total protein, and serum albumin provide information on the relative hemoconcentration or dilution.

Symptoms are not always reliable in ECV assessment. A patient can be saline overloaded and yet experience cramps and hypotension during dialysis if the ultrafiltration rate exceeds plasma refilling capacity. The patient may be normotensive or hypotensive while saline overloaded if in heart failure or, in some cases, even without heart failure [14]. On the other hand, the patient can be hypertensive with a low or normal ECV (for example, high renin state, white-coat effect, or hypercalcemia). Symptoms also lack sensitivity, as they occur relatively late when the patient is hypovolemic, and very late when the patient is hypervolemic.

The only systematic prospective study of symptoms in relation to ECV in HD, by Wizemann *et al.* [15], showed that, to correct symptoms of hypovolemia, the dialysis target weight had to be augmented by a mean of 940 g. To correct the symptoms of hypervolemia, a mean reduction in target weight of 2409 g was needed. These corrections leave a wide, clinically mute fluctuation zone of approximately 3.4 kg around the ideal DW. Therefore, in DW evaluation, one should not wait for clinical manifestations of ECV overload or depletion, but check ECV systematically and regularly.

The frequency of systematic ECV (DW) evaluation is a crucial issue, although it is seldom mentioned in the literature. Besides, the ideal frequency of DW evaluation is variously appreciated by various authors. Some recommend an evaluation when volume [15] or hypertension [16] is not controlled; others advise a monthly [17] or even less frequent evaluation. In Tassin, DW is re-evaluated after each and every session by the physician or the person in charge of the patient.

The clinical DW method consists of using the ECV indicators together with BP values to estimate the DW. If BP is normal throughout the interdialytic cycle and the patient is comfortable, that patient is probably at DW. But only "probing" for DW will turn the clinical estimate into a reasonable certainty [13]. At the start of HD, and often during the maintenance phase, whenever one cannot determine the patient's ECV, one must use the "probe" for DW.

### The probe for dry weight

The probe consists of a systematic, step-by-step, progressive, trial-and-error lowering of post-HD weight to reach the point where hypotension appears. During the probe, intense, carefully monitored ultrafiltration, combined with a strict low sodium diet, permits a gradual reduction in pre-dialysis weight over several sessions. The actual rate of decrease is determined strictly by trial-and-error, governed by the patient's tolerance, to reduce to a minimum episodes of muscle cramps and hypotension. The occurrence of hypotension at the end of dialysis is the major proof that the patient has achieved a low-normal or low ECV. When this point has been reached, the target post-dialysis weight must be increased by a few hundred grams.

The clinical practice of routinely and deliberately bringing the patient to the edge of hypovolemia and to hypotension is questioned by some authors on the basis that patients who become hypotensive on dialysis may be intravascularly hypovolemic, euvoletic, or hypervolemic. This discrepancy between the interstitial and plasma compartments is explained by the fluid kinetics between the two compartments. The transfer of fluid from the interstitium to the plasma is delayed for a few hours. The shorter the dialysis and the higher the UF rate, the less the ability of plasma refilling to catch up with the speed of ultrafiltration. It is, therefore, true that hypotension on dialysis is not, *per se*, evidence that the patient has achieved DW. In practice, to distinguish DW achievement from hypotensive episodes due to rapid ultrafiltration, post-dialysis weight must be reduced progressively over several sessions.

#### *Practical implementation of clinical DW assessment*

In practice, the clinical DW method requires a dialysis log chart that summarizes the pre- and post-dialysis weight, BP values, and intradialytic events. These data are summarized on a single line for each dialysis (Appendix: Table I). Browsing through three or four weeks of HD data gives a one-glance, dynamic picture of the weight/blood pressure (WT/BP) relationship. This picture cannot be achieved using separate HD flow sheets that one must shuffle through, or a dialysis book that one must flip through. The lack of such a comprehensive display of recent dialysis data makes the use of the clinical DW method almost impossible; but, a BP/WT flow sheet as part of the current bedside record is an essential tool in maintaining DW.

#### *Long-term relationship between ECV and BP*

The long-term relationship between ECV and BP is well illustrated by looking at the entire Tassin experience of patients in their first year of dialysis (Fig. 1). The ECV expressed by post-dialysis weight drops sharply during the first month owing both to vigorous UF and to a strict low-salt diet. Anti-hypertensive drugs are stopped in more than 95% of patients. Over time, BP continues to decrease progressively; but, after

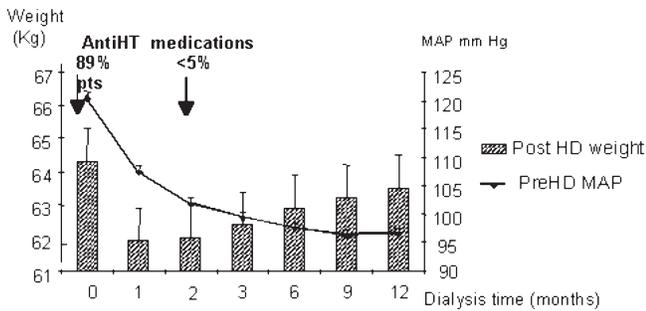


FIGURE 1 Evolution of post-dialysis weight (standard error of mean) and pre-dialysis mean arterial pressure (MAP; standard error of mean) in first hemodialysis (HD<sub>0</sub>) year. HT = hypertensive.

two months, weight begins to increase, and weight and pressure curves cross over. The weight gain does not reflect a change in ECV, but an increase in lean and fat body mass owing to anabolism. After one year or so, weight and BP stabilize.

#### Does ECV control allow for controlling BP in almost all patients?

In Tassin, using thrice weekly 8-hour dialysis sessions, the achievement of DW leads to BP normalization in more than 95% of cases. The cases failing to respond are due either to the patient's lack of compliance, or to non volume related hypertension that may require treatment of the other causative factor of hypertension or the use of antihypertensive medications. The mean casual pre-dialysis BP in the population (calculated from all values for each patient) is 128/79 mmHg, which is within the normal range according to the Sixth Joint National Committee on BP evaluation [18].

This result is not a "center effect." The same good control of BP without drugs was achieved in 90% of patients in the late 1960s and early 1970s [19,20] and is still achieved by those who continue to use long HD [21,22] and also very often by those who use daily dialysis, whether short [23] or long [24]. Furthermore, the same goal can be achieved in standard dialysis, but only if the diet is low enough in sodium to limit interdialytic weight gain [25].

Clinical achievement of DW requires that certain conditions be met. First and foremost, the degree of dietary sodium restriction must match the time available for ultrafiltration. Thus, in the case of standard 3 – 4 hour thrice-weekly dialysis sessions, the low-sodium diet must be very strict; however, this restriction is often forgotten today [26].

Dialysate sodium activity, rather than concentration, governs overall sodium balance [27]. Dialysate sodium must be set so that the movement of sodium by diffusion is either nil or slightly outward (that is, 138 mmol/L in Tassin). Paradoxically, dialysate sodium has been increasing over the years as dialysis has been shortened [28]. With a thrice-weekly 8-hour schedule, these same conditions apply, but to a lesser degree.

With every-other-day dialysis or quotidian dialysis, the interdialytic interval is shorter; and, with nocturnal dialysis, the time for ultrafiltration is so long that less dietary sodium restriction is usually needed.

Convection associated with UF is the most common method of sodium removal. Technically, it is limited primarily by the plasma refilling capacity of the patient. The increasing entry into dialysis treatment of fragile patients (elderly, cardiovascular disease, diabetes), who are particularly susceptible [29] to side effects of UF, poses insurmountable difficulties in fluid removal in short dialysis.

#### Failure of the clinical dry weight estimation

In spite of wide acknowledgment of the importance of DW, most HD patients in 2001 are hypertensive. One possible explanation of this troubling paradox is that the DW is adequately estimated, but cannot be achieved owing to too-short HD sessions and poor UF tolerance by patients. Even more often, this failure seems to be due to inaccurate estimation of DW. Why?

The difficulty may arise from the use of either (or both) of the two indexes used for DW assessment, BP and ECV.

##### BP target assessment

To clinically assess DW, we rely mainly on BP. If the BP value that we use as our guide is wrong, our DW estimation will be wrong. There are two sources of error.

The first source of error is the BP measurement itself. There are nowadays so many methods and circumstances of BP measurement (systolic, diastolic, mean, differential, day, night, before or after dialysis, pulse pressure, ambulatory blood pressure, etc.) that it is difficult to know which figure to rely on, and which measurement correlates best with ECV. Ambulatory BP monitoring is probably the most accurate, but it has its own limitation (that is, patient acceptance), and it provides many different BP figures (mean, change, variations, nocturnal, etc.) among which, again, a choice must be made. In clinical practice, pre- and post-dialysis values are sufficient.

A second source of confusion is the use of antihypertensive medications. If they work, they create a "fake" normotension, owing to vasodilatation rather than to restoration of adequate ECV. On the other hand, antihypertensive drugs very often fail to work in the presence of ECV excess, but they still potentiate hypotension during UF, making achievement of DW extremely difficult.

##### ECV target assessment (DW assessment)

Four reasons can possibly explain the failure of the DW method. The first reason may be that DW is a mobile target that keeps changing. Because weight is the mirror of ECV, any cause of weight variation (especially changes in lean and fat body mass) must be taken into account in ECV assessment. Recognition of DW change is easy in specific circumstances, such as catabolism following surgery or intercurrent disease, or anabolism after HD initiation. But recognizing the

very common progressive changes in DW is much more difficult. So, clinical circumstances, together with food intake and physical activity changes, must be monitored in a cautious and systematic manner.

A second possible cause of failure of the clinical DW method is that clinical symptoms are often discordant, unreliable, or unspecific. They also may lack sensitivity.

The third problematic issue in using the clinical DW method is the existence of a time lag between ECV and BP changes [30]. In the first month, ECV drops sharply, achieving its nadir in a few weeks; meanwhile, pre-dialysis mean arterial pressure (MAP) decreases slowly over months. We don't know why BP lags behind the volume change, but the answer is probably that vascular remodeling [31,32] takes about that long. It is of utmost importance that clinicians, nurses, and patients themselves understand the lag time phenomenon, so that they can accept that weight or ECV reduction (accompanied by hypotension or cramps) will not immediately result in BP normalization, and so that they won't become discouraged by the probe process.

The fourth and final difficulty in the DW clinical method comes from the common confusion between DW and interdialytic weight change. Reports on the association between pre-dialysis blood pressure and interdialytic weight gain conflict [33–37]. Findings are biased by the use of antihypertensive medications in most patients [38–40]. In 1962, Scribner observed [41] that “It is the average level of sodium in the body over a long period of time that correlates with blood pressure,” rather than the acute variations of weight. In other words, interdialytic weight gain does not, *per se*, directly affect BP; but, when it leads to a progressive increase in ECV, it results in hypertension. These two quite different concepts should not be confused.

Acute interdialytic weight changes are highly variable. First-order ECV oscillations are due to the intermittent nature of HD, while DW is the stable ECV level needed to achieve normal BP. In our opinion, much energy has been uselessly devoted toward discussing the relationship between interdialytic weight and BP. The issue is not the variation of weight around DW, but the DW itself, which is associated with good BP control.

#### **Dry weight assessment: clinical or not?**

Based on the fact that clinical determination of dry weight was judged insensitive [3,11,42,43], unreliable [44,45], or too difficult [46], several so-called objective methods for measurement of ECV or DW in HD patients have been proposed.

The cardiothoracic ratio on chest x-ray [47,48] and its more sophisticated form, the electron beam CT scan of lung density [49], are used to anatomically assess volume status.

Vena cava diameter and collapsibility were first proposed by Ando *et al.* [50]. The method has found many advocates [51–57] because of its simplicity and good correlation with intravascular volume and CVP, but it has also been criticized [58–63] because it is measured at the end of the HD session

before plasma volume rebound is complete. This timing leads to a systematic overestimate of the UF and an underestimate of the ECV. Other critics point to the fact that the technique is operator-dependent and of limited use in heart failure or heart valve diseases.

The use of serum ANP [64–70] and cGMP [69–71] levels has also found defenders; but, again, critics point to their unknown low-level values (they detect only overload, and with questionable sensitivity) and the marked interpatient variability [3,62,72–79].

The first proposals to use bioimpedance analysis (BIA) [11,43,80–83], and the first criticisms [46], were directed toward the monofrequency mode. Monofrequency BIA is not able to differentiate intracellular and extracellular compartments [63,84]. Multifrequency (BIA spectroscopy) is able to differentiate them adequately [85], but multifrequency BIA has its own limitations. Conductivity is measured on limbs, ignoring the eventual changes in the central part of the body [86], leading to an underestimation of fluid removal of up to 30% [86]. Besides, patients' postural changes profoundly affect the results [87]. This situation has led to the proposal of segmental BIA [88].

The segmental approach uses the sum of arm, trunk, and leg measurements. The correlation between BIA and clinical DW assessed by repeated challenges to achieve a lower DW is not excellent [89]; a normal Ri:Re ratio (Ri and Re stand for resistivity of intra and extracellular compartments) does not exclude fluid excess. The extrapolation of Ri and Re in volumetric terms has been derived from non uremic populations, so that BIA cannot be considered validated in uremic patients [63]. Furthermore, short-term and long-term changes in body composition probably alter the conductivity properties of uremic tissues [90].

Continuous blood volume monitoring (BVM) was initially designed [91] and used for UF kinetic analysis [92]. It was then developed as a tool for preventing intradialytic morbid events [93,94], leading eventually to biofeedback blood volume control loops [95,96]. Because refilling depends on fluid overload [6,97], BVM has been suggested for use in DW determination—more specifically, in detecting fluid overload [98,99]. This use has been experimentally verified in patients considered at their DW on standard clinical assessment [98,100]. But the value of BV monitoring as a tool for DW determination has limitations: refilling rates have wide inter- and intra-individual variability [63,101]. Besides, BV during dialysis is not an accurate reflection of the entire ECV [102].

None of these methods is perfect, and so a suggestion is often made to combine two or more of them [71,103,104], and eventually to associate them with clinical DW assessment [3,63,70]. However, combinations of methods increase the costs, complexity, and complications of dialysis even more.

In fact, for clinical purposes, we do not really need a very precise absolute evaluation of ECV. The relative evaluation provided by clinical estimation suffices, as long as it allows for the normalization of ECV and of blood pressure. The ad-

vantages of the clinical DW concept are its simplicity, cost effectiveness, immediacy, and universal availability. We therefore agree with the authors who conclude that the so-called objective methods can be of help, but that they do not replace clinical judgment [71,102].

On the other hand, the clinical method of DW assessment relies heavily on BP measurements. Ambulatory BP monitoring sharpens our estimation of the “true” BP. It is the best way to diagnose silent hypervolemia [63]. It allows us to recognize the rather common situation in which a patient who is normotensive throughout the interdialytic period has an elevated BP just before dialysis (lack of resting conditions, white-coat hypertension). Accordingly, we use it in patients when the simple pre- and post-dialysis BP approach may be questioned.

Studies have shown that the best estimate of the BP obtained by 24-hour automated blood pressure monitoring is given by pre-dialysis BP [2,105], by post-dialysis BP immediately [33] or 20 minutes later [106], or by a mean value of the latter two [35]. In fact, it seems reasonable to say that nothing really replaces the full automated blood pressure monitoring report.

**Discussion**

One is amazed by the fact that, in the literature concerning DW assessment in dialysis, BP control is seldom mentioned [70]. The invisibility of BP control is especially true for reports of “objective methods” of DW assessment.

For this report, we reviewed 42 papers analyzing DW assessment tools. Only one quarter of them (10/42) mentioned BP values, only one third (14/42) mentioned hypertension or hypotension, and almost half of them (20/42) did not mention BP at all. This divorce between volume control and BP *per se* is extremely revealing. Much effort must still be made to promote the concept that in HD patients, BP is strongly volume-dependent.

For those who are convinced and who use BP as the primary index of adequate volume, the main difficulty remains that, in the clinical method, BP is at the same time the index and the target (normotension). This situation is what makes the use of trial and error essential and mandatory.

In practice, the problems encountered in achieving DW using clinical assessment are relieved by simple means: regularly and systematically evaluating appetite and salt intake; regularly using a dialysis log to summarize several weeks of dialysis; and accounting for the lag time. Each time confusion arises in DW evaluation, one should use the probe, which almost always gives the answer.

Clinical assessment of DW is feasible and reliable in dialysis patients. But with standard 3 – 4 hour thrice-weekly dialysis, it is possible only if the patient restricts salt intake. Assessment is much easier if the sessions are not too short, and if the patients are free of antihypertensive drugs.

The main goal in controlling ECV is to achieve BP control. But other conditions are also dependent on ECV, and independent of BP control: left ventricular hypertrophy [107,108], congestive heart failure [109,110], and nutrition [111,112].

**Appendix**

The computer screen (Table I) presents data from 4 weeks (12 sessions). From left to right, the columns show the session identification number (column 1), date (column 2), pre-dialysis weight (column 3), post-dialysis weight (column 4), pre-dialysis BP (column 5), post-dialysis BP (column 6), and weight loss (column 7). Column 8 summarizes the events occurring during the session (for example, cramps, hypotension, vomiting, saline infusion) and the comment of the physician on the symptoms and the achieved post-dialysis weight in relation to the newly estimated DW. This latter figure can be positive, nil, or negative: that is, +0.3 means that the patient remained 300 g over the estimated DW; -0.8 means that

APPENDIX: TABLE I Tassin dialysis log sheet (computer screen).

(1) HD #	(2) Date	(3) Weight pre-HD	(4) Weight post-HD	(5) Blood pressure pre-HD	(6) Blood pressure post-HD	(7) ΔW (kg)	(8) Observations	(9) Validation (MD)
1097	5/05/94	65.9	64.5	145/82	128/77	1.4	—	Dr. C
1098	7/05/94	65.5	64.3	128/80	90/60	1.2	Cramps -0.7	Dr. C
1099	9/05/94	66.9	65.3	142/82	130/80	1.6	+0.3	Dr. C
1100	14/05/94	66.2	64.7	145/82	105/70	1.5	-0.3	Dr. C
1101	16/05/94	65.9	64.8	136/79	124/73	1.1		BC
1102	19/05/94	66.9	64.0	125/82	90/60	2.9	Hypotension NaCl 250 mL -1.5	BC
1103	21/05/94	65.2	64.2	98/50	95/60	1.0	Vomiting -1.3	JCT
1104	23/05/94	65.5	65.3	125/65	112/65	0.2	-0.2	JCT
1105	26/05/94	66.9	65.5	125/60	108/67	1.4		JCT
1106	28/05/94	66.3	65.7	122/70	110/75	1.2	+0.2	Dr. C
1107	30/05/94	67.0	65.5	138/82	128/77	1.5		CC
1108	02/06/94	67.2	65.5	138/82	128/77	1.7		BC

HD = hemodialysis; ΔW = weight loss during the session.

the patient lost 800 g too much; and no comment means that the post-dialysis weight corresponded to the estimated ideal DW. The physician comment on the achieved post-dialysis weight made after each session is a vital point. It is taken into account by the computer program to reset the ideal DW for the next session. Column 9 identifies the physician who validated the session.

As an example, dialysis sessions #1097 – #1108 for patient X are reported here (Table I). At the end of session #1097, the weight is 64.5 kg, and Dr. C makes no comment, considering 64.5 kg to be the correct dry weight. After the following session, because the patient had cramps and a low post-dialysis BP, Dr. C increased the DW by 500 g (DW = 65 kg) by indicating that post weight was 700 g below estimated DW. This DW is maintained for sessions #1099 and #1100. During session #1101, the patient lost 200 g too much, but the physician in charge, BC, given the BP figures, considers this weight (64.8 kg) to be the true DW. It therefore becomes the new DW. The next session, #1102, is marked by a low post-dialysis BP and by intradialytic hypotensive episodes requiring infusion of 250 mL of saline; BC therefore increases the DW by 700 g (DW = 65.5 kg) by writing that the post weight was –1.5 kg below the true DW. Unfortunately, the next dialysis session is again troubled: the patient vomits and loses too much weight. The physician in charge, JCT, maintains the DW at 65.5 kg indicating that post weight was 1.3 kg below DW. At the end of the next uneventful session, #1104, the DW is almost reached (–200 g), and the BP comes to a correct level. In the four subsequent sessions, the DW is maintained at 65.5 kg.

## References

- 1 Mailloux LU. Hypertension in the dialysis patient. *Am J Kidney Dis.* 34(2):359–61, 1999.
- 2 Zucchelli P, Santoro A. How important is volume excess in the etiology of hypertension in dialysis patients? *Semin Dial.* 12(5):304–6, 1999.
- 3 Jaeger JQ, Mehta RL. Dry weight and body composition in hemodialysis: A proposal for an index of fluid removal. *Semin Dial.* 12(3):164–74, 1999.
- 4 Guyton AC. *Arterial Pressure and Hypertension.* Philadelphia, PA: W.B. Saunders Company, 1980.
- 5 Kim KE, Neff M, Cohen B, Somerstein M, Chinitz J, Onesti G, Swartz C. Blood volume changes and hypotension during hemodialysis. *ASAIO Trans.* 16:508–14, 1970.
- 6 Koomans HA, Geers AB, Dorhout Mees EJ. Plasma volume recovery after ultrafiltration in patients with chronic renal failure. *Kidney Int.* 26(3):845–54, 1984.
- 7 Fagugli RM, Buoncrisiani U, Ciao G, Pasticci F, Pasini P, Cicconi B. *et al.* Blood pressure reduction in hypertensive patients on daily hemodialysis (abstract). *J Am Soc Nephrol.* 9:233A, 1998.
- 8 Leyboldt JK, Cheung AK. Extracellular volume in nocturnal hemodialysis. *Semin Dial.* 12(suppl 1):S51–4, 1999.
- 9 Guyton AC. Renal function curve—A key to understanding the pathogenesis of hypertension. *Hypertension.* 10(1):1–6, 1987.
- 10 Thomson GE, Waterhouse K, McDonald HP Jr, Friedman EA. Hemodialysis for chronic renal failure. *Arch Intern Med.* 120(2):153–67, 1967.
- 11 Kouw PM, Olthof CG, ter Wee PM, Oe LP, Donker AJM, Schneider H, de Vries PM. Assessment of post-dialysis dry weight: An application of the conductivity measurement method. *Kidney Int.* 41(2):440–4, 1992.
- 12 Lazarus JM, Denker BM, Owen WF. Hemodialysis. In: Brenner BM. ed. *The Kidney.* Philadelphia, PA: W.B. Saunders Company, 1996; 2424–506.
- 13 Charra B, Chazot C, Laurent G, Calemard E, Terrat JC, Vanel T, Jean G, Ruffet M. Clinical assessment of dry weight. *Nephrol Dial Transplant.* 11(suppl 2):16–19, 1996.
- 14 Katzarski KS, Charra B, Luik A, Nisell J, Divinho Filho JC, Leyboldt JK, Leunissen KML, Laurent G, Bergström J. Fluid state and blood pressure control in patients treated with long and short hemodialysis. *Nephrol Dial Transplant.* 14(2):369–75, 1999.
- 15 Wizemann V, Schilling M. Dilemma of assessing volume state—The use and the limitations of a clinical score. *Nephrol Dial Transplant.* 10(11):2114–7, 1995.
- 16 Roy LF, Leenen FHH. Therapy of hypertension in end-stage renal disease. In: Parfrey PS, Harnett JD. eds. *Cardiac Dysfunction in Chronic Uremia.* Dordrecht, Netherlands: Kluwer Academic Publishers, 1992; 247–66.
- 17 Daugirdas JT, Dumler F, Zasuwa G, Levin NW. Chronic hemodialysis prescription. In: Daugirdas JT, Ing TS. eds. *Handbook of Dialysis.* Boston, MA: Little, Brown and Company, 1988; 72–86.
- 18 The sixth report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. *Arch Intern Med.* 157(21):2413–46, 1997.
- 19 Vertes V, Cangiano JL, Berman LB, Gould A. Hypertension in end-stage renal disease. *N Engl J Med.* 280(18):978–81, 1969.
- 20 Mailloux LU, Levey AS. Hypertension in patients with chronic renal disease. *Am J Kidney Dis.* 32(5 suppl 3):S120–41, 1998.
- 21 Goldsmith DJA, Covic AA, Venning MC, Ackrill P. Ambulatory blood pressure monitoring in renal dialysis and transplant patients. *Am J Kidney Dis.* 29(4):593–600, 1997.
- 22 McGregor DO, Buttimore AL, Nicholls MG, Lynn KL. Ambulatory blood pressure monitoring in patients receiving long, slow home haemodialysis. *Nephrol Dial Transplant.* 14(11):2676–9, 1999.
- 23 Buoncrisiani U, Fagugli R, Pinciaroli MR, Kulurianu H, Bova C. Optimal blood pressure control with daily hemodialysis. *Perit Dial Int.* 16(suppl 2):S99, 1996.
- 24 Pierratos A. Nocturnal home haemodialysis: An update on a 5-year experience. *Nephrol Dial Transplant.* 14(12):2835–40, 1999.
- 25 Özkahya M, Töz H, Ünsal A, Özerkan F, Basci G, Gürgün C, Akcicek E, Dorhout Mees EJ. Treatment of hypertension in dialysis patients by ultrafiltration: Role of cardiac dilatation and time factor. *Am J Kidney Dis.* 34(2):218–21, 1999.
- 26 Mailloux LU. The overlooked role of salt restriction in dialysis patients. *Semin Dial.* 13(3):150–1, 2000.
- 27 Locatelli F, Ponti R, Pedrini LA, Costanzo R, Di Filippo S, Marai P, Pozzi C. Sodium kinetics across dialysis mem-

- branes. *Nephron* 38(3):174–7, 1984.
- 28 Port FK, Johnson WJ, Klass DW. Prevention of dialysis disequilibrium syndrome by use of high sodium concentration in dialysate. *Kidney Int.* 3(5):327–33, 1973.
  - 29 Raine AEG. The susceptible patient. *Nephrol Dial Transplant.* 11(suppl 2):6–10, 1996.
  - 30 Charra B, Bergström J, Scribner BH. Blood pressure control in dialysis patients. The importance of the lag phenomenon. *Am J Kidney Dis.* 32(5):720–4, 1998.
  - 31 Gibbons GH, Dzau VJ. The emerging concept of vascular remodeling. *N Engl J Med.* 330(20):1431–8, 1994.
  - 32 Mulvany MJ. Resistance vessel structure in hypertension: Growth or remodeling? *J Cardiovasc Pharmacol.* 22(suppl 5):S44–7, 1993.
  - 33 Kooman JP, Gladziwa U, Böcker G, Wijnen JAG, van Bortel LMA, Luik AJ, deLeeuw P, van Hoof JP, Leunissen KML. Blood pressure during the interdialytic period in hemodialysis patients: Estimation of representative blood pressure values. *Nephrol Dial Transplant.* 7(9):917–23, 1992.
  - 34 Fishbane S, Natke E, Maesaka JK. Role of volume overload in dialysis-refractory hypertension. *Am J Kidney Dis.* 28(2):257–61, 1996.
  - 35 Coomer RW, Schulman G, Breyer JA, Shyr Y. Ambulatory blood pressure monitoring in dialysis patients and estimation of mean interdialytic blood pressure. *Am J Kidney Dis.* 29(5):678–84, 1997.
  - 36 Ifudu O, Dawood M, Homel P, Friedman EA. Excess interdialytic weight gain provokes antihypertensive drug therapy in patients on maintenance hemodialysis. *Dial Transplant.* 26(8):541–59, 1997.
  - 37 Rahman M, Fu P, Sehgal AR, Smith MC. Interdialytic weight gain, compliance with dialysis regimen, and age are independent predictors of blood pressure in hemodialysis patients. *Am J Kidney Dis.* 35(2):257–65, 2000.
  - 38 Luik AJ, Gladziwa U, Kooman JP, van Hoof JP, de Leeuw PW, van Bortel LMA, Leunissen KML. Influence of interdialytic weight gain on blood pressure in hemodialysis patients. *Blood Purif.* 12(4–5):259–66, 1994.
  - 39 Sherman RA, Daniel A, Cody RP. The effect of interdialytic weight gain on pre-dialysis blood pressure. *Artif Organs.* 17(9):770–4, 1993.
  - 40 Chazot C, Charra B, Laurent G, Didier C, Vo Van C, Terrat JC, Caemard E, Vanel T, Ruffet M. Interdialysis blood pressure control by long hemodialysis sessions. *Nephrol Dial Transplant.* 10(6):831–7, 1995.
  - 41 Scribner BH. Discussion. *ASAIO Trans.* 8: 298, 1962.
  - 42 Kinet JP, Soyeur D, Balland N, Saint-Rémy M, Collignon P, Godon JP. Hemodynamic study of hypotension during hemodialysis. *Kidney Int.* 21(6):868–76, 1982.
  - 43 de Vries JP, Kouw PM, Van der Meer NJ, Olthoff CG, Oe LP, Donker JM, de Vries PMJM. Non invasive monitoring of blood volume during hemodialysis: Its relation with post-dialytic dry weight. *Kidney Int.* 44(4):851–4, 1993.
  - 44 Leunissen KM, Kouw P, Kooman JP, Cheriex EC, de Vries PM, Donker AJ, Van Hoof JP. New techniques to determine fluid status in hemodialyzed patients. *Kidney Int.* 43(suppl 41):S50–6, 1993.
  - 45 Dionisio P, Valenti M, Bergia R, Caramello E, Stramignoni E, Berto IM, Pellerey M, Bajardi P. Influence of hydration state on blood pressure values in a group of patients on regular maintenance hemodialysis. *Blood Purif.* 15(1):25–33, 1997.
  - 46 Kong CH, Thomson CM, Lewis CA, Hill PD, Thomson FD. Determination of total body water in uremic patients by bioelectrical impedance. *Nephrol Dial Transplant.* 8(8):716–19, 1993.
  - 47 Poggi A, Maggiore Q. Cardiothoracic ratio as a guide to ultrafiltration therapy in dialyzed patients. *Int J Artif Organs.* 3(8):332–7, 1980.
  - 48 Don C, Burns KDA, Levine DZ. Body fluid volume status in hemodialysis patients: The value of chest radiograph. *J Can Assoc Radiol.* 41(3):123–6, 1990.
  - 49 Metry G, Wegenius G, Wikström B, Källskog V, Hansell P, Lindgren PG, Hedenström H, Danielson BG. Lung density for the assessment of hydration status in hemodialysis patients using the computed tomographic densitometry technique. *Kidney Int.* 52(6):1635–44, 1997.
  - 50 Ando Y, Tabei K, Shiina A, Asano Y, Hosoda S. Ultrasonographic evaluation of changes in the inferior vena cava configuration during hemodialysis: Relationship between the amount of water removed and the diameter of the inferior vena cava. *J Jpn Soc Dial Ther.* 18:173–9, 1985.
  - 51 Leunissen KM, Cheriex EC, Janssen JHA, Mooy JM, Van Hoof JP. The inferior vena cava diameter and collapsibility index and the optimal dry weight in chronic hemodialysis patients (abstract). *Nephrol Dial Transplant.* 2(5):438, 1987.
  - 52 Cheriex EC, Leunissen KML, Janssen JHA, Mooy JM, Van Hoof JP. Echography of the inferior vena cava is a simple and reliable tool for estimation of “dry weight” in hemodialysis patients. *Nephrol Dial Transplant.* 4(6):563–8, 1989.
  - 53 Horejs J, Ort J, Nemecek K, Straková M. Echography of the inferior vena cava is a simple and reliable tool for estimation of dry weight in haemodialysis patients (letter). *Nephrol Dial Transplant.* 5(3):238, 1990.
  - 54 Assanelli D, Cannella G, Guerra UP, Cuminetti S, Gagiotti M, Sandrini M. Effects of intravascular volume loading and unloading on atrial sizes and left ventricular function in dialyzed uremic man. *Am J Noninvasive Cardiol.* 4:53–9, 1990.
  - 55 Katzarski KS, Bergström J, Danielsson A, Radmaa I, Freyschuss U. Vena cava inferior diameter in blood pressure control of hypertensive hemodialysis patients (abstract). *Blood Purif.* 11(3):197, 1993.
  - 56 Kouw PM, Kooman JP, Cheriex EC, Olthoff CG, de Vries PM, Leunissen K. Assessment of postdialysis dry weight: A comparison of techniques. *J Am Soc Nephrol.* 4(1):98–104, 1993.
  - 57 Sakurai T, Ando Y, Masunaga Y, Kusano E, Asano Y. Diameter of inferior vena cava as a useful marker of dry weight in continuous ambulatory peritoneal dialysis patients (abstract). *J Am Soc Nephrol.* 5(3):526, 1994.
  - 58 Katzarski KS, Nisell J, Bergström J. Relationship between blood volume and inferior vena cava diameter during and after short and long hemodialysis (abstract). *J Am Soc Nephrol.* 5(3):4, 1994.
  - 59 Tetsuka T, Ando Y, Ono S, Asano Y. Change in inferior vena caval diameter detected by ultrasonography during and after hemodialysis. *ASAIO J.* 41(1):105–10, 1995.
  - 60 Ando Y, Yanagiba S, Asano Y. The inferior vena cava diam-

- eter as a marker of dry weight in chronic hemodialyzed patients. *Artif Organs*. 19(12):1237–42, 1995.
- 61 Mandelbaum A, Ritz E. Vena cava diameter measurement for estimation of dry weight in haemodialysis patients. *Nephrol Dial Transplant*. 11(suppl 2):24–7, 1996.
  - 62 Katzarski KS, Nisell J, Randmaa I, Danielsson A, Freyschuss U, Bergström J. A critical evaluation of ultrasound measurement of inferior vena cava diameter in assessing dry weight in normotensive and hypertensive hemodialysis patients. *Am J Kidney Dis*. 30(4):459–65, 1997.
  - 63 Jaeger JQ, Mehta RL. Assessment of dry weight in hemodialysis: An overview. *J Am Soc Nephrol*. 10(2):392–403, 1999.
  - 64 Rascher W, Tulassay T, Lang RE. Atrial natriuretic peptide in plasma of volume-overloaded children with chronic renal failure. *Lancet*. 2(8450):225–8, 1985.
  - 65 Kojima S, Inoue I, Hirata Y, Kimura G, Saito F, Kawano Y, Satani M, Ito K, Omae T. Plasma concentration of immunoreactive-atrial natriuretic peptide in patients on hemodialysis. *Nephron*. 46(1):45–8, 1987.
  - 66 Deray G, Maistre G, Basset JY, Cacoub P, Carayon A, Anouar M, Beaumelou A, Legrand JC, Ancrì D, Jacobs C. Plasma levels of atrial natriuretic peptide in chronically dialyzed patients. *Kidney Int*. 34(suppl 25):S86–8, 1988.
  - 67 Ando R, Matsuda O, Miyake S, Yoshiyama N. Plasma levels of human atrial natriuretic factor in patients treated by hemodialysis and continuous ambulatory peritoneal dialysis. *Nephron*. 50(3):225–8, 1988.
  - 68 Anderson U, Sylven C, Lindvall K, Theodorsson E, Noree LO. Cardiac function and cardiovascular hormone balance during hemodialysis with special reference to atrial natriuretic peptide. *Clin Nephrol*. 30(6):303–7, 1988.
  - 69 Lauster F, Gerzer R, Weil J, Fülle HJ, Schiffel H. Assessment of dry body weight in haemodialysis patients by the biochemical marker cGMP. *Nephrol Dial Transplant*. 5(5):356–61, 1990.
  - 70 Wolfram G, Sitter T, Gottsmann M, Gerzer R, Schiffel H. Assessment of dry weight in haemodialysis patients by the volume markers ANP and cGMP. *Nephrol Dial Transplant*. 11(suppl 2):28–30, 1996.
  - 71 Franz M, Pohanka E, Tribl B, Woloszczuk W, Hörl WH. Living on chronic hemodialysis between dryness and fluid overload. *Kidney Int*. 51(suppl 59):S39–42, 1997.
  - 72 Shiota J, Kubota M, Hamada C, Koide H. Plasma ANP during hemodialysis with or without fluid removal. *Nephron*. 55(3):283–6, 1990.
  - 73 Kooman JP, Kouw PM, Cheriex EC, Menheere P, Van Hoof JP, Peltenburg HG, Leunissen KML. Is c-GMP a reliable tool to assess dry weight in haemodialysis patients? *Nephrol Dial Transplant*. 6(10):803, 1991.
  - 74 Katzarski KS, Randmaa I, Danielsson A, Freyschuss U, Bergström J. Is atrial natriuretic peptide a reliable marker for “dry weight” in hemodialysis patients? *Nephrol Dial Transplant*. 7(7):681, 1992.
  - 75 Lauster F, Fülle HJ, Gerzer R, Schiffel H. The postdialytic plasma cyclic guanosine 3':5'-monophosphate level as a measure of fluid overload in chronic hemodialysis. *J Am Soc Nephrol*. 2(9):1451–4, 1992.
  - 76 Niwa T, Fujishiro T, Uema K, Tsuzuki T, Tominaga Y, Emoto Y, Miyazaki T, Maeda K. Effects of hemodialysis on plasma levels of vasoactive peptides: Endothelin, calcitonin gene-related peptide, and human natriuretic peptide. *Nephron*. 64(4):552–9, 1993.
  - 77 Heintz B, Königs F, Dakshinamurty KV, Kierdorf H, Gladziwa U, Kirsten R, Nelson K, Wieland D, Riehl J, Mann H. Response of vasoactive substances to intermittent ultrafiltration in normotensive hemodialysis patients. *Nephron*. 65(2):266–72, 1993.
  - 78 Iimura O, Tabei K, Nagashima H, Asano Y. A study of regulating factors of plasma refilling during hemodialysis. *Nephron*. 74(1):9–25, 1996.
  - 79 Ojanen S, Pietilä K, Pasternack A. Plasma atrial natriuretic peptide, body weight and twenty-four-hour blood pressure monitoring in chronic hemodialysis patients. *Nephron*. 73(2):174–8, 1996.
  - 80 Bolot JF, Bernard C, Laurent G, Robert A, Calemard E, Jenin P, Lenoir J, Thomasset A. Appréciation des mouvements liquidiens sectoriels par mesure de l'impédance globale du corps. Étude faite au cours d'hémodialyses et de traitement par diurétiques majeurs. *Nouv Presse Med*. 4(24):1787–91, 1975.
  - 81 de Vries PM, Meijer JH, Oe PL, Van Bronswijk H, Schneider H, Donker AJM. Conductivity measurements for analysis of transcellular fluid shifts during hemodialysis. *ASAIO Trans*. 33:554–6, 1987.
  - 82 de Vries PMJM. Plasma volume changes during hemodialysis. *Semin Dial*. 5(1):42–7, 1992.
  - 83 Olthof CG, de Vries PMJM, Kouw PM, Oe PL, Gerlag PGG, Schneider H, Donker AJM. The recovery of the fluid balance after haemodialysis and haemofiltration. *Clin Nephrol*. 37(3):135–9, 1992.
  - 84 Sinning WE, De Oreo PB, Morgan AL, Brister EC. Monitoring hemodialysis changes with bioimpedance: What do we really measure? *ASAIO J*. 39(3):M584–9, 1993.
  - 85 Katzarski KS, Charra B, Laurent G, Lopot F, Divino-Filho JC, Nisell J, Bergström J. Multifrequency bioimpedance in assessment of dry weight in hemodialysis. *Nephrol Dial Transplant*. 11(suppl 2):20–3, 1996.
  - 86 Zaluska WT, Schneditz D, Kaufman AM, Morris AT, Levin NW. Relative underestimation of fluid removal during hemodialysis hypotension measured by whole body impedance. *ASAIO J*. 44:823–7, 1998.
  - 87 Scharfetter H, Monif M, Laszlo Z, Lanbauer T, Hutten H, Hinghofer-Szalkay H. Effect of postural changes on the reliability of volume estimations by bioimpedance spectroscopy data. *Kidney Int*. 51(4):1078–87, 1997.
  - 88 Zhu F, Schneditz D, Levin NW. Sum of segmental bioimpedance analysis during ultrafiltration and hemodialysis reduces sensitivity to changes in body position. *Kidney Int*. 56(2):692–9, 1999.
  - 89 Spiegel DM, Bashir K, Fisch B. Bioimpedance resistance ratios for the evaluation of dry weight in hemodialysis. *Clin Nephrol*. 53(2):108–14, 2000.
  - 90 de Vries PMJM, Meijer H, Vlaanderen K, Visser V, Oe LP, Donker JM, Schneider H. Measurement of transcellular fluid shift during haemodialysis. *Med Biol Eng Comput*. 27(2):152–8, 1989.
  - 91 Stiller S, Mann H, Byrne T. Continuous monitoring of blood volume during dialysis. *Proc Eur Soc Artif Organs*. 7:167–71, 1980.

- 92 Mann H, Ernst E, Gladziwa U, Schallenberg U, Stiller S. Changes in blood volume during dialysis are dependant upon the rate and amount of ultrafiltrate. *ASAIO Trans.* 35:250–2, 1989.
- 93 Röckel A, Abdelhamid S, Fiegel P, Menth M, Walb D, Schneditz D. Characterization of “refilling types” by continuous blood volume monitoring during hemodialysis. *Kidney Int.* 43(suppl 41):S67–9, 1993.
- 94 Steuer RR, Leypoldt JK, Cheung AK, Harris DH, Conis JM. Hematocrit as an indicator of blood volume and a predictor of intradialytic morbid events. *ASAIO J.* 40:M692–6, 1994.
- 95 Ishihara T, Igarashi I, Kitano T, Shinzato T, Maeda K. Continuous hematocrit monitoring method in an extracorporeal circulation system and its application for automatic control of blood volume during artificial kidney treatment. *Artif Organs.* 17(8):708–16, 1993.
- 96 Santoro A, Mancini E, Paolini F, Spongano M, Zucchelli P. Automatic control of blood volume trends during hemodialysis. *ASAIO J.* 40(3):M419–22, 1994.
- 97 Wizemann V, Leibinger A, Mueller K, Nilson A. Influence of hydration state on plasma volume changes during ultrafiltration. *Artif Organs.* 19(5):416–19, 1995.
- 98 Wilkie ME, Lindley EJ, Edwards L, Roebuck AF, Warwick G, Brown CB. Improved ultrafiltration control using an online blood volume monitor (abstract). *Nephrol Dial Transplant.* 11(6):A202, 1996.
- 99 Lopot F, Koryk P, Blaha J, Forejt J. Use of continuous blood volume monitoring to detect inadequately high dry weight. *Int J Artif Organs.* 19:411–14, 1996.
- 100 Steuer RR, Germain M, Leypoldt JK, Cheung AK. Enhanced fluid removal guided by blood volume monitoring during chronic hemodialysis (abstract). *ASAIO J.* 42(2):83, 1996.
- 101 Krepel HP, Nette RW, Akcahüseyin E, Zietse R. Variability of relative blood volume during haemodialysis. *Nephrol Dial Transplant.* 15(5):673–9, 2000.
- 102 Leypoldt JK, Cheung AK. Evaluating volume status in hemodialysis patients. *Adv Ren Replace Ther.* 5(1):64–74, 1998.
- 103 Wann GL, Tsai CS, Lin SH, Huang WD, Chu P, Chen GS, Lin YF. Prediction of dry weight through changes in blood volume and plasma cyclic 3',5'-guanosine monophosphate in patients under maintenance hemodialysis. *ASAIO J.* 44(5):M569–73, 1998.
- 104 Bogaard HJ, de Vries JPPM, de Vries PMJM. Assessment of refill and hypovolemia by continuous surveillance of blood volume end extracellular fluid. *Nephrol Dial Transplant.* 9(9):1283–7, 1994.
- 105 Conlon PJ, Walshe JJ, Heinle SK, Minda S, Krucoff M, Schwab SJ. Predialysis systolic blood pressure correlates strongly with mean 24-hour systolic blood pressure and left ventricular mass in stable hemodialysis patients. *J Am Soc Nephrol.* 7(12):2658–63, 1996.
- 106 Mitra S, Chandna S, Farrington K. What is hypertension in chronic hemodialysis? The role of interdialytic blood pressure monitoring. *Nephrol Dial Transplant.* 14(12):2915–21, 1999.
- 107 Schmieder RE. Dietary intake and left ventricular hypertrophy. *Nephrol Dial Transplant.* 12(2):245–8, 1997.
- 108 Özkahya M, Ok E, Cirit M, Akcicek E, Basci A, Dorhout Mees EJ. Regression of left ventricular hypertrophy in hemodialysis patients by ultrafiltration and reduced salt intake without antihypertensive drugs. *Nephrol Dial Transplant.* 13(6):1489–93, 1998.
- 109 Harnett JD, Parfrey PS. The management of congestive heart failure in uremic patients. In: Parfrey PS, Harnett JD, eds. *Cardiac Dysfunction in Chronic Uremia.* Dordrecht, Netherlands: Kluwer Academic Publishers, 1992; 221–46.
- 110 Töz H, Özerkan F, Ünsal A, Soydas C, Dorhout Mees EJ. Dilated uremic cardiomyopathy in a dialysis patient cured by persistent ultrafiltration. *Am J Kidney Dis.* 32(4):664–8, 1998.
- 111 Chazot C, Charra B, Vo Van C, Jean G, Vanel T, Caemard E, Terrat JC, Ruffet M, Laurent G. The Janus-faced aspect of “dry weight”. *Nephrol Dial Transplant.* 14(1):121–4, 1999.
- 112 Ortega Marcos O, Rodriguez I, Gallar P, Oliet A, Vigil A. Importance of dry weight assessment in well-being, appetite, nutritional status, and anemia correction in haemodialysis patients (letter). *Nephrol Dial Transplant.* 13(9):242, 1998.