Impaired sympathetic response before intradialytic hypotension: a study based on spectral analysis of heart rate and pressure variability

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ABSTRACT

The purpose of this study was to evaluate the autonomic response to standard haemodialysis and the changes associated with the onset of intradialytic hypotension in 12 normotensive patients with uraemia. Power spectra of R–R interval and of blood pressure fluctuations were obtained during a standard dialysis session and estimated in the low-frequency (LF, 30–150 mHz) and high-frequency (HF, 150–400 mHz) range. The absolute power of the LF component of blood pressure variations and the LF/HF ratio of R–R interval were assumed as indexes of sympathetic activity. Standard haemodialysis induced hypotension in six patients (unstable) while a minor pressure decline was present in the other six (stable). Normalized blood volume before dialysis and percentage volume reduction were similar in the two groups. Tachycardia in response to pressure and volume decrease was more pronounced in stable than in unstable patients, as evidenced by a higher slope of the relation between R–R interval and systolic blood pressure (7.9 versus 0.9 ms/mmHg, \( P < 0.01 \)). Sympathetic tone was enhanced during early dialysis in all patients (\(+2.1\) for R–R LF/HF ratio, \(+2.4\pm0.6\) mmHg\(^2\) and \(+7.2\pm2.0\) mmHg\(^2\) for absolute LF power of diastolic and of systolic blood pressure respectively, \( P < 0.05 \)), compared with baseline predialysis values. During late dialysis, unstable patients showed an impairment of sympathetic activation which preceded hypotension and was maximal during the crisis (\(-2.9\pm1.4\) for R–R LF/HF ratio, \(-2.7\pm1.4\) mmHg\(^2\) and \(-8.6\pm4.0\) mmHg\(^2\) for absolute LF power of diastolic and of systolic blood pressure respectively, \( P < 0.05 \)). On the contrary, stable patients showed constantly elevated indexes (\(+3.7\pm1.4\) for R–R LF/HF ratio, \(+5.9\pm2.7\) mmHg\(^2\) and \(+13.3\pm6.2\) mmHg\(^2\) for LF of diastolic and of systolic blood pressure, \( P < 0.05 \)). Values returned to predialysis levels after the end of the dialysis session in all patients. We conclude that standard haemodialysis activates a marked and reversible sympathetic response in both stable and unstable uraemic patients. However, in unstable patients, such activation is impaired in late dialysis, therefore contributing to the onset of the hypotensive crisis.

Key words: intradialytic hypotension, spectral analysis, sympathetic activity.
Abbreviations: HF, high frequency; LF, low frequency.
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INTRODUCTION

In spite of technological progress, intradialytic hypotension remains the most common acute complication of haemodialysis [1].

The removal of body fluid through blood ultrafiltration causes a relative hypovolaemia and a decrease in venous return leading to a reduction in stroke volume. The unloading or inhibition of central volume and arterial pressure receptors is an appropriate stimulus for sympathetic activation, which tends to preserve blood pressure by peripheral vasoconstriction and heart rate acceleration [1–4].

Although several treatment-specific factors such as dialysate composition, dialysate temperature and ultrafiltration rate might interfere with cardiovascular adjustments [1,2,5], intradialytic hypotension can be broadly viewed as the result of the imbalance between the degree of central hypovolaemia and the adequacy of sympathetic mediated haemodynamic response. The imbalance may result from excessive hypovolaemia, or an impaired autonomic response, or both [1,2,4,5].

The role of a dysfunction of the autonomic nervous system in the genesis of intradialytic hypotension is controversial, with some studies claiming [6–8], and others denying [9–13], its importance.

A limitation common to several previous studies is that the standard tests used for exploring autonomic function were not suitable for monitoring the rapid changes of the autonomic state throughout the dialysis session. Spectral analysis of heart rate and blood pressure variability has been proven to be a useful non-invasive tool for monitoring the variations of sympatho-vagal balance controlling heart rate and vasomotor tone [14]. The spectrum of oscillations of physiological signals contains two main components, centred in the low-frequency (LF) domain ranging from 30 to 150 mHz and in the high-frequency (HF) domain, at about 250 mHz. Since the HF component (respiratory related) of heart rate is of purely vagal origin, the ratio LF/HF provides an indirect estimate of the sympatho-vagal balance, whereas the LF component of blood pressure oscillations is thought to be related to the sympathetic control of vasomotion [15].

In this study we investigated the autonomic regulation of the haemodynamic response to standard haemodialysis in patients with chronic uraemia. For this purpose a non-invasive continuous monitoring of both ECG and peripheral blood pressure with spectral analysis of their oscillations was carried out. Haemodynamic changes during dialysis treatment were related to the corresponding variations of the spectral indexes of the autonomic state, especially to those preceding and/or associated with the intradialytic hypotensive crisis.

METHODS

Study population

Twelve normotensive uraemic patients, aged 48–78 years (mean 60 ± 10 years), maintained on three weekly standard dialysis sessions were studied. Blood pressure measured before each dialysis session was less than 140/90 mmHg. Patients were on no hypotensive medication for at least 1 month before the study. End-stage renal failure was due to chronic glomerulonephritis (n = 3 patients), interstitial nephritis (n = 3) and polycystic kidney disease (n = 6). All of them were virtually anuric, none passing more than 100 ml of urine per day. Dialysis treatment duration ranged from 24 to 120 months.

Patients included in the study were free from heart disease, arrhythmia, diabetes mellitus and symptomatic peripheral neuropathy. Six of them had not experienced any episode of intradialytic hypotension during the month preceding the study, the other six (hypotension prone) had manifested intradialytic hypotension in at least 2 out of 3 dialysis treatments in the week before the study. Patients were studied during the midweek session, at the same time of day (14:00 to 19:00 hours). All medications were discontinued at least 1 week before the study. The details of the study were accurately explained to the patients, who gave their informed consent. The study protocol was carried out in accordance with the Declaration of Helsinki of the World Medical Association (1989) and approved by the Institutional Ethics Committee.

Study protocol

Patients were studied while lying in bed in a quiet, air-conditioned room at constant temperature (25 °C). Signal recording was started after 10–30 min of supine rest. The ECG was continuously monitored from standard lead II. Arterial pressure was monitored non-invasively from the middle finger of the hand opposite the fistula using a digital plethysmographic device (Finapres 2300, Ohmeda, Italy). The cuffed finger was kept at heart level. During dialysis, blood pressure recordings were generally discontinued for 10 to 15 min every 30 to 40 min of monitoring; in four cases, however, a continuous pressure recording during dialysis was obtained by using alternately two fingers of the same hand with a comparable pressure signal on Finapres. Recording duration was 240 ± 13 min.

For the purpose of this study dialysis treatments were carried out according to the usual schedule, including target body weight, which was estimated from clinically based standard criteria. Dialysate contained 35 mmol/l bicarbonate, 135 mmol/l sodium, 1.5 mmol/l potassium and 1.5 mmol/l calcium and was kept at a mean temperature of 37 ± 0.5 °C. Mean ultrafiltration rate was 0.9 ± 0.05 litres/h and treatment was maintained for
Sympathetic impairment in intradialytic hypotension

The time series of beat-to-beat R–R interval, systolic blood pressure (SBP) and diastolic blood pressure (DBP) are shown in the upper panel. Hypotensive crisis (arrow) is characterized by a marked pressure decline associated with heart rate decrease (R–R interval increases). The corresponding spectra of consecutive intervals of 256 data points of the time series of R–R interval and systolic blood pressure are shown in the bottom panel (arrow at hypotension).

Power spectrum analysis
The analogue ECG and blood pressure signals were digitized on a personal computer at 250 samples per
second with 12 bit per sample precision, stored in a binary format (2 bytes per sample). The ECG was processed by using extensively tested algorithms [16] in order to detect the QRS complex and the R wave reference point by a derivative/amplitude criterion, without interpolation of the original signal. Using the R wave reference point, the blood pressure signal was also processed by an appropriate algorithm in order to detect diastolic and systolic blood pressure values.

To obtain a spectral representation of the R–R interval, and of diastolic and systolic blood pressure variability, the autoregressive technique was found to be appropriate, because of the non-deterministic behaviour of the time series [17]. The time series were analysed in consecutive intervals of 256 data points. The intervals were processed by the Levinson–Durbin recursive algorithm [18] to generate the autoregressive coefficients: the number of coefficients for the analysis was set to 12 on the basis of previous studies [17]. The goodness of the model was tested by evaluating the normality of the distribution of the resulting white noise: the series without normal white noise distribution (because of either artefacts or non-stationarities) were discarded. For each 256-data-point interval, the power spectral function was evaluated, and the most significant spectral components were extracted according to a spectral decomposition algorithm [19]. Two major frequency components were considered in the R–R power spectrum: a low-frequency (LF) component (30–150 mHz), thought to be related to the sympatho-vagal baroreflex control of arterial pressure and to sympathetic activity, and a high-frequency (HF) component (150–400 mHz), ascribed to the vagally mediated respiratory sinus arrhythmia for R–R interval. For blood pressure power spectrum the LF component was considered, which is thought to be related to the adrenergic control of vasomotion (Meyer’s waves) [15].

For each spectrum the mean R–R interval, the mean of diastolic and systolic blood pressure values, and the spectral indexes – i.e. the total spectrum power, the absolute values of the power and the frequency of each component and the LF/HF ratio of R–R interval – were stored for statistical analysis.

The average values of R–R interval, pressure and relative spectral parameters were obtained from at least 15 min of recording in each patient, starting 30 min before dialysis until 15 min after the end of dialysis. Figure 1 shows an example of the original computer output (ECG and blood pressure time series and corresponding spectra) of the entire monitoring period in an unstable patient experiencing a hypotensive episode.

**Statistical analysis**

Data are expressed as means ± S.E.M. Comparisons of both haemodynamic and spectral parameters were performed by ANOVA using Sheffé F-test of significance; $P < 0.05$ was considered to be significant. Linear regression was also performed to correlate data.

**RESULTS**

All six hypotension-prone patients experienced one episode of hypotension during the study (unstable patients), while only a minor pressure decrease at the end of dialysis was observed in the other six patients who had not manifested hypotension during the last month (stable patients): the episode initiated 180–200 min after the start of dialysis and was monitored for no longer than 3 min, provided that blood pressure did not fall below 60/40 mmHg, before specific interventions were made. These consisted of arresting ultrafiltration and infusing saline until symptoms disappeared and blood pressure was restored to the pre-crisis values.

Stable and unstable uraemic patients did not differ significantly in terms of age, sex, time of onset of uraemia, dialysis duration or body weight.

Predialysis and end-dialysis haemodynamic and corresponding spectral indexes are summarized in Table 1.

None of the predialysis haemodynamic parameters was significantly different between stable and unstable patients. Predialysis blood volume normalized for patient height was $3.12 ± 0.23$ litres/m in the unstable patients and $3.57 ± 0.15$ litres/m in the stable patients, a difference which does not reach statistical significance. The absolute amount of volume removal during dialysis was lower in unstable compared with stable patients ($0.29 ± 0.07$ litres/m versus $0.59 ± 0.03$ litres/m, $P < 0.05$), but percentage blood volume reduction at the onset of hypotension was comparable in the two groups ($86 ± 2\%$ versus $84 ± 2\%$, $P$ not significant).

During dialysis, patients displayed a tachycardic response which was significant in stable patients only and reached its peak at the lowest blood pressure. In contrast, no significant change in R–R interval was present during intradialytic hypotension in the unstable patients, although heart rate tended to decrease. The heart rate response to intradialytic pressure changes was evaluated by relating the R–R interval duration to the corresponding systolic blood pressure changes of at least six intradialytic periods of approximately 15 min duration. Stable patients showed a direct and significant linear relation between the changes in R–R interval duration and systolic pressure, whereas unstable patients displayed either no relation ($n = 3$) or a poor relation ($n = 3$), as shown in Figure 2. On average, the two groups differed for regression significance ($R = 0.92 ± 0.02$ in the stable group and $R = 0.59 ± 0.07$ in the unstable group) and for the slopes of linear regression ($7.9 ± 1.3$ ms/mmHg in stable and $0.9 ± 0.1$ ms/mmHg in unstable patients, $P < 0.01$) (Figure 3).
Table 1  Haemodynamic and spectral parameters in stable and unstable uraemic patients during predialysis and at maximal blood pressure (BP) decrease

Values are means ± S.E.M. Abbreviations: DBP, diastolic blood pressure; SBP, systolic blood pressure. *P < 0.05 compared with predialysis values. §P < 0.05 compared with stable patients at maximal BP decrease (ANOVA, Sheffe F-test).

<table>
<thead>
<tr>
<th></th>
<th>Stable patients (n = 6)</th>
<th>Unstable patients (n = 6)</th>
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<tbody>
<tr>
<td></td>
<td>Predialysis</td>
<td>Max BP decrease</td>
</tr>
<tr>
<td>Normalized blood volume (litres/m)</td>
<td>3.6 ± 0.1</td>
<td>3.0 ± 0.1</td>
</tr>
<tr>
<td>R–R interval (ms)</td>
<td>805 ± 38</td>
<td>639 ± 35*</td>
</tr>
<tr>
<td>R–R total power (ms²)</td>
<td>297 ± 111</td>
<td>204 ± 151</td>
</tr>
<tr>
<td>R–R LF power (ms²)</td>
<td>150 ± 60</td>
<td>187 ± 124</td>
</tr>
<tr>
<td>R–R HF power (ms²)</td>
<td>112 ± 63</td>
<td>32 ± 16</td>
</tr>
<tr>
<td>R–R LF/HF ratio</td>
<td>4.3 ± 2.6</td>
<td>8.0 ± 3.5</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>67 ± 3</td>
<td>65 ± 4</td>
</tr>
<tr>
<td>DBP total power (mmHg²)</td>
<td>3.1 ± 1.0</td>
<td>11.6 ± 4.0*</td>
</tr>
<tr>
<td>DBP LF power (mmHg²)</td>
<td>1.6 ± 0.4</td>
<td>7.5 ± 2.7*</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>125 ± 6</td>
<td>97 ± 4*</td>
</tr>
<tr>
<td>SBP total power (mmHg²)</td>
<td>12 ± 3</td>
<td>29 ± 7*</td>
</tr>
<tr>
<td>SBP LF power (mmHg²)</td>
<td>4.4 ± 0.8</td>
<td>18 ± 6*</td>
</tr>
</tbody>
</table>

Figure 2  R–R interval response to intradialytic systolic blood pressure (SBP) changes in one stable (●) and one unstable patient (□).

A highly significant and linear relation is evident in the stable patient, indicating an intact and constant tachycardic response to volume and pressure decline; in the unstable patient the linear relation is lost at end-dialysis, with no change in heart rate when SBP drops below 100 mmHg (deficient baroreflex sensitivity).

Figure 3  Relation between R–R interval and systolic blood pressure (SBP) changes during dialysis, expressed as differences relative to basal.

A lower regression R and a markedly lower slope is present in the unstable (□) compared with stable patients (●), indicating a reduced baroreflex function.

The sequence of changes of spectral indexes of sympathetic activity (average of three spectra, 15 min period), and of the corresponding haemodynamic parameters recorded during the dialysis session, is illustrated in Figure 4. The periods shown are basal predialysis, early dialysis (40 ± 12 min of dialysis treatment), late dialysis (140 ± 18 min of treatment), lowest blood pressure recorded (which coincided in all but one patient with end-dialysis, at 200 ± 6 min of treatment) and post dialysis (immediately after extracorporeal blood resti-
systolic blood pressure respectively, compared with baseline predialysis values).

After the end of the dialysis session, spectral indexes of sympathetic activation were restored to predialysis levels. In stable patients, who displayed only minor changes in blood pressure and heart rate, sympathetic activation disappeared. In unstable patients, spectral indexes increased to predialysis levels together with the partial recovery of heart rate and blood pressure.

Table 2 summarizes the changes in haemodynamic and spectral parameters in stable and unstable patients. At the lowest blood pressure value recorded during dialysis, the LF/HF ratio of R–R interval and the absolute power of the LF component of systolic and diastolic pressure fluctuations increased in stable patients and decreased in unstable patients ($P < 0.05$).

A significant correlation was found between the maximal diastolic blood pressure decrease observed...
Table 2 Changes in haemodynamic and autonomic parameters at maximal blood pressure (BP) decrease (absolute differences relative to predialysis)

Values are means ± S.E.M. Abbreviations: DBP, diastolic blood pressure; SBP, systolic blood pressure. ＊P < 0.05 compared with unstable patients (ANOVA, Sheffe F-test)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Stable patients (n = 6)</th>
<th>Unstable patients (n = 6)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normalized blood volume (litres/m)</td>
<td>–0.6 ± 0.03*</td>
<td>–0.4 ± 0.07</td>
</tr>
<tr>
<td>R–R interval (ms)</td>
<td>–140 ± 60*</td>
<td>–60 ± 36</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>–2 ± 3*</td>
<td>–28 ± 4</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>–25 ± 6*</td>
<td>–50 ± 13</td>
</tr>
<tr>
<td>R–R LF power (ms²)</td>
<td>+37 ± 90</td>
<td>–56 ± 26</td>
</tr>
<tr>
<td>R–R HF power (ms²)</td>
<td>–80 ± 68</td>
<td>+10 ± 6</td>
</tr>
<tr>
<td>R–R LF/HF ratio</td>
<td>+2.7 ± 1.4*</td>
<td>–2.9 ± 1.4</td>
</tr>
<tr>
<td>DBP LF power (mmHg²)</td>
<td>+5.9 ± 2.7*</td>
<td>–2.7 ± 1.4</td>
</tr>
<tr>
<td>SBP LF power (mmHg²)</td>
<td>+13.3 ± 6.2*</td>
<td>–8.6 ± 4.0</td>
</tr>
</tbody>
</table>

Figure 5 Relations between the maximal diastolic blood pressure decrease (ΔDBP) observed during dialysis in all patients studied and the change in spectral indexes of sympathetic activation (ΔLF/HF ratio of R–R interval and ΔLF power of diastolic blood pressure oscillations) measured on the same time interval during dialysis in all patients and the changes in spectral indexes of sympathetic activation (ΔLF/HF ratio of R–R interval and ΔLF power of diastolic pressure oscillations) measured at that time. Data suggest that the extent of blood pressure decrease is directly related to the severity of sympathetic impairment at end-dialysis (Figure 5).

DISCUSSION

Haemodynamic and autonomic changes during standard haemodialysis

The standard haemodialysis procedure associated with ultrafiltration is a potent stimulus for the sympathetic system, which can be activated by central hypovolaemia through the unloading of cardiopulmonary receptors [3,20]. The increase in heart rate in response to autonomic tests exploring baroreflex sensitivity is blunted in patients with uraemia, although the response to tests exploring the efferent sympathetic pathway such as the cold pressor test is normal [9]. A defect of the afferent limb of the baroreceptor reflex arc has been hypothesized [21,22], although its role in the pathogenesis of dialysis hypotension is still questionable [23].

Volume removal and blood pressure decline elicit tachycardia both in stable and unstable patients; in the latter, however, heart rate increases less during dialysis and it even decreases before hypotension, suggesting that baroreflex sensitivity might be deficient. The impaired tachycardic response during dialysis in the unstable patients suggests a deranged function of pressure and/or volume receptor afferent pathways of the baroreceptor reflex, which can contribute to the occurrence of the hypotensive episode.

Even a small volume removal at the beginning of dialysis elicits a marked sympathetic response in all patients: blood pressure variability increases, due to an increase in the LF component of pressure oscillations, and the LF/HF ratio of R–R interval variability also increases, due to a decrease in HF and to the increase in the LF component. In late dialysis, sympathetic activation drops in hypotension-prone patients, whereas stable patients maintain a sympathetic tone higher than basal. Since sympathetic activation increases in stable as
well as in unstable patients within the first hour of dialysis and is restored to predialysis levels at the end of treatment, it is unlikely that sympathetic withdrawal before and during the hypotensive crisis is due to a dysfunction of the efferent sympathetic pathway, as hypothesized by others [8,10,24]. A marked and sustained hypovolaemia can lead to the withdrawal of the sympathetic reflex response even in healthy subjects [25].

Moreover, a brief sympathetic stimulus, such as a short cold stress, well tolerated in normal conditions, can trigger a subsequent acute sympatho-inhibition when applied during reduced cardiac output both in normal subjects and during dialysis [26]. Using a different technique, namely the direct measurement of sympathetic nerve discharge, Converse et al. [23] found that the hypotensive crisis is caused by an acute, paradoxical withdrawal of the sympathetic vasoconstrictor drive elicited by the dialysis treatment.

Therefore, the hypotensive episode could be the consequence of the withdrawal of a normal sympathetic response to volume removal in patients who undergo dialysis treatment with a relatively low predialysis total blood volume and with a reduced baroreflex sensitivity.

Some authors report that dialysis restores to normal an altered autonomic function at predialysis [27–29]. Weise et al. [27] performed spectral analysis in stable uraemic patients and in normal subjects and found that the overall variability of heart rate and blood pressure was reduced before dialysis and restored to normal when overhydration was corrected by treatment. On the contrary, we found that spectral indexes are normal in stable patients before dialysis and variability increases at end-dialysis as a consequence of the sustained but reversible sympathetic activation. These discrepancies can be accounted for by the older age of the patients we examined as well as by the different range that we used for the estimate of LF band; moreover, spectral analysis limited to pre- and post-dialysis periods, as performed in the above studies, may fail to capture the dynamic changes of autonomic activity occurring during the procedure.

Recently, Cavalcanti et al. [30] performed spectral analysis of R–R variations during dialysis in patients who did not experience hypotension but had a previous history of haemodynamic instability. At variance with our results – which demonstrate an acute impairment of an initially intact sympathetic response to dialysis in the unstable patients – these authors observed a systematically lower LF/HF ratio during dialysis in the hypotension-prone patients and concluded that they have a chronically reduced efficiency of the autonomic system. There are major differences between our study and theirs: they did not monitor the hypotensive episode, their unstable patients are much older than the stable ones, a factor which per se affects variability, they used a higher threshold (60 mHz) for the lower range of the LF component [31], possibly underestimating the absolute value of LF component and the LF/HF ratio and, especially, they did not evaluate the LF component of blood pressure oscillations, directly related to sympathetic control of vasomotion. In fact, LF/HF ratio of R–R interval variations should not be taken as the only index of sympathetic activity: as recently stated, both autonomic impairment as well as a saturating level of sympathetic input can lead to a reduced heart rate variability [31].

On the other hand, our results fully agree with a recent report [32] on heart rate variability during dialysis hypotension: the LF/HF ratio of R–R interval variations increases before hypotension, in the compensated phase, and it suddenly decreases below predialysis values during hypotension, due to a marked decrease of the LF and to an increase in the HF component of R–R variations. Although we could not document a vagal activation associated with sympathetic inhibition in all patients, the time course of the spectral indexes derived from both heart rate and blood pressure oscillations is very similar to that reported by Barnas et al. [32].

Limitations of the study
Some limitations of this study must be acknowledged.

A relatively small number of patients was studied, due both to the selection criteria and to the difficulties of the methodology employed. Therefore, our results should not be generalized as they are not likely to include every form of intradialytic hypotensive episode. The low number of cases has reduced the statistical significance of differences in spectral parameters – which generally have a great variability – between the stable and unstable group.

Blood volume changes were not continuously monitored during dialysis in these patients, due to the difficulties of the methodology employed; preliminary results on the changes in cardiac output and in cardiac filling by Echo-Doppler monitoring during the hypotensive episode have been reported previously [5].

We did not measure plasma levels of catecholamines as an additional index of sympathetic activity because they are markedly affected by the variations in spillover, regional blood flow and clearance, which is also influenced by the dialysis treatment. Conflicting data have been reported on plasma levels of catecholamines in patients on maintenance haemodialysis [2,24] and, furthermore, plasma noradrenaline concentrations have been shown to be independent of sympathetic nerve discharge [23].

Finally, we did not perform a separate evaluation of ultrafiltration and dialysis so we cannot differentiate the autonomic changes related to blood volume from those related to increase in body temperature, decrease in osmolality, removal of circulating noradrenaline, etc. [1,2,11].
CONCLUSIONS

A sympathetic activation is elicited in early dialysis in all patients with uremia and intradialytic hypotension is preceded by and associated with the withdrawal of this response. The reduction in all spectral indexes of sympathetic activity, derived both from heart rate and blood pressure oscillations, precedes and parallels the reduction in blood pressure. These findings indicate that the sympathetic efferent pathway is intact in both stable and unstable uremic patients and that intradialytic hypotension occurs during hypovolemia as a consequence of impairment or inhibition of the autonomic response to volume removal in the presence of a reduced baroreflex function. Since baroreflex sensitivity plays an important role in buffering the changes in blood pressure during dialysis, its reduction may also contribute to the occurrence of the hypotensive episode in the unstable group. Further studies are needed to differentiate the role in buffering the changes in blood pressure during dialysis, its reduction may also contribute to the occurrence of the hypotensive episode in the unstable group. Further studies are needed to differentiate the effects of volume depletion per se from those of a loose control of sympathetic response to pressure and volume decline during standard haemodialysis treatment.

REFERENCES