Categorization of sodium sieving by 2.27% and 3.86% peritoneal equilibration tests—a comparative analysis in the clinical setting

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Abstract

Background. Analysis of the dialysate sodium concentration during a peritoneal equilibration test (PET) provides information on the rates of water and solute transport through different membrane pathways. A hypertonic (3.86%) glucose-based dialysate may enhance the accuracy of analysis. There are still gaps in our knowledge regarding this question, in the clinical setting.

Objective. The aim of this study was to compare the categorization of the sodium sieving effect in peritoneal dialysis (PD) patients by 2.27% and 3.86% PETs, and to disclose clinical correlates of this phenomenon.

Method. Ninety PD patients underwent prospectively 2.27% and 3.86% modified (dialysate samples at 0, 60, 90, 120 and 240 min) PETs, in a random order. We searched for differences in the time profiles of sodium sieving and its categorization. We correlated sodium sieving with ultrafiltration (UF) and solute transport capacity, as also with selected clinical and demographic variables, using a multivariate approach.

Results. The maximum dip in the dialysate sodium concentration **(** 11.1 mM/L, 3.86% versus 7.1 mM/L, 2.27%, $P < 0.001$) was most common after 90 min in the 3.86% PET, with the 2.27% test somewhere between 60 and 90 min. Low sodium sieving (defined by a dip <5 mM/L at 60 min) was observed in 8.9% of the patients in the 3.86% test. The same limit categorized 34.4% of the patients as low sieving in the 2.27% test (100.0% sensitivity and 72.0% specificity, using 3.86% as a reference). UF and $D/P_{240\ min}$ creatinine were independent predictors of the sodium sieving effect in both tests. Moreover, multivariate analysis disclosed a consistent inverse correlation between GFR and sodium sieving in both the 2.27% ($B = -0.23$, 95% CI −0.40, −0.07, $P = 0.006$) and 3.86% PET ($B = -0.46$, 95% CI −0.65, −0.26, $P < 0.0005$).

Conclusions. The standard 2.27% PET permits some categorization of sodium sieving in PD patients. However, the information provided by this test lacks the discriminatory capacity of the 3.86% PET, which should be considered the one for reference for this purpose. GFR keeps a

consistent inverse correlation with the intensity of sodium sieving in both the 2.27% and 3.86% PET.

Keywords

Peritoneal equilibration test, residual renal function, sodium sieving, ultrafiltration

Introduction

A significant fraction of ultrafiltration (UF) during peritoneal dialysis (PD) occurs as free water transport (FWT) through ultrasmall, solute-impermeable transcellular water channels [1,2]. Aquaporin-1 (AQP1) is the essential molecular correlate of these channels [3]. FWT increases proportionally to the osmolality of the dialysate, driven by the gradient generated by the high reflection coefficient of AQP1 to glucose. The net contribution of FWT to final UF decreases with dwell time, as the osmotic gradient is gradually dissipated by dilution and absorption [4,5]. On the other hand, a polyglucose-based dialysate brings colloid osmotic UF through small intercellular pores, but does not generate significant FWT, due to its isotonicity [6].

Sodium sieving, expressed as a decrease in the dialysate sodium concentration, is the cardinal manifestation of FWT during the early phases of glucose-based PD exchanges. As such, the variation in the dialysate sodium concentration during a peritoneal equilibration test (PET) permits an assessment of FWT and, indirectly, of the AQP1 functional status [7–10]. Use of a hypertonic (3.86%) glucose-based dialysate during a PET enhances FWT, improving potentially the characterization of this phenomenon [7–9,11,12]. Disorders of FWT may contribute to a minor, yet significant, fraction of the cases of UF failure during PD therapy [11–15].

Although the standard test is performed with a 2.27% glucose-based dialysate, this increasing support for the use of a 3.86% solution for routine PET evaluation, as the latter has been claimed to permit a more precise categorization of both the UF capacity and FWT. The compared behaviour of 1.36%, 2.27% and 3.86% PETs has been reported previously [7–9,11,12], but there are still gaps in the knowledge of factors that may influence the results of sodium sieving analysis in both tests, in the clinical setting. We have performed a prospective study with the aims of comparing the behaviour of this phenomenon in 90 PD patients who underwent 2.27% and 3.86% glucose-based PETs, as well as to disclose clinical and demographic correlates of the results of sodium sieving analysis in both tests.

Population and method

General design

Following a prospective design, 90 PD patients underwent, in a random order, two modified PETs using 2.27% and 3.86% dextrose solutions. The median delay between both tests was 4 months (range 0, 6). We compared the saturation profiles for glucose, creatinine and sodium during both tests, with a particular interest in the behaviour of the dialysate sodium concentration. We analysed the correlation between sodium sieving characteristics and selected demographics (age, gender, time on dialysis, PD modality, diabetes, ongoing drug therapy), and laboratory results [residual renal function, haemoglobin, plasma albumin, C-reactive protein (CRP)] and functional peritoneal variables (creatinine transport rates, UF).

Population

We considered for the study all the patients from our PD Unit fulfilling two conditions, namely a stable clinical condition without peritonitis or any significant intercurrence during the previous 2 months, and willingness to cooperate. On the other hand, patients suffering from peritonitis, haemoperitoneum or peritoneal catheter malfunction, undergoing catheter removal or, in general, presenting any significant clinical event during the study period were excluded from analysis.

Study protocol

All patients underwent the estimation of residual renal function (mean of urea and creatinine clearances) during the month preceding each PET. All the PET studies followed the general schedule for these tests, with small modifications. In brief, after an overnight dwell with a 2.27% dialysate, 2 L of the 2.27% or 3.86% dialysate was infused into the peritoneal cavity. Dialysate samples were collected, according to standardized procedures, after 0, 60, 90, 120 and 240 min. At the end of the 4-h dwell, complete emptying of the abdominal cavity was allowed. We retrieved blood samples for analysis after 120 min of the test.

Sample processing and secondary calculations

Plasma and dialysate sodium concentrations were estimated using an indirect ion-selective electrode method. Plasma and dialysate levels of glucose, creatinine and albumin were determined with a standard autoanalyser. Dialysate creatinine levels were corrected for simultaneous glucose levels. Residual renal function was computed as the mean of urea and creatinine clearances. Plasma CRP was estimated using a high-sensitivity immunoturbidimetric assay (Roche Diag., Mannheim, Germany).

Peritoneal transport (D/P creatinine) was categorized according to the results of the 2.27% PET. D/P for sodium and creatinine was calculated as the ratio between dialysate sodium/creatinine concentration at any point and plasma sodium/creatinine concentration in the midpoint of the test. Sodium sieving was estimated by subtracting the sodium dialysate concentration at time 0 from the sodium dialysate concentration at each sampling point. The nadir point was defined as the lowest concentration of sodium in the dialysate during the test. In the case of equal concentrations in two or more samples, the earlier one was considered the nadir point. We arbitrarily defined low sodium sieving as a fall in the dialysate sodium concentration of <5 mM/L at 60 min, in the 3.86% PET. UF during the test was estimated as the difference between the weights of the dialysate bag before infusion and after final drainage.

The main dependent variables of the study were the 60 min and maximum (nadir) dips in the dialysate sodium concentration. We compared the performance of these variables in the 2.27% and 3.86% PET. We also searched for correlations between the main variables on one side, and the demographic and clinical variables scrutinized, including the solute transport characteristics (as estimated from $D/P_{240\ min}$ creatinine) and UF during the corresponding PET. UF failure was defined by an UF < 400 mL during the 3.86% PET.

Statistics

Numerical variables are presented as mean or median values (range), as appropriate. Direct comparisons between variables were produced according to Student's *t* -test (paired and unpaired), ANOVA (numerical variables) and χ^2 distribution (categorical variables). Univariate correlation between numerical variables was calculated according to Spearman's correlation coefficient. Multivariate correlates of sodium sieving were scrutinized by multiple regression analysis. We used SPSS 15.0 for data processing.

Results

Overview

The main baseline characteristics of the study group are presented in Table 1 . Twenty-three patients (25.5%) had minor changes in the PD prescription between the first and second PET performed. The peritoneal glucose load delivered to the patients was 87.2 ± 47.0 g/day (27.2, 233.7) at the time of the 2.27% PET, and 90.4 ± 55.9 g/day (13.6, 236.0) at the time of the 3.86% PET ($P = 0.41$). Icodextrin was started in two cases, and amino acid-based solution in four cases, between the first and second PET. Two patients received low-dose prednisone therapy during the study period.

From PET results, mean D/P creatinine profiles were found to be very similar in 2.27% and 3.86% PETs (Figure 1). The sample included 17 slow (18.9%), 63 average (70.0%) and 10 fast transporters (11.1%). Mean UF values were 131.4 ± 256.7 mL (−400, 600) during the 2.27% PET and 542.0 ± 344.5 mL (−150, 1300) during the 3.86% PET. Twenty-six patients (28.9%) fulfilled the diagnostic criteria for UF failure. Mean plasma sodium levels were 138.4 ± 3.6 mM/L (131, 144) in the 2.27% PET and 137.9 ± 4.5 mM/L (129, 144) in the 3.86% PET ($P = 0.66$).

Comparison of sodium sieving

As expected, the sodium sieving effect was more pronounced during the 3.86% PET (Figure 2). Mean decrements in the dialysate sodium concentration at 60 min were 5.9 ± 3.1 mM/L (−1, 15) in the 2.27% PET, as compared with 9.1 ± 3.7 mM/L (1, 21) in the 3.86% PET ($P < 0.001$). The maximal dips in the sodium concentration were 7.1 ± 3.7 mM/L (−1, 18) and 11.1 ± 4.7 mM/L (1, 27), respectively ($P < 0.001$). The nadir point was somewhat delayed in the 3.86% PET, as compared with the 2.27% PET (Figure 3), although the difference did not reach statistical significance. More than half of the patients

showed their maximal sodium dip at 90 min, during the 3.86% PET. There was a strong correlation between the sieving effects at 60 min ($r = 0.49$, $P < 0.001$), as also between the maximal falls in the sodium concentration (Figure 4), in both PETs. This correlation standed after stratified analysis do different peritoneal transport categories, although it was relatively poor in patients with UF failure ($r = 0.32$, $P = 0.11$).

Categorization of sodium sieving defects

Eight patients (8.9%) showed a low sodium sieving effect, during the 3.86% PET, as compared with 31 patients (34.4%) in the 2.27% PET, using a dip <5 mM/L at 60 min as the limit for categorization. The latter group included the 8 patients (25.8%) with a low sieving effect, as also 18 patients (58.1%) with a sodium dip between 5 and 9 mM/L, and only 5 patients (16.1%) with a dip ≥10 mM/L, in the 3.86% test. The categorization of sodium sieving from the nadir rather than the 60 min point rescued one patient in the 3.86% PET, and five in the 2.27% PET, from the low sieving group. Remarkably, the latter five patients were those presenting a dip ≥10 mM/L at 60 min in the 3.86% test.
Table 2 provides some insight into the performance of the 2.27% PET for the diagnosis of defective sodium sieving, using three different cut-off points of sodium dip. As previously stated, in the absence of a gold standard diagnostic test for defective FWT, we arbitrarily assigned the reference status to a sodium dip <5 mM/L at 60 min during the 3.86% PET.

PET correlates of sodium sieving

All patients. The correlation between UF and $D/P_{240\ min}$ creatinine was significant, but relatively poor ($r = -0.37$, $P < 0.001$ for the 2.27% PET and $r = -0.23$, $P = 0.003$ for the 3.86% PET). On the other hand, the magnitude of sodium sieving correlated more

significantly with both UF during PET and solute transport characterization. The correlation between UF and sodium sieving was similar whether sodium dip at 60 min or nadir levels (Figure 5) were used for analysis. In contrast, D/P $_{240\ min}$ creatinine showed a better correlation with nadir levels (Figure 6) than with 60 min levels ($r = -0.34$, $P = 0.001$ for the 2.27% PET and $r = -0.20$, $P = 0.056$ for the 3.86% PET).

Multivariate analysis confirmed UF during the PET and, to a lesser extent, D/P creatinine at 240 min as independent predictors of the sodium sieving effect (Table 3).

A higher UF capacity [$r = 0.22$ ($P = 0.035$) for the 2.27% test versus $r = 0.25$ ($P = 0.019$) for the 3.86% test] and a slower solute transport category (D/P $_{240\ min}$ creatinine) [$r = -0.26$ ($P = 0.015$) for the 2.27% test versus $r = -0.37$ ($P < 0.001$) for the 3.86% test] were associated with a later appearance of the sodium nadir point in both PETs.

Patients with UF failure. In this subgroup, the correlation between nadir or 60 min levels of sodium on one side and solute transport or UF on the other was relatively poor, both in the 2.27% and the 3.86% PET. Six of the eight patients (75.0%) with a sodium dip <5 mM/L in the 3.86% test, and 17 of the 31 patients (54.8%) with the same characteristics in the 2.27% test had UF failure. Conversely, 23.1% of the patients with UF failure showed a low sodium dip at 60 min in the 3.86% PET, and 65.4% did so in the 2.27% test (Figure 7). In this subpopulation, multiple regression identified UF as a weak predictor of the maximum fall in the concentration of sodium in the dialysate in the 2.27% PET ($B = 0.005$, 95% CI 0.000, 0.010, $P = 0.04$) (D/P $_{240\ min}$ creatinine, NS), while in the 3.86% PET, neither of the variables was able to predict the fall in the sodium concentration.

Extreme solute transporters. Subanalyses of slow (n = 17) and fast (n = 10) transporters disclosed a good correlation between the sodium dip in the dialysate and UF (r = 0.56, P = 0.02 in the 3.86% PET and r = 0.46, P = 0.05 in the 2.27% PET) only in the former group, while we observed no significant correlation between UF and changes in the sodium concentration in fast transporters.

Demographic correlates of sodium sieving

Table 4 depicts univariate correlations between sodium sieving estimations and the different demographic variables scrutinized. Icodextrin therapy, daily peritoneal glucose load, residual renal function and, less consistently, age, diuresis, urinary sodium and CRP correlated with sodium sieving. When multiple regression was applied to control for the UF capacity and solute transport characteristics, only GFR stood as a consistent inverse correlate of the sodium sieving effect (Table 5).

Discussion

A PET using a 2.27% glucose dialysate is the standard approach to routine clinical evaluation of peritoneal solute transport characteristics. In the last years, a modified test using a 3.86% dialysate has gained wide acceptance, due to its improved ability to define the UF capacity of PD patients [7,9,11,12]. In fact, the most widely used criterion for the definition of UF failure is based on the latter test [8]. Occasional hyperglycaemia or haemodynamic instability in some patients represents minor arguments against the routine use of a 3.86% glucose dialysate for PET analysis.

As expected [9], the dialysate sodium concentration fell more markedly in the 3.86% than in the 2.27% PET, in our study. The nadir point tended to be later in the 3.86% than in the 2.27% test, as also in slow transporters and patients with a high UF capacity.

According to our results, the sodium sieving effect should be scrutinized at 90 min, rather than the conventional 60 min [7,10], in the 3.86% PET. Establishing the time point for a maximum dip in the dialysate sodium concentration may not be trivial, particularly in patients undergoing automated PD, as the bulk of this therapy is delivered in a short-dwell schedule. In fact, automated PD is less effective than CAPD to remove sodium, even at similar UF rates [16]. The assessment of the dialysate sodium concentration at 60, 90 and 120 min (thus covering the usual dwell time span in automated PD) may help to individualize therapy, in these patients.

The categorization of sodium sieving in clinical practice remains largely empiric, even if only data from 3.86% PET studies are considered. The correlation of this phenomenon with FWT is likely continuous [12], and no clear cut-offs for defining deficient sodium sieving have been established. This limitation is favoured by several methodologic heterogeneities. Some studies present their results as D/P sodium [8,13,17–19], while others advocate the absolute dip in the dialysate sodium concentration [9,12]. Also, some groups scrutinize the dialysate sodium concentration at 60 min [12,19], while others prefer nadir values [8,9] or a continuous variable approach [13,17]. A correction of the dialysate sodium concentration for diffusion (using mass transfer coefficient of creatinine or urate) has been recommended to define as much as possible its association with FWT [8,19,20], but the impact of this measure may be limited, at least in the first hours of the test [8,20], and other groups prefer not to apply this correction [9,12,17]. The method of estimation of sodium concentration in the dialysate may also hamper the comparisons between different studies. Mean reported values for sodium sieving show a significant variability [8,12,13,17–19]. A D/P of sodium <0.04 mM/L [8] and a maximum dip <5 mM/L [11] have been empirically proposed as limits for the definition of low sodium sieving.

In our study, the pre-selected reference value to define low sodium sieving was a dip in the dialysate sodium concentration <5 mM/L at 60 min in the 3.86% PET, and both the same and lower cut-off points were also explored for the 2.27% PET (Table 2). Categorizations did not differ markedly whether the 60 min or maximal dip was used for analysis, in either the 2.27% or 3.86% PET. Interestingly, the standard 2.27% PET appeared to be able to detect sodium sieving defects. However, the diagnostic ability of this test was seemingly poor, when compared with the 3.86% PET (Table 2). The main drawbacks of this approach were a limited specificity and a low positive predictive value, the second related to the relatively low prevalence of defective FWT, according to the 3.86% PET. It may be argued that the designation of the latter as the reference category is arbitrary and that, when applying a <5 mM/L limit for the dip in sodium concentration, it may have a low sensitivity to disclose subtle defects of FWT. This notwithstanding, the 3.86% PET is reasonably standardized and yields more discriminatory and easy-to-interpret results than the 2.27% PET. For these reasons, it should be considered the one for reference this purpose, until other approach may be found to be more accurate.

UF and solute (creatinine) transport characteristics showed an expected independent correlation with the sodium sieving effect [17,21]. The correlation was equally patent during the 2.27% and the 3.86% PET, whether the 60 min or maximum sodium dip was used for analysis (Table 3). Fast peritoneal transport rates blunt the decrease in the D/P of sodium, hampering the assessment of FWT [14]. The design of our study does not permit to disclose the role of defective FWT in the pathogenesis of UF failure, but the sodium sieving patterns varied markedly whether the 2.27% or the 3.86% PET was used for analysis, in these patients (Figure 7).

One of the most striking findings in our study was the consistent, independent, inverse correlation between GFR and sodium sieving (Table 5). This finding has not been

reported previously, to our knowledge. In a previous study, peritoneal AQP1 expression did not appear to correlate with time on PD [22], but the latter factor is not a surrogate of GFR, as shown by our own data (Table 4). Clerbaux *et al*. [18] could not disclose a correlation between sodium sieving and renal function, but the latter was estimated from creatinine clearance, and multivariate analysis was not attempted. The explanation for our finding is not clear. Patients with a lower GFR demand higher peritoneal glucose loads, to prevent volume overload. This could result in an up-regulation of peritoneal AQP1 expression [23], thus establishing an indirect inverse link between GFR and the sodium dip during the PET. However, the results of multivariate analysis were not consistent with this hypothesis, as the GFR stood as a negative correlate of the sodium dip, after controlling for the contemporaneous, daily peritoneal glucose load (Table 5). Alternatively, the renal expression of AQP1 has been reported to increase in parallel with the intensity of kidney damage [24], and a similar mechanism could affect the peritoneal membrane.

We could not find consistent correlations between sodium sieving and inflammatory markers (CRP, albumin) (Table 5), in agreement with previous reports [18]. Acute inflammation does not appear to modify the peritoneal expression of AQP1 [25,26], but the effect of chronic inflammation has not been specifically assessed so far, to our knowledge. Neither did ACEI–ARA influence sodium sieving, in our study (Tables 4 and 5). These drugs have been claimed to decrease peritoneal expression of AQP1 [27]. Previous reports indicate that they do not modify peritoneal transport in the short term [28,29], while sustained therapy may help to preserve solute transport in PD patients, but without an apparent effect on FWT [18,30].

In summary, the standard PET using a 2.27% glucose dialysate permits some categorization of defective sodium sieving in PD patients. However, the diagnostic

performance of this test is seemingly poor, when compared with the 3.86% PET, which represents a better standardized and more efficient tool, for this purpose. Besides UF and solute transport characteristics, GFR shows a consistent inverse correlation with the intensity of sodium sieving. Further studies will be necessary to better define this association.

Conflict of interest statement . None declared.

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Table 1. Study population

Age (years)	57.4 ± 14.2 (20, 84)
Gender (males/females) (%)	48/42 (53.3/46.7)
Diabetes (%)	33 (36.7)
PD modality (CAPD/automated PD) (%)	68/22 (75.6/24.4)
Time on PD (months)	20.4 ± 22.0 (2.107)
Patients with peritonitis (none/1/>1) (%)	61/15/14 (67.8/16.7/15.5)
Icodextrin for long dwell (%)	41 (45.6)
ACEI–ARA drugs (%) [a]	44 (48.9)
Calcium antagonists (%) [b]	45 (50.0)
Furosemide (%) [c]	47 (52.2)
PD system (Baxter-Deerfield, Illinois; Fresenius-Heildelberg, Germany) (%)	81/9 (90.0/10.0)
Glomerular filtration rate (mL/min)	6.1 ± 4.2 (0.16)
Diuresis (mL/day)	1117 ± 651 (0, 2500)
Urinary sodium (mM/day)	53.0 ± 54.1 (0, 272)
Albumin (g/L)	37.3 ± 3.5 (29, 46)
Haemoglobin (g/dL)	12.0 ± 1.6 (7.0, 16.7)
C-reactive protein (mg/L)	2.6 (1, 117)

[a] Enalapril and/or losartan in 40/44 cases.

[b] Included amlodipine (n = 19), nifedipine (n = 14) and diltiazem (n = 9).

[c] Mean dose 97.0 mg/day (20, 240).

Figures denote values recorded at the initiation of the study. Numerical variables presented as mean values ± SD (range), except for C-reactive protein, presented as median (range).

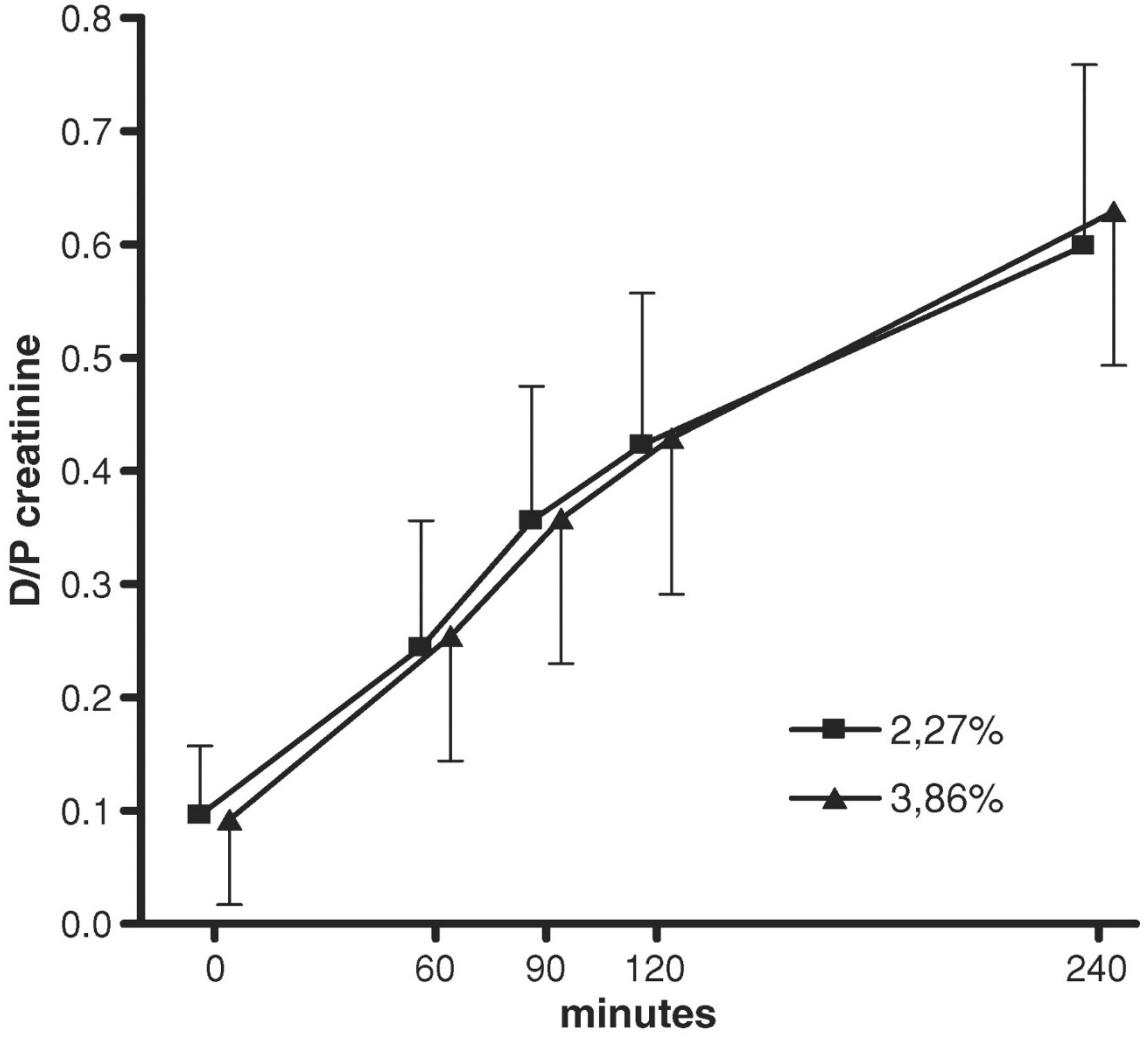

Fig. 1. Solute transport characterization (as estimated from $D/P_{240\ min}$ creatinine) in the 2.27% and 3.86% PET (differences not significant).

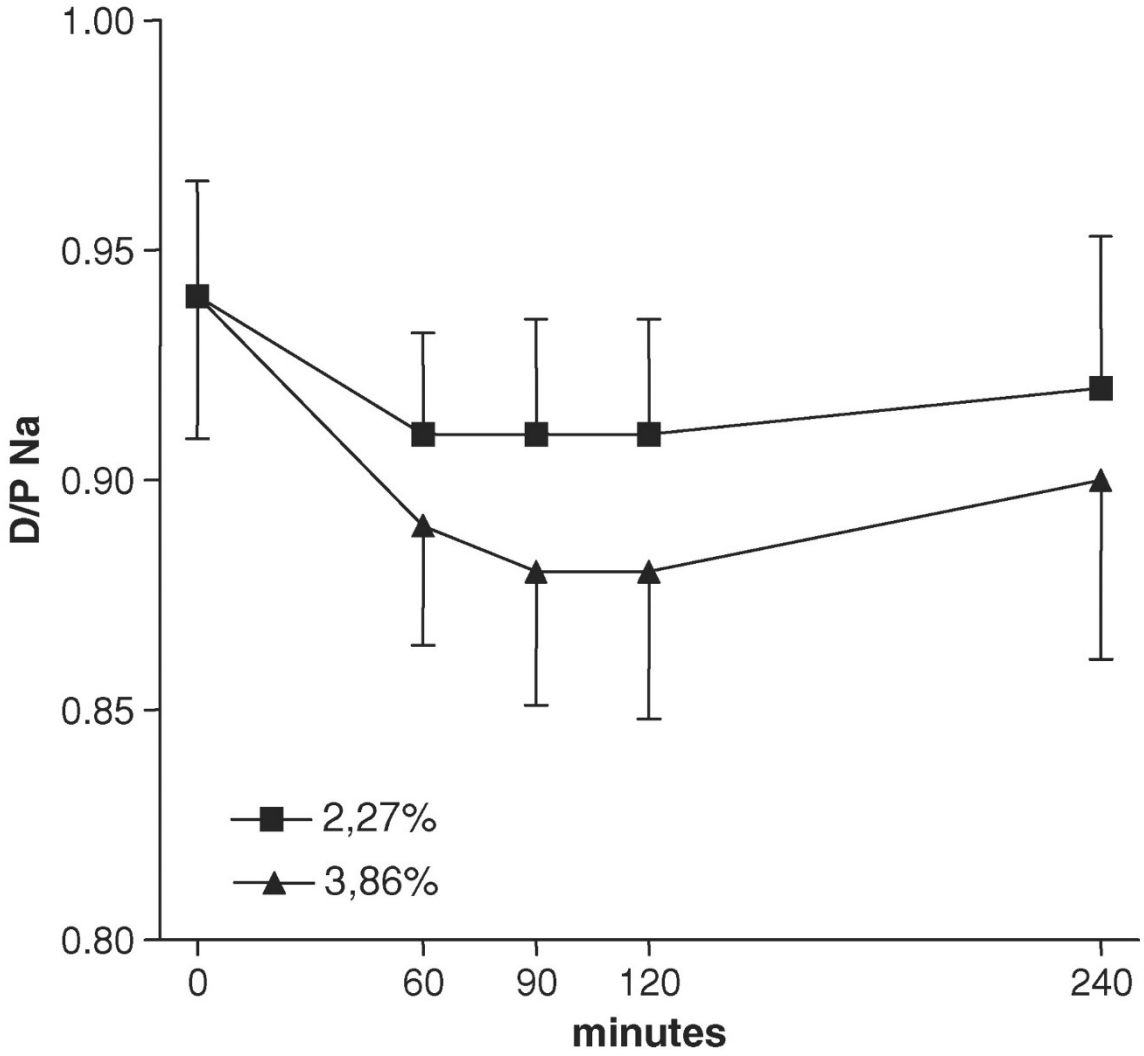

Fig. 2. D/P sodium in the 2.27% and 3.86% PET ($P < 0.001$, at any point beyond 0 min).

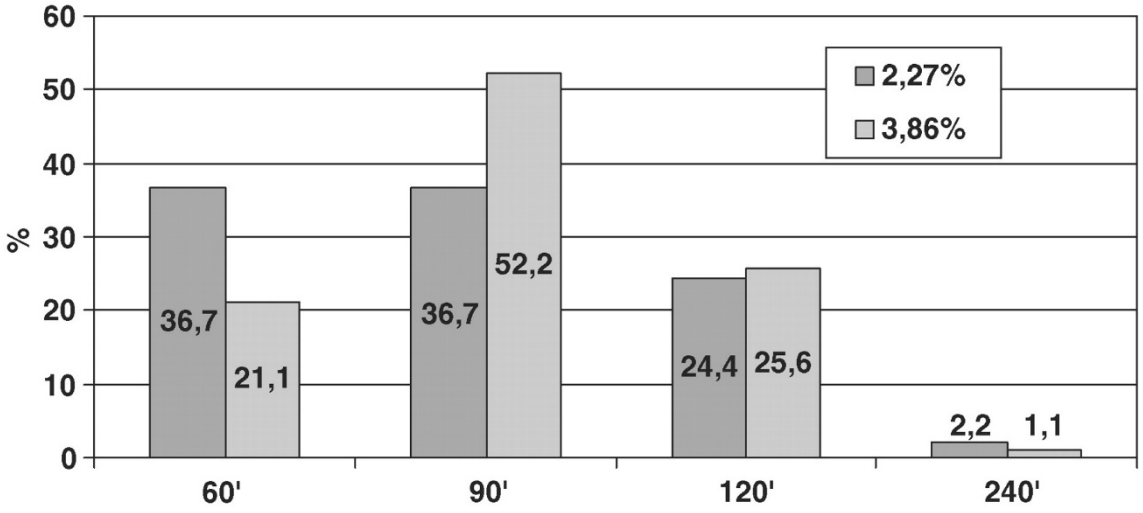

Fig. 3. Time point for sodium nadir estimation in the 2.27% and 3.86% PET ($P = 0.14$, χ^2 analysis).

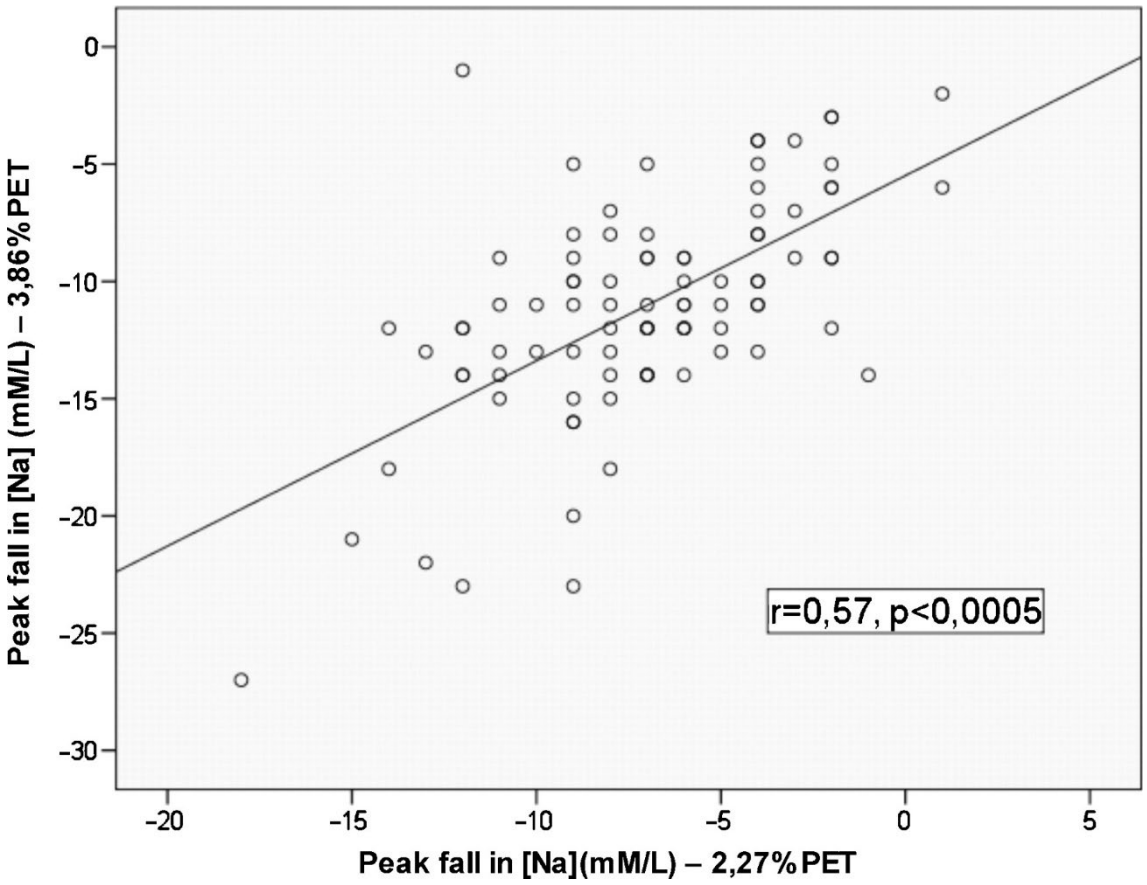

Fig. 4. Univariate correlation between sodium sieving in the 2.27% and 3.86% PET (Spearman's correlation coefficient).

Table 2. Diagnostic performance of the 2.27% PET for defective sodium sieving at different cut-off points of sodium dip

Sodium dip (mM/L)	Sensitivity	Specificity	Positive predictive value	Negative predictive value
60 min				
<3	37.5	87.9	23.1	93.5
<4	62.5	79.3	22.7	95.6
<5	100	72.0	21.9	98.3
Nadir				
<3	42.9	90.4	27.3	94.9
<4	71.4	88.0	28.6	96.1
<5	100	75.9	23.1	98.4

Figures express values in %. A sodium dip < 5 mM/L (60 min or nadir) during the 3.86% PET was used as the reference value for categorization of defective sodium sieving.

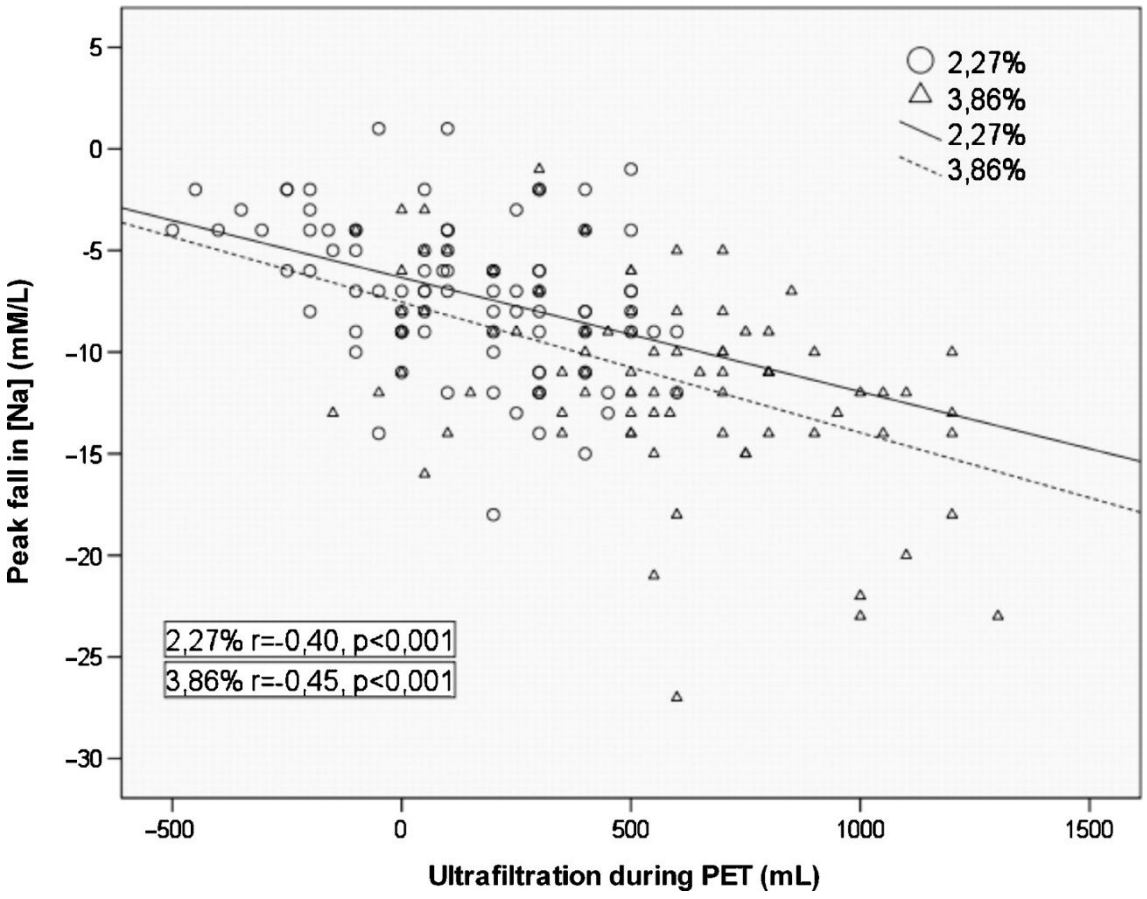

Fig. 5. Univariate correlation between sodium sieving and UF in the 2.27% and 3.86% PET (Spearman's correlation coefficient).

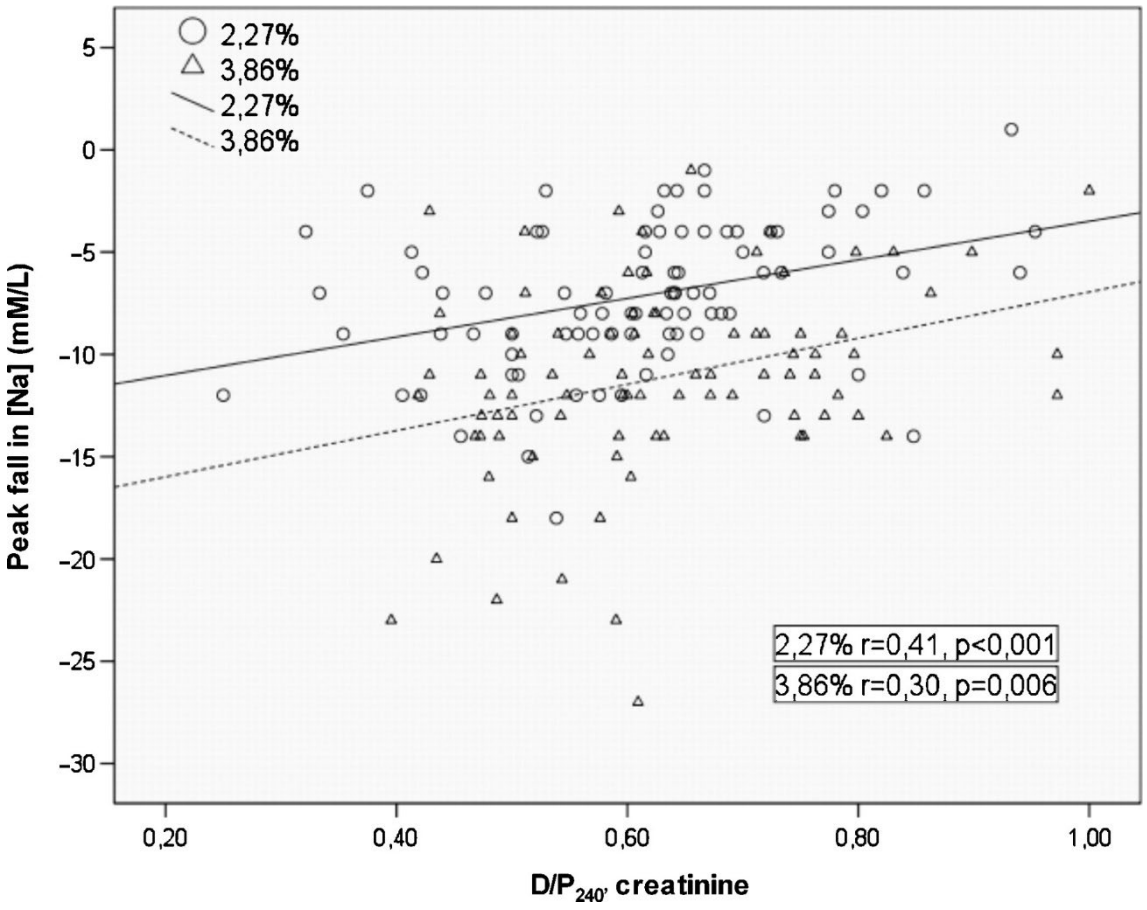

Fig. 6. Univariate correlation between sodium sieving and solute transport (as estimated from $D/P_{240\ min}$ creatinine) in the 2.27% and 3.86% PET (Spearman's correlation coefficient).

Table 3. PET correlates of sodium sieving (multivariate analysis)

Dependent variable	Covariates	*B*	95% CI of *B*	*P*
Sodium dip at 60 min	Ultrafiltration (× mL)	0.003	0.001, 0.006	0.009
2.27% PET	D/P_{240} min creatinine	−4.42	−8.90, 0.06	0.053
Maximum sodium dip	Ultrafiltration (× mL)	0.004	0.001, 0.007	0.006
2.27% PET	D/P_{240} min creatinine	−6.14	−11.34, −0.95	0.02
Sodium dip at 60 min	Ultrafiltration (× mL)	0.005	0.003, 0.007	0.0005
3.86% PET	D/P_{240} min creatinine	−3.79	−9.08, 1.49	0.16
Maximum sodium dip	Ultrafiltration (× mL)	0.006	0.003, 0.009	0.0005
3.86% PET	D/P_{240} min creatinine	−7.90	−14.54, – 1.26	0.02

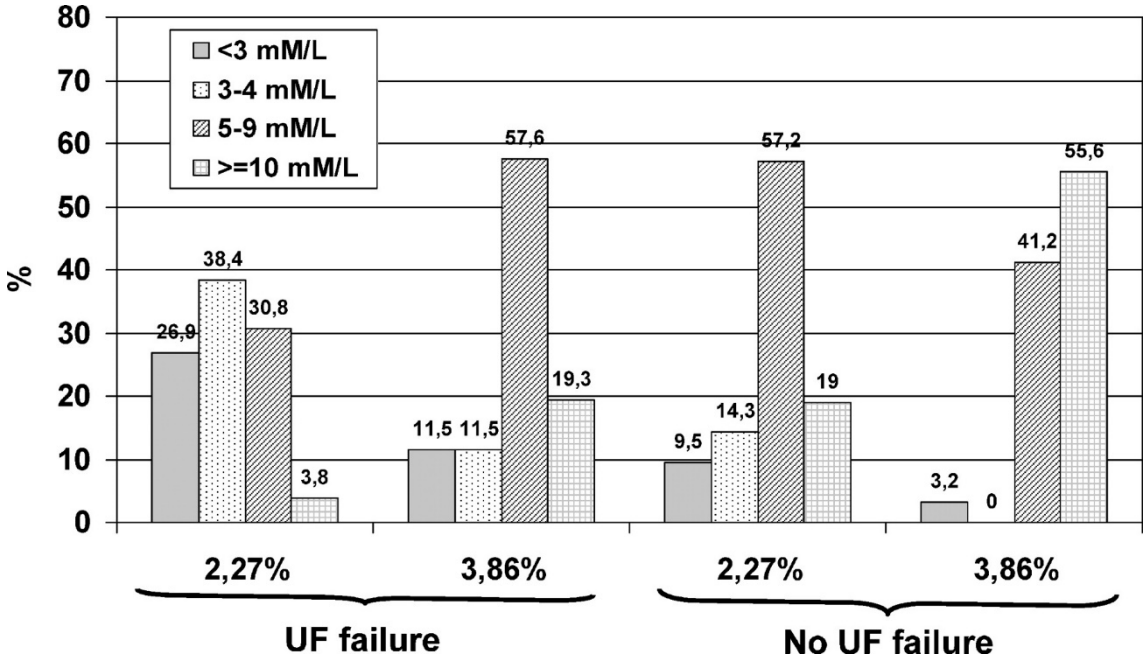

Fig. 7. Distribution of the dip in the dialysate sodium concentration at 60 min during 2.27% and 3.86% PET in patients with ($n = 26$) and without ($n = 64$) ultrafiltration (UF) failure.

Table 4. Demographic correlates of sodium sieving (univariate analysis)

	Sodium dip at 60 min 2.27% PET	Maximum sodium dip 2.27% PET	Sodium dip at 60 min 3.86% PET	Maximum sodium dip 3.86% PET
Age (years)	−0.34 (0.001)	−0.27 (0.01)	−0.20 (0.06)	−0.14 (0.18)
Gender	0.14 (0.19)	0.12 (0.25)	0.09 (0.39)	0.09 (0.42)
Diabetes	0.16 (0.13)	0.18 (0.09)	0.12 (0.28)	0.13 (0.24)
Mode of PD	0.06 (0.60)	0.01 (0.90)	0.16 (0.14)	0.10 (0.37)
Time on PD (months)	0.01 (0.90)	0.08 (0.46)	0.08 (0.45)	−0.01 (0.93)
Peritonitis (number)	0.06 (0.56)	0.08 (0.48)	0.08 (0.48)	0.07 (0.52)
ACEI–ARA therapy	0.17 (0.12)	0.14 (0.18)	0.13 (0.21)	0.07 (0.53)
Calcium antagonists	0.008 (0.94)	0.001 (0.94)	0.18 (0.09)	0.09 (0.40)
Loop diuretic therapy	−0.13 (0.20)	−0.13 (0.22)	−0.15 (0.15)	−0.11 (0.32)
Peritoneal glucose load (g/day)	0.22 (0.04)	0.23 (0.028)	0.28 (0.008)	0.29 (0.005)
Icodextrin for long dwell	−0.24 (0.025)	−0.26 (0.015)	−0.21 (0.06)	−0.24 (0.027)
Glomerular filtration rate (mL/min)	−0.23 (0.03)	−0.24 (0.025)	−0.36 (0.001)	−0.36 (0.001)
Diuresis (mL/day)	−0.06 (0.57)	−0.11 (0.29)	−0.29 (0.005)	−0.31 (0.003)
Urinary Na (mM/day)	−0.05 (0.62)	−0.11 (0.30)	−0.24 (0.023)	−0.28 (0.008)
C-reactive protein (mg/L)	−0.29 (0.009)	−0.31 (0.006)	−0.15 (0.18)	−0.17 (0.13)
Albumin (g/L)	0.11 (0.32)	0.19 (0.08)	0.18 (0.10)	0.19 (0.07)
Haemoglobin (g/dL)	0.07 (0.51)	0.06 (0.61)	−0.06 (0.58)	−0.10 (0.36)

Figures express Spearman's correlation coefficient (*P* -values in parantheses).

Table 5. Demographic and clinical correlates of sodium sieving (multivariate analysis)

Dependent variable	Covariates	*B*	95% CI of *B*	*P*
Maximum dip in sodium (mM/L) 2.27% PET	Age (years)	−0.04	−0.09, 0.008	0.09
	GFR (mL/min)	−0.23	−0.40, −0.07	0.006
	Peritoneal glucose load (g/day)	−0.003	−0.019, 0.013	0.69
	C-reactive protein (mg/dL)	−0.31	−0.70, 0.08	0.11
	Albumin (g/L)	1.49	−0.56, 3.55	0.15
	Icodextrin	0.16	−1.52, 1.84	0.88
	ACEI–ARA therapy	1.24	−0.16, 2.64	0.08
Maximum dip in sodium (mM/L) 3.86% PET	Age (years)	−0.03	−0.08, 0.01	0.16
	GFR (mL/min)	−0.46	−0.65, −0.26	0.0005
	Peritoneal glucose load (g/day)	0.003	−0.012, 0.018	0.70
	Diuresis (mL/day)	0.000	−0.002, 0.003	0.81
	Urinary sodium (mM/day)	−0.002	−0.026, 0.022	0.87
	C-reactive protein (mg/dL)	−0.37	−0.88, 0.13	0.14
	Albumin (g/L)	2.17	−0.10, 4.44	0.06
	Icodextrin	0.10	−1.95, 2.15	0.93
	ACEI–ARA therapy	1.46	−0.30, 3.22	0.10

Figures express multivariate coefficients for each variable, after controlling for D/P_{240} min creatinine, UF during the 3.86% PET and, when appropriate, other scrutinized variables.