Improvement in Sleep Apnea during Nocturnal Peritoneal Dialysis Is Associated with Reduced Airway Congestion and Better Uremic Clearance

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Background and objectives: Among peritoneal dialysis (PD) patients, nocturnal PD (NPD) is known to improve sleep apnea compared with continuous ambulatory peritoneal dialysis (CAPD), but the contributing factors are unclear.

Design, setting, participants, and measurements: Thirty-eight incident ESRD patients underwent overnight polysomnography (PSG) during NPD and CAPD. Bioelectrical impedance analysis, magnetic resonance imaging of the upper airway, and urea kinetics (Kt/V) during sleep were measured on both occasions.

Results: The prevalence of severe sleep apnea (apnea-hypopnea index, AHI ≥ 15/h) was 21.1% during NPD, and 42.1% during CAPD. Mean AHI increased from 9.6 ± 2.7/h during NPD to 21.5 ± 4.2/h during CAPD. Both obstructive and central apnea worsened after conversion to CAPD. NPD achieved greater reductions in total body water, hydration fraction, and net ultrafiltration than CAPD during sleep. Overnight peritoneal Kt/V and creatinine clearance were lower after conversion. Both peritoneal Kt/V and peritoneal creatinine clearance correlated with AHI, as did their changes after conversion. Volumetric magnetic resonance imaging revealed reduced pharyngeal volumes and cross-sectional area, and tongue enlargement after conversion.

Conclusions: Improvement in sleep apnea during NPD versus CAPD is associated with better fluid and uremic clearance and reduced upper airway congestion during sleep.


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leep apnea has been reported in up to 50% to 70% of patients with end-stage renal disease (ESRD) (1), a value at least 10 times higher than the prevalence reported in the general population (2–4). The pathogenesis of sleep apnea in patients with ESRD remains unclear. Unruh et al. (5) recently reported that patients on hemodialysis had a fourfold increase in prevalence of sleep-disordered breathing and nocturnal hypoxemia even after adjusting for cardiovascular morbidity and diabetic status, compared with participants from the Sleep Heart Health Study matched for age, gender, body mass index, and race, indicating that the pathophysiology of sleep apnea is uniquely associated with the development of chronic renal failure. Previous investigators have observed features of both central and obstructive sleep apnea (OSA) in patients with ESRD (6–10), which suggest that its pathogenesis is related both to destabilization of central respiratory control and upper airway occlusion. Moreover, nocturnal hypoxemia is a strong predictor for incident cardiovascular complications in the dialysis population (11).

Although sleep apnea is not corrected by conventional hemodialysis or peritoneal dialysis (9,10), it has been reversed both by nocturnal hemodialysis (7) and kidney transplantation (6,8). Recently, we also reported improvement of sleep apnea by nocturnal cycler-assisted peritoneal dialysis (NPD) compared with conventional continuous ambulatory peritoneal dialysis (CAPD) (12) through better fluid clearance during sleep. However, a direct proof of whether a reduction in total body water (TBW) content would alleviate airway occlusion is still lacking. Furthermore, improved fluid removal per se remains an inadequate explanation of why central sleep apnea is also improved by NPD. In this study, we hypothesized that NPD, in comparison to CAPD, promotes better fluid and solute clearance during sleep and this may be associated with improvements of both the obstructive and central components of sleep apnea, respectively.

Materials and Methods

Study Design

We performed a modified cross-over study. We made use of the fact that in Hong Kong, all incident patients who required CAPD had to undergo, after Tenckhoff catheter placement, a temporary period of mandatory cycler-assisted NPD for approximately 8 wk while awaiting their turn for CAPD training. Such a design circumvents the logistic difficulties in performing cross-over studies in PD subjects caused by the inherent complexity in establishing a patient on a particular system of NPD or CAPD, as each system has its own hardware, dialysis solution, and connective methodology, together with the possible training needed for the patient, family member, helper to use the particular system. Consenting adult subjects underwent one PSG study toward the end of their 6 to 8 wk of

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NPD treatment. They then underwent training for CAPD, and a second PSG study was performed as soon as they had been established on stable CAPD. All subjects had to have been deemed clinically euolemic, with serum sodium between 135 and 145 mmol/L on study entry and before PSG. The study protocol was reviewed and approved by the Institutional Review Board and Clinical Research Ethics Committee of the University of Hong Kong, and all patients gave written informed consent to participate in the study.

**Patients**

The PSG data in 24 incident PD subjects and body water composition data in 15 of these 24 subjects during NPD and CAPD were reported previously (12). Here, we extended these data by recruiting another 22 incident patients to undergo PSG and magnetic resonance imaging (MRI) of the upper airway during NPD and CAPD. Among them, 8 completed the first PSG and MRI study but declined to participate in the second measurement, and their data were not included in the analyses. The final number of patients with evaluable PSG data was 38, of whom 14 were new. The final numbers of patients with evaluable body water composition and MRI data were 29 and 14, respectively.

The underlying renal disease of the 38 patients (19 men; mean age, 54.2 ± 13.3 y) were diabetes mellitus in 14, IgA nephropathy in four, membranous nephropathy in one, lupus nephritis in one, focal segmental glomerulosclerosis in two, pauci-immune crescentic glomerulonephritis in two, anti-GBM disease in one, polycystic kidney disease in two, and unknown in 11. The average interval between the two sets of PSG recordings was 4.12 ± 0.63 mo, and there was no appreciable change in residual renal function, neck circumference, neckheight ratio, body mass index, and other biophysical parameters over this period (Table 1).

**PSG**

Comprehensive overnight PSG was performed in hospital with the Alice 3 or Alice 5 apparatus (Healthdyne, Atlanta, GA). All PSGs were scored manually according to standard criteria by an independent expert in sleep medicine who was unaware of the mode of dialysis (13). The average number of episodes of apnea and hypopnea per hour of sleep (apnea-hypopnea index [AHI]) was calculated as the summary measurement of sleep-disordered breathing.

Significant sleep apnea was defined as an AHI of ≥ 15 events per hour of sleep. Apnea were classified as central if there was no chest and abdominal movement, or as obstructive if chest and abdomen moved paradoxically, and as mixed if an initial absence of ventilatory effort was followed by an obstructive apnea pattern on resumption of effort. For practical purposes, all hypopneic or mixed events were classified as obstructive events.

**Dialysis Protocol**

During NPD, patients performed overnight exchanges of 10 to 12 L of peritoneal dialysis fluid (PDF) at 2.0 to 2.2 L cycle for 5 to 6 cycles by means of an automated cycler (HomeChoice, Baxter Healthcare, McGaw Park, IL). For CAPD, patients performed 3 to 4 daily exchanges of 2 L PDF using either the UltraBag (Baxter Healthcare, Guangzhou, China), StaySafe or AndyDisk (Fresenius Medical Care, Bad Homburg, Germany), or Gambrosol Trio (Gambro Lundia AB, Lund, Sweden) system. The number of exchanges was clinically determined to achieve euolemic.

**Residual Renal Function, Overnight Kt/V, and Creatinine Clearance (CrCl) Assessment**

Residual GFR was calculated according to the four-variable MDRD equation at the point of Tenckhoff catheter placement (14). The adequacy of dialysis indices were assessed by measuring total, peritoneal, and renal Kt/V and CrCl. The peritoneal component (nightly peritoneal Kt/V [pKt/V] and peritoneal CrCl [pCrCl]) was estimated solely from the overnight effluent dialysate on the night of PSG examination, whereas the renal component was estimated from 24-h urine collection.

For nightly pKt/V and pCrCl during NPD, all spent dialysate drained from the PD cycler was collected in a bucket. The volume of the dialysate was accurately measured, and the dialysate was stirred vigorously before a representative aliquot was obtained for calculation of urea and creatinine concentrations. Because NPD patients did not undergo dialysis during the day, the nightly pKt/V and pCrCl was also the 24-h pKt/V and pCrCl. Renal clearance was derived from 24-h urine urea and creatinine divided by plasma urea and creatinine concentrations, respectively. The nightly renal component of Kt/V and CrCl was estimated from the 24-h urine result normalized for time in bed (24-h result × time in bed in h/24 h).

For nightly pKt/V and pCrCl during CAPD, the spent dialysate from the overnight dwell of PDF was used for calculation of nightly clearance in a fashion similar to that for NPD. For 24-h clearance, all spent dialysate from day and night exchanges were used for calculation. Renal clearance (24-h and nightly result) was estimated as stated above. The crude body weight was used to calculate V according to the method of Watson (15).

**Table 1. Biophysical and clinical parameters while on NPD or CAPD (n = 38)**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>On NPD</th>
<th>On CAPD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body mass index (kg/m², abdomen emptied)</td>
<td>23.2 ± 3.6</td>
<td>23.9 ± 4.2</td>
</tr>
<tr>
<td>Neck circumference (cm)</td>
<td>37.1 ± 3.4</td>
<td>36.6 ± 3.1</td>
</tr>
<tr>
<td>Neckheight ratio</td>
<td>0.219 ± 0.024</td>
<td>0.222 ± 0.022</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>133 ± 16</td>
<td>131 ± 17</td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>83 ± 10</td>
<td>86 ± 12</td>
</tr>
<tr>
<td>No. of antihypertensive drugs</td>
<td>2.4 ± 0.68</td>
<td>2.3 ± 0.54</td>
</tr>
<tr>
<td>Hemoglobin (g/dl)</td>
<td>9.5 ± 1.3</td>
<td>10.1 ± 1.9</td>
</tr>
<tr>
<td>Serum albumin (g/L)</td>
<td>34.8 ± 4.9</td>
<td>34.5 ± 3.7</td>
</tr>
<tr>
<td>Plasma bicarbonate (mmol/L)</td>
<td>24.0 ± 3.2</td>
<td>26.4 ± 3.6</td>
</tr>
<tr>
<td>Residual urine output (L/24 h)</td>
<td>0.88 ± 0.13</td>
<td>0.84 ± 0.10</td>
</tr>
<tr>
<td>Residual GFR (ml/min/1.73 m² BSA)</td>
<td>8.77 ± 2.2</td>
<td>8.69 ± 1.9</td>
</tr>
</tbody>
</table>

Data are presented as mean (± SD). P > 0.05 for all comparisons during NPD or CAPD. GFR, glomerular filtration rate; BSA, body surface area.
Bioelectrical Impedance Analysis

Bioelectrical impedance analysis (BIA) to assess body water composition using Nutrigard-M Bioelectrical Impedance Analyzer (Data Input GmbH, Darmstadt, Germany) was performed on the night of PSG before starting NPD or CAPD night dwell, and in the morning after completing NPD or the CAPD night dwell, with the peritoneum emptied. All measurements were made with the patient in the euvoletic state, i.e., after the patients' condition had become stabilized while they were undergoing a particular mode of PD. Formulas for deriving body water compositions were described in our recent report (12).

Magnetic Resonance Imaging (MRI)

Each patient underwent an MRI scan of the upper airway while awake, within 6 h after completing NPD or the night dwell of CAPD. The protocol was modified from that reported by Ryan et al. (16) Briefly, the patient's head was placed in a holding frame, and scans were performed during tidal breathing using a GE MRI 3T scanner. Sagittal and axial scans were acquired from T1-weighted images.

All MRI images were recorded digitally and interpreted by two independent observers who were unaware of the patient’s mode of dialysis. Five anatomical sites were measured (Figure 1) and were defined as follows:

1. Nasopharynx was airway bordered by the soft palate anteriorly, the nasal turbinates anteriorly and superiorly, and the adenoids posteriorly and superiorly. The inferior border was at the level of the inferior tip of the uvula.

2. Oropharynx was the aerated space bordered by the hard palate superiorly, the tongue inferiorly, and the soft palate posteriorly.

3. Hypopharynx was the aerated space bordered by the posterior aspect of the tongue anteriorly, the posterior pharyngeal wall posteriorly, and the inferior aspect of the soft palate superiorly. The inferior border was defined by the inferior extent of the base of the tongue.

4. Minimal pharyngeal cross-sectional area was taken from the axial slice at which the pharyngeal area was the smallest.

5. Volume of the tongue measured on sagittal images with axial correlation.

Volume measurements were then calculated on the basis of the sagittal areas of a series of contiguous slices and on the thickness of the scan slices.

Statistical Analyses

Data were expressed as means ± SD unless otherwise specified. Statistical analyses were performed using SPSS for Windows software version 14.0 (Statistical Package for the Social Sciences Inc., Chicago, IL). Comparisons between groups were performed by χ² test for categorical data, and Mann-Whitney U test for continuous data. Because of individual variations in the volumetric analyses of the upper airway parameters as shown on MRI, all numeric changes were expressed as percentage variation with respect to values obtained during NPD. Nonparametric paired-sample Wilcoxon signed rank test was used to determine changes in sleep disturbance parameters, nightly and 24-h Kt/V and creatinine clearances, and volumetric measurements on MRI studies during NPD and CAPD. Comparison of within-group differences in prevalence of sleep apnea during NPD or CAPD was performed using the McNemar test. Relationships between absolute or differential nightly pKt/V or pCrCl and AHI were analyzed using Spearman rank correlation. A P value of < 0.05 was considered significant. All probabilities were two-tailed.

Results

The prevalence of sleep apnea (AHI ≥ 15/h) was 21.1% (n = 8) during NPD and 42.1% (n = 16) during CAPD (Table 2).
When the AHI cutoff was lowered to 10/h, the prevalence was 26.3% (n = 10) during NPD, and 50% (n = 19) during CAPD. As a group, the absolute AHI values increased from 9.6 ± 2.7/h during NPD to 21.5 ± 4.2/h during CAPD (P < 0.001 for comparison before and after transfer to CAPD). Both obstructive and central components of sleep apnea became significantly more severe, and the individual changes in AHI values during the two different modes of PD are shown in Figure 2. The overall sleep pattern during NPD or CAPD was summarized in Table 2. Although there was no significant difference in the total sleep time during NPD or CAPD, the duration of sleep with hypoxia (oxygen saturation < 90%) was substantially longer during CAPD (P = 0.025). In addition, there were significantly more frequent arousals after conversion to CAPD (P < 0.001).

Multifrequency BIA revealed no difference in absolute body water content before NPD or the night dwell of CAPD (Figure 3), alleviating concerns that there may be significant change in body water composition in the brief interval between NPD and CAPD. Applying BIA just before and after NPD or the night dwell of CAPD to calculate the change in body fluid composition during sleep, NPD achieved a significantly larger volume of fluid removal, as reflected by greater reductions in absolute body water contents (Figure 4A). Compared with CAPD, NPD led to a 2.2-fold (P = 0.003), 1.9-fold (P = 0.005), and 1.6-fold (P = 0.003) more intense reduction in total body water, extracellular body water, and intracellular body water, respectively, during sleep. When total body water was expressed as hydration fraction (percentage of body weight due to water only), there was a sixfold difference in reduction achieved by NPD versus CAPD (−3.77 ± 0.49 versus −0.62 ± 0.41%, P < 0.001). Similarly, the percentage change in hydration before and after NPD or CAPD was also significantly higher in the former (P < 0.001) (Figure 4B).

Volumetric MRI measurements showed that there were significant reductions in the nasopharyngeal (−24.5 ± 9.6%) and oropharyngeal (−34.0 ± 11.6%) volumes, minimal pharyngeal cross-sectional area (−16.8 ± 4.7%), and increase in tongue volume (+8.22 ± 2.4%) after conversion from NPD to CAPD (Figure 5). There were also numerical reductions in the hypopharyngeal volume (−21.6 ± 11.9%), albeit not reaching statistical significance. When the contiguous volumes of the naso-, oro-, and hypopharynxes were examined in aggregate, there was a 30.8 ± 9.2% reduction (P = 0.004) after conversion. The reduction in this aggregate volume also correlated with the change in the indices of obstructive sleep apnea (r = −0.565, P = 0.035).

Urea kinetics analyses of the overnight spent dialysate during the night of PSG examination revealed that nightly pKt/V and pCrCl were both significantly higher during NPD than CAPD (pKt/V: 0.289 ± 0.070 versus 0.065 ± 0.023 per night, P < 0.001; pCrCl: 4.87 ± 1.22 versus 1.84 ± 0.67 L per night, P < 0.001) (Figure 6). The difference in nocturnal clearance between NPD and CAPD remained significant (total Kt/V: 0.338 ± 0.078 versus 0.118 ± 0.051 per night, P < 0.001; total CrCl: 6.42 ± 1.50 versus 3.22 ± 1.20 L per night, P < 0.001) even after taking into account the estimated endogenous component of Kt/V and CrCl that was derived from 24-h urine and normalized against the time in bed for each patient (Table 3). To further substantiate the impact of peritoneal clearance on sleep apnea, we examined the relationship between overnight pKt/V or pCrCl and AHI in all subjects during NPD and CAPD. There were significant negative correlations between pKt/V and AHI (r = −0.332, P = 0.003), and between pCrCl and AHI (r = −0.319, P = 0.005). The difference in overnight pKt/V or pCrCl between each mode of PD was significantly correlated with the difference in AHI for each individual patient (Figure 7).

**Discussion**

We have extended our fixed intervention study cohort to investigate the mechanisms through which NPD improves sleep apnea compared with CAPD. Our results reaffirmed a

*Table 2. Polysomnographic data while on NPD or CAPD (n = 38)*

<table>
<thead>
<tr>
<th></th>
<th>On NPD</th>
<th>On CAPD</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total sleep time (h)</td>
<td>5.0 ± 1.60</td>
<td>5.55 ± 1.41</td>
<td>NS</td>
</tr>
<tr>
<td>Sleep efficiencya</td>
<td>57.4 ± 14.4</td>
<td>62.8 ± 12.7</td>
<td>NS</td>
</tr>
<tr>
<td>Stage of sleep (% of total sleep time)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>rapid eye movement</td>
<td>18.2 ± 10.5</td>
<td>16.7 ± 8.6</td>
<td>NS</td>
</tr>
<tr>
<td>stage 1, 2</td>
<td>78.8 ± 22.1</td>
<td>79.3 ± 20.0</td>
<td>NS</td>
</tr>
<tr>
<td>slow wave (stage 3, 4)</td>
<td>1.9 ± 3.2</td>
<td>4.0 ± 3.6</td>
<td>NS</td>
</tr>
<tr>
<td>AHI (no./h)</td>
<td>9.6 ± 2.74</td>
<td>21.5 ± 4.15</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>subjects with AHI &gt; 15 (n, %)</td>
<td>8 (21.1%)</td>
<td>16 (42.1%)</td>
<td>0.008</td>
</tr>
<tr>
<td>subjects with AHI &gt; 10 (n, %)</td>
<td>10 (26.3%)</td>
<td>19 (50%)</td>
<td>0.004</td>
</tr>
<tr>
<td>subjects with AHI &gt; 5 (n, %)</td>
<td>11 (28.9%)</td>
<td>22 (57.9%)</td>
<td>0.001</td>
</tr>
<tr>
<td>Duration with oxygen saturation &lt; 90% (min)</td>
<td>64.3 ± 15.8</td>
<td>94.8 ± 20.5</td>
<td>0.025</td>
</tr>
<tr>
<td>Arousals (no./h)</td>
<td>16.6 ± 9.8</td>
<td>26.6 ± 16.4</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Periodic leg movement (no./h)</td>
<td>0.5 ± 0.21</td>
<td>1.4 ± 0.35</td>
<td>0.007</td>
</tr>
</tbody>
</table>

aRatio of total sleep time to total time in bed. Data are presented as mean (± SD). AHI, apnea-hypopnea index (i.e., average number of episodes of apnea and hypopnea per hour of sleep).
significant worsening of sleep apnea after conversion from NPD to CAPD, as reflected by substantial increases in AHI, increased prevalence of sleep apnea, prolongation of the duration of hypoxia, and more frequent arousals during sleep. On a mechanistic basis, we showed that a major component that alleviated the severity of sleep apnea during NPD in comparison to CAPD was more intensive ultrafiltration, which led to larger reductions in total body water and hydration fraction during sleep, as indicated by BIA measurements. This may translate indirectly to reduced airway edema and therefore less severe OSA.

Here, we performed MRI of the upper airway to detect any subtle changes in the airway during each of the two modes of PD. Volumetric MRI is a powerful tool for detecting changes in these structures (17). A practical disadvantage of MRI is patient-perceived discomfort and claustrophobia. A considerable proportion (up to 36%) of patients in this study, having undergone the first MRI, declined to undergo a second.

Among patients who underwent MRI during NPD and CAPD, there were reduced aggregate pharyngeal volumes and
minimal pharyngeal cross-sectional area, together with concomitant increase in tongue volume after switching from NPD to CAPD. Tongue enlargement may also represent fluid accumulation in other soft tissues bordering the airway such as the nuchal and peripharyngeal areas, which are difficult to assess in an objective manner because of the lack of clear-cut anatomical demarcations. It is conceivable that such anatomic alterations favoring narrowing of the upper airway may give rise to OSA during sleep in predisposed patients when there is a reduced tone of the supporting airway musculature. This is evidenced by the correlation between the reduction in the aggregate pharyngeal volume and the worsening of the obstructive component of sleep apnea after conversion to CAPD. Furthermore, it is envisaged that fluid accumulation in the upper airway will increase airflow resistance of the pharynx, as reflected by the decrease in minimal pharyngeal cross-sectional area on switching to CAPD. In support of this notion, Chiu et al. (18) recently applied lower body positive pressure using anti-shock trousers to displace fluid from the legs to the upper body, and found that the maneuver resulted in increased pharyngeal airflow resistance. Beecroft et al. (19) recently showed that pharyngeal narrowing contributes to the pathogenesis of OSA in dialysis-dependent patients. Furthermore, in patients converted from conventional to nocturnal hemodialysis, there was an increase in pharyngeal size together with improvement in sleep apnea (20). This study is the first to show a reduction in airway diameter on conversion from NPD to CAPD.

Apart from improving OSA, our data showed that NPD also improved the central component of sleep apnea. It remains unclear why a modality switch that principally affects fluid balance...
and airway patency, and hence resistance, should affect central sleep apnea. One possible explanation, learned from cross-over studies in hemodialysis subjects (7), is through correction of metabolic acidosis and hypocapnia using nocturnal hemodialysis. The attenuation of hypocapnia may then disinhibit the central respiratory center that became suppressed in the face of hypocapnia. However, this is not a likely explanation for NPD, which is a biochemically modest procedure compared with HD in terms of acidosis correction, as reflected by similar serum bicarbonate levels during NPD or CAPD. An alternative mechanism that we contemplated was a difference in the rate of removal of uremic waste products. Because residual renal function remained unchanged during NPD or CAPD, we focused on the peritoneal component of solute clearance during sleep and calculated the nightly $K_t/V$ and $CrCl$ contributed solely by PD during sleep. The only demonstrated difference between NPD and CAPD, apart from fluid status, was the much higher nightly $pK_t/V$ and $pCrCl$ during NPD, which could represent a circumstantial evidence of the effect of uremic toxins on sleep apnea. Even after taking into account the estimated contribution of endogenous clearance during the night, the total nighttime clearance was still significantly higher during NPD than during CAPD. Although it could be argued that the daytime component of CAPD that has to confer a significantly higher clearance than that of nocturnal hemodialysis still had significantly worse sleep apnea during daytime versus nocturnal hemodialysis (7); and (4) the decrease in chemoreflex responsiveness to carbon dioxide after conversion from conventional to nocturnal hemodialysis, which is associated with increased uremic clearance during sleep (21).

There are several limitations to this study. First, we did not measure airway pressure and pharyngeal resistance, because it

<table>
<thead>
<tr>
<th>Parameter</th>
<th>On NPD</th>
<th>On CAPD</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td>24-h $K_t/V$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>total</td>
<td>0.427 ± 0.101</td>
<td>0.358 ± 0.097</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>peritoneal$^a$</td>
<td>0.289 ± 0.070</td>
<td>0.207 ± 0.071</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>renal$^b$</td>
<td>0.138 ± 0.062</td>
<td>0.151 ± 0.121</td>
<td>0.908</td>
</tr>
<tr>
<td>24-h $CrCl$ (L)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>total</td>
<td>9.20 ± 2.64</td>
<td>8.32 ± 2.01</td>
<td>0.024</td>
</tr>
<tr>
<td>peritoneal$^a$</td>
<td>4.87 ± 1.22</td>
<td>4.36 ± 1.16</td>
<td>0.069</td>
</tr>
<tr>
<td>renal$^b$</td>
<td>4.33 ± 2.38</td>
<td>3.96 ± 2.51</td>
<td>0.233</td>
</tr>
<tr>
<td>Nightly $K_t/V$ (time in bed), L</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>total</td>
<td>0.338 ± 0.078</td>
<td>0.118 ± 0.051</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>peritoneal$^c$</td>
<td>0.289 ± 0.070</td>
<td>0.065 ± 0.023</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>renal$^d$</td>
<td>0.049 ± 0.023</td>
<td>0.053 ± 0.043</td>
<td>0.523</td>
</tr>
<tr>
<td>Nightly $CrCl$ (time in bed), L</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>total</td>
<td>6.42 ± 1.50</td>
<td>3.22 ± 1.20</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>peritoneal$^c$</td>
<td>4.87 ± 1.22</td>
<td>1.84 ± 0.67</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>renal$^d$</td>
<td>1.55 ± 0.89</td>
<td>1.38 ± 0.91</td>
<td>0.179</td>
</tr>
<tr>
<td>Net ultrafiltration (ml per night)</td>
<td>1,729 ± 799</td>
<td>394 ± 315</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Body weight (kg)

night before PSG: 56.2 ± 9.8
morning after PSG: 54.1 ± 9.7
% change: −3.7 ± 1.7%

Peritoneal transport status$^e$

overall $D/P_{Cr}$ at 4 h (n, %) N/A 0.66 ± 0.093
low
low average 21 (55.2%)
high average 13 (34.2%)
high 4 (10.5%)

Data are presented as mean (± SD).

$^a$Based on overnight peritoneal effluent for NPD and 24-h effluent for CAPD.

$^b$Based on 24-h urine collection.

$^c$Based on overnight peritoneal effluent.

$^d$Estimation based on 24-h result × time in bed (h) /24 h.

$^e$According to the method of peritoneal equilibration test and classification of transport status by Twardowski (31).
induces too much discomfort in a patient undergoing CAPD and PSG monitoring to place another two catheter transducers at the naso/hypopharynxes and a tightly fitting face mask for flow recording during the same night (18). Second, the number of subjects is small. It was difficult to recruit subjects to undergo PSG and MRI in succession on two occasions, as reflected by the high drop-out rate among consenting subjects. Third, our results suggest, but do not directly prove, that better nocturnal clearance improves apnea. To achieve this goal will require adding day exchanges to the NPD phase to match the daytime clearance during the subsequent CAPD phase, which was not feasible in our institutional setting. Likewise, reduced airway congestion on NPD, as shown on MRI, provides only circumstantial evidence that this is linked to improved sleep apnea. Nevertheless, this contention is supported by the close association between airway edema and sleep apnea (22–25). Finally, we have not studied the changes in intraperitoneal pressure and the correlation with oxygen saturation. This arises mainly from our concern with introducing a potential source of contamination during PD caused by extra manipulation and connection to a pressure transducer for continuous pressure recording (26). Nevertheless, time-averaged intraperitoneal pressure may be higher during CAPD than during NPD because of the permanent load of peritoneal dialysate during the night in CAPD. Theoretically, this may have two effects: (1) changes in respiratory mechanics, such as reduction in vital capacity (27), although recent studies showed that sleep apnea is unrelated to pulmonary function measurements [12,28] and that flow-volume curve indices have no value in predicting sleep apnea (29); (2) more pressure effect on the diaphragm. To maintain the same tidal volume, a more negative intrathoracic pressure has to be generated, which may cause arousal and airway collapsibility, which could in turn lead to sleep apnea. Determining whether the difference in intraperitoneal pressure during CAPD and NPD causes a large enough change in intrathoracic pressure to bring about worsening of sleep apnea is beyond the scope of the present study.

In conclusion, our results suggest that while many factors including body habitus, comorbidities, and psychosocial circumstances (30) contribute to the development sleep apnea in patients with chronic renal failure, fluid status and solute clearance rates may be associated with the development of sleep apnea in PD patients (Figure 8).

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Disclosures
None.

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