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Editorial Comment

Does CAPD guarantee adequate dialysis delivery and nutrition

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Despite the vast experience gained over 15 years of CAPD practice there is growing concern about the long-term viability of this therapy and its ability to deliver sufficient dialysis to maintain patient well-being and survival [1]. With reduction in peritonitis and the subsequent improvement in technique survival the issue of long-term dialysis adequacy becomes increasingly important. The purpose of this editorial is to evaluate normalized clearance as a measure of dialysis adequacy, and the nature of the relationship between dialysis dose and nutrition. Is it possible to determine the priorities in optimizing CAPD therapy?

How can one quantify delivered dose of dialysis in the CAPD patient?

Attention has focused on quantifying dialysis dose in terms of small solute clearance in order to adequately prescribe and monitor CAPD therapy [2]. The concept of defining targets for dialysis adequacy in CAPD patients in terms of normalized clearance—that is, a dose of dialysis appropriate for the size of the patient—has been adopted from the haemodialysis practice [3]. However, though mass removal of urea and creatinine is similar for both haemodialysis and CAPD, CAPD clearance is at most only 60% that of thrice-weekly haemodialysis [2]. In addition dialysis clearance exhibits a narrow interpatient variation. In practice, in our study of 147 CAPD patients [4], total urea clearance ranged from 3.5 to 10.2 litres/day, and creatinine clearance from 3.2 to 8.8 litres/day. It is questionable whether the narrow variation in dialysis dose possible on a standard CAPD regime will result in a clinically significant difference between patients, given the impact of other comorbid conditions. The quantifying of this dose has been further complicated by adjusting this value for patient size.

Dialysis adequacy targets i.e. Kt/V or normalized creatinine clearance represent the actual clearance value adjusted by a size component. Argument reigns as to whether actual or ideal body weight should be used. In order to justify normalization of clearance by a size component it is logical to assume that there should be a constant relationship between these two variables, and furthermore an optimal dialysis dose for a given patient size. This relationship has not been validated for dialysis patients. Using more precise techniques to measure body composition (dual energy X-ray absorptiometry) we observe only weak correlations (r = 0.3) between both urea and creatinine clearance and their normalizing body components [5]. This subsequently results in adequacy values exhibiting negative correlations with nutritional markers. This phenomenon is aggravated in patients who progressively lose weight. As a consequence of the ‘shrinking’ denominator the patients adequacy value will actually increase. Evidence suggesting the inappropriateness of using normalized clearance as an index of dialysis adequacy can be found in studies showing that patients attaining targets for normalized urea and creatinine clearance have significantly lower values for individual nutritional parameters [4-6].

The use of an ‘ideal’ body rather than the patient’s actual weight to normalize both adequacy targets and the protein catabolic rate has been proposed. This is an attempt to overcome both reductions in patient weight and the inaccuracy of determining the volume of urea distribution in obese patients. However, potential problems with this approach include the lack of ideal weights for growth-restricted patients and the failure of ideal weights to reflect anabolic weight gain.

The optimal normalization factor should reflect the actual patient size and thus peritoneal surface area without exhibiting a significant bias against well-nourished patients. In addition it should not be adversely affected by dramatic fluctuations in patient weight. One possibility is that of normalizing to a function of height; however, such approaches need prospective evaluation.

What is the role of residual renal function?

Residual renal function plays an important role in determining dialysis adequacy targets [5,7]. It is known that an increase in residual renal urea clearance of only 1 ml/min increases the overall clearance value by up to 9.8 litres/week. Lameire et al. [7] report that in

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the first 2 years of CAPD, endogenous renal urea clearance contributed over 20% to the total Kt/V (urea) value, and that the decline in renal function was the most important factor contributing to the fall in Kt/V (urea) with time. Blake et al. [8], found that in 76 CAPD patients, 45% of total creatinine clearance came from residual function. In our cross-sectional study [4] loss of the renal component would have resulted in only 16% attaining the target value of 1.7 or greater for Kt/V (urea) and only 7% the proposed target of 50 litres/week for creatinine clearance. In light of the substantial renal contribution to current adequacy values and given the limited potential to increase clearance on CAPD it would appear impractical to advocate greater values for dialysis adequacy targets without resorting to automated peritoneal dialysis.

It is noteworthy that studies suggesting that targets for dialysis adequacy reflect patient outcome base this conclusion on adequacy values which reflect both dialysis and residual renal clearance [9]. No study to date has shown a relationship between clinical outcome and dialysis dose independent of the renal contribution to the adequacy value.

We recommend regular evaluation of residual renal function with subsequent adjustment of dialysis dose to compensate for its loss. This does not commit one to a specific target but, given the link between loss of residual renal function and malnutrition, should compensate for the impact of declining renal function on nutritional state.

**Does dialysis efficacy influence protein intake and nutrition?**

The evidence that dialysis adequacy is an important determinant of nutritional status is largely based on the correlation between dialysis dose as expressed by Kt/V (urea) and dietary protein intake calculated from the normalized protein catabolic rate. In contrast there is evidence to suggest that Kt/V (urea) has an inverse relationship with other markers of nutrition, in particular serum albumin [4,8].

The paucity of the relationship between Kt/V (urea) and formal measures of nutrition contrasts markedly with the strong correlation coefficients demonstrated between Kt/V (urea) and the NPCR. It must, however, be realized that both Kt/V and the NPCR are calculated in part from shared parameters and correlations of a similar magnitude to that seen in 'real life' can be obtained using randomly generated numbers [10]. This finding does not negate a relationship between dialysis dose and protein intake but rather indicates that the magnitude of the correlation is in part due to commonality between the shared parameters from which Kt/V and NPCR are derived. This phenomenon is known as mathematical coupling and contributes considerably to the strength of the observed correlation. This is borne out by the weaker correlation coefficients ranging from 0.2 to 0.3, observed when non-coupled markers of nutrition and dialysis (actual dietary protein intake and non-normalized urea and creatinine clearance) are related [4].

**Adequacy and outcome**

There are obvious temptations to define a minimal level for dialysis dose in terms of urea and creatinine clearance. Some authorities advocate attaining a total Kt/V (urea) of 1.5 or greater and a weekly total creatinine clearance of more than 35 litres/week. However it must be emphasized that no randomized study has yet validated the concept of encapsulating dialysis adequacy in terms of small-molecular-weight clearance. Common sense tells us that this approach is unlikely to succeed. One of the strengths of CAPD is that overall patient outcome is similar to that of haemodialysis despite 40% less small solute clearance. Given the relative inflexibility of modifying the standard CAPD prescription, adherence to a rigid policy of such targets is likely to lead to increasing numbers of patients being denied this therapy. We believe that factors such as blood-pressure control, treatment of hyperparathyroidism, correction of anaemia, and improvement in peritonitis rate are more likely to benefit patient outcome than blind manipulation of prescription volume solely to reach any given clearance target.

**Conclusions**

We believe that targets based on normalized clearance, i.e. Kt/V, are inappropriate markers for dialysis adequacy. Such values are inversely related to the patients nutritional state and will increase with progressive wasting and malnutrition. Similarly it has been shown that patients remain well nourished despite their values for Kt/V and normalized creatinine clearance falling below conventional target levels. Use of ideal body weight may safeguard against the 'shrinking denominator' but again forces the imposition of a target dialysis dose based solely on body size. Given the limitations of increasing CAPD clearance, inflexible adoption of targets for dialysis adequacy based on size-adjusted clearance would result in large numbers of patients being denied CAPD.

Residual renal function is undoubtedly important in achieving adequacy targets; however, given that this function is often insufficient to sustain life, its relevance remains unclear. Prospective studies which are able to determine the outcome of different doses of dialysis and discriminate between dialysis and residual renal clearance are called for. At present rather than rigorously attempting to increase dialysis prescription to offset loss of renal clearance, patients should be more closely monitored in terms of nutrition, fluid balance and uraemic symptoms. More use should be made of food diaries coupled with dietetic interviews rather than sole reliance on the normalized protein catabolic
rate as an index of dietary protein intake. Finally, given the weak relationship between nutrition and dialysis dose, careful consideration should be given to investigation of other causes of malnutrition in the CAPD population.

References