and abundant electron-dense mesangial deposits ultra-structurally are consistent with HSP and not MPA. The anti-neutrophil cytoplasm antibodies seronegativity is an additional factor against MPA. (3) MPA and PN are aggressive conditions that without treatment are associated with high mortality and morbidity rates. Our patient was treated with steroids (pulse methylprednisolone for 3 days followed by oral prednisone) and angiotensin converting enzyme inhibitor. He showed a significant decrease in proteinuria and maintained normal renal function on follow-up at 2 months, which would be unexpected if he had MPA or PN. Thus, based on the clinical and pathologic findings, we believe that the patient has HSP with multi-organ involvement and not POS.


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The use of online clearances in dialysis


To the Editor: Real-time measurement of dialysis efficacy is sought by all practising renal physicians in charge of dialysis patients. Thus, we were very interested in the article by Gross et al.,1 entitled ‘Online clearance measurement in high-efficiency haemodiafiltration’. We were surprised to discover that the paper reported the use of ionic dialysance to estimate urea clearance, as we consider this method to be widely accepted and that it was made commercially available quite a long time ago.

Ionic dialysance as commented by Gross et al. was first proposed to estimate urea clearance by Polaschegg and Petitclerc et al.3 in 1993. Given the simplicity of the method, many investigators have adopted it. We used ionic dialysance for evaluating the efficacy of different types of online haemodiafiltration after validating the results with blood measurements of urea clearances in vivo and reported it in the late nineties.4

Based on the accuracy of ionic dialysance in estimating urea clearance, we further used this method to evaluate the expected modifications in clearances by varying the convection flows in pre- and post-dilutional high-efficiency haemodiafiltration. Our studies allowed us to define a simplified formula to predict the clearance changes linked to convection. We tested these formulas in vivo in dialysis patients and we proposed their use in clinics. These studies were reported in the journal in 2000.5

Although we do not know the reasons that led the authors to overlook our reports when stating ‘a direct comparison of ionic dialysance with blood-side clearance has been made only in standard haemodialysis’, we were disappointed to discover that no new findings were presented in the current paper. We would have been interested to know whether the results of Gross et al. fit with the formulas we proposed in our Technical Note of 2000 in Kidney International.5 This would have represented a step forward in using ionic dialysance in estimating the efficacy of high-efficiency haemodiafiltration. Unfortunately, the current paper reports a high rate of discarded measurements due to ‘technical problems’ (48 out of 108 online measurements had to be excluded for this reason), and adds little to the existing literature.


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Response to ‘The use of online clearances in dialysis’


We have read with interest the letter submitted by Arglé’s et al.1 concerning our article on online clearance measurement in high-efficiency haemodiafiltration.

Ionic dialysance measurement is a surrogate method to evaluate urea clearance that has been extensively validated in mainly diffusive haemodialysis. To perform in vivo measurements of ionic dialysance, a transient conductivity change of the dialysate is automatically applied and dialysance is calculated from the time courses of the conductivities at the dialysate inlet and outlet. In high-efficiency haemodiafiltration, convection becomes the predominant part of the cleansing process.
We have extended the existing method of ionic dialysance measurement in the diffusive regime to the highly convective online hemodiafiltration regimen by developing a method that is applicable to various modes of substitution (post-, pre-, and mixed dilution). In contrast to other theoretical methods, all required quantities can be measured to a high precision without the knowledge of blood and dialyzer properties. In our in vivo study, we have validated our approach by taking the blood-side urea clearance as a reference. We did not intend to do a systematic analysis of the dependence of the dialysance on the dialysis conditions.

In their in vitro experiment, Ficheux et al.1,2,3 evaluated the impact of increasing ultrafiltration flow and varying site of substitution on total ionic clearance. They nicely illustrated that ionic dialysance is linearly correlated with ultrafiltration flow. In contrast to our study, they did not provide a technically feasible method to measure ionic dialysance in vivo as their model either requires the measurement of blood-side conductivity or the a priori knowledge of the dialysance without online substitution.

In our study, 26 measurements have been omitted due to errors in the sampling or processing of the corresponding blood reference samples. During our experimental study, size and shape of the transient conductivity change were varied to determine their optimal values. From the statistical analysis of the measurement, error threshold values for these parameters were fixed. In 22 cases, the conductivity change was suboptimal and the corresponding measurements were excluded from the final analysis. No individual measurements were excluded a posteriori as statistical outliers.

We conclude that in contrast to what the authors of this letter claim, our modeling approach is quite original. It allows the determination of the ionic dialysis in online hemodiafiltration treatments by measurements on the dialysate side alone. This approach provides a safe and reliable tool to evaluate routinely in clinic the dialysis dose delivered by online HDF.


Thiazide diuretics: rat versus human

To the Editor: I read with interest the study by Reungjui et al.1,2,3 and the accompanied editorial published in the same issue of Kidney International. The study by Reungjui et al. included 20–24 rats divided into four groups, with two of them being subjected to thiazide diuretics. The authors convincingly show that low kidney function and glomerular and tubular injury manifest more commonly in thiazide-treated rats, an effect that cannot be explained by hypokalemia alone. Various credible explanations were also provided and are all well taken. However, in the conclusion section as well as in the editorial that accompanied this article, the authors make a connection between the pathological findings in rats and the results of various clinical hypertensive studies done in humans. Although I do not disagree or agree with the authors’ interpretations and arguments regarding the design and outcome of these cited clinical trials, I find it very puzzling to make a link between pathological findings in rats taking thiazide diuretics and clinical studies that used thiazides in humans. Current knowledge indicates that rat kidneys differ not only from humans but there is also intraspecies variability.1,2,3,4 A few other questions come to mind. Assuming that the rats actually drank all the thiazide-loaded water and accordingly received 1.5–1.75 mg thiazide/day, how does this dose translate to clinical practice or to the dose used in the ALLHAT or AASK trials? What is the equivalent of 20 weeks of rat age in human life? Can the same findings be reproduced if another rat species was used?

Basic science research continues to be the best tool to build breakthrough clinical research, but until studies dedicated to examine the effect of chronic thiazide use on histological findings and/or kidney function in humans become available, I think extrapolating from animal studies risks unwarranted changes in clinical practices and public confusion.


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