and T-MARE regulatory sequences of *HNF1*β was evidenced.

In conclusion, although mutations in RARE and T-MARE regulatory sequences of *HNF1*β may nevertheless be implicated in some renal congenital disorders, we show here that mutations in these sequences are not a frequent cause of CAKUT. More experiments are required to assess the role of the RA-MAFB-HNF1β pathway in kidney development. Moreover, the molecular basis of these renal malformations is still poorly understood, and further works remain to be done to identify new CAKUT genes. Delineation of transcriptional networks involved in early human metanephros development may be a way to identify these new genes.

Conflict of interest statement. None declared.

[1]Service de Néphrologie et Immunologie clinique, Centre de référence des maladies rénales rares, CHU Toulouse F-31400
[2]INSERM U563, Centre de Physiopathologie de Toulouse Purpan, Toulouse F-31300
[3]Université Toulouse III Paul-Sabatier, Toulouse F-31400
[4]Service de Néphrologie-Médecine interne et INSERM U588 (I2MR équipe 5)
[5]Service de Génétique médicale CHU Toulouse F-31300, France
E-mail: stanislas.faguer@inserm.fr

Stanislas Faguer[1,2,3]
Dominique Chauveau[1,2,3]
Stéphane Decramer[3,4]
Nicolas Chassaing[2,3,5]

1. Weber S, Moriniere V, Knuppel T *et al*. Prevalence of mutations in renal developmental genes in children with renal hypodysplasia: results of the ESCAPE study. *J Am Soc Nephrol* 2006; 17: 2864–2870
2. Bellanne-Chantelot C, Chauveau D, Gautier JF *et al*. Clinical spectrum associated with hepatocyte nuclear factor-1beta mutations. *Ann Intern Med* 2004; 140: 510–517
3. Bellanne-Chantelot C, Clauin S, Chauveau D *et al*. Large genomic rearrangements in the hepatocyte nuclear factor-1beta (TCF2) gene are the most frequent cause of maturity-onset diabetes of the young type 5. *Diabetes* 2005; 54: 3126–3132
4. Pouilhe M, Gilardi-Hebenstreit P, Desmarquet-Trin Dinh C *et al*. Direct regulation of vHnf1 by retinoic acid signaling and MAF-related factors in the neural tube. *Dev Biol* 2007; 309: 344–357

doi: 10.1093/ndtplus/sfp039

Advance Access publication 27 May 2009

Conductivity pulses needed for Diascan® measurements: does it cause sodium burden?

Sir,

Recent, New techniques based on conductivity measurement enable physicians to evaluate the adequacy of haemodialysis dose (HDD) on all haemodialysis treatments by on-line monitoring of Kt/V. Ionic dialysance (Diascan® Hospal-Gambro, Mirandola, Italy) is a parameter calculated from the dialysate conductivity at the dialyser inlet and outlet. Every half hour, the inlet dialysate conductivity increases by 1 ms/cm during 5 min, while the outlet dialysate conductivity-cell measures the effect of this increase. The calculated ion transfer across the membrane (largely sodium) is almost equivalent to urea transfer and therefore ionic dialysance reflects the urea clearance. This method has been shown to have a good correlation with Kt/V measured by the mathematical urea equations in several studies [1–4].

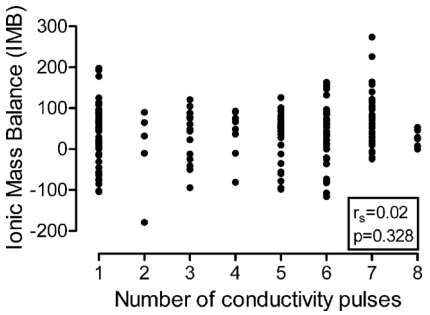

Fig. 1. Correlation between IMB during isovolaemic haemodialysis and number of conductivity pulses.

However, some investigators suggest that during the conductivity pulse of 5 min, significant amounts of sodium may be transferred into the patient [5]. In our centre, we studied that effect. Diascan® also measures ionic mass balance (IMB) and plasma conductivity (PC) that are likely to represent sodium balance and plasma sodium, respectively (plasma sodium in mmol/l ≈ plasma conductivity in ms/cm × 10). This is performed by constant measurements of the conductivity in the dialysate inlet and outlet, according to the formulas

$$\mathrm{PC} = [\mathrm{Cd_{out}} - (1 - \mathrm{D}/\mathrm{Qd_{in}}) \times \mathrm{Cd_{in}}]/(\mathrm{D}/\mathrm{Qd_{in}}),$$

where $\mathrm{Qd_{in}}$ and $\mathrm{Qd_{out}}$ are dialysate flow at, respectively, inlet and outlet, $\mathrm{Cd_{in}}$ and $\mathrm{Cd_{out}}$ are dialysate conductivity at, respectively, inlet and outlet and D is ionic dialysance, and

$$\mathrm{IMB} = (\mathrm{Qd_{in}} \times \mathrm{Cd_{in}} - \mathrm{Qd_{out}} \times \mathrm{Cd_{out}}) \times 10 \times \mathrm{time(min)}.$$

A positive IMB means sodium removal from the patient. A negative IMB means sodium transport to the patient.

In patients with zero inter-diaytic weight gain, IMB was measured in 200 isovolaemic haemodialysis sessions by Diascan®, 137 sessions were performed with four to eight conductivity pulses. A total of 63 sessions was performed with only one conductivity pulse.

The results showed a highly significant correlation between pre-dialytic plasma conductivity and IMB (Spearman rank $r_s = 0.902$, $P < 0.005$), in agreement with previous studies [6]. There was no correlation between IMB and the number of conductivity pulses (Spearman rank $r_s = 0.02$, $P = 0.328$) (Figure 1).

In conclusion, Diascan® is a useful tool to assess HDD without evidence of an increased sodium load related to the conductivity pulses during haemodialysis treatment.

Conflict of interest statement. None declared.

[1]Department of Internal Medicine Máxima Medical Centre, Veldhoven
[2]Department of Clinical Epidemiology, Leiden University Medical Center
[3]Department of Internal Medicine University Hospital, Maastricht The Netherlands
E-mail: K.Moret@mmc.nl

Karin Moret[1]
Diana C. Grootendorst[2]
Charles Beerenhout[1]
Jeroen P. Kooman[3]

1. Mercadal L, Petitclerc T, Jaudon MC *et al*. Is ionic dialysance a valid parameter for quantification of dialysis efficiency? *Artif Organs*1998; 22: 1005–1009
2. Lindsay R, Bene B, Goux N *et al*. Relationship between effective ionic dialysance and *in vivo* urea clearance during haemodialysis. *Am J Kidney Dis* 2001; 38: 565–574
3. McIntyre CW, Lambie SH, Taal MW *et al*. Assessment of haemodialysis adequacy by ionic dialysance: intra-patient variability of delivered treatment. *Nephrol Dial Transplant* 2003; 18: 559–563
4. Moret K, Beerenhout CH, Van Den Wall Bake AWL *et al*. Ionic dialysance and the assessment of Kt/V: the different estimates of V on method agreement. *Nephrol Dial Transplant* 2007; 22: 2276–2282
5. Gotch FA, Panlilio FM, Buyaki RA *et al*. Mechanisms determining the ratio of conductivity clearance to urea clearance. *Kidney Int Suppl* 2004; 89: S3–S24
6. Moret K, Hassell D, Kooman JP *et al*. Ionic mass balance and blood volume preservation during a high, standard, and individualized dialysate sodium concentration. *Nephrol Dial Transplant* 2002; 17: 1463–1469

doi: 10.1093/ndtplus/sfp059

Advance Access publication 3 June 2009

Assessment and reduction of fluid overload using a body composition monitor

Sir,

Assessment and reduction of fluid overload is a major clinical problem in haemodialysis (HD) patients which should be assessed by objective methods [1]. Non-invasive bioimpedance spectroscopy with a body composition model has been validated against other methods to assess fluid status [2,3] and fluid changes accurately [4]. Our study investigated whether the application of a new bed-side bioimpedance spectroscopy device BCM (body composition monitor, Fresenius Medical Care, Germany) was feasible in a normal clinical setting. A single pre-dialytic measurement provides the body composition of the patient and quantifies his fluid overload.

The fluid status of HD patients from three centres was measured with BCM. The study consisted of a cross-sectional study (fluid status was assessed once) and a longitudinal study (fluid status was measured repeatedly, and potential fluid overload reduced following the target defined by BCM).

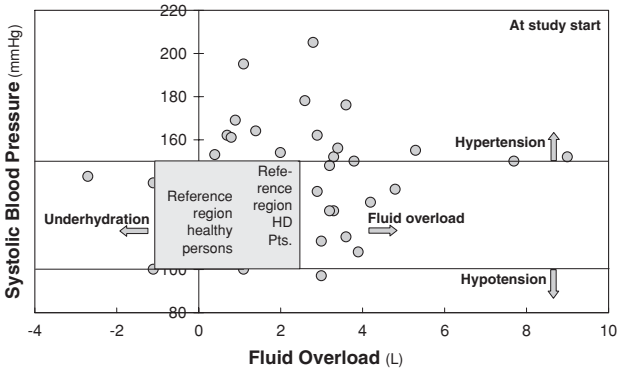

Fig. 1. Pre-dialytic systolic blood pressure and fluid overload in the 34 patients of the longitudinal study at the start of the study.

A total of 139 HD patients were investigated with BCM. The patients were grouped concerning their pre-dialytic fluid overload in quartiles with -0.14 ± 1.04 L in the lowest quartile (Q1) and 4.13 ± 1.50 L in the highest quartile (Q4).

In Q4 we found predominantly men (77% versus 43% in Q1, $P < 0.01$). The incidence of hypertension was at maximum in Q4 (94% versus lowest in Q2 with 76%, $P < 0.05$). The highest ultrafiltration volumes were observed in Q4 (3.1 ± 0.8 L versus the lowest in Q2 with 2.6 ± 0.8 L, $P < 0.02$). Patients with a high fluid overload had a lower body mass index (25.0 ± 4.3 kg/m^2 in Q4 versus 27.8 ± 4.5 kg/m^2 in Q1, $P < 0.01$).

For the longitudinal study, a sub-group of 34 patients was selected predominantly according to fluid overload and blood pressure values outside the reference region for healthy persons at the start of the study (fluid load <-1.1 L or >1.1 L and systolic blood pressure values <100 mmHg or >140 mmHg; see Figure 1); they were repeatedly investigated with BCM during 5.9 ± 1.7 months. The mean fluid overload was reduced by 0.62 L; in patients with fluid overload >1.1 L, it even was reduced by 0.81 L (see Table 1).

The observed mean pulse pressure decrease of 3 mmHg did not reach statistical significance ($P = 0.146$); pulse pressure has been associated with risk of death [5].

Changes in the prescription of antihypertensive medication were not significant either.

In the sub-group of fluid overloaded patients with high blood pressure, a non-significant reduction of blood pressure was observed (BP, pre-dialytic systolic/diastolic BP: $165 \pm 18/77 \pm 13$ mmHg at the start of the study versus $157 \pm 26/73 \pm 11$ mmHg at the end of the study, $P = 0.140/P = 0.286$). Moreover, a significantly higher UF volume was observed (2.5 ± 1.0 L at the start of the study versus 2.8 ± 0.9 L at the end of the study, $P = 0.013$).

Fluid overload is present in many HD patients, often unexpected. The analysis shows that special patient groups