Letters and Replies

Fluid restriction in dialysis patients

Sir,

Dr Tomson [1] made an excellent analysis of the (lack of a) physiological basis for the wide-spread habit to force fluid restriction on dialysis patients. It is amazing, though, that such a warning is at all necessary, because this habit reveals ignorance or negligence of basic physiological principles well known for more than half a century [2]. The NDT editors and Dr Tomson are to be congratulated for this timely challenge. I would like to comment on two issues.

First, the low peripheral resistance in patients becoming normotensive with good volume control need not be ‘paradoxical’. In fact, nearly all stable hypertensive patients, including those with ‘volume induced’ hypertension, have elevated peripheral resistance. When their blood pressure (BP) is lowered by volume reduction, resistance decreases. The reasons for this phenomenon (autoregulation of tissue perfusion) have been explained by Guyton [3]. Changes in vascular wall sodium content may not necessarily be responsible. However, patients whose BP has been kept normal by volume control for long periods, sometimes develop lower than normal BP and low peripheral resistance. This event is not fully understood but may be related to decreased responsiveness to vasoconstrictive stimuli [4].

My second comment concerns a crucial question: Why is it so difficult to persuade patients to limit the salt content of their diet? Salt consumption is a (mild) form of addiction, and time is needed for the taste threshold to adapt. Thus persuasion takes time, a rare commodity nowadays. Moreover, as Dr Tomson points out, obtaining low-salt products is becoming increasingly difficult. Thus doctors may be tempted to believe that the well-known principles are not that important after all. This mistaken belief is strengthened by the present tendency in the literature and among nutritional advisers to play down the importance of sodium in hypertension in general. But it is clear that, while the advantages of salt restriction in a general population may be debatable, there is no alternative for this measure in patients without renal function.

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Reply

Sir,

I thank Professor Dorhout Mees for his constructive comments. We agree that the regulation of peripheral vascular resistance is poorly understood, particularly in long-term haemodialysis patients. My use of the term ‘paradoxical’ was intended to refer to the paradox that hypertension in renal disease is usually thought to be due to a combination of factors, including saline overload for certain but also including increased sympathetic tone and increased secretion of vasopressor hormones such as angiotensin II arising from diseased kidneys. These mechanisms operate independently of the local autoregulation of tissue blood flow described by Guyton [1]. On simple physiological principles, one would not expect correction of saline overload to remove these powerful vasoconstrictor stimuli, yet a very high proportion of patients undergoing long slow dialysis have low peripheral vascular resistance. The causes of salt appetite are complex. While some salt ingestion may be habitual and some blamed on the food industry, it is clear that spontaneous salt intake amongst humans with free access to salt is often much higher than that needed to maintain homeostasis. These observations are consistent with a genetic contribution to salt appetite as a result of evolution in conditions of salt scarcity [2]. In addition, studies in animals point to powerful biological determinants of salt-seeking behaviour, including angiotensin, aldosterone, recurrent hypovolaemia and decreased plasma osmolality [3]. These observations may help to explain why it is so difficult to persuade patients to reduce salt intake. However, my point remains that without reduction of salt intake, advice to restrict water intake is illogical and pointless.

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