The Heart is a Muscle, the Kidney is a Vessel
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To the Editor We read with interest the article by Kawamoto et al in a recent issue of the Journal (1). Their study showed a significant association between renal function and arterial stiffness. Since it was a cross-sectional study, it does not definitely prove a cause-effect relationship. It is pertinent to note that the kidney is intimately connected to other vascular disorders as well. Hypertension is a silent killer, and an optimal blood pressure prevents ischemic heart disease, stroke and renal disease. Studies since the early 20th century have shown that even in ‘essential’ or primary hypertension the basic pathology is an inability of the kidney to excrete the salt load adequately. In such patients a higher BP is required to excrete a given sodium load (2). A reduced nephron number due to low birth weight also predisposes to development of hypertension later in life (3, 4). Increased peripheral resistance due to arterial stiffness also causes essential hypertension. This results in systolic hypertension especially in the elderly. The study by Kawamoto et al implicates the kidney here as well. Hence subtle renal dysfunction is actually responsible for a majority of cases of what are thought to be primary hypertension. It is well known that amongst the secondary causes of hypertension, renal parenchymal disease accounts for more than 80% of all cases. Unfortunately the kidney is also a victim of the effects of hypertension. Obviously then, management of hypertension should include appropriate investigations directed at the kidney. The other manifestation of vascular disease is endothelial dysfunction, which has been implicated in the pathophysiology of cardiovascular disease. The kidney has a vast endothelial capillary network. The functioning of this endothelium is regulated and determined by the nephron number, which is reduced in low birth weight babies. It is not surprising that one of the manifestations of endothelial dysfunction is microalbuminuria. Studies have shown that microalbuminuria is predictive of cardiovascular disease events and mortality not only in diabetics and hypertensives, but also in the general population (5). The normal urinary albumin excretion is as low as 5 mg/day, and the risk is linear (just as the risk of hypertension is linear starting at 115/70 mmHg). So the divisions: microalbuminuria and macroalbuminuria are in fact redundant and misleading (6). It is important to correctly evaluate albuminuria, which can be done conveniently on a spot urine sample (preferably morning) as the albumin-creatinine ratio. Periodic screening of (micro) albuminuria could allow early identification of vascular disease (5). Microalbuminuria and arterial stiffness have also been correlated (7). Thus, hypertension, arterial stiffness, (micro) albuminuria and chronic kidney disease are all interrelated.

The time has come to recognize the role of the kidney beyond its conventionally accepted functions as an excretory and endocrine organ. It plays a vital role in maintaining blood pressure and mirrors vascular disease. Measurement of blood pressure, albuminuria and glomerular filtration rate not only helps in detecting kidney disease, but also gives a clue to the presence of underlying vasculopathy.

References