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Long-term longitudinal study of renal failure progression

Although the progression of renal failure is known to be inexorable, the rate of decline of the glomerular filtration rate (GFR) remains a subject of controversy. Previous studies of the prognosis of chronic kidney disease do not provide a clear answer, as the largest epidemiological surveys were not longitudinal and the smaller longitudinal ones did not provide estimates of change in GFR. Importantly, it is necessary to prove that patients participating in these studies actually have chronic renal failure. This can be demonstrated by persistent decreases in GFR. In a 58,000-person community in Norway, Eriksen and Ingebretsen identified patients with low filtration rates and ascertained that low GFRs remained 3 months later. In a subgroup of 3,000 patients, nine had low GFRs measured over the follow-up period, which averaged 44 months. Almost three-fourths of the patients had a decline in GFR. The cumulative incidence of renal failure averaged 0.04%, well below what had been previously reported. It is worth noting that women had a slower rate of renal failure progression than did men. **See page 375.**

Carbon monoxide: poison or treatment?

One of the surprising findings of modern cell biological research is that carbon monoxide (CO), long known as a potent toxic gas, is actually produced in mammalian tissues and has some potent biological effects that may be beneficial in important disease settings. CO is produced by catabolism of heme by the enzyme heme oxygenase-1. This enzyme is often induced in situations of injury, especially in ischemia. Several studies have shown that infusion of CO reduces apoptosis and in flammation associated with ischemic states in the kidney and other organs. One of the major problems in kidney transplantation is the ischemic injury that the kidney suffers after being removed from the donor. Several solutions are available that allow minimization of this injury, but they still require improvement. Sandouka *et al.* delivered low levels of CO to isolated perfused rabbit kidneys and tested its effect on ischemic injury. Their innovation was to perfuse the kidney with compounds that spontaneously hydrolyze to release CO under physiological conditions of pH and temperature. The kidneys were flushed with these solutions and then stored at 4 °C for 24 hours. The perfusion rates, glomerular filtration rates and sodium and glucose absorptive capacities of the kidneys were then evaluated. Isolated perfused kidneys pre-treated with CO-releasing compounds had higher rates of perfusion and filtration than those stored after flushing with standard solutions. Studies of their oxygen consumption and mitochondrial function also showed excellent preservation. Although this study will need to be supplemented by *in vivo* methods, it provides a novel and intriguing avenue of renal protection. **See page 239.**

Cystatin C as a measure of glomerular filtration rate

In epidemiological studies, the use of serum creatinine has been fraught with some difficulty. There is a continuing need to find a useful substitute, the most recent of which is plasma cystatin C. Cystatin C is a small protein that is filtered and degraded completely by the proximal tubule in one pass. In a study reported in this issue, Rule *et al.* systematically evaluated serum creatinine, cystatin C and iothalamate clearance (as a measure of GFR) in 460 adults. Remarkably, the relationship between cystatin C and GFR differed across clinical presentations. Patients with kidney disease had the strongest correlation between cystatin C and GFR, and healthy individuals had a weaker correlation. GFR was higher in transplant recipients than in patients with kidney disease for any given cystatin C level. In patients with chronic kidney disease, the correlation between cystatin C and GFR was slightly higher than when the serum creatinine equation was used. This research emphasizes the need for a better marker of GFR that requires a single blood test. **See page 399.**

Home blood pressure measurements in renal failure

There is increasing evidence that blood pressure measurement in the clinic does not reflect the patient's actual blood pressure. In part this is due to the anxiety that patients experience when going to clinics. Agarawl and Andersen studied home blood pressure monitoring as a predictor of renal failure. They found that blood pressure measured at home was lower than that measured in the clinic. Patients were followed for more than 3 years. The development of renal failure or other serious end points was correlated with either clinic or home blood pressure. To test which method of measuring blood pressure more accurately predicted end-stage renal failure or mortality, the authors evaluated the contribution of an increase of blood pressure equivalent to 1 standard deviation of that measured in the clinic or at home. They found that the increase in event rate (or risk) was significantly greater for blood pressure measured at home than for that measured in the clinic. As risk is presumably related to the effect of the integrated pressure on the circulation, these findings demonstrate that measurement of blood pressure at home is the right way to go. **See page 406.**