**Solutions to *e*AJKD's Test Your Knowledge: The Liver and the Kidney**

**1B. False**

This statement is false. Since creatine (the precursor of creatinine) production in patients with cirrhosis is reduced, both serum creatinine and any creatinine-based equation would be an unreliable tool to estimate the true GFR. The 6-variable MDRD equation, although not perfect for the same reasons, is a better choice. 24-hour creatinine clearance might be considered, but tubular secretion of creatinine also increases in cirrhosis and creatinine clearance will not just be a function of glomerular filtration, but also of tubular secretion.

**2B. Free water restriction and a loop diuretic**

Free water restriction with or without a loop diuretic is the best approach in this scenario. In the absence of neurologic changes/seizures, there is no reason to use 3% hypertonic saline immediately given the risk of central pontine myelinosis with overcorrection. Dialysis is not warranted given the preserved kidney function. In a patient with kidney failure on hemodialysis, the dialysate sodium should be within 10 mEq/L of the patient's serum sodium to prevent rapid correction. V1a *agonists* (terlipressin) has been shown to improve kidney function is cases of hepatorenal syndrome (HRS), and conivaptan use is not recommended to treat hyponatremia in cirrhosis given the risks of hypotension and HRS. Finally, tolvaptan was previously used to treat hyponatremia in cirrhosis, however the FDA has recently advised against its use in cirrhosis due to the risk of worsening liver function.

**3B. False**

This statement is false. Even though HRS is instinctively thought of as the most common reason for AKI in cirrhosis, it actually constitutes less than 10% of cases. Volume depletion and acute tubular necrosis are the two major causes of AKI in cirrhosis. This fact also underlines an important fact about treatment: volume expansion (with albumin and holding diuretics) must be tried for two days before making a diagnosis of HRS.

**4G. Combination of D, E, and F**

The important point to remember is that HRS is a *functional* form of AKI with no intrinsic structural kidney damage. In fact, a diagnosis of HRS should not be made if there is evidence of prior structural kidney damage (proteinuria >500 mg/day, hematuria >50 RBC/hpf). The mechanism of HRS involves intense renal vasoconstriction due to an

imbalance between splanchnic vasodilatory (nitric oxide) and renal vasoconstrictive (RAAS system, reduced prostaglandins) stimuli.

**5E. Transjugular Intrahepatic Portosystemic Shunt (TIPS)**

All of the options could be reasonable choices in treatment of HRS. However, TIPS insertion is contraindicated in cases of hepatic encephalopathy as it could potentially worsen the confusion. Terlipressin is a vasoconstrictor that acts by binding to the V1 receptor. It is approved for this use in Europe, but not yet in North America. Midodrine and norepinephrine are alpha-1 adrenergic agonists. Octreotide, which inhibits glucagon (that mediates splanchnic vasodilatation), has also been shown to be effective in treatment of HRS.