private schools, but all the other schools in the survey had had students with the disorder in the recent past.

The usual age at entry to secondary schools in South Australia is 12 or more years. The one-year prevalence of anorexia nervosa was 1.05 per 1000 female secondary-school students. Anorexia nervosa proved to be a fairly rare disorder among South Australian schoolgirls. We wonder whether that might be the case elsewhere.

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POLYCYSTIC KIDNEY DISEASE

To the Editor: An increased prevalence of aortic incompetence and mitral-valve prolapse has been described previously in patients with polycystic kidney disease.1 Hossack et al. (Oct. 6 issue)2 reported a large-scale study done on a prospective basis and employing Doppler echocardiography. The authors contend that they found no association between mitral-valve prolapse and hypertension, a frequent complication of polycystic kidney disease. However, there was a history of hypertension in 56 percent of their subjects with polycystic kidney disease, 12 percent of the unaffected family members, and 12 percent of the controls (P<0.0005). Furthermore, their Table 3 showed a systolic blood pressure of over 140 mm Hg in 29 percent of their patients with polycystic kidney disease, 11 percent of the unaffected family members, and 9 percent of the controls — an obvious difference. Therefore, it appears that the increased prevalence of mitral-valve prolapse found by Hossack et al. in their patients with polycystic kidney disease may well have been related to the associated hypertension in these patients rather than to the polycystic kidney disease itself. Roberts3 reported an increased prevalence of mitral-valve prolapse in patients with hypertension and postulated that the mitral valve is prone to prolapse under elevated left ventricular systolic pressure.

The fact that in the study of Hossack et al. the patients with polycystic kidney disease were older than the members of the other two groups also suggests that elevated blood pressure, which is more frequent in older subjects, may be responsible for the increase in the prevalence of mitral-valve prolapse in these subjects. Naggar et al.4 showed an increase in mitral regurgitation with advancing age in patients with mitral-valve prolapse. More recently, Wijckien and Hickey5 also found a much greater frequency of severe mitral regurgitation in men than in women, and attributed the difference to higher blood pressure in men.

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To the Editor: Hossack and colleagues reported high prevalences of mitral-valve and tricuspid-valve prolapse and of mitral, tricuspid, and aortic regurgitation in patients with autosomal dominant polycystic kidney disease. However, in their control population the prevalence of mitral and tricuspid regurgitation detected by pulsed-wave Doppler examination seems to us to be extraordinarily low (9 and 4 percent, respectively). We believe that the techniques they used and their reported incidence of valvular regurgitation, well below that found in more detailed Doppler studies of normal populations, raise a question about the conclusions of this study.

In our experience, the detection of trivial or mild amounts of valvular regurgitation by any Doppler method (continuous-wave, pulsed-wave, or color-flow imaging) depends not only on the sensitivity of the test but also, as much if not more importantly, on the skill, effort, and time spent by the operator. Earlier studies in our laboratory showed tricuspid regurgitation demonstrable by continuous-wave Doppler in 84 percent of the patients examined and analyzable tricuspid regurgitant velocities for the determination of right ventricular systolic pressure in over 70 percent.1,2 In 13 echo-Doppler studies reviewed,3 the detection of tricuspid regurgitation by various Doppler methods ranged from 0 to 96 percent. In a prospective echo-Doppler study4 at our institution, the prevalences of tricuspid, mitral, and aortic regurgitation in normal subjects (mean age, 47 years) were 61, 47, and 10 percent, respectively, by color-flow imaging.

Another recent study5 using color-flow imaging also demonstrated a high prevalence of valvular regurgitation in normal persons. Because of the high prevalence of valvular regurgitation noted in normal subjects, some echocardiographers are including a statement in their echocardiographic reports emphasizing this fact.6 Although Hossack and colleagues quoted a previous pulsed-wave Doppler study7 from Japan that suggested a low prevalence of valvular regurgitation in patients 40 to 49 years of age, we believe that they underestimated markedly the true prevalence of valvular regurgitation that can be detected with current technology.

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The above letters were referred to the authors of the article in question, who offer the following reply:

To the Editor: Dr. Cheng suggests that the higher incidence of mitral-valve prolapse in our patients with polycystic kidney disease may have been due to hypertension. Table 3 of our article1 shows that as the level of blood pressure rises, the incidence of mitral-valve prolapse decreases in patients with polycystic kidney disease. Furthermore, the patients with polycystic kidney disease whose blood pressure was normal had a much higher incidence of mitral-valve prolapse than the controls with similar blood pressures (28 percent vs. 2 percent). Neither Roberts2 nor Wilcken and Hickey3 suggested that hypertension caused mitral-valve prolapse, but instead they speculated that hypertension may lead to severe mitral incompetence in patients with mitral-valve prolapse.

Lavie and colleagues are critical of our Doppler technique because the frequency of mitral and tricuspid regurgitation that we report in control subjects is lower than that reported in studies using color-flow mapping.4 Current echocardiographic equipment is more sensitive in detecting small regurgitant lesions than the equipment we used. Nevertheless, our study design involved echocardiographic examination of the subjects and interpretation of the results without knowledge of group assignment. Thus, any difference in the incidence of valvular regurgitation between the controls and the patients with polycystic kidney disease could not be accounted for by differences in echocardiographic equipment or technique. Moreover, our conclusion that patients with polycystic kidney disease have a high incidence of valvular abnormalities is supported by a recent autopsy study5 that indicated valvular abnormalities in 25 of 86 patients with polycystic kidney disease (29 percent).

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To the Editor: In their paper on the linkage heterogeneity of autosomal dominant polycystic kidney disease (Oct. 6 issue),1 Kimberling et al. state that phosphoglycerate kinase, together with the alpha-hemoglobin gene complex, is linked to autosomal dominant polycystic kidney disease on the short arm of chromosome 16. However, the authors appear to have confused phosphoglycerate kinase with phosphoglycerate phosphatase. In fact, only phosphoglycerate phosphatase has been shown to be linked to autosomal dominant polycystic kidney disease on chromosome 16.5 In contrast, phosphoglycerate kinase has been assigned to the long arm of the X chromosome,6 whereas the polymorphism at the phosphoglycerate phosphatase locus detected by starch-gel electrophoresis is a useful marker in the investigation of the inheritance of adult polycystic kidney disease in families at risk, in combination with the flanking probes 3′HVR7 and 24.1.8

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To the Editor: To the Editor: Professor Carbonara and Dr. Restagno correctly point to a clerical error in our paper on the linkage heterogeneity of autosomal dominant polycystic kidney disease. Phosphoglycerate phosphatase is on chromosome 16 and linked with the autosomal dominant polycystic kidney disease. Phosphoglycerate phosphatase was referred to in the original manuscript, but unfortunately it was mutated9 to phosphoglycerate kinase during one of our various revisions.

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To the Editor: Grantham's otherwise lucid and accurate editorial (Oct. 6 issue)1 raises the issue of identifying autosomal dominant polycystic kidney disease as "the most common inherited disorder that leads to renal failure in adults." Without question, diabetic nephropathy heads the list of known genetic causes of end-stage renal disease in the United States,2 Canada,3 and since 1987, Japan. In 1984, diabetest not only led all other primary diagnoses in the Medicare end-stage renal disease registry, with an incidence of 24 per million population, but had the highest annual compound rate of growth—25.9 percent.2 Like polycystic kidney disease, diabetic nephropathy still requires exhaustive study before its pathogenesis, natural history, and culprit gene or genes are understood. What deepens the mystery of diabetic microvasculopathy is the unknown factors that distinguish afflicted persons who are likely to have nephropathy and retinopathy from others with apparently equivalent metabolic perturbations who never have kidney or eye disease. Admittedly, the elucidation of the etiologic and relative importance of genetic predisposition and environment-induced expression in insulin-dependent and non-insulin-dependent diabetes requires much investigation. Nevertheless, diabetes is the genetic disease that ranks first as a cause of end-stage renal disease in adults.

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The above letter was referred to Dr. Grantham, who offers the following reply:

To the Editor: Friedman points out that diabetic nephropathy, the end stage of a group of devastating disorders characterized by hyperglycemia, glycosuria, and vasculopathy, is associated with more chronic renal failure than is autosomal dominant polycystic kidney disease. However, his assertion that diabetic nephropathy is a more common hereditary cause of chronic renal failure is arguable. Autosomal dominant polycystic kidney disease has a clear mendelian pattern of inheritance that can be genetically defined in over 95 percent of the cases that are linked to markers on...
chromosome 16. On the other hand, diabetes mellitus is the phenotype of several disorders. Some are familial, but none have been genetically defined with certainty. 1 For that matter, glomerulonephritis and hypertensive renal disease—two other large, heterogeneous groups of disorders listed in Medicare reports on end-stage renal disease—may also have roots in mutated DNA. 2 In the strictest sense, however, autosomal dominant polycystic kidney disease appears for the moment to be the most common uniquely defined hereditary disease that leads to chronic renal failure in adults.

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RIGHT RENAL TRAUMA: A SIDE EFFECT OF BILIARY LITHOTRIPSY

To the Editor: Macrohematuria or microhematuria occurs frequently as a result of the treatment of biliary stone disease by extracorporeal shock-wave lithotripsy.1,2 This effect has been attributed to the inclusion of the right kidney in the path of the shock waves.3 Although evidence of renal trauma has been detected by magnetic resonance imaging (MRI) or CT,1,4,5 in nearly all patients undergoing lithotripsy for renal stone disease, we are not aware of any radiographic documentation of right renal trauma after biliary lithotripsy.

Recently, an 84-year-old man received 2000 shocks to a stone in the common bile duct during an investigational study of the efficacy of this form of treatment. On the day after lithotripsy, the patient underwent CT scanning for evaluation of biliary colic. Images obtained before contrast medium was administered disclosed a marked enlargement of the right kidney and a subcapsular hematoma. Images obtained after contrast medium had been administered showed multiple areas of hemorrhage throughout the renal cortex and medulla.

Lithotripsy for renal stone disease causes multiple small or large hemorrhages in the kidney, and these hemorrhages may resorb completely or result in the formation of scar tissue.2,6 Such changes may cause reduced blood flow to the kidney. Thirty percent of our patients had a decreased blood flow to the treated kidney immediately after lithotripsy,4 and a similar proportion of patients (25 percent) have been noted to have decreased blood flow 3 weeks8 and 18 months10 after treatment. In most instances such reductions have been permanent. A sustained marked increase in blood pressure requiring antihypertensive medication has occurred within a few days to as long as a year after lithotripsy in approximately 8 percent of patients.10,11 There appears to be a correlation between the extent of the reduction in blood flow to the kidney and the development of high blood pressure, but the precise relation has not yet been determined.10

Thus, patients undergoing lithotripsy for biliary stone disease are at risk for damage to the right kidney and the possible subsequent development of sustained hypertension. For this reason, we believe that during the investigational phase of the study of biliary lithotripsy it is appropriate for patients to have renograms before and after lithotripsy, and a similar proportion of patients (25 percent) should also have their blood pressure checked every two to three months for the first year after lithotripsy.

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VALVULAR HEART DISEASE IN SYSTEMIC LUPUS ERYTHEMATOSUS

To the Editor: Galve et al. (Sept. 29 issue) 1 reported on their experience with cardiac valvular involvement in 74 patients with systemic lupus erythematosus. They studied their patients with echocardiography and reported an 18 percent prevalence of valvular involvement, including lesions suggestive of Libman-Sacks endocarditis, and valvular thickening with both stenosis and regurgitation, especially of the mitral valve.

We have also prospectively looked for cardiac valvular lesions in Chinese patients with systemic lupus erythematosus. We studied 50 female patients with lupus and 43 matched normal controls with echocardiography and continuous-wave Doppler analysis with simultaneous imaging. All the patients studied met the revised diagnostic criteria of the American Rheumatism Association for the classification of systemic lupus erythematosus, 5 and the majority had received steroid therapy. One patient had thickening of the mitral and aortic valves, whereas another had thickening of the mitral valve only. However, preliminary analysis Doppler echocardiography showed a very high prevalence of mitral regurgitation (46 percent in the patients with lupus vs. 9 percent in the controls), whereas we found aortic incompetence in 4 percent of the patients but none of the controls.

We believe the high prevalence of mitral incompetence detected by Doppler analysis in our study does reflect endocardial involvement, since mitral incompetence was less common in the control group and was usually associated with mitral-valve prolapse. The prevalence of mitral incompetence in our study is comparable to the prevalence of endocardial involvement documented in autopsy studies. 3 However, we cannot exclude the possibility that racial factors contributed to the differences between our two studies.

Although we agree with Galve et al. about the high prevalence of endocardial involvement in lupus and of mitral incompetence in particular, we observed no stenotic valve lesions, whereas Galve et al. reported four cases of mitral or aortic stenosis. The difference may reflect the differing ages of the members of the study groups, since our patients were between 15 and 46 years of age, whereas those with valvular lesions studied by Galve et al. were somewhat older (mean age, 48 years). Again, racial differences may also be important. Although valvular stenosis has been reported in systemic lupus erythematosus, it is rare, 3 and it is possible that