We were also surprised to find in Bennett and Porter's data that clearances repeated one hour after an initial study during constant plasma inulin concentration and urine flow showed marked discrepancy between initial clearance and subsequent clearance in some patients. For example, one normal subject's inulin clearance went up from 105 ml/min to 133 ml/min. The other patient's inulin clearance went down from 101 ml/min to 76 ml/min. Glomerular filtration in man was found to be relatively constant throughout the 24-hour period when patients were confined to bed rest,⁸ and glomerular filtration rate in normal man is found to be remarkably constant on repeated measurement from day to day and from month to month over years.⁹

If we believe the data reported by Bennett and Porter with hour-to-hour variations physicians will be hard put to follow the change of glomerular filtration rate of patients with renal disease by either inulin clearance or endogenous creatinine clearance.—We are, etc.,

KWAN E. KIM

GADDO ONESTI

CHARLES SWARTZ

Hahnemann Medical College and Hospital,
Philadelphia, Pa., U.S.A.

- 1 Kim, K. E., Onesti, G., Ramirez, O., Brest, A. N., and Swartz, C., *British Medical Journal*, 1969, 4, 11.
- 2 Onesti, G., Kim, K. E., Swartz, C., and Brest, A. N., *Postgraduate Medicine*, 1966, 40, 327.
- 3 Smith, H. W., *Principles of Renal Physiology*, p. 32. New York, Oxford University Press, 1956.
- 4 Shock, N. W., *Geriatrics*, 1946, 1, 232.
- 5 McCance, R. A., in *Renal Disease*, ed. D. A. K. Black, p. 166. Oxford, Blackwell, 1962.
- 6 Berlyne, G. M., Varley, H., Nilwarangkur, S., and Hoerni, M., *Lancet*, 1964, 2, 874.
- 7 Reubi, F. C., *Clearance Tests in Clinical Medicine*, p. 13. Springfield, Charles C. Thomas, 1963.
- 8 Sirota, J. H., Baldwin, D. S., and Villarreal, H., *Journal of Clinical Investigation*, 1950, 29, 187.
- 9 Pitts, R. F., *Physiology of the Kidney and Body Fluids*, p. 68. Chicago, Yearbook Medical Publishers, 1968.

Myasthenia and Systemic Lupus Erythematosus

SIR,—Dr. Peter Isaacs (6 November 1971, p. 339), in his report of a patient with myasthenia gravis and systemic lupus erythematosus, argues that thymectomy would best be avoided in patients with both these diseases. I do not feel that this conclusion has either logical or empirical validity. It is based upon a report¹ of two myasthenic patients who developed lupus erythematosus several years after thymectomy, which had been followed by a remission of the myasthenia. Since, however, these two diseases have usually occurred together in non-thymectomized patients, one is not justified in concluding that thymectomy is unwise in the patient who already has both diseases, or in depriving the patient of the possibility of benefit to the myasthenia from thymectomy.

The interrelationship between myasthenia gravis and lupus is complex.² As would be expected from the known female preponderance in both diseases, almost all the reported patients are women. Either condition can occur first, and the interval between them has ranged from one month to 20 years. The effect of the development of lupus on the severity of the myasthenia has been quite variable. In several cases there were remissions of the myasthenia prior to or with the development of systemic lupus erythema-

tosis. There are other cases where the myasthenia was unaffected or worse as systemic lupus erythematosus evolved. In another case³ an excellent remission of the myasthenia occurred following thymectomy, despite antecedent lupus.—I am, etc.,

SHELDON M. WOLF

Southern California Permanente Medical Group,
Los Angeles,
California, U.S.A.

- 1 Alarcón-Segovia, D., Galbraith, R. F., Maldonado, J. E., and Howard, F. M., jun., *Lancet*, 1963, 2, 662.
- 2 Wolf, S. M., and Barrows, H. S., *Archives of Neurology*, 1966, 14, 254.
- 3 Mäkelä, P. E., Ruosteenoja, R., Wager, O., Wallgren, G. R., and Jokinen, E. J., *Acta Medica Scandinavica*, 1964, 175, 777.

Raised Elbows and Coronaries

SIR,—I have been interested for some years as a general practitioner to note how frequently it is possible to diagnose coronary artery disease in the preinfarction stage or when there is a small infarction. Fibrositis or gastric upset may well be explanations given to the patient—particularly the young patient—when family history, stress, blood pressure, cholesterol, and cardiographs are not abnormal. It is my experience that when findings are normal but suspicion remains, or when there is no other positive explanation, then if the patient is an "arm-mover" he or she has coronary artery disease, often proved later.

I define an "arm-mover" as one moving the elbows excessively or keeping them raised during conversation, relaxation, or sleep. I would be interested to hear of others finding this a significant diagnostic sign, as I am in no doubt as to its value.—I am, etc.,

ALAN N. RENNIE

Busby,
Glasgow

Caroli's Disease

SIR,—It is misleading, we feel, to dissociate Caroli's syndrome completely from choledochal cyst, as suggested by Dr. J. G. McNulty (4 December 1971, p. 623), for there is a spectrum of developmental abnormalities affecting the intrahepatic and extrahepatic biliary tree. A classical choledochal cyst may be associated with non-obstructive cystic or saccular dilatation of the left or right hepatic ducts and sometimes diverticula may arise from these ducts.¹ Cases of non-obstructive dilatation of the smaller intrahepatic ducts (Caroli's syndrome) may be associated with dilatation of the common bile duct² or even a choledochal cyst.³ Though the latter is usually an isolated finding, the presence of additional proximal biliary tree dilatation is of some clinical importance, for, as in Dr. Kelly's case (13 November 1971, p. 407), recurrent bouts of cholangitis are likely to follow removal or drainage of the choledochal cyst.

In Caroli's syndrome the hepatic parenchyma is normal, apart from some fibrosis and inflammation immediately adjacent to the dilated bile ducts, and portal hypertension has not been recorded. However, an identical pattern of dilatation of the intrahepatic biliary tree also occurs in some cases of congenital hepatic fibrosis,⁴ and in these cases portal hypertension with bleeding oesophageal varices is common but not in-

variable.⁵ We have recently seen two patients with congenital hepatic fibrosis in whom gross non-obstructive dilatation of the intrahepatic biliary tree with intractable cholangitis dominated the clinical picture and led to death. One of these cases also had unexplained dilatation of the common bile duct, again emphasizing the association of developmental abnormalities of the intrahepatic and extrahepatic biliary tree.—We are, etc.,

IAIN M. MURRAY-LYON

ROGER WILLIAMS

Liver Unit,
King's College Hospital,
London S.E.5

- 1 Tsuchida, Y., and Ishida, M., *Surgery*, 1971, 69, 776.
- 2 Caroli, J., Couinaud, C., Soupault, R., Porcher, P., and Etévé, J., *Semaine des Hôpitaux de Paris*, 1958, 34, 496.
- 3 Gots, R. E., and Zuidema, G. D., *American Journal of Surgery*, 1970, 119, 726.
- 4 Grumbach, R., Bourillon, J., and Auvert, J.-P., *Archives d'Anatomie Pathologique*, 1954, 30, 74.
- 5 Erlinger, S., Sakellariadis, D. A., Maillard, J.-N., and Benhamou, J.-P., *Presse Médicale*, 1969, 77, 1189.

Leicester Royal Infirmary

SIR,—Leicestershire doctors will be delighted to read the excellent review of Mr. Frizelle and Miss Martin's book *The Leicester Royal Infirmary* (15 January, p. 187). There are, however, two small errors. C. J. Bond was a very famous Leicester surgeon, but the matron referred to in the review was a Miss Burt (not Bond), and her memory is still revered in both Leicester and at Guy's Hospital. Sir George McRobert's Aberdonian upbringing, together with his delight to discover that the founder of the Leicester Royal Infirmary was educated at Aberdeen, must have carried him away, so that he allocated Dr. William Watts to the Scottish clan of Watt. Personally I would like to think that we belong to the same clan, but south of the border we add a significant S.—I am, etc.,

CUTHBERT WATTS

Ibstock, Leics

Representation on the Joint Consultants Committee

SIR,—The report (*Supplement*, 1 January, p. 1) of the decision by the Central Committee for Hospital Medical Services acting on the advice of its executive to oppose the admission of the Regional Hospitals' Consultants and Specialists Association to the Joint Consultants Committee in no way underlines the importance of this decision, and I trust that you will allow me the courtesy of your columns to do so.

It may not be known that the R.H.C.S.A. has been applying for admission to the Joint Consultants Committee for over 20 years, and the recent attempt to do so again was entirely in accordance with the B.M.A. policy determined by the resolutions of successive Annual Representative Meetings in 1970 and 1971 (*Supplement*, 4 July 1970, p. 15; and 7 August 1971, p. 90). These called for an increase in regional consultant representation and a democratic system of election, but both have been disdainfully ignored by the Joint Consultants Committee.

The recent invitation by the chairman of the Joint Consultants Committee for Mr. R. E. Jowett and myself to journey to Edinburgh and give evidence in support of our application for membership of the J.C.C.