(Rose et al., in press). When measures are taken to remove free paraquat from the gut the concentration of paraquat in the plasma is considerably reduced, the lung no longer accumulates paraquat to high levels, and rats survive a dose of paraquat which is normally lethal. Hence, paraquat concentrations in the lung of 10-15 nmol/g do not cause sufficient damage to kill rats and it is the accumulation of paraquat to concentrations greater than these values which leads to severe pulmonary damage and death. It can also be concluded that it is not only the peak plasma concentration which is responsible for determining the lung level but also maintenance of plasma concentrations from which the lung can accumulate large amounts of paraquat. The maintenance of such plasma concentrations in the rat has been shown to be the result of continued absorption of paraquat from the gut over the first 30 hours after administration by mouth.

We have shown that slices of human lung accumulate paraquat in vitro and the kinetics of the process are very similar to those for the rat (V max for both about 300 nmol of paraquat/g tissue/ hour; K_M for both in the region $4 \times 10^{-5}\text{-}8 \times 10^{-5}$ mol/l.). Clearly, human lung has the capacity to accumulate paraquat from relatively low plasma concentrations and this process may be prolonged, as in the rat. That this might be the case is suggested by the delay of several days often seen in patients before signs of lung damage occur. In the treatment of cases of human poisoning measures which remove paraquat from the gastrointestinal tract should be effective in reducing plasma levels and thus might prevent the accumulation of damaging amounts of paraquat in the lung. In rats treatment consisting of a stomach wash followed by repeated administration of cathartics, together with large volumes of fuller's earth or bentonite, has been shown to be effective, and we suggest that this type of treatment continued for several days might be beneficial in cases of human poisoning. Other measures, such as haemoperfusion or haemodialysis, which act directly to reduce the plasma concentration of paraquat should also be considered since in man impairment of renal function is frequently an additional complication (Bullivant, 1966).

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MEDICAL MEMORANDA

Horseshoe Kidney: A Report of One Family

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This paper is a report of a family in which all three siblings had horseshoe kidneys, while the mother had a rotational abnormality of one kidney. X-ray examination showed other congenital bony abnormalities.

Case Reports

Case 1.—The youngest in the family had a lumbar meningo-myelocoele and gross congenital deficiency of the left iliac bone. She was referred at the age of two for assessment of her urinary tract because of recurrent urinary infections and continuous dribbling incontinence. Intravenous pyelography showed a typical horseshoe kidney (fig. 1), and an expression cystogram showed gross reflux to a dilated pelvis and ureter on the right (fig. 2). Her problems were due to her neurogenic bladder rather than to her kidneys and she was managed accordingly.

Case 2.—The boy, aged six, was referred after a single episode of vague abdominal pain and haematuria lasting two days. His intravenous pyelogram (fig. 3) again showed a horseshoe kidney,

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with slight fullness of the left pelvis and ureter. The 12th ribs were seen to be short. A cystogram was normal, while a left retrograde pyelogram did not show any obstruction. He remained symptom free.

Case 3.—This 10-year-old girl had had no symptoms at all. but her intravenous pyelogram was again characteristic, and she had a short left 12th rib (fig. 4).

Case 4.—The mother, who was the only other member of the family to be x-rayed, had a different renal pattern. While her right kidney was entirely normal the left was abnormally rotated, with the lowest calyx pointing medially in typical horseshoe fashion. On her x-ray film six lumbar vertebrae are seen (fig. 5).

Comment

The frequency with which horseshoe kidney occurs, according to different estimates, varies between one in 400 (Glenn, 1959) and one in 1,000 (Walters and Priestley, 1932). A large post-mortem series (Bell, 1950) gives probably the most accurate estimate of frequency in the general population—one in 497.

The family reported here illustrates some of the clinical pictures. Many patients like the girl in case 3, are symptom free and may remain so throughout life, their horseshoe kidney being discovered accidentally at routine medicals, laparotomy, or necropsy. This must in part account for the variation in estimates of the frequency of the condition. As in case 1, many patients have associated congenital anomalies. A recent series (Kölln et al., 1972) gives a figure of 30%, with 51 anomalies in 32 patients; of these 16 were urogenital, 11 musculoskeletal, and 10 cardiovascular, and seven were in the central nervous system, including three meningomyelocoeles.

Those horseshoe kidneys that cause symptoms do so usually either because of hypertension or because of partial obstruction to the ureters as they cross the bridge of kidney tissue. No

FIG. 1—Case 1. Typical horseshoe kidney. FIG. 2—Case 1. Expression cystogram showing gross reflux to dilated pelvis and ureter on right. FIG. 3—Case 2. Horseshoe kidney.
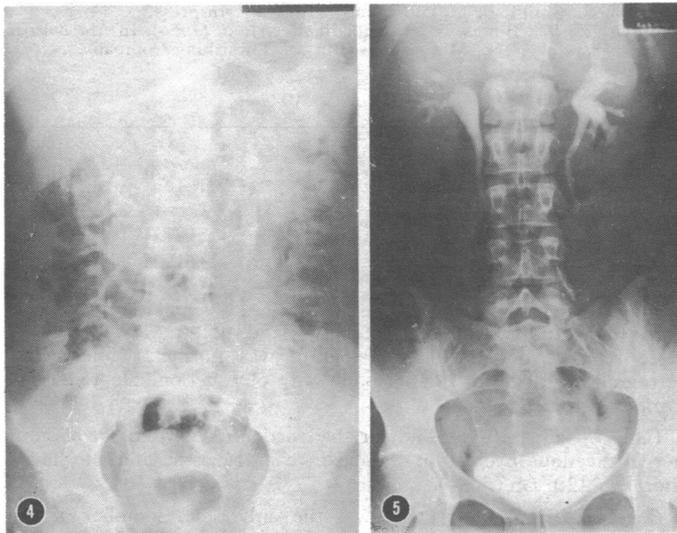

FIG. 4—Case 3. Short left 12th rib. FIG. 5—Case 4. X-ray picture showing six lumbar vertebrae.

member of this family was hypertensive at the time of writing, but the haematuria in case 2 was possibly secondary to an episode of partial ureteric obstruction.

Horseshoe kidney is caused by the fusion of the renal blastomas at a very early stage in fetal development when the primitive kidneys are in close proximity and still anteriorly rotated. Fusion prevents further normal rotation. The situation in case 4, where the lowest calyx of the left kidney was directed medially, suggests that this explanation is not complete.

When one considers the familial incidence of other renal abnormalities such as polycystic kidneys and duplications it is surprising that there is no previous record of such a tendency with regard to horseshoe kidney. One study of 100 relatives of a patient (Robertson, 1968) showed many hypertensive patients but no other horseshoe kidneys. One other paper (Bridge, 1960) describes identical twins, one with a horseshoe kidney the other with the very similar abnormality of a crossed ectopic kidney.

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