[6] Powell, H, *et al*, *Journal of Pediatrics*, 1973, **82**, 976.
[7] Shuman, R M, Leech, R W, and Alvord, E C, *Pediatrics*, 1974, **54**, 689.
[8] Vaterlaus, B P, and Hostynek, J J, *Journal of the Society of Cosmetic Chemists*, 1973, **24**, 291.
[9] Lockhart, J, and Simmons, H E, *Pediatrics*, 1973, **51**, no 2 pt 11, 430.
[10] Plueckhahn, V D, and Banks, J, *Medical Journal of Australia*, 1972, **1**, 897.
[11] Light, I J, and Sutherland, J M, *Pediatrics*, 1973, **51**, no 2 pt 11, 345.
[12] Shuman, R M, Leech, R W, and Alvord, E C, *Archives of Neurology*, 1975, **32**, 320.
[13] Plueckhahn, V D, and Collins, R B, *Medical Journal of Australia*, 1976, **1**, 815.
[14] Towfighi, J, Gonatas, N K, and McCree, L, *Laboratory Investigation*, 1974, **31**, 712.
[15] Udall, V, and Malone, J C, *Proceedings of the European Society for Study of Drug Toxicity*, 1970, **11**, 233.
[16] Kimbrough, R D, and Gaines, T B, *Archives of Environmental Health*, 1971, **23**, 114.
[17] Plueckhahn, V D, *Medical Journal of Australia*, 1973, **1**, 93.
[18] Alder, V D, *et al*, *Lancet*, 1972, **2**, 384.
[19] Mullick, F G, *Pediatrics*, 1973, **51**, 395.
[20] Cunningham, M D, and Tsoulos, N G, *Pediatric Research*, 1972, **6**, 431.

Diabetic feet

Lesions of the feet in middle-aged and elderly diabetics are usually caused by the combined effects of sepsis, neuropathy, and ischaemia. Before treatment, every patient must be assessed to decide the relative importance of these factors. In the absence of severe ischaemia conservative medical and surgical management may produce spectacular results.

Diabetic neuropathy affects both the somatic and the autonomic nerves in the feet. The patient often complains of paraesthesiae, and diminished sensation and absent ankle jerks are found on physical examination. Weakness of the small muscles of the foot leaves the pull of the long muscles of the toes unopposed, producing crumpling and clawing, especially of the 3rd, 4th, and 5th toes. Weight distribution in the foot is disturbed, which leads to excessive pressure on a small area; and because of the diminished sensation the patient may be unaware of any skin damage. Callosities develop and may progress to penetrating ulcers, and healing may be impaired because local vascular reflexes are disturbed by autonomic neuropathy. Osteomyelitis of the metatarsal heads and phalanges is common, and gross cellulitis of the whole foot and infective gangrene may result. The foot is hot, red, and swollen and the pulses are usually bounding. Unfortunately, the condition is often well advanced before the patient seeks advice.

Pure ischaemia in the diabetic patient is no different from that seen in the non-diabetic individual. Absent pedal pulses associated with coldness, persistent colour change, rest pain, and the presence of dry gangrene are sinister symptoms. Unless the limb can be salvaged by direct arterial surgery a major amputation is usually necessary.

A comprehensive retrospective analysis of the records of 172 diabetic patients who had had operations on their feet has recently been published from the USA.[1] The important finding was that operations carried out in the presence of severe active infection—high fever, leucocytosis, high blood sugar, and subcutaneous gas on the radiograph—gave poor results. This conclusion confirms the orthodox British teaching that rest, raising of the limb, and antibiotics may often transform a clinical picture that seemed a lost cause.[2] Urgent surgical drainage of a fluctuant abscess may be required, but other surgery should be delayed until the maximum benefit has been obtained from conservative treatment. Great care must be taken to prevent the development of pressure sores on the heels of both the good and the bad leg, and the use of padded canvas slings has been recommended.[2] Bone lesions may not show on the radiograph for two to three weeks, so conservative treatment also allows the physician to make a more precise assessment of the extent of the osteomyelitis. Once the cellulitis has been controlled then debridement is required to remove infected bone. Surgical technique is important: no tourniquet should be used and the wounds should not be sutured, a technique which prevents any dead space forming and so lessens the opportunity for infection to persist. Furthermore, any postoperative swelling of the wound does not imperil the viability of the skin edges. Simple neuropathic lesions usually heal without operation provided that attention is given to detail in the conservative treatment and that both the doctor and the patient are prepared to wait.

Every diabetic patient should be taught the importance of foot hygiene, sensible footwear, and regular chiropody. For the elderly this will usually mean the services of a chiropodist. The use of orthopaedic footwear to minimise the pressure effects produced by the foot deformities is also important in helping to prevent the development of these lesions. Prevention is better than cure.

[1] Goodman, J, *et al*, *Surgery, Gynecology and Obstetrics*, 1976, **143**, 587.
[2] Catterall, R C F, in *Clinical Diabetes*, eds W G Oakley, D A Pyke, and K W Taylor. Oxford, Blackwell, 1968.

Asymptomatic proteinuria in the preschool child

What is the significance of detection of proteinuria at a routine preschool clinic or as a chance finding when a child has come to the surgery or health centre for an unrelated reason? What course of action should be taken to exclude serious renal disease? These questions must face doctors frequently, yet the answers are far from clear-cut. The association between proteinuria and almost all types of nephropathy has been well recognised for many years, even though in severe obstructive nephropathy or chronic pyelonephritis protein may be absent from the urine. Urinary screening of school children has shown, however, that, though asymptomatic proteinuria and bacteriuria are both common, especially in adolescent girls, their correlation is poor.[1] Proteinuria is not a good indicator of urinary tract infections.

Normally between 50 and 150 mg of protein is excreted in the urine in 24 hours. The amount in a single specimen varies with the output, the concentrating ability, and the time of day. Chemically impregnated dipsticks are sensitive to 10-20 mg/dl, so from time to time the test is likely to detect physiological amounts as a "trace." If the reading is +(30 mg/dl) this may represent clinically significant proteinuria. Even so, false-positives are frequent—for example, in alkaline specimens—and the presence of proteinuria must be confirmed by the salicylsulphonic acid test. This is simply performed by adding 0·5 ml of 3% SSA to an equal volume of urine and observing turbidity. Quantitation is possible using a spectrophotometer and a standardised curve.

Once proteinuria has been confirmed a detailed history and examination are needed either to establish a simple explanation or to exclude obvious renal causes. Transient proteinuria may follow exercise, emotional stress, exposure to cold, or fever of

any aetiology. One in every 20 patients admitted to hospital with febrile non-renal conditions has proteinuria.[2] The mechanism is incompletely understood. However, the state of hydration, plasma volume, adrenaline release from stress, renal blood flow changes, or parenchymal inflammation by the infecting agent are all possible factors. A family history of nephropathy or deafness is at least as important as symptoms suggestive of renal or hypertensive disease. Examination should include growth and stature, blood pressure, and tests of vision and fundoscopy and hearing, as well as a search for oedema, purpura, and microscopic haematuria.

If the results of that investigation prove negative the parents may be reassured that renal disease is unlikely but that the child should be re-examined periodically to establish whether the proteinuria is transient, intermittent, or constant and to ensure that haematuria is excluded. When there is both haematuria and proteinuria the combination is often indicative of glomerular lesion,[3] and a biopsy should probably be carried out to settle both the diagnosis and prognosis.

Once persistence of the proteinuria is established, further investigations should proceed after paediatric referral. An attempt should be made to establish any postural element. Accurate quantitation is difficult in the preschool child. One good method is to compare the protein content of 12-hour collections by night and day. Orthostatic proteinuria is, however, unlikely in this age group, being much more a feature of adolescents, though pathological proteinuria may have a postural component. More detailed investigations ought to include 24-hour urine collection for measurement. In both orthostatic and persistent isolated proteinuria the range is 1-1·5 g protein per 24 hours. Measurement of the blood urea and creatinine and the glomerular filtration rate is essential, but further investigation is unlikely to contribute much. Urinary protein electrophoresis and immunochemical clearances for selectivity may help to separate normal, glomerular, or tubular leaks, but variation and overlap exist. Normal proteinuria is unselective and reflects the plasma proteins,[4] possibly indicating failure of tubular reabsorption. Additional low molecular weight globulins probably come from the kidney, urinary tract, or seminal glands.

Epidemiological studies have mostly been on school-children.[1 6] One recent survey from Galveston, Texas,[6] showed a steady rise in the prevalence of asymptomatic proteinuria from age 6 to 12, and found it more frequent in girls—perhaps in consequence of the later onset of adolescence in boys. Certainly the curves closely follow those for rate of growth and puberty changes. Children with proteinuria as a group are taller, which again may be an association with growth rate or puberty. A study[7] of over 2000 infants and preschool children found no persistent or postural proteinuria and transient proteinuria in roughly 2% boys and 6% girls. This research group believed that recurrent transient proteinuria is not a normal finding: indeed, 91% of children later found to have urinary tract disease had proteinuria, and they suggest that it always merits investigation, in particular to exclude obstructive uropathy. The increasing recognition of the value of a routine preschool screening examination at age 4, as practised in Sweden, may help to evaluate this approach epidemiologically.

Considerable doubt still exists about the value of proteinuria screening as a routine, even in adolescent girls. The yield from investigation and biopsy is low. Histological abnormalities generally amount to minor segmental or focal changes in a few cases: the vast majority have normal biopsy findings. Almost always, too, neither management nor prognosis is affected by the results of biopsy. Follow-up studies suggest that proteinuria persists for at least three years in half of asymptomatic cases.[5] Long-term studies are in progress to evaluate the natural history of both isolated proteinuria and chronic renal disease. Meanwhile, though all the evidence suggests that proteinuria is benign, a suspicion remains that it may be a signal of early renal disease.

[1] Wagner, M, *et al*, *Journal of Pediatrics*, 1968, **73**, 825.
[2] Marks, M I, *Archives of Disease in Childhood*, 1970, **45**, 250.
[3] Habib, R, in *Pediatric Nephrology*, ed Roger, P, *et al*. Philadelphia, Saunders, 1974.
[4] Rowe, D F, and Soothill, J F, *Clinical Science*, 1961, **21**, 87.
[5] Dodge, W F, *et al*, *Journal of Pediatrics*, 1976, **88**, 327.
[6] McLaine, P, and Drummond, K N, *Pediatrics*, 1970, **46**, 548.
[7] Randolph, M F, and Greenfield, M, *American Journal of Diseases of Children*, 1967, **114**, 631.

Mental disorder and mental handicap

The distinction between mental illness and mental retardation has long been recognised in law. The statute *De praerogativa regis* in 1325 provided for the protection of lands of idiots (natural fools) and the rendering of their lands on their death to their right heirs. In contradistinction, the same statute provided that the lands of lunatics might be restored to them on their recovery. This legal distinction was then important only for the well-to-do and would have been limited in practice to the more severely affected.

The Elizabethan Poor Law provided a workhouse structure into which some of both the mentally ill and handicapped were fitted, but it was not until the middle of the last century that charitable asylums for idiots began to be provided. On the other hand the Lunatic Asylum Act (1853) emphasised that the concept of lunatic included every person of unsound mind and every idiot. The separate Idiots Act (1886) did something to lessen the legal overlap, but the Lunacy Act (1890) again included idiot in the definition of lunacy. It was not until the Mental Deficiency Act (1913) that vigorous efforts began to be made to find separate provision for the mentally handicapped. This measure, which was in part motivated on eugenic grounds, began to take effect only after the first world war, when there was a dramatic increase in the number of the mentally handicapped in hospital.

In our own time social services have become available for both groups, but the question of formal detention or other encroachment on civil liberty is arousing concern. The National Society for Mentally Handicapped Children, which expresses the views of a large number of their parents, believes it desirable so far as is practicable to separate legislation on mental illness and mental handicap so as to avoid public confusion.[1] Many members of the public do have difficulties in distinguishing the two concepts, and parents of mentally handicapped children have been embarrassed and saddened when others reacted to their handicapped child as though he were mentally ill.

Until the 1959 Mental Health Act (which replaced both the Lunacy Act and the Mental Deficiency Act) all the mentally handicapped in hospital were committed there by legal process and were subject to detention. In 1973 there were 11 501 admissions in England to hospitals and units for the mentally