

## Dialysis dementia

The recognised neurological complications<sup>1</sup> of acute and chronic renal failure are usually reversible by efficient dialysis. Nevertheless, in 1972 Alfrey *et al*<sup>2</sup> described a new syndrome of unknown aetiology, progressive dialysis encephalopathy, and as confirmatory reports<sup>3-8</sup> accumulated it has become clear that it is now one of the causes of death in patients treated with haemodialysis.<sup>5</sup>

The syndrome of progressive dialysis encephalopathy may occur at any time after 15 months of haemodialysis—sometimes as long as seven years—and may be insidious or rapid in onset. The first sign is usually a speech disorder, most commonly stuttering or slurring of speech followed later by dysarthria, dysphasia, and sometimes mutism. Myoclonic jerks are constant, and all patients progress to global dementia. Psychological changes may also be present characterised by agitation, delirium, paranoia, and hallucinations. Some patients have focal neurological deficits, most commonly a facial weakness, and some have convulsions. Early in the disorder the symptoms may be intermittent, being worse at the end of a dialysis and improving before the next. In all cases reported the symptoms and signs have been progressive over periods ranging from 3 to 15 months ending in death from suicide, pneumonia, septicaemia, or uraemia. Recently Ward<sup>6</sup> has included metabolic bone disease in the syndrome, while Platts<sup>9</sup> has described three cases associated with osteomalacia, but other authors have not described the association.

Investigations have shown that the cerebrospinal fluid, cerebral arteriogram, pneumoencephalogram, and brain scan are normal. The electroencephalogram is always abnormal, showing paroxysmal high voltage theta and delta waves with spike-and-slow wave bursts, more definite after dialysis. These changes have also been recorded some months before the clinical onset of the syndrome.<sup>5</sup> Histological changes in the central nervous system are not characteristic.

Treatments that have been tried include renal transplantation, increased frequency of dialysis, and the administration of dimercaprol, penicillamine, dexamethasone, levodopa, anti-convulsants, and vitamins; nothing has influenced the course of the disease.

This syndrome, then, occurs in well-dialysed patients 15 months or more after starting haemodialysis, but it is not improved by increasing the frequency of dialysis. Its progressive nature argues against its being due to disequilibrium or underdialysis and suggests either a toxic encephalopathy or

a deficiency syndrome. There is no clear evidence in favour of a specific deficiency, and despite Wardle's<sup>10</sup> suggestion of dopamine deficiency in combination with enzyme inhibition treatment with L-dopa has not altered the course of the illness.<sup>5</sup> No apparent differences in dialysis techniques, hours of dialysis, diet, and serum electrolytes, creatinine, urea, and acid-base balance have been found between patients with and without the syndrome.

Platts<sup>9</sup> and Lyle<sup>11</sup> suggested that the condition might be a toxic encephalopathy perhaps due to trace elements such as copper, zinc, lead, or cadmium. (Patients being treated with haemodialysis may be susceptible to trace element poisoning<sup>12 13</sup> because of the large volume of water used in single pass systems combined with selective binding of elements by red cells and plasma.<sup>14 15</sup>) Aluminium toxicity has received much attention,<sup>16</sup> and increased serum and bone concentrations have been found in patients on haemodialysis, directly proportional to the total length of treatment.<sup>17-19</sup> The source of the aluminium has usually been aluminium hydroxide, though Flendrig<sup>7</sup> found high concentrations in a dialysis system using aluminium-coated heating elements. Alfrey *et al*<sup>16</sup> found that the aluminium content of bone, muscle, and brain was increased in patients with and without the encephalopathy syndrome. Those with progressive encephalopathy showed raised muscle and brain concentrations compared with normal controls, grey matter being especially affected. All their patients had been taking oral aluminium hydroxide for hyperphosphataemia.

An encephalopathy similar to progressive dialysis encephalopathy has been described in a normal patient exposed to high concentrations of aluminium.<sup>20</sup> Nevertheless, the incidence of the syndrome is low by comparison with the number of patients being treated with aluminium hydroxide.

Most studies of this encephalopathy have not reported the patients' serum-phosphate concentrations. Pathological changes in the central nervous system may follow phosphate depletion in animals,<sup>21</sup> and electroencephalographic changes and mental changes similar to those of progressive encephalopathy occur in man.<sup>22 23</sup> Hypophosphataemia decreases ATP in the red cell<sup>24</sup> and possibly in tissue generally. Certainly phosphate absorption is variable in patients treated with dialysis, and if exacerbated by administration of phosphate binders may cause severe bone disease.<sup>25</sup> The unpredictable combination of high aluminium absorption and severe

hypophosphataemia needs to be examined in patients with progressive encephalopathy. Moreover, since hypophosphataemia responds to treatment, it should not be difficult to test the hypothesis.

- <sup>1</sup> Strauss, M B, and Welt, L G, *Diseases of Kidney*, 2nd edn, p 335. Boston, Little Brown, 1971.
- <sup>2</sup> Alfrey, A C, et al, *Transactions of the American Society for Artificial Internal Organs*, 1972, **18**, 257.
- <sup>3</sup> Barratt, L J, and Lawrence, J R, *Australian and New Zealand Journal of Medicine*, 1975, **5**, 62.
- <sup>4</sup> Mahurka, S D, et al, *Lancet*, 1973, **1**, 1412.
- <sup>5</sup> Burks, J S, et al, *Lancet*, 1976, **1**, 764.
- <sup>6</sup> Ward, M K, et al, *Abstracts 13th Congress European Dialysis and Transplant Association*, Hamburg, 1976, p 34.
- <sup>7</sup> Flendrig, J A, Kruis, H, and Das, H A, *Abstracts 13th Congress European Dialysis and Transplant Association*. Hamburg, 1976. p 35.
- <sup>8</sup> Chokroverty, S, et al, *Journal of Neurology, Neurosurgery, and Psychiatry*, 1976, **39**, 411.
- <sup>9</sup> Platts, M M, Moorhead, P J, and Grech, P, *Lancet*, 1973, **2**, 159.
- <sup>10</sup> Wardle, E N, *Lancet*, 1973, **2**, 47.
- <sup>11</sup> Lyle, W H, *Lancet*, 1973, **2**, 271.
- <sup>12</sup> Manzler, A D, and Schreiner, A W, *Annals of Internal Medicine*, 1970, **73**, 409.
- <sup>13</sup> Gallery E D M, Blomfield, J, and Dixon, S R, *British Medical Journal*, 1972, **4**, 331.
- <sup>14</sup> Blomfield, J, McPherson, J, and George, C R P, *British Medical Journal*, 1969, **2**, 141.
- <sup>15</sup> Blomfield, J, Dixon, S R, and McCredie, D A, *Archives of Internal Medicine*, 1971, **128**, 555.
- <sup>16</sup> Alfrey, A C, LeGendre, G R, and Kaehny, W D, *New England Journal of Medicine*, 1976, **294**, 184.
- <sup>17</sup> Berlyne, G M, et al, *Lancet*, 1972, **1**, 564.
- <sup>18</sup> Parsons, V, et al, *British Medical Journal*, 1971, **4**, 273.
- <sup>19</sup> Berlyne, G M, et al, *Lancet*, 1970, **2**, 494.
- <sup>20</sup> McLaughlin, A I G, et al, *British Journal of Industrial Medicine*, 1962, **19**, 253.
- <sup>21</sup> *Bergengram Abstract 2nd International Workshop on Phosphate*. Heidelberg, 1976.
- <sup>22</sup> Lotz, M, Zisman, E, and Bartter, F C, *New England Journal of Medicine*, 1968, **278**, 409.
- <sup>23</sup> Boelens, P A, et al, *American Journal of Diseases of Children*, 1970, **120**, 350.
- <sup>24</sup> Travis, S F, et al, *New England Journal of Medicine*, 1971, **285**, 763.
- <sup>25</sup> Ahmed, K Y, et al, *Lancet*, 1976, **2**, 439.

## Are dietitians a luxury?

The NHS employs about 700 dietitians, but their scatter over the country is extraordinarily uneven: fifty districts employ none at all; some have as many as ten. Most work in hospitals, but about 100 district dietitians have recently been appointed to co-ordinate and advise on dietetic policy. These statistics, presented last week at an informal colloquium on dietitians of the future, show the lack of any clear consensus on their place in the Health Service. Eighteen months ago Professor Macdonald's working party<sup>1 2</sup> argued a case for a reappraisal of their function, but the response was muted. Since then economic circumstances have discouraged health authorities from recruiting any new staff, and newly trained dietitians are having difficulty in finding work.

No one doubts the importance of expert advice on nutrition in health and disease, but much of the traditional work of the hospital dietitian has become obsolete in recent years. No longer are scrupulous dietary regimens prescribed for peptic ulcer, and much of the obsessional attention to detail has gone from the control of diabetics' diets. The Macdonald report suggested that dietitians should spend less time in one-to-one consultation with patients and in preparing individual diets. Speakers at the colloquium agreed that more priority should be given to advice to health educators and others concerned with changing public attitudes and to the food industry. There was no clear agreement, however, on the balance that

should be drawn between therapeutic dietetics and more general advice on hospital catering and general nutrition. Yet—as was emphasised by several speakers—if dietitians themselves do not specify the areas in which their expertise is essential then hard-pressed health authorities will not employ them. Only the profession itself can agree what its future should be (though there is a large fund of medical good will from which advice can be drawn), and it needs to do so urgently.

<sup>1</sup> *Dietitians of the Future*. A report by a working party of the Dietitians Board. London, The Council for Professions Supplementary to Medicine, 1975.

<sup>2</sup> *British Medical Journal*, 1976, **2**, 134.

## Boots, boots, boots

As we all know, disabilities arising from disease or deformities of the feet are extremely common; patients are often (for a variety of reasons) reluctant to remove their socks or stockings. When they do so various abnormalities may be disclosed, the commonest being osteoarthritis of the first metatarsophalangeal joint with considerable hallux valgus, the other toes being displaced in a number of different directions. This is essentially a disease of the western world, where boots and shoes are constantly worn, and it is much less common where sandals are the fashion or people walk barefoot. Women suffer more than men, as their footwear is usually more constricting, and they will suffer discomforts for years in the name of fashion that no man would tolerate for a day. It is no wonder that chiropody is so popular and so necessary.

The Royal National Hospital for Rheumatic Diseases in Bath has long been interested in and working on this problem and recently Dixon and his colleagues<sup>1</sup> completed a study on the great toe as a clinical problem in rheumatoid arthritis. Two hundred consecutive inpatients with rheumatoid arthritis were assessed for pain or deformity of the feet and of the great toe in particular. Rheumatoid arthritis may present in the metatarsal heads, and radiological signs are often seen first here; and, as Dixon *et al* point out, surgery on the big toe before the true diagnosis has become apparent may cause more trouble and more deformity as the disease progresses. Of these 200 rheumatoid patients some deformity occurred in 104; 12 feet were excluded because of previous foot surgery, leaving 196 great toes for detailed examination. There were 114 with hallux valgus, defined as deviation of the great toe laterally from the long axis of the metatarsal bone by over 20°; and 99 had shoe-pressure lesions.

The patients with rheumatoid disease, in contrast to those with non-rheumatoid hallux valgus, rarely showed bony exostoses or bursae, and their shoe pressure lesions affected the skin only, except in four cases where sepsis and sinus formation had occurred. Medial drift of the first metatarsal (metatarsus primus varus) was seen most commonly with severe valgus deformity of over 40°; in 32 feet bony erosions were present at the base of the metatarsal. This metatarsal medial drift is a substantial factor in the forefoot spread seen so often in rheumatoid arthritis, and this in turn renders pressure lesions over the medial aspect of the metatarsophalangeal joint more likely to occur unless particular care is taken with footwear. The great toe showed rotation deformity (hallux tortus) in 56 feet, producing painful pressure under the medial aspect of the interphalangeal joint; this could be eased