(600-700 mg) phosphorus a day. Dietary advice was reinforced at regular clinic visits, and adherence to the diet was assessed by a combination of dietary checks and measurement of ratios of urea to creatinine concentrations. The rate of progression of renal function before and during the diet was assessed by plotting the reciprocal of serum creatinine concentrations against time.³ Paired Student's *t* test was used for statistical analysis.

Full data were available on 31 patients (age range 17-65 (mean 47)), who were studied for three to 62 (mean 18) months and had a mean (SEM) serum creatinine concentration of 819 (45) $\mu\text{mol/l}$ (9.3 (0.5) mg/100 ml) and a mean serum urea concentration of 37 (2) mmol/l (222 (12) mg/100 ml) at the start of dietary treatment. Mean serum phosphate concentrations remained stable at 2.17 (0.12) mmol/l (6.7 (0.4) mg/100 ml) before the diet and 1.95 (0.08) mmol/l (6.0 (0.2) mg/100 ml) during the diet.

For the whole group there was a highly significant ($p < 0.001$) change in the slopes showing progression of disease from -1.17 (0.17) ($\times 10^{-4}$) before the diet to -0.5 (0.11) ($\times 10^{-4}$) during the diet. Twenty seven patients responded to dietary treatment, as shown by a change in the angle of the slope. The figure shows individual progression slopes for those who responded according to the serum creatinine concentrations at the start of the diet and for those who did not respond. There was also a highly significant ($p < 0.001$) change in the mean ratio of urea to creatinine concentrations from 0.049 (0.002) before the diet to 0.041 (0.001) during the diet. Only one patient with proteinuria (> 5 g/24 h) showed evidence of protein depletion with persistently reduced total serum protein and serum albumin concentrations.

Effect of low protein diet on progression of renal function in patients with chronic renal failure. Patients are grouped according to serum creatinine concentrations at the start of the diet: (a) initial concentration < 650 $\mu\text{mol/l}$ (7.4 mg/100 ml); (b) initial concentration < 850 $\mu\text{mol/l}$ (9.7 mg/100 ml); (c) initial concentration ≥ 851 $\mu\text{mol/l}$ (9.7 mg/100 ml).

Comment

This study shows that deterioration in renal function can be slowed significantly by a diet containing 35-45 g protein, which is double the amount allowed in a previous diet.⁴ The exact mechanism of the effect has not been determined and may depend on several factors including control of blood pressure and serum phosphate concentrations.¹

There are no recent guidelines on when to restrict protein intake in chronic renal failure, although the onset of uraemic symptoms used to be a criterion. Our data (figure) suggest that dietary modifications should be introduced early—that is, when serum creatinine concentrations are less than 650 $\mu\text{mol/l}$ (7.4 mg/100 ml)—to obtain maximum response. Some patients were given a long time (up to several years) free from dialysis. It has never been claimed that a low protein diet should be a substitute for efficient dialysis when that is indicated. Dietary modification early in the course of chronic renal failure, however, can delay the start of dialysis, thereby allowing more efficient use of existing dialysis facilities.

As we had sufficient data for fewer than half the patients treated further controlled studies of the role of dietary protein are required to ascertain the best method of treatment and to avoid protein malnutrition. The study highlights the need for early patient referral to specialist renal units to obtain maximum benefits from dietary modifications.

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² Maschio G, Oldrizzi R, Tessitore N, et al. Effects of dietary protein and phosphorus restriction on the progression of early renal failure. *Kidney Int* 1982;22:371-6.

³ Mitch WE, Walser M, Buffington GA, Lemann J. A simple method of estimating progression of chronic renal failure. *Lancet* 1976;ii:1326-8.

⁴ Giovannetti S, Maggiore Q. A low nitrogen diet with proteins of high biological value for severe chronic uraemia. *Lancet* 1964;i:1000-3.

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Paget's disease of bone in Ireland

Little is known about the occurrence of Paget's disease of bone in Ireland. Recent radiological surveys in 31 British towns and 13 towns in nine other European countries showed wide variations in prevalence.^{1,2} The highest values were in Britain: only in France did the prevalences reach the lowest values among the British towns.

Although Ireland was not included in the radiological surveys in Europe, information was obtained from responses to a preliminary postal questionnaire sent to a sample of radiologists in every Western European country. The questionnaire inquired about the average frequency with which Paget's disease was seen as either the principal abnormality or, more often, as an incidental one. A map based on the 1416 responses showed a pattern of distribution similar to that obtained in the subsequent radiological surveys.

Replies from four centres in Eire suggested that the frequency in Dublin was somewhat lower than that in Britain and that the frequency in Cork, Limerick, and Galway was much lower. Replies from Belfast (received during a pilot study of the questionnaire) indicated a frequency similar to that in Dublin.

Present study and results

In order to corroborate the findings in Eire radiological surveys were carried out in Dublin and Galway. The method used was identical with that in previous surveys.² A consecutive sample of abdominal radiographs of people aged 55 and over was taken from films stored in hospitals. The selected films showed the entire pelvis and sacrum, the femoral heads, and all lumbar vertebrae—sites that are affected in 95% of patients with Paget's disease. The numbers of radiographs for men and women were similar. Standardised criteria were used for diagnosing the disease.

The table gives the results. The prevalence rates in each sex were directly age standardised. As in other surveys the combined population of the 14 British towns initially studied was used as the standard, so that the results of all subsequent surveys were directly comparable. The prevalence

Prevalence of Paget's disease among hospital patients aged 55 years and over

	No of patients	No with disease	Prevalence (%) of Paget's disease		
			Men* (n = 884)	Women* (n = 768)	Both sexes†
Dublin	938	15	1.8	1.6	1.7
Galway	714	4	0.6	0.7	0.7

*Age standardised rates.

†Age and sex standardised rates.

of 1.7% in Dublin was lower than those in any of the 31 British towns surveyed, where the range was 8.3% to 2.3%. It was also lower than the values recorded in the three French towns surveyed (range 2.7% to 2.0%). The prevalence of 0.7% in Galway was comparable to values in European towns outside Britain and France (range 1.3% to 0.4%). The numbers of cases in Dublin (15) and Galway (4) were too small to give reliable age specific rates, but there was no evident difference in the age distribution of the disease between Eire and Britain.

Comment

The low prevalence in Dublin, situated so close to Britain, is remarkable. It is difficult to reconcile with the recent hypothesis that the disease is due to infection with a virus,³ since the virus must be

widespread in Britain. It seems necessary to postulate the existence of cofactors. There is no evident explanation for the rarity of the disease in western Eire.

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Perforation of a bronchus due to electrical injury

We report a case of electrical injury that occurred under unusual circumstances and led to bronchial perforation, mediastinal emphysema, and pneumothoraces. Despite severe bronchial mucosal damage the patient quickly made a full recovery. Pulmonary complications after electrical injury are well documented,^{1,2} but this pattern of injury has not, to our knowledge, been noted before.

Case report

A previously fit 21 year old motor mechanic was admitted to a casualty department with severe chest pain and respiratory distress. He later described the events that had led to his injury. While working with a grinding tool he salivated through an air vent in the machine casing on to a live terminal. He noticed a blue flash that entered his mouth; he was then thrown away from the tool and was immediately aware of chest pain, difficulty in breathing, and swelling of his face.

On admission to the casualty department cyanosis, haemoptysis, and surgical emphysema of head and neck were apparent. Fresh blood was noted in his mouth. The upper airway was patent, and bilateral pneumothoraces were confirmed by x ray examination. Circulation was stable, and no other injuries were apparent. Intercoastal drains were inserted, with some improvement in breathing pattern. He was then transferred to this intensive care unit.

Repeat x ray examinations showed partial re-expansion of both lungs, severe bilateral pulmonary shadowing suggesting pulmonary oedema, mediastinal emphysema, and massive gastric dilatation. Artificial ventilation was necessary because of respiratory failure due to severe pulmonary oedema and the large quantity of analgesia required for adequate pain relief. The programme of intermittent positive pressure ventilation followed methods that are standard to this unit.³ Over the next 48 hours the hypoxaemia worsened and lung compliance decreased. The alveolar arterial pressure gradient for oxygen was 45.7 kPa (343 mm Hg) (10 times the control value of 2.9-5.9 kPa (22-44 mm Hg)).

Copious amounts of bloodstained tracheal aspirate were produced. Fibreoptic bronchoscopy showed blackened and haemorrhagic mucosa with necrotic slough. A perforation in the wall of the left main bronchus was noted, 2 cm distal to the carina. This had a base of necrotic tissue, suggesting that the perforation had sealed off.

The tracheal aspirate decreased over the next six days, and gas exchange improved. The lung fields cleared, and the mediastinal emphysema resolved. Repeat bronchoscopy showed healthy regenerating bronchial mucosa with healing at the site of the perforation. On day 10 he was successfully weaned from the ventilator. A further problem was hypercatabolism with a urinary nitrogen loss in excess of 3.2 mol (45 g) a day; this was managed with intravenous feeding. He was discharged 12 days after admission and remained well, with no evidence of bronchial stenosis.

Comment

Pulmonary complications of electrocution are well known and include haemorrhage necrotising tracheobronchitis,⁴ staphylococcal pneumonia,¹ pneumonitis, atelectasis, and pleural effusions.² Although one case of bronchopleural fistula with empyema has been described,¹ the findings in our case are new.

We believe that electrical current (110 volts, alternating current, delivered through a transformer) passed up the salivary stream into the mouth and continued down the trachea into the left main bronchus. There a perforation was formed as it entered the mediastinum. This produced mediastinal emphysema, bilateral pneumothoraces, and surgical emphysema of head and neck.

We accept that this is an unusual injury and that the current usually delivered by such a machine is not of the magnitude associated with severe electrical injury. We have, however, sought alternative explanations and fully discussed the matter with the medical adviser of the Health and Safety Executive. The extremely old machine may possibly have been delivering a current in excess of that calculated, but unfortunately it was disposed of after the initial factory inspection and this theory cannot be tested. The patient's account was substantiated by the foreman who found him, and as neither stood to gain financially we see no reason why they should invent this story.

In addition, the unusual features of this case are the presence of a spontaneously healing bronchial perforation and severe pulmonary parenchymal damage that resolved rapidly with artificial ventilation. This was without recourse to specialised techniques such as surgical repair of the bronchus or differential lung ventilation.

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Acne vulgaris and hidradenitis suppurativa as presenting features of acromegaly

Production of sebum is of major importance in the pathogenesis of acne vulgaris¹ and is increased in acromegaly.² The two diseases have only rarely been associated, but we report on two patients in whom acne vulgaris was the presenting feature of acromegaly; both also developed hidradenitis suppurativa.

Case reports

Case 1—A 30 year old man presented with a two year history of severe nodulocystic acne of the face, with milder acne of the back and chest, and hidradenitis suppurativa affecting the groin and left axilla. He had had mild acne as an adolescent but had subsequently been free from active disease for several years. Besides these skin changes and mild Raynaud's phenomenon he had obviously acromegalic features and his shoe size had increased from 10 to 12 over the previous two years. Acromegaly was confirmed, and there was evidence of a small pituitary tumour. Growth hormone concentrations (19.2 mU/l) failed to decrease appreciably after oral glucose. Basal gonadotrophin concentrations were normal but did not rise in response to gonadotrophin releasing hormone. Serum concentrations of testosterone and prolactin were 10 nmol/l (2.9 ng/ml) (normal 10-30 nmol/l (2.9-8.7 ng/ml)) and 166 mU/l (normal <425 mU/l) respectively. As he was unable to tolerate bromocriptine, pergolide mesylate 50 µg daily was administered and suppressed growth hormone concentrations satisfactorily (to 2.2 mU/l) while hypophysectomy was considered. Minocycline controlled the acne, but the hidradenitis remained active.

Case 2—A 24 year old man had developed mild acne at the age of 14.