pyramidal signs.⁴ Delayed myelopathy has been described as a complication of severe electrical burns.⁴ Spastic paraplegia⁵ and quadriplegia have been recorded secondary to electrical injury but not after lightning injury. We ascribe the delay of 36 hours before the onset of symptoms in our patient to oedema of the spinal cord.

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Amanita phalloides poisoning treated by early charcoal haemoperfusion

Treatment of *Amanita phalloides* poisoning remains disappointing. Prednisone, thioctic acid, vitamin C, penicillin, cytochrome-C, and haemodialysis alone or in combination have been used with variable success.¹⁻² Perfusion of blood over coated activated charcoal has also been tried in a few cases, but always after the onset of acute liver failure.³⁻⁴ We have used charcoal haemoperfusion as early as possible after the ingestion of *A phalloides* to try to fix the toxins before they caused hepatic or renal damage.

Patients, methods, and results

Four men and four women, aged 16 to 55, were admitted to the emergency room. All had eaten mushrooms 16 hours earlier. Seven had eaten more than three—a supposedly lethal quantity. They all had severe gastrointestinal symptoms. There were no clinical or biochemical signs of hepatic failure (serum alanine transaminase (serum ALT; SGPT) concentration <40 IU/l, blood ammonia <45 µg/100 ml (32 µmol/l)). The mushrooms remaining after the meal were identified as *A phalloides*. All the patients except the one who had eaten only a small amount were treated by haemoperfusion. In three patients this was started immediately. In the remainder it was begun a few hours after an initial dialysis. All patients were given penicillin 1 000 000 U hourly and vitamin C 1 g six-hourly intravenously. Each haemoperfusion lasted three hours, and they were continued daily until there was clinical and biochemical improvement. One patient had five haemoperfusions, one had four, three had three each, and two patients had two each.

There were no technical problems. Platelet counts dropped during most of the perfusions to a mean of 80% of the starting value. The lowest count observed after one hour of haemoperfusion was $76 \times 10^9/l$ ($76\ 000/mm^3$). White blood cell counts did not change. The clinical course in each case was uneventful. Vomiting and diarrhoea subsided within 24 to 36 hours. No clinical signs of liver failure were observed, even in the two patients with high ALT concentrations. Four of the patients left hospital after three days. The gastrointestinal symptoms in the patient who was not haemoperfused were more severe and lasted longer than those in any of the others. Serum ALT concentrations responded in two different ways (table). In two patients they rose to 1920 and 310 IU/l on the third day. In the remainder there were only slight and transient rises. Changes in aspartate transaminase (AST; SGOT) concentrations closely paralleled those of ALT. In contrast, in all eight patients there were sharp changes in blood urea nitrogen (BUN) and blood ammonia concentrations. BUN fell from 21.5 ± 1.1 mg/100 ml (7.6 ± 0.4 mmol/l) to reach its lowest level of 3.5 ± 0.8 mg/100 ml (1.2 ± 0.2 mmol/l) (mean $\pm$ SEM) on day four, while blood ammonia concentrations rose from 34.9 ± 2.9 µg/100 ml (25.9 ± 2.1 µmol/l) to 56 ± 3.9 µg/100 ml (42.1 ± 2.7 µmol/l). These changes, suggestive of impaired hepatic synthesis activity,

Serum alanine transaminase (serum ALT; SGPT) concentrations (IU/l) in eight patients with mushroom (*A phalloides*) poisoning

Day No	Case No:								Mean $\pm$ SEM
	1	2	3	4	5	6	7	8	
1	51	15			10		5		
2	179	180	33	8	18	7	6	8	55 ± 27
3	1920	312	17	24	18	6	6	8	289 ± 236
4	990	142	14	5	9	5	4	4	147 ± 122
5	600	105	31	10	10	3	6	19	98 ± 73
6	460	76	47	29	10	8	12	11	82 ± 55
7	360	65	48	33	8	14	14	4	68 ± 42
8	265		32			30	13		
9	155								
10	105	40	22		30	27	14		40 ± 14
11	60	20							

were confirmed by a fall in serum cholesterol to 116 ± 11 mg/100 ml (3.0 ± 0.2 mmol/l) and a prolonged thromboplastin time (quick level 55% of normal value) four days after ingestion. The following serum concentrations remained unchanged: alkaline phosphatase, bilirubin, lactic and pyruvic acid, fibrinogen, coagulation factors V, VII, and X, calcium, and creatinine. Follow-up examination two months later showed no sequelae in any of the patients.

Comment

Since there was no way of measuring the dose of toxin we could not know whether it was lethal. But all except one patient had eaten more than three mushrooms (>50 g), which is generally accepted as a lethal dose. The wife of one patient (case 2), who had also taken the meal, was treated in another hospital by exchange transfusion and peritoneal dialysis. She was in hepatic coma for eight days, with serum ALT concentrations of 4000 IU/l, and remained in hospital for more than three months. Her husband, who had eaten the same quantity and was treated by early haemoperfusion, left hospital after six days. Seeger and Bartels have since reported that in-vitro charcoal haemoperfusion removes alpha-amanitine from water and protein solutions.⁵ Our cases provide strong circumstantial evidence that charcoal can effectively remove toxins from the blood even 24 hours after eating *A phalloides*. Further clinical trials of charcoal haemoperfusion seem justified.

We thank Dr H R Brunner for his critical review of this report.

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Effect of 24,25-dihydroxycholecalciferol on calcium absorption in proximal small intestine in uraemia

In patients who have undergone nephrectomy and patients with chronic renal failure the available 25-hydroxycholecalciferol is preferentially converted to 24R,25-dihydroxycholecalciferol (24R, 25(OH)₂D₃), the biological activity of which is uncertain, rather than to 1α,25-dihydroxycholecalciferol (1,25-(OH)₂D₃), the most potent hormonally active form of vitamin D₃. Despite the generally suppressed production of both 1,25-(OH)₂D₃ and 24R,25-(OH)₂D₃, which is reflected by, respectively, their absence¹ or low plasma

concentrations,² the patients continue to absorb appreciable amounts of calcium from the gut.

Kanis *et al*³ reported recently that 24R,25-(OH)₂D₃ at low daily doses increased intestinal absorption of calcium in anephric subjects and patients with advanced chronic renal failure, as measured by total body counter. The method reflects absorption throughout the entire intestine and does not discriminate between active transport and passive diffusion. On the other hand, Walling *et al*⁴ reported that in nephrectomised rats the duodenal, largely active, transport response was equivalent for equimolar doses of either 1,25-(OH)₂D₃ or 1α,24,25-trihydroxycholecalciferol while for 24R,25-(OH)₂D₃ it was none. We decided therefore to find out whether 24R,25-(OH)₂D₃ stimulates absorption in the proximal small intestine in man.

Patients, methods and results

Twelve patients with chronic renal failure who were not undergoing dialysis were investigated. None had been treated with vitamin D. For two months six patients were each given 1.3 μg daily of 24R,25-(OH)₂D₃ and the other six 1.0 μg daily of 1α-hydroxy vitamin D₃ (1α-OHD₃). Before and immediately after treatment serum and urinary calcium, phosphorus, and creatinine were measured. Intestinal calcium absorption was measured by concurrent use of oral and intravenous calcium tracers and calculation by deconvolution, as described by Szymendera *et al*⁵ but modified in that the oral dose of the tracer was given with 198 mg of calcium carrier as glucoheptonate instead of a test breakfast. This method, whose reproducibility exceeds 94%, measures absorption in the proximal small intestine, where calcium is taken up largely by active transport.

The table summarises the results. After small doses of 24R,25-(OH)₂D₃ the absorption increased in two patients, remained unchanged in two, and fell in two patients. Thus, the observed differences of paired results represented the natural variability, and the mean change (±SD), 2.20 ± 3.56% of the test dose, was not significantly different from zero.

The other agent, 1α-OHD₃, failed to act in one patient with polycystic kidneys but increased the intestinal absorption of calcium in the remaining five patients, who had chronic glomerulonephritis. This response was significant by the Wilcoxon signed rank test (*P* < 0.05, one tail). The increased absorption was accompanied by a rise in the serum concentration and urinary excretion of calcium. These related changes were significant (*P* < 0.05).

Comment

Our results show that treatment of uraemic patients with small doses of 1α-OHD₃ increased calcium absorption in the proximal small intestine and in turn raised serum calcium concentrations and the urinary excretion of calcium. These results are presented merely to show that the applied test showed changes that occurred after administration of an agent known to be active in chronic renal failure.

On the other hand, 24R,25-(OH)₂D₃ had no demonstrable effect on calcium absorption tested in this way. Thus, our results agree with those of Walling *et al*⁴ in that 24R,25-(OH)₂D₃ does not stimulate active calcium transfer in duodenum and proximal jejunum. The mode of action of this vitamin D₃ metabolite on calcium absorption therefore remains to be elucidated.

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Social class, smoking, and obesity

Previous studies of the effects of smoking on obesity in British men have been based on surveys of either predominantly social classes III, IV, and V¹ or classes I and II.^{2,3} In the former, smokers were consistently less obese than the non-smokers, though the smallest difference between smokers and non-smokers was in the small proportion of the sample in classes I and II.¹ In the surveys of mainly upper class men there was no difference between smokers and non-smokers.^{2,3} We have studied the relationship between smoking and obesity in working populations in North-west London with an adequate proportion of members of classes I and II as well as of III, IV, and V. Data on women as well as on men are available.

Methods and results

Information on age, social class, and smoking habits was obtained from 1339 men (aged 18-64) and 582 women (aged 18-59) interviewed during a study of ischaemic heart disease.⁴ Smokers were defined as those regularly smoking at least one cigarette, cigar, or pipe a day. The obesity index of weight/height² (kg/m²) was used (weight in standard light gown; height without shoes). Skinfold thicknesses were measured at forearm, triceps, subscapular, and suprailiac sites. In both sexes there was a lower proportion of smokers and higher proportion of non-smokers in classes I/II than in IV/V.

The results (figure) showed that in men in classes I/II smokers were significantly more obese than non-smokers (smokers mean (±SD) W/H² = 25.1 ± 2.77; non-smokers mean W/H² = 23.8 ± 2.73). In classes IV/V the reverse was seen (smokers mean W/H² = 24.7 ± 3.46; non-smokers mean W/H² = 26.3 ± 3.76). There was no difference in class III (smokers mean W/H² = 24.8 ± 3.15; non-smokers mean W/H² = 24.8 ± 3.40). Ex-smokers (not shown in figure) were significantly more obese than smokers in all classes (*P* < 0.05). In women there were no significant differences in W/H² between smokers and non-smokers in classes I/II (smokers mean W/H² = 23.1 ± 2.77; non-smokers mean W/H² = 24.0 ± 3.48) or class III (smokers mean W/H² = 24.4 ± 4.00; non-smokers mean W/H² = 24.5 ± 4.04). In classes IV/V, however, non-smokers were more obese than smokers (smokers mean W/H² = 24.1 ± 3.86; non-smokers mean W/H² = 25.8 ± 2.37). Ex-smokers (not shown in figure) were significantly more obese than smokers

Responses of uraemic patients to two-months' treatment with 24R,25-(OH)₂D₃ or 1α-OHD₃

Case No	Plasma creatinine (μmol/l)		Calcium absorption (% oral dose)			Plasma calcium (mmol/l)			Urinary calcium (mmol/day)		
	Before	After	Before	After	Difference	Before	After	Difference	Before	After	Difference
<i>Responses to 24R,25-(OH)₂D₃</i>											
1	1255	1361	8.25	8.24	0	1.82	1.77	-0.05	1.9	1.7	-0.2
2	1273	1202	23.07	24.28	0	2.02	2.02	0	2.6	2.7	+0.1
3	884	1140	12.34	7.55	-4.8	2.42	2.52	+0.10	2.0	2.9	+0.9
4	1096	1052	5.72	14.34	+8.6	2.12	2.37	+0.25	2.0	2.8	+0.8
5	707	1008	14.06	3.80	-10.3	2.40	1.97	-0.43	1.6	1.5	-0.1
6	583	610	9.76	28.19	+18.4	2.10	2.25	+0.15	3.6	4.1	+0.5
<i>Responses to 1α-OHD₃</i>											
7	707	884	9.90	15.00	+5.1	1.80	2.02	+0.22	2.7	3.3	+0.6
8	619	716	19.21	83.15	+63.9	2.40	2.52	+0.12	1.9	3.2	+1.3
9	566	619	14.59	19.18	+4.6	2.25	2.40	+0.15	1.6	2.1	+0.5
10	469	513	26.01	40.51	+14.5	1.92	2.22	+0.30	2.4	2.9	+0.5
11	380	495	13.65	51.00	+37.4	2.50	2.64	+0.14	2.4	4.0	+1.6
12	318	283	23.30	23.09	0	2.25	2.45	+0.20	1.8	1.8	0

Conversion: SI to traditional units—Creatinine: 100 μmol/l = 1.13 mg/100 ml. Calcium: 1 mmol/l = 4 mg/100 ml; 1 mmol/day = 40 mg/day.