pyramidal signs. Delayed myelopathy has been described as a complication of severe electrical burns.[4] Spastic paraplegia[5] 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 76×10^9/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±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, 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.

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

Day No	Case No: 1	2	3	4	5	6	7	8	Mean ± SEM
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							

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.

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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$(OH)_2D_3$), the biological activity of which is uncertain, rather than to 1α,25-dihydroxycholecalciferol (1,25-$(OH)_2D_3$), the most potent hormonally active form of vitamin D_3. Despite the generally suppressed production of both 1,25-$(OH)_2D_3$ and 24R,25-$(OH)_2D_3$, which is reflected by, respectively, their absence[1] or low plasma