Our patient had airways obstruction that improved after treatment. She also had diaphragmatic weakness of moderate severity, which can produce dyspnoea on exertion[4]; this may improve as the diaphragmatic weakness resolves in cases in which a treatable cause for respiratory muscle dysfunction exists.[5] In our patient the strength of quadriceps muscle[5] and function of expiratory and diaphragmatic muscles returned to within the normal range after treatment; low values for maximal static inspiratory mouth pressures seemed to be due to poor technique, a not uncommon finding with this manoeuvre. We conclude that Addison's disease can cause myopathy which may affect the respiratory muscles.

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(*Accepted 17 March 1988*)

Carotenaemia in Alzheimer's disease

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Alzheimer's disease is the commonest type of dementia, during which patients may lose a considerable amount of weight.[1] Anorexia nervosa is a disease of the young in which there is also appreciable weight loss. Patients who have anorexia nervosa have hypercarotenaemia. There is no blood test for Alzheimer's disease. If plasma carotene concentrations were increased in this condition this would be of considerable interest.

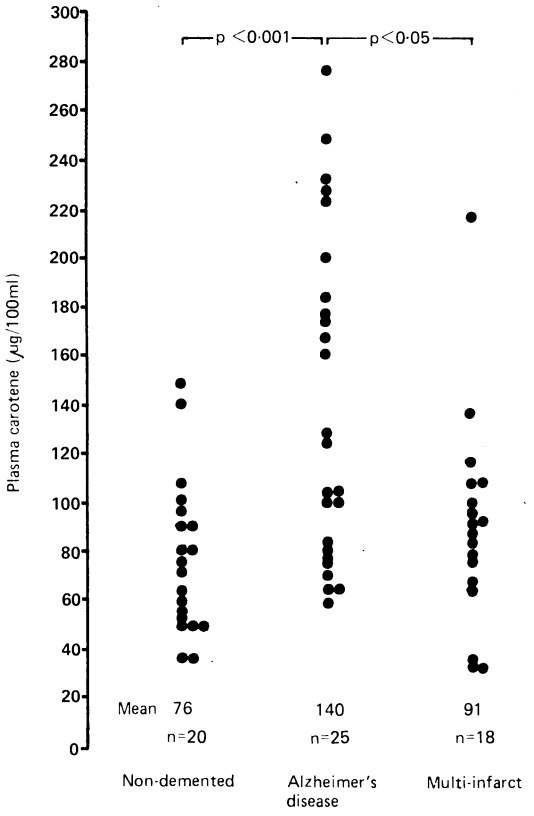
Patients, methods, and results

We studied plasma carotene concentrations in a group of elderly women who had Alzheimer's disease (mean age 86 (range 71-94)). Two control groups—namely, women who had multi-infarct dementia (mean age 83 (72-94)) and non-demented long stay elderly women (mean age 81 (70-89))—were also studied. Altogether there were 63 patients. Dementia was diagnosed from clinical criteria, and the Hachinski score[2] was used to differentiate patients who had multi-infarct dementia from those who had Alzheimer's disease. We measured plasma carotene concentrations spectrophotometrically and also studied the relation between plasma carotene and plasma vitamin A concentrations, as dietary carotene is a source of vitamin A. In addition, thyroid function and plasma lipid concentrations[3] were studied. Tests of thyroid function were performed on patients in all three groups, and lipid profiles were determined in the groups who had Alzheimer's disease and multi-infarct dementia.

A considerable difference in carotene concentrations was found among the Alzheimer group and the control groups (figure), but no significant differences in vitamin A concentrations were found. Lipid profiles were available for 12 patients in the Alzheimer group and 15 patients in the multi-infarct group. There was no significant difference between the mean values for the two groups. There were, however, significant differences ($p<0.05$, Mann-Whitney U test) between the Alzheimer group and the non-demented group in total thyroxine concentrations (mean (SEM) 86·7 (3·4) nmol/l *v* 97·6 (4·9) nmol/l) and free thyroxine indices (168·2 (4·4) *v* 194·0 (10·3)).

Comment

Many patients who have Alzheimer's disease have increased plasma concentrations of carotene. Carotene is stored in adipose tissue, and it may be thought that weight loss itself leads to the carotenaemia. Patients who have other wasting illnesses, however—for example, disseminated malignancy—tend to have low plasma carotene concentrations.[4] Carotenaemia may arise as a consequence of increased carotene intake or be associated with diabetes mellitus, hypothyroidism, or hyperlipidaemia.[3]



Plasma carotene concentrations in patients who had Alzheimer's disease and two control groups

There is no reason to suspect an increased carotene intake in the patients with Alzheimer's disease; on observation their ward diet was no different from that of fellow patients. The difference in thyroid function was small and at most was likely to contribute only partly to the carotenaemia.

Dietary carotene is a precursor of vitamin A, the conversion occurring in the gut wall. Accordingly, a positive correlation between plasma carotene and plasma vitamin A concentrations would be expected. This was seen in the non-demented group (Spearman correlation coefficient 0·51; $p<0.05$) but not in the Alzheimer group (Spearman coefficient 0·30), suggesting an abnormality in the conversion of carotene to vitamin A. The vitamin A concentration was not, however, lower than expected in the Alzheimer group, but the above hypothesis may still be

correct, as the plasma concentration of vitamin A is closely controlled.

The multi-infarct group had an intermediate mean plasma carotene concentration and showed no significant correlation between plasma carotene and plasma vitamin A concentrations (Spearman coefficient 0·32). These findings very probably result from including patients who also have Alzheimer's disease in the multi-infarct group.[5]

The cause of carotenaemia in Alzheimer's disease is unclear. Although there is considerable overlap with other groups, increased plasma carotene concentrations may be useful as a diagnostic criterion for Alzheimer's disease.

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(Accepted 12 April 1988)

Fatal pulmonary aspiration of oral activated charcoal

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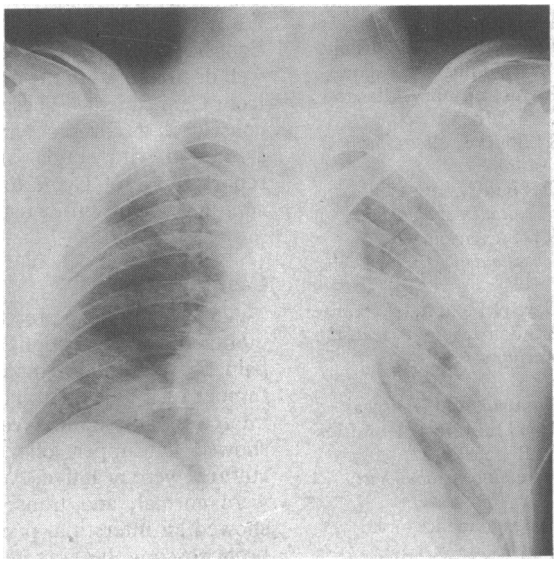
Oral activated charcoal is used to limit drug absorption after overdose, and repeated administration may enhance the removal of drugs already absorbed.[1,2] Such treatment is normally considered to be safe, and little information is available about complications. We report a case of fatal respiratory failure after pulmonary aspiration of oral activated charcoal.

Case report

A 58 year old man with a long history of epilepsy, disturbed behaviour, depression, and admissions for self poisoning was found unrousable. He took phenobarbitone, carbamazepine, and clonazepam regularly and was presumed to have taken an overdose. On examination his temperature was 33°C, pulse rate 60 beats/minute, and blood pressure 110/60 mm hg, and his chest was clear. Response to stimulation was minimal, the gag reflex was present, and there were no focal neurological signs. The arterial oxygen and carbon dioxide tensions while he was breathing 30% oxygen were 17·8 and 5·5 kPa respectively. A chest *x* ray film and an electrocardiogram were normal. Plasma concentrations of phenobarbitone and carbamazepine were reported as >220 μmol/l (>50 mg/l) and 81 μmol/l (19 mg/l) respectively.

Gastric lavage was performed. Unfortunately, the molar units in which the phenobarbitone concentration was reported were misinterpreted and a standard regimen of forced alkaline diuresis was started and activated charcoal (Medicoal) administered by nasogastric tube. The position of the tube was checked by aspiration of gastric acid, the head of the bed was raised, and a loading dose of 50 g charcoal in 200 ml water was given. Subsequently, further doses of 12·5 g charcoal were administered hourly after aspiration of the stomach contents. Vomiting occurred 12 hours later with pulmonary aspiration of gastric contents. The trachea was intubated and charcoal suctioned out of the upper airways. With the airway protected further charcoal was given through the nasogastric tube. Chest physiotherapy was given and intravenous flucloxacillin and metronidazole started. His condition improved, and at 48 hours he was alert and orientated.

Over the next 24 hours fever and tachycardia developed, and his arterial oxygen and carbon dioxide tensions were 7·7 kPa and 5·3 kPa respectively while he was breathing 40% oxygen. A chest *x* ray film showed generalised bilateral opacities (figure), and lung function deteriorated progressively. Assisted ventilation was started on day 8 but was withdrawn five days later after he suffered a major stroke. The neurological deficit and severe respiratory impairment persisted, and chest *x* ray films showed severe abnormalities; he died 15 days after admission.



Chest X ray film showing bilateral pulmonary infiltrates 48 hours after aspiration of activated charcoal

Necropsy findings included recent cerebral infarction, aspiration pneumonia, diffuse pulmonary thromboembolism, and chronic obstructive airways disease. Microscopic examination of the lungs showed inflammatory changes with charcoal and crystalline material lying free within alveoli, in alveolar macrophages and histiocytes, and in the sinusoidal histiocytes of the regional lymph nodes.

Comment

Repeated oral administration of activated charcoal is considered to be a simple, safe, and effective means of increasing the elimination of many drugs taken in overdose. The charcoal is, however, unpalatable and must be given by nasogastric tube in unconscious patients. Vomiting can occur,. and although pulmonary aspiration of charcoal is rare, it can cause obstruction, shunting of blood, and hypoxaemia, which may be exacerbated by aspiration of gastric acid.[3,4]

Medicoal contains povidone as a non-absorbable suspending agent. This can cause chronic inflammatory reactions and has been implicated in pneumonitis after inhalation of hairsprays.[5] In our patient aspiration of activated charcoal resulted in progressive and ultimately fatal respiratory failure. The pronounced reaction in the pulmonry alveoli and associated lymph nodes presumably accentuated pre-existing damage and precipitated respiratory failure. The airway should be protected when activated charcoal is given, par-