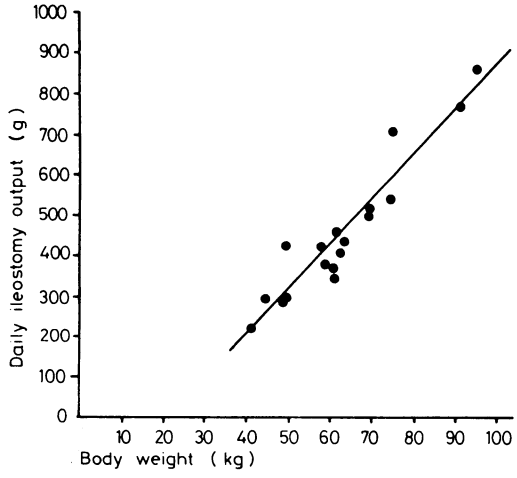
and the thickness of the mid-biceps, mid-triceps, and subscapular skinfolds were measured three times each with a Holtain skinfold calliper. Body density was calculated from the mean of the three measurements at each site by the method described by Durnin and Womersley.[3] Body fat was subsequently derived by Siri's equation,[4] and the fat-free mass calculated by subtracting the weight of body fat from body weight. Total body nitrogen was measured by in-vivo neutron activation using a sealed tube neutron generator producing 14 MeV neutrons.[5]

The mean weight of ileostomy discharge for the whole group was 459 ± 172 g/day (range 222 ± 60 g/day to 861 ± 198 g/day). Each patient had small daily variations in the weight of discharge (mean coefficient of variation 18·9%). The relation between the mean of the five daily collections of ileostomy fluid and body weight is shown in the figure. The regression equations, together with the standard error of the estimate, for the prediction of mean daily ileostomy output (IO) and the various anthropomorphic measurements (that is, height (Ht), body weight (BW), fat-free mass (FFM), and total body nitrogen (TBN)) are:

IO (g) = 10·8 Ht (cm) − 1280 ± 116 r = 0·75
IO (g) = 11·1 BW (kg) − 240 ± 57 r = 0·95
IO (g) = 12·9 FFM (kg) − 135 ± 97 r = 0·84
IO (g) = 0·42 TBN (g) − 171 ± 78 r = 0·90



Relation between daily ileostomy output and body weight.

Comment

The results show a close relation between the size of a patient and the ileostomy output. The relation is closest for body weight and the lean body mass (fat-free mass and total body nitrogen), but there is also a relation between height and the daily output of ileostomy fluid. According to these equations a so-called normal patient of 70 kg should have an average daily output from his ileostomy over five days of around 540 g (95% confidence limits are ±114 g).

These results put a new light on past and present studies of ileostomy function in health and disease. Probably they will also have important consequences for our understanding of gastrointestinal physiology.

We thank Professor R E Ellis and Drs L Burkinshaw and C B Oxby for the total body nitrogen measurements.

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Neurological manifestations and mycoplasma pneumoniae infection

We report three patients presenting with acute neurological symptoms associated with mycoplasma pneumoniae infection.

Case reports

Case 1—A pharmacist aged 31 developed an influenza-like illness with a cough productive of yellow sputum seven days before hospital admission. He did not respond to treatment with trimethoprim and sulphamethoxazole, and three days later noted paraesthesiae and progressive weakness of both legs. On admission his temperature was 37·5°C and he had an obvious spastic paraparesis, extensor plantar responses, sensory level at D4, and retention of urine. Haemoglobin was 14·3 g/dl, white cell count 19·5 × 10^9/l (polymorphs 84%), erythrocyte sedimentation rate (ESR) 12 mm in 1 hour. A chest radiograph showed consolidation of the right upper lobe. A myelogram was normal. Cerebrospinal (CSF) contained 25 polymorphs and 12 lymphocytes/μl and protein 4 g/l; no organisms were seen or cultured. Serum mycoplasma antibody titres were raised (see table). He was treated with tetracycline and physiotherapy. His paraparesis improved gradually and he recovered completely within eight months.

Case 2—A 41-year-old woman was admitted with a 24-hour history of increasing restlessness and confusion after a short influenza-like illness with sweating and dry cough. She was afebrile and could move all four limbs but was drowsy with bilateral extensor plantar responses. Blood pressure was 140/80 mm Hg and there were no other systemic signs. Haemoglobin was 14·5 g/dl, white cell count 16·5 × 10^9/l, ESR 25 mm in 1 hour. A chest radiograph was normal. A computerised tomography (CT) scan showed areas of reduced attenuation in the white matter of both cerebral hemispheres. A brain biopsy specimen showed normal appearances. CSF contained 2 lymphocytes/μl and no organisms were seen or cultured. Serum mycoplasma antibody titres were raised (see table). She improved after treatment with tetracycline, and after five months her only deficit was slight expressive dysphasia and right hemiparesis.

Mycoplasma antibody titres in the three patients (measured by complement fixation test)

Case No	Neurological complications	On admission	One month later	Four months later
1	Transverse myelitis	4096	2048	256
2	Encephalitis	1080	640	80
3	Cerebellar syndrome	2048	512	256

Case 3—A 46-year-old man developed an influenza-like illness with headaches and myalgia. Two weeks later he became dysarthric and unsteady on walking with a tendency to fall to his right. On admission he was afebrile and fully conscious but dysarthric and had inco-ordination of both arms and legs with definite ataxia of gait. There was no meningism and examination was otherwise normal. Haemoglobin was 16·9 g/dl, white cell count 7·7 × 10^9/l, ESR 15 mm in 1 hour. Chest radiograph and CT scan were both normal. CSF protein concentration was 0·85 g/l and there was no pleocytosis. Serum mycoplasma antibody titres were raised (see table). No specific treatment was given and his cerebellar signs improved gradually. Six months after the onset he had only slight residual ataxia.

Comment

These three patients presented with acute neurological syndromes, the first affecting the spinal cord, the second the cerebral hemispheres, and the third the cerebellum. Although each had an initial influenza-like illness, there were no other specific clinical features to indicate a primary mycoplasma infection.

The neurological complications of mycoplasma pneumoniae include meningoencephalitis, cerebellar syndromes, cranial and spinal polyradiculoneuritis, and transverse myelitis.[1 2] Few such cases have been reported in Britain[3 4] and only one with transverse myelitis.[5] Antecedent or concurrent chest symptoms are not invariable. Nevertheless, a causal relationship between mycoplasma pneumoniae and the neurological complications seems likely either with direct infection of the nervous system or possibly with a secondary immunological reaction. Although death and persistent neurological deficits have been reported, the prognosis is often favourable.

Our experience supports the view that tests for mycoplasma pneumoniae should be included in the investigation of patients

presenting with otherwise unexplained acute neurological symptoms, and the association may be more common than is generally recognised.

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Sialochemistry in evaluating bromhexine treatment of Sjögren's syndrome

Frost-Larsen *et al*[1] found that lacrimal gland secretion, measured by the Schirmer test, increased during bromhexine treatment for Sjögren's syndrome (SS). They did not find that bromhexine had any effect on salivary gland function, but their methods of estimating salivary secretion "were crude and of doubtful value." Changes in the composition of saliva are a sensitive indicator of salivary gland disease in SS. Significantly raised concentrations of Na, IgA, and IgG in saliva have been reported.[2 3] We therefore decided to study the effect of bromhexine on the quantity and quality of saliva in patients with SS.

Patients, methods, and results

Twenty patients under the age of 60 were divided into two groups. Group 1 (SS group) consisted of five patients with sicca syndrome who had no associated disease and who had been followed up for at least 18 months, and seven patients with Sjögren's syndrome associated only with seropositive rheumatoid arthritis. Group 2 (control group) consisted of eight patients with seropositive rheumatoid arthritis without sicca complex. The criteria for sicca and Sjögren's syndrome were decreased tear flow to less than 5 mm/min by Schirmer's test, staining of the cornea with rose bengal dye, diminished salivary flow, and abnormal salivary composition. All patients with rheumatoid arthritis fulfilled the American Rheumatism Association's criteria for either definite or classical rheumatoid arthritis.

Bromhexine 16 mg three times daily was given for four weeks. The medical treatment remained unchanged during this period. Saliva was collected before and at the end of the course of bromhexine. Total mixed unstimulated saliva was collected for 10 minutes. The rate of flow was measured and the saliva analysed for Na, IgA, and IgG as described.[2] Student's *t* test was applied for statistical analysis. Symptoms were alleviated to a varying degree during bromhexine treatment. They recurred when bromhexine was discontinued and improved when bromhexine was again given. None of the control group had sialorrhoea or excessive lacrimation. No side effects were recorded even during prolonged treatment. The SS patients had significantly higher initial concentrations of Na, IgA, and IgG when compared with the controls, whose saliva was normal (table). Bromhexine had no effect on salivary composition in the control group, but in the SS group concentrations of Na, IgA, and IgG were significantly reduced towards normal without an increase in salivary flow. Concentrations were not always uniformly reduced in all patients. In a few the concentration of only one component was significantly lower. Four SS patients took bromhexine continuously for months and a further gradual lowering of Na, IgA, and IgG concentrations was noted. Two SS patients took part twice in the trial, with a two-weeks interval between courses of treatment. The concentrations of Na, IgA, and IgG had returned to their original levels after the interval. Further treatment lowered them, as before.

Comment

The change in salivary composition towards normal without a significant increase in salivary flow raises the question whether the clinical improvement with bromhexine treatment could be due to the change in the quality of the saliva. Bromhexine reduces sputum viscosity in chronic bronchitis.[4] A reduction in sodium concentration may affect salivary viscosity, which changes with the cationic concentration.[5] How bromhexine alters salivary composition in SS is unknown. Since it lowers the concentrations of IgG and IgA perhaps it inhibits the local transformation of B lymphocytes.

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Effect of PUVA on serum 25-OH vitamin D in psoriatics

The action of ultraviolet radiation (UVR) on 7-dehydrocholesterol in the epidermis is one of the main sources of vitamin D in man. It is important to know whether treatment of psoriasis with PUVA (8-methoxypsoralen and long-wave ultraviolet light) may lead to excessive production of vitamin D and to toxic concentrations in the blood.

Patients, methods, and results

Twenty-five patients with chronic plaque psoriasis were studied. They had never had PUVA treatment and had not recently had UVR. They were irradiated two hours after taking the 8-methoxypsoralen (8-MOP), when the peak concentrations of the drug are believed to occur in blood and skin. Further details of treatment, which was given three times a week till the rash was clear and approximately weekly after that, are described elsewhere.[1]

Mean (± SD) flow rate and composition of saliva before and after bromhexine treatment in a control group and a group of patients with Sjögren's syndrome (SS group)

	Rate of flow (ml/min)		Na (mmol(mEq)/l)		IgA (mg/l)		IgG (mg/l)	
	Before	After	Before	After	Before	After	Before	After
Control group (n = 8)	0·30	0·31	4·6 ± 1·4	4·3 ± 1·3	97 ± 52	105 ± 61	< 10	< 10
SS group (n = 12)	0·08	0·07	22·6 ± 17·1	13·6 ± 12·3	423 ± 205	296 ± 174	133 ± 42	45 ± 39

Statistical analysis
(1) Pretreatment control/pretreatment SS: Na $P < 0.01$; IgA and IgG $P < 0.001$ (Student's *t* test).
(2) Pretreatment/on treatment. A control group—no significance. B SS group—Na and IgA $P < 0.001$; IgG (n = 6) $P < 0.01$; rate of flow, no significance (*t* test for paired comparison).