high risk of infection to which such children are exposed. A policy of BCG vaccination at birth for the children of these ethnic groups born in this country, as recommended by the Department of Health and Social Security* and the joint tuberculosis committee of the British Thoracic Society,⁹ would probably protect most immigrants' children. It is equally important to reduce the likelihood of exposure to infection by earlier diagnosis and prompt treatment of the index cases, as it is those with pulmonary tuberculosis positive on smear testing who are the main source of infection. Clearly, exposure to the risks of infection in the Indian subcontinent, which occurs when immigrants visit their country of origin¹⁰ or when their relatives visit this country, will contribute to the high level of infection in this ethnic group.

The white and Indian subcontinent patients also differed in the proportion of previously untreated patients with initial resistance to one or more antituberculous drugs in 1983. The very low rates (1.6%) in both surveys for the white population reflect the high standards of chemotherapeutic practice over many years in the indigenous population of the United Kingdom. In contrast, 12.8% of the strains from the Indian subcontinent patients were resistant in 1983, a higher proportion than in 1978-9 (7.5%). Initial resistance was therefore more common in the Indian subcontinent patients in both surveys and had increased in the second, although in most cases resistance was to a single drug, usually streptomycin. Resistance to streptomycin alone, however, is probably of little clinical importance as use of this drug in primary chemotherapy has declined in the United Kingdom.¹¹

This report highlights the main findings of the survey but does not explore many important aspects in detail—for example, the non-respiratory lesions—and further reports are in preparation.

This survey succeeded because of the enthusiastic cooperation of all (over 200) medical officers for environmental health in England and Wales, all (nearly 400) chest physicians and general physicians with a special interest in chest diseases, and over 400 clinicians in other specialties who provided information on the patients; the nursing staff, secretaries, and records officers of many hospitals and chest clinics; and the infectious diseases clerks and other local authority staff. We thank the Department of Health and Social Security, the Welsh Office, the British Thoracic Society, and several other specialist groups for their help. Dr Ian Sutherland and Dr Victor Springett gave valuable advice, particularly during the analysis.

References

- 1 Fox W, Wiener A, Mitchison DA, Selkon JB, Sutherland I. The prevalence of drug-resistant tubercle bacilli in untreated patients with pulmonary tuberculosis: a national survey 1955-56. *Tubercle* 1957;38:71-84.
- 2 Miller AB, Tall R, Fox W, Lefford MJ, Mitchison DA. Primary drug resistance in pulmonary tuberculosis in Great Britain: second national survey. *Tubercle* 1966;47:92-108.
- 3 Medical Research Council Tuberculosis and Chest Diseases Unit. National survey of tuberculosis notifications in England and Wales 1978-9. *Br Med J* 1980;281:895-8.
- 4 British Tuberculosis Association. Tuberculosis among immigrants to England and Wales: a national survey in 1965. A report from the research committee of the British Tuberculosis Association. *Tubercle* 1966;47:145-56.
- 5 British Thoracic and Tuberculosis Association. A tuberculosis survey in England and Wales 1971: the influence of immigration and country of birth upon notifications. A report from the research committee of the British Thoracic and Tuberculosis Association. *Tubercle* 1973;54:249-60.
- 6 Department of the Environment. *National dwelling and housing survey*. London: HMSO, 1979.
- 7 British Thoracic and Tuberculosis Association. Tuberculosis among immigrants related to length of residence in England and Wales. *Br Med J* 1975;iii:698-9.
- 8 Department of Health and Social Security. *Immunisation against infectious disease*. London: HMSO, 1984.
- 9 Joint Tuberculosis Committee of the British Thoracic Society. Control and prevention of tuberculosis: a code of practice. *Br Med J* 1983;287:1083-156.
- 10 McCarthy OR. Asian immigrant tuberculosis—the effect of visiting Asia. *Br J Dis Chest* 1984;78:248-53.
- 11 Medical Research Council Tuberculosis and Chest Diseases Unit. Treatment of pulmonary tuberculosis in patients notified in England and Wales in 1978-79: chemotherapy and hospital admission. *Thorax* 1985;40:113-20.

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Lesson of the Week

Non-oliguric renal failure during treatment with mefenamic acid in elderly patients: a continuing problem

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Mefenamic acid, a non-steroidal anti-inflammatory drug of the anthranilic acid group, is widely used for the relief of mild to moderate pain. Like other non-steroidal anti-inflammatory agents, mefenamic acid has been reported as having a range of nephrotoxic effects,¹ particularly in the elderly.² None the less, the drug continues to be widely used at high dosage in elderly patients. We describe five patients referred to a geriatric medicine unit during one month with non-oliguric renal failure developing during treatment with mefenamic acid.

Mefenamic acid should be prescribed with great caution, if at all, to old people, particularly those receiving diuretics

Case histories

All patients were receiving mefenamic acid 500 mg three times a day. Tables I and II show their clinical and biochemical details. All the patients had developed diarrhoea and vomiting after beginning mefenamic acid, leading to depletion of fluid and electrolytes. Two patients (cases 1 and 2) had very poor oral intake of fluid, while four (cases 2-5) were also receiving diuretics, which were continued despite their deteriorating clinical state. In case 4 the serum potassium concentration at presentation was 8.1 mmol(mEq)/l. Discontinuation of mefenamic acid in all cases, and also the diuretic in cases 2-5, together with appropriate fluid replacement was associated with complete biochemical and clinical recovery in four patients. In case 4 the patient improved initially but died six weeks later from ischaemic heart disease. Postmortem examination showed changes in the kidneys compatible with chronic pyelonephritis and sustained hypertension, but no interstitial nephritis or papillary necrosis was noted.

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Discussion

Non-steroidal anti-inflammatory agents inhibit the production of renal prostaglandins which appear to have important autoregulatory effects on renal blood flow, particularly in states where the plasma renin activity is increased, such as dehydration and diuretic treatment.¹ Mefenamic acid commonly causes gastrointestinal disturbances,³ which may include vomiting, diarrhoea, and even steatorrhoea.⁴ Gastrointestinal disturbances, particularly diarrhoea, occurred in all of our patients, and despite the manufacturer's

despite the manufacturer's recommendation to the contrary.⁵ It would seem prudent to advise any patient receiving the drug of the importance of gastrointestinal upset as a side effect and to warn the patient to discontinue treatment immediately should side effects develop. It is also noteworthy that all of our patients were receiving mefenamic acid for conditions that would have been more appropriately treated by other methods. The prescription of mefenamic acid in all of these cases was an attempt to control symptoms only, without any effort being made to treat the primary disorder.

TABLE 1—Clinical details of patients studied

Case No	Age (years)	Sex	Daily dose of mefenamic acid (g)	Underlying diseases	Duration of treatment	Other drugs	Presentation
1	78	M	1.5	Gangrenous toe; peripheral vascular disease	12 Months	Triazolam, inositol nicotinate, chlordiazepoxide	Depression, nausea, vomiting, drowsiness, dehydration
2	75	M	1.5	Lumbar spondylosis; depression	12 days	Moduretic (amiloride, hydrochlorothiazide)	Vomiting, diarrhoea, depression
3	83	M	1.5	Ischaemic heart disease causing shoulder pains	30 days	Oxprenolol, isosorbide mononitrate, bendrofluazide	Nausea, vomiting, diarrhoea, dehydration
4	74	M	1.5	Burns and cellulitis of legs	47 days	Frusemide, spironolactone	Vomiting, diarrhoea, heart failure, leg ulcers
5	84	F	1.5	Steroid induced osteoporotic vertebral collapse	4 days	Frusemide, ferrous sulphate, prednisolone	Vomiting, diarrhoea, dehydration

TABLE II—Biochemical details of patients studied

Case No	Premorbid			Morbid			After treatment			Duration of recovery phase
	Urea (mmol/l)	Creatinine (μmol/l)	HCO ₃ (mmol/l)	Urea (mmol/l)	Creatinine (μmol/l)	HCO ₃ (mmol/l)	Urea (mmol/l)	Creatinine (μmol/l)	HCO ₃ (mmol/l)	
1	7.0	140	28.1	68.5	650	12.8	6.6	176	25.1	10 days
2		Not available		36.0	268	18.5	6.1	97	30.0	12 days
3	6.3	130	26.0	27.3	270	14.2	8.4	184	16.5	6 weeks
4	14.1	161	27.0	49.0	213	19.0	25.0	153	29.0	Until death 6 weeks after admission
5	10.0	166	27.2	27.9	662	15.0	3.6	70	24.1	9 days

Conversion: SI to traditional units—Urea: 1 mmol/l≈6.0 mg/100 ml. Creatinine: 1 μmol/l≈0.01 mg/100 ml. Bicarbonate: 1 mmol/l≈1mEq/l.

recommendation in the data sheet⁵ the drug was not discontinued. We therefore believe that the gastrointestinal toxicity of mefenamic acid in our patients caused fluid and electrolyte depletion, sensitising the patients to the nephropathic effects of the drug mediated by inhibition of intrarenal prostaglandin synthesis. In addition, four of our patients were receiving diuretics, which may potentiate the nephropathic effects of the drug by contributing to extracellular fluid depletion. Two of the patients were receiving potassium conserving diuretics, and it has been suggested that triamterene in particular may predispose to the nephrotoxic effects of non-steroidal anti-inflammatory drugs.⁶ Concomitant diuretic treatment was also being given in several reported cases of mefenamic acid nephropathy.^{7,8} Robertson *et al*² considered that renal papillary necrosis was the potential mechanism of nephropathy induced by mefenamic acid, but in two patients renal biopsy disclosed interstitial nephritis.^{7,9} Our fourth patient who had serious pre-existing renal disease showed neither of these renal conditions. It is not clear whether the gastrointestinal and renal toxicity of mefenamic acid is dose related. In the absence of good evidence that the pharmacokinetics of mefenamic acid are unchanged in elderly subjects it seems wise to recommend the use of lower doses of this drug in old people. Another cause for concern is that our patients with vomiting and diarrhoea continued to receive mefenamic acid

These five cases emphasise the dangers of using mefenamic acid in elderly patients. We conclude that if mefenamic acid is to be used at all as an analgesic in elderly subjects, then it must be used in the minimum effective dose, and that patients should be instructed to discontinue the drug should any evidence of gastrointestinal upset occur.

References

1 Clive DM, Stoff JS. Renal syndromes associated with non-steroidal anti-inflammatory drugs. *N Engl J Med* 1984;310:563-72.
2 Robertson CE, Ford MJ, Van Someren V, Dlugolecka M, Prescott LF. Mefenamic acid nephropathy. *Lancet* 1980;ii:232-4.
3 Reynolds JEF, ed. *Martindale; the extra pharmacopoeia*. 28th edition London: Pharmaceutical Press, 1982: 262.
4 Marks JS, Gleeson MH. Steatorrhoea complicating therapy with mefenamic acid. *Br Med J* 1975;iv:442.
5 Parke-Davis & Co. *ABPI data sheet compendium*. Datapharm Publications Ltd, 1984:5.
6 McCarthy JT, Torres VE, Romero JC, Woehor DN, Velosen JA. Acute intrinsic renal failure induced by indomethacin: role of prostaglandin synthetase inhibition. *Mayo Clin Proc* 1982;57:289-96.
7 Woods KL, Michael J. Mefenamic acid nephropathy. *Br Med J* 1981;282:1471.
8 Drury PL, Asirdas LG, Bulger GV. Mefenamic acid nephropathy: further evidence. *Br Med J* 1981;22:865-6.
9 Venning V, Dixon A, Oliver DO. Mefenamic acid nephropathy. *Lancet* 1980;iii:745-6.

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What is wry neck?

Torticollis, or in common parlance "wry neck," means simply a curvature of the cervical spine. This may occur after birth injury, sternomastoid tumour, or inflammatory cervical adenopathy, especially in children. It may also happen spasmodically from a variety of neurological causes and may become spastic, or fixed. Acute torticollis is analogous to acute lumbago and is

probably due to an acute derangement of a joint in the cervical spine, though seldom can the doctor be certain whether this originates from a disc or facet joint lesion. Root symptoms and signs occasionally follow repeated attacks but myelopathy is extremely rare. Manipulative treatment is often successful provided that there is no question of a sinister pathology or vascular insufficiency.—J A MATHEWS, consultant physician (rheumatology), London.