of hypertension (2.2) and heart disease (1.2). If all three factors were present in the man the increased risks were 4.2 and 2.0. For women similar increases in prevalence of disease occurred with even higher risks.

Discussion

In this study we found that 42% of all people were reported to snore: over 70% of husbands and just over 50% of wives. The prevalence of snoring increased with age up to the seventh decade and then decreased slightly. These findings are similar to those of Lugaresi *et al*,¹ but we found a higher prevalence of

asleep⁴ could be the causative factors for the associations that we have shown.

Our findings reproduce and enlarge on those of Lugaresi *et al* and give strong evidence that snoring is associated with an increased risk for cardiovascular diseases, especially hypertension. We found that men who snored, smoked, and were obese had a risk of hypertension 4.2 times greater than non-snorers. Berry and Block have recently shown that positive pressure on the nasal airway reduces both the frequency of snoring and the desaturation of oxygen in heavy snorers.¹⁷ Case reports and personal experience of the authors indicate that to stop smoking or lose weight, or both, can reduce snoring considerably in some

TABLE IV—Hypertension and heart disease by decade and sex. (Values are percentages)

Decade	Men				Women			
	Hypertension		Heart disease		Hypertension		Heart disease	
	Snorers	Non-snorers	Snorers	Non-snorers	Snorers	Non-snorers	Snorers	Non-snorers
Fifth	7		5	3	12	5		2
Sixth	22	13	8		10		12	4
Seventh	28	14	17	14	15	27	7	4
Eighth	27	13	29	20	40	25	29	25
Ninth	12	11	43	33	40	17	40	25

TABLE V—Prevalence of disease compared with smoking, obesity, and snoring. (Values are % (numbers) of cases)

	Men		Women	
	Hypertension	Heart disease	Hypertension	Heart disease
None	8 (60)	13	7 (149)	8
Smoking	11 (17)	5	37 (16)	6
Obesity	12 (8)		22 (9)	
Smoking and obesity	0 (2)	0	0 (0)	0
Snoring	19 (187)	15	20 (138)	13
Snoring and smoking	15 (85)	9	10 (39)	13
Snoring and obesity	29 (51)	16	39 (28)	11
All	35 (23)	26	33 (12)	25

snoring in all categories. As reported by Lugaresi *et al* we found that snoring is associated with an increase in cardiovascular disease (especially hypertension)^{2,11} and most other common chronic medical conditions. Snoring is particularly associated with obesity and smoking, both of which have been implicated in the prevalence of cardiovascular and other medical problems. When we analysed our data those subjects who were not overweight and did not smoke but snored continued to have an increased prevalence of cardiovascular disease. Thus we concluded that an association exists between snoring and cardiovascular and other medical conditions.

Several authors have speculated on the reasons why snoring might be associated with increased risks of morbidity and mortality.⁵⁻⁸ It has been shown that some snorers have pulmonary hypertension during sleep¹⁰ and that some children who snore have clinical and electrocardiographic findings of pulmonary hypertension.¹² This pulmonary hypertension and the excess desaturation of oxygen found in some snorers while they are

people. Similarly, corrections of anatomical abnormalities have been reported to relieve some snorers. In the light of our findings investigation of the effects of these and other corrective measures for snoring with respect to people's health is warranted.

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SHORT REPORTS

Carbamazepine induced systemic lupus erythematosus: case report

Anticonvulsants (phenytoin and ethosuximide) have been implicated in drug induced systemic lupus erythematosus.¹ Carbamazepine (Tegretol), however, which is widely used in the treatment of grand mal and complex partial seizures, has not been definitely incriminated in this disease. I report a case of systemic lupus erythematosus associated with carbamazepine which resolved rapidly on withdrawal of the drug.

Case report

The patient, a 30 year old housewife, initially presented with complex partial seizures. Investigation disclosed a space occupying lesion in the right temporal lobe and at operation a grade II astrocytoma was partially removed. Her seizure disorder persisted postoperatively and was treated with carbamazepine 1 g and phenobarbitone 120 mg daily.

A year later she developed pain and stiffness of her joints, affecting hands, feet, wrists, elbows, knees, and hips. She noticed a blotchy rash on her hands and feet and her eyes became sore and itchy. During hospital admission she developed pleuritic chest pain. Examination findings were normal apart from a vasculitic rash on hands and feet. There was no joint swelling and results of an ophthalmological examination were normal. Investigations

showed a haemoglobin concentration of 124 g/l; white cell count $2.4 \times 10^9/l$ (76% neutrophils, 20% lymphocytes, 4% monocytes); platelet count $200 \times 10^9/l$; sedimentation rate 44 mm in first hour; normal urea and electrolytes and liver function values; antinuclear factor titre positive at 1/1280; rheumatoid arthritis haemagglutination titre positive at 1/80; and double stranded deoxyribonucleic acid (DNA) binding 94 U/ml (normal 25 U/ml). She was a slow acetylator and her HLA type was A1 AW24 B7 B17 DR2, DR3. Chest x ray appearances were normal.

In view of the polyarthritis, cutaneous vasculitis, pleurisy, leucopenia, positive antinuclear factor titre, and raised DNA binding value, a diagnosis of carbamazepine induced systemic lupus erythematosus was made. The carbamazepine was withdrawn but the phenobarbitone continued. Prednisolone 30 mg daily was added. The patient improved rapidly and prednisolone was stopped after six months. Subsequently she remained well for 12 months. The DNA binding returned to normal but her antinuclear factor titre remained positive at 1/160.

Comment

The rapid resolution of this patient's illness on withdrawal of carbamazepine and lack of recurrence over the next year strongly suggest that the systemic lupus erythematosus was drug induced. The patient was a slow acetylator and possessed HLA-DR3, both of which may predispose to drug induced lupus erythematosus.² Several case reports suggestive of carbamazepine induced systemic lupus erythematosus have appeared^{3,4} or been reported to the Committee on the Safety of Medicines and Ciba-Geigy Pharmaceuticals, but many have lacked confirmatory laboratory investigations. This case report shows that tests for antinuclear factor and DNA binding should be done when there is multiorgan disease associated with idiosyncratic reactions to drugs including carbamazepine. A small study of antinuclear factor in nine epileptics receiving only carbamazepine showed that seven had antibodies to soluble nucleoprotein.⁵ Carbamazepine should therefore be added to the list of anticonvulsants which may induce systemic lupus erythematosus, though the risk is considerably less than with phenytoin.

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Polymicrobial infection in campylobacter enteritis

Campylobacter subsp *jejuni* has recently been recognised as a common cause of bacterial gastroenteritis in children. We have determined the prevalence of polymicrobial enteritis with *Campylobacter* compared with other enteric pathogens in our hospital.

Prevalence of *C jejuni* among enteric pathogens isolated from cases of polymicrobial enteritis during December 1980 to October 1983

	Organisms isolated from mixed infections with <i>Campylobacter</i>					Organisms isolated from mixed infections without <i>Campylobacter</i>			
	<i>Salmonella</i>	<i>Shigella</i>	<i>E coli</i> *	<i>Salmonella</i> + <i>E coli</i> *	<i>Shigella</i> + <i>E coli</i> *	<i>Salmonella</i> + <i>Shigella</i>	<i>Salmonella</i> + <i>E coli</i> *	<i>Shigella</i> + <i>E coli</i> *	Total
No of patients	8	8	2	2	2	1	4	2	7

*Enteropathogenic *E coli*.

Materials, methods, and results

All stool specimens submitted to the bacteriology laboratory from December 1980 to October 1983 were cultured for *Campylobacter* and other gastrointestinal pathogens. Specimens were inoculated on to a selective plating medium (modified Skirrow's medium; Skirrow's medium and amphotericin B 2 mg and novobiocin 5 mg/l) and the plates incubated at 42 C for 48 hours in 5% oxygen, 10% carbon dioxide, and 85% nitrogen. *Campylobacter* was identified by Gram's stain and biochemical characteristics.

A retrospective survey was made of the records of all children with enteric pathogens, including *C jejuni*, cultured in stools.

A total of 945 specimens from 661 children were tested during the three years. *C subsp jejuni* was isolated from the stools of 131 children. During the same period *Shigella* was found in 354 children, *Salmonella* in 122, and enteropathogenic *Escherichia coli* in 54. In 22 of the 131 cases (3.3%) *Campylobacter* was found together with other enteric pathogens—namely, *Salmonella* (eight cases), *Salmonella* and *E coli* (two), *Shigella* (eight), *Shigella* and *E coli* (two), and *E coli* (two) (see table). Mixed infections without *C jejuni* were found in five patients with *Salmonella* (0.8%) and two with *Shigella* (0.3%).

Comment

Since Skirrow devised a selective medium for identifying *C subsp jejuni* this organism has come to be recognised as an important enteric pathogen in man, causing acute and subacute illness. Many clinical microbiology laboratories are now using methods for isolating this organism from faecal culture, and many studies have been performed evaluating the clinical and epidemiological features of infection with *Campylobacter*, *Salmonella*, enteropathogenic *E coli*, and *Shigella*. Lassen and Kapperud reported four years' experience with *C jejuni* in Norway. Out of 249 patients infected with *C jejuni*, *Salmonella*, or *Shigella*, 40 (16.1%) had mixed infections—25 with *C jejuni* and *Salmonella*, 13 with *C jejuni* and *Shigella*, and two who were excreting all pathogens.¹

Pitkanen *et al* summarised the clinical picture and epidemiological characteristics of infection with *C jejuni* in 186 patients admitted to hospital in Finland during a three year period.² A mixed infection was detected in 14 patients, and the most frequent pathogen in these was *Salmonella*, which was isolated in 11 cases. In Israel Shmilovitz *et al* succeeded in isolating *C jejuni* from 2430 patients in two years. In 176 of them (7.2%) *C jejuni*, *Salmonella*, or *Shigella* was isolated concomitantly—*C jejuni* and *Salmonella* in 104 patients, *C jejuni* and *Shigella* in 69, and *C jejuni*, *Salmonella*, and *Shigella* in three.³

In our series we checked for mixed infections with other enteric pathogens and found a surprisingly low incidence of mixed infections with all other pathogens compared with those with *C jejuni* (Student's *t* test: *p* < 0.005). This has not been noted before and may have therapeutic implications. Robins-Browne *et al* found that erythromycin was ineffective in campylobacter associated enteritis and attributed this in part to the fact that all but eight of their 26 patients were infected with at least one erythromycin resistant enteropathogen in addition to *C jejuni*.⁴ The high prevalence of polymicrobial enteritis in their group is considered a normal phenomenon in developing countries.

Many mixed infections appear to show microbial synergism, mediated by any of four basic types of interaction. A micro-organism may lower host resistance and thereby increase the likelihood of invasion by another micro-organism, facilitate colonisation of potential hosts by providing elements essential to the growth of other micro-organisms, or increase the virulence of other micro-organisms.⁵

Campylobacter may diminish the ability of the host to resist invasion by other pathogens. It may achieve this by altering cellular immunity, humoral immunity, or the anatomy of the host. In a previous study we found that *Campylobacter* may facilitate colonisation by enteric pathogens, especially in immunocompromised patients.

Little is known about the specific mechanism of synergistic infection, and the pathogenicity of campylobacter infection is not clear. Further work is needed to elucidate the exact mechanism of campylobacter polymicrobial infections.