especially about drinking and driving, might have influenced this more impressionable age group. Another possible explanation might be the increase in the unemployment rate among young people between 1978 and 1985. Some of this newly formed section of the unemployed might be expected to drink less because they could not afford it.

Among women the picture was different. In 1985 women consumed on average more alcohol than in 1978 (5·1 units *v* 4·2 units). Although the percentage of non-drinkers rose significantly (51% *v* 42%), the average alcohol consumption per woman drinker was significantly higher in 1985 than in 1978 (10·4 units *v* 7·2 units). Thus women seem to have "polarised" to some degree. More women were not drinking, but those who were drinking drank more. Apart from the current vogue for "healthy living," one factor influencing the increase in the numbers of non-drinkers may have been government sponsored negative advertising. This, however, was having no effect on those women who were drinking. For them the relative cheapness of alcohol in 1985 compared with 1978 could well have been a more relevant factor.[6,7] Likewise, the pressure of positive advertising from the Brewers Society plus the liberation of women, such that their lifestyles (and therefore patterns of drinking) more closely resembled those of men, may also have contributed to this increase.[8]

Whatever the reason for this increase, it is important to reverse this trend. Women are more sensitive to alcohol damage,[9] and the inherent risk of increased consumption must therefore be made clear. Perhaps further advertising campaigns should highlight this risk.

The findings on regional variations must be considered with caution because of the small sample size. Nevertheless, rather broad agreement was seen between our survey and that of Wilson.[1] Both tended to show regional variation in consumption, with the northern areas, London, and Wales consuming the most, while East Anglia, the south west, and the south east consumed the least.

Like Wilson, we found that social class had little effect on alcohol consumption. Because of differences in definitions no formal comparisons were made between results from 1978 and 1985. These findings are, however, at variance with some others. A survey undertaken in a single London suburb suggested that men in the higher social classes drank less than those in the lower classes.[10] This effect was reversed in women. Possible reasons for these different findings include great differences in the geographical spread of the work and a large time interval between the surveys.

Marital status, in both this and the 1978 survey, had some influence on alcohol consumption. Living with (or having lived with) another person seemed to moderate drinking.

Review of the limited data for underage drinking (16-17 years) did not suggest particularly heavy consumption in this group. Nevertheless, many people (65%) under the legal age limit were drinking. This underage group showed similar consumption patterns to the other age groups, except for those over 65. Given the relative immaturity of 16 and 17 years olds such adult consumption patterns are likely to produce an increased incidence of alcohol related problems.

Consumption per head of alcohol correlates well with certain markers of alcohol morbidity and mortality (drunkenness convictions, drinking and driving offences, first admissions to hospital for alcohol dependence, death from cirrhosis of the liver).[11] Therefore we hope the fact that overall alcohol intake did not change between 1978 and 1985 heralds an eventual downturn in consumption with a resulting reduction in alcohol related harm.

Such a pattern was in fact seen over the same seven years in several European countries. Per capita consumption in litres of absolute alcohol fell between 1978 and 1985 in France (16·3 to 13·9 litres), Germany (12·9 to 11·3 litres), Italy (12·0 to 9·4 litres), and Spain (14·3 to 11·8 litres). Consumption in these countries in 1978 was, however, considerably greater than in the UK (7·2 litres). For those European countries with a more equivalent 1978 consumption a different pattern was seen. Thus intake increased in Belgium (10·2 to 10·8 litres), Denmark (8·9 to 9·8 litres), and Portugal (9·9 to 13·1 litres), dramatically in the last case; the Netherlands showed a small decline (8·9 to 8·7 litres). When compared with other European countries, Britain's performance was middling.

Probably the single most important reason why alcohol intake remained stable between 1978 and 1985 was the relative cost of alcohol. Over the period the "work equivalent" of a bottle of whisky changed from 2 hours 24 minutes to 2 hours 11 minutes and the percentage of disposable income spent on alcohol increased from 7·3 to 7·4%.[7] The relative cost of alcohol thus changed little, and this might well account for the observed stability in alcohol consumption.

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SHORT REPORTS

First multiresistant pneumococcus in Britain

Multiresistant pneumococci were first detected in clinical specimens in South Africa in 1977 (types 6A and 19A)[1] from severe infections such as pneumonia and meningitis. Though pneumococci moderately resistant to penicillin (minimal inhibitory concentration 1 mg/l) have been isolated in Britain for years, no multiresistant isolate similar to the South African strains has been reported. We report the isolation of a type 6 strain showing multiple antibiotic resistance from the eye of a neonate in Bristol.

Case report

An eye swab was received in December 1986 from a 17 day old neonate with moderate conjunctivitis. The neonate had been delivered at home, had not received any antibiotics, and was otherwise well. Culture of the swab produced a moderate growth of *Streptococcus pneumoniae*, identified by standard methods.[2] Minimal inhibitory concentrations to various antibiotics (table) were determined by a microdilution method (Sensititre APO1, Seward Laboratories) using Isosensitest agar (Oxoid) and 3% lysed horse blood or by broth dilution (for trimethoprim and sulphamethoxazole). The isolate was found to be sensitive to rifampicin and mupirocin on disc testing by a modified Stokes method.[3] Capsular

Antimicrobial sensitivity of pneumococcus

Antimicrobial	Minimal inhibitory concentration	Antimicrobial	Minimal inhibitory concentration
Penicillin	1	Trimethoprim	16
Erythromycin	>32	Sulphamethoxazole	>64
Chloramphenicol	32	Vancomycin	0·5
Tetracycline	16		

typing was performed and the multiple antibiotic resistance confirmed by the Streptococcus Reference Laboratory, Colindale.

Nose and throat swabs were obtained from all the immediate family—father, mother, a 3 year old brother, a 2 year old sister—and the index patient. The resistant strain was grown from the nose swabs of the index patient and the 3 year old sibling. A sensitive pneumococcus was grown from the nose of the 2 year old, whereas no pneumococci were grown from the parents. The neonate's infected eye was treated for three weeks with topical penicillin (10×10^6 U/l) with clinical and bacteriological cure.

Comment

The family's travel history is of note. Though the father had made business trips to Johannesburg for two weeks in 1976 and again in 1984, of more interest was a two year residence in Spain by the family between 1983 and 1985. In a recent survey of pneumococcal carriage in children aged 4-5 years in Barcelona[4] two of 159 pneumococcal isolates were type 6 and possessed the same multiple resistance pattern as the Bristol isolate. Probably, therefore, the 3 year old sibling had acquired carriage during his time in Spain.

This infection was treatable because of the high concentration of penicillin obtainable at the site of infection with topical application. Systemic infection with this strain would be much more difficult to treat adequately, and treatment with penicillin might well fail. Eradication of carriage of multiply resistant pneumococci would not be easy; using a combination of rifampicin and fusidic acid in Johannesburg had only 65% efficacy.[5] Eradication of carriage was not attempted in our case in view of the mild nature of the infection. Evidence of spread in a susceptible population such as immunocompromised children would require a more aggressive approach. It is to be hoped that such strains do not become more widespread in Britain.

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Department of Microbiology, Southmead Hospital, Bristol BS10 5NB
J H PATON, MB, MRCPATH, senior registrar
D S REEVES, MB, FRCPATH, consultant

Correspondence to: Dr Paton.

Acquired factor VIII inhibitor associated with lung abscess

Haemoptysis in chronic bronchitis is common and may be worsened by any generalised bleeding disorder. We report on a patient who developed a lung abscess and was found to have a coagulopathy due to an acquired factor VIIIC inhibitor.

Case report

A 70 year old man with chronic bronchitis was admitted with haemoptysis; he had not noted abnormal bleeding previously. On admission his sputum was bloodstained and he had signs of consolidation of the right middle lobe, which was confirmed by chest radiography. Despite antibiotic treatment cavitation developed in the consolidated lobe; a lung tumour was excluded at bronchoscopy. Extensive superficial bruising and petechias and prolonged bleeding from venepuncture sites were noted.

Platelet count and morphology were normal. Activated partial thromboplastin time was prolonged to 84 s (normal range 21-37 s) and bleeding time to >20 minutes (normal range 2-9 minutes). Prothrombin time, thrombin time, and concentrations of fibrinogen and fibrin degradation products were within the normal range. Von Willebrand factor was 368% (normal range 60-160%); platelet aggregation and von Willebrand factor multimer pattern were normal. The prolonged activated partial prothrombin time was caused by a low factor VIIIC concentration (1%) (normal range 60-160%). His plasma contained a factor VIIIC inhibitor at a concentration of 37 Bethesda units.[1] The inhibitor had no activity against porcine factor VIIIC. He was treated with continuous antibiotics for two months; the haemoptysis resolved in six weeks, his temperature returned to normal in eight weeks, and clearance of the abscess took 23 weeks.

One week after abnormal bleeding was first noted he had an episode of profuse haematuria that required treatment with cryoprecipitate. This increased his factor VIIIC concentration to 10%, which was one fifth of the maximum rise predicted in the absence of a factor VIIIC inhibitor. The factor VIIIC concentration declined with a half life of one hour, compared with the normal half life of infused factor VIIIC of over eight hours. The bleeding time, however, was corrected for 12 hours, and the haematuria stopped immediately after the infusion. Eleven months later the bleeding disorder was not clinically evident and he was well. Factor VIIIC inhibitor was still detectable although much reduced. The table shows changes in the variables associated with clotting.

Changes in variables associated with clotting over the 11 months after illness

No of weeks after admission*	Bleeding time (min)	Activated partial thromboplastin time (s)	Factor VIIIC (%)	Factor VIIIC inhibitor (Bethesda units)
4	>20	84	1	37
8	8	49	5	19
13	8	50	5	3
23	5·5	54	8	12
48	5	49	12	2

*Abscess recognised at week 2; temperature settled at week 8; discharge from hospital at week 13; resolution of abscess by radiography at week 23.

Comment

Acquired inhibitors to factor VIIIC in non-haemophiliacs are rare but may develop in association with autoimmune disorders, malignancy, and pregnancy. Nearly half of all patients have no detectable underlying disorder.[2] In most cases the pattern of spontaneous haemorrhage is similar to that in patients with inherited factor VIIIC deficiency (haemophilia A).[3] The decline in factor VIIIC inhibitor concentration in our patient as the lung abscess resolved suggested an association, although, interestingly, the inhibitor persisted at a lower concentration when he was well.

A prolonged bleeding time is not characteristic of factor VIIIC deficiency even if inhibitors are present: in this our patient's illness resembled von Willebrand's disease, although the antigen structure and activity of von Willebrand factor were normal. This is analagous to the bleeding disorder in renal failure, in which the bleeding time may be corrected with cryoprecipitate,[4] although the plasma concentration of von Willebrand factor is usually already increased.[5] The mechanism whereby cryoprecipitate corrects bleeding time in renal failure is not fully understood, although it may increase the ratio of high to low molecular weight oligomers of von Willebrand factor. This mechanism may have operated in our patient, although as in renal failure the changes in the multimer pattern of von Willebrand factor were too subtle to be picked up by sodium dodecylsulphate electrophoresis.

Although haemoptysis is well recognised in patients with lung abscesses, basic coagulation screening should be carried out to exclude a generalised bleeding disorder. Identification of an associated coagulopathy in patients with other medical problems may substantially alter management.

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Departments of Haematology and Medicine, Royal Postgraduate Medical School, London W12 0HS
PETER S GANLY, BA, MRCP, registrar in haematology
JOHN D ISAACS, BSC, MRCP, registrar in medicine
MICHAEL A LAFFAN, BA, MRCP, registrar in haematology
CHRISTOPHER HASLETT, MRCP, senior lecturer in medicine
JILL M HOWS, MD, MRCPATH, senior lecturer in haematology

Correspondence and requests for reprints to: Dr Ganly.