Letters to the Editor

Preference is given to letters commenting on contributions published recently in the JRSM. The usual length of letters is up to 400 words (one table or illustration may be included) and they should be typed in double spacing.

Alcohol consumption as a cause of emergency general medical admissions

Sir, We were interested to read the report by Lockhart et al. (March JRSM, p. 132) in which 27% of acute admissions to a medical unit were attributed to alcohol consumption. One of us (RAS) has recently completed a survey of alcohol and smoking habits in 100 consecutive emergency medical admissions under a single consultant. The admissions were from October to December 1985 to Dudley Road Hospital, an inner city district general hospital in Birmingham. In this survey alcohol abuse was considered to have been the principal cause of 5% of admissions. In a further 6% alcohol was considered a probable factor leading to admission. In 13% a history of excessive alcohol consumption was obtained (>4 pints of beer/equivalent per day; 56 units of alcohol per week). The 13% who were heavy drinkers were divided into 10% who smoked heavily (>10 cigarettes per day) and 3% who did not.

We suspect that, countrywide, 27% is an overestimate of the influence of alcohol causing medical admission. Were none of the 6 patients admitted with chest disease not also smokers? Was it considered whether, in all the alcohol-associated overdoses, alcohol was taken before the decision to overdose? Were those with peptic ulcers non-smokers?

We were, furthermore, interested that alcohol was not thought by Lockhart et al. to have caused any strokes, as it is probably a significant risk factor.

Whatever the exact extent of alcohol-related admissions, it is certainly a considerable burden, and as Lockhart et al. point out, a burden which health education can hopefully reduce.

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Reference

*Drs Lockhart and Baron reply below:
Sir, We were interested to read the results of Shinton and Gill. In a survey similar to our own they consider 10% of patients to have been admitted as a result of alcohol, whereas we found a figure of 27%. The proportion of patients drinking more than about 50 units per week is 13% in their study compared to 12% in ours, which might suggest that in terms of drinking behaviour the two groups were similar. Anyone working in this field will be fully aware, as was eloquently emphasized by Wallace1, that there is a serious methodological limitation in studying alcohol as a cause of medical admission. There is no 'gold standard', no objective indicator of causation. Studies in which only one investigator classifies all patients, as in the study of Shinton and Gill, are very susceptible to the effects of individual variation in clinical judgment. This difficulty also leads to the use of vague definitions. Shinton and Gill looked for patients principally or probably admitted as a result of alcohol consumption. We looked for patients probably or possibly admitted as a result of alcohol consumption. If we had used only the first category our figure would have been 20%. Work in Manchester suggested that 27% of general medical admissions were due to alcohol2 and in Glasgow (male admissions) the figure was 19%.3 These studies are all subject to the same methodological problems, but suggest that alcohol consumption places a considerable burden upon general medical facilities in the units in which they were undertaken.

All these studies have been in hospitals in large cities. Shinton and Gill suggest that we attempt to extrapolate our figures to a 'countrywide' estimate. This would be epidemiologically indefensible and we would not wish to do so. We do, however, hope that these studies will provide a stimulus to further work on a national scale.

We are grateful to Shinton and Gill for emphasizing that heavy drinkers are often also heavy smokers. This is a further mechanism by which an alcoholic lifestyle may lead to physical ill health. We felt that reiteration of the relationship between smoking and chest infection and peptic ulceration was unnecessary. Greater suspicion of the effects of an alcoholic way of life, however, still requires emphasis.

As Shinton and Gill will realize from their clinical experience, alcohol is involved in self-poisoning in several ways. Some patients take overdoses because of problems associated with chronic alcoholism, some decide to take an overdose because of impaired judgment during acute intoxication (which may be part of a drinking problem), and some take alcohol after deciding to take an overdose to make it easier to injure themselves. All of these patients provide a burden upon the Health Service, and the prevention of the first two groups provides an opportunity for individual intervention.

We are grateful to Shinton and Gill for bringing their work on alcohol as a risk factor in stroke to our attention.

These points should not obscure our common conclusion, that alcohol consumption places a considerable burden upon general medical facilities within our particular hospitals, and that this problem requires more definitive study and intervention on a national scale.

S P Lockhart
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References
2 Jariwalla AG, Adams PH, Hore BD. Alcohol and acute general medical admissions to hospital. Health Trends 1979;11:95-7
3 Quinn MA, Johnston RV. Alcohol problems in acute male medical admissions. Health Bull (Edinb) 1976; 34:253-6
The silent metastasis from breast cancer

Sir, It has for long been known that the marrow of the sternum is a site for breast cancer metastasis. Tumour cells reach it by way of the efferent lymphatic of the internal mammary artery. They do not give rise to any swelling or pain but replicate in situ and pass into the general circulation, thus possibly causing further metastases throughout the life of the patient. One would have thought the sternum would be a prime site for irradiation. Yet, according to much discussion and comment in The Lancet in the past year and in JRSM Supplement No. 9, 1985 (Bone Metastases from Breast Carcinoma), sternal metastasis is not so much as mentioned. All attention for treatment is given to the primary site, axillary nodes and other bone metastases.

B D PULLINGER
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*Dr Yarnold replies below:

Sir, In response to Dr Pullinger's query, I would point out that tumour cells from the breast do not pursue a single lymphatic or arterial pathway to any single local site, but disseminate widely in the lymphatic and vascular system at any early stage. I am afraid that there is no evidence to suggest that the marrow of the sternum is ever a sole site of metastasis. This speculative hypothesis has never been tested formally but, as it happens, the sternum has often received full exposure by radiotherapy delivered to the underlying internal mammary chain, especially when an anterior portal has been used to irradiate both sides. In the context of randomized clinical trials evaluating this mode of treatment, no survival advantage has ever been noted for the irradiated group.

J R YARNOLD
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Infections caused by opportunistic mycobacteria

Sir, We read with interest the review paper by Grange and Yates (April JRSM, p 226). Although they referred to open heart surgery they omitted any reference to implanted valve endocarditis2-4. These infections may be attributed to contamination of the tissue at source5 with inadequate sterilization and screening procedures.

The advent of a new all-British porcine bioprosthesis6 required careful examination of the levels of tissue contamination, the microbiological screening procedure and the sterilization procedure, since the contamination posed by mycobacteria spp. is compounded by their slow growth and difficulty in detection7.

Tissue from five hearts collected on any day were stored overnight at 4°C in 0.9% NaCl and screened on the following day for mycobacteria spp. on a weekly basis to indicate the level of tissue contamination. Aortic samples from each porcine bioprosthesis accompany the valve through all the manufacturing and sterilization procedures, including 0.2% and 1% glutaraldehyde and 4% formaldehyde. These aortic samples are used in the microbiological screening.

In order to assess the sterilization, 8 samples were deliberately contaminated with a suspension of mycobacteria at a concentration of 1 x 106 organisms/ml and incubated at 22°C for 20 minutes. They were then sterilized in the same way as the bioprostheses, followed by 96 hours in 4% formaldehyde. All samples were then routinely screened by placing them with an aseptic technique into:
(a) Robertson Cooked Meat Medium which was sub-cultured after 3 and 6 days onto blood agar plates and incubated aerobically and anaerobically
(b) Sabouraud Liquid Medium
(c) 8% Glycerol in Nutrient Broth
(d) Lowenstein-Jensen slope.

All cultures were incubated at 37°C for 6 weeks and observed weekly.

A total of 225 random pig hearts had no positive culture for mycobacteria spp., showing that the tissue contamination is extremely low, and abattoir managers suggest that overt porcine thoracic tuberculosis is very rare. All 8 samples contaminated in the laboratory with Mycobacterium tuberculosis and Mycobacterium xenopi yielded mycobacteria spp. on culture. The 8 samples contaminated in the laboratory and then treated with the sterilization procedures all failed to yield growth. A total of 2910 bioprostheses have been sterilized and there were no positive cultures of mycobacteria spp.

The review by Grange and Yates emphasizes that we should not be complacent about the possibility of mycobacterial contamination, and there will be a continuing policy of screening for mycobacteria spp.

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References
3 O'Rourke MF, Shanahan MX, Harkness JL. Endocarditis with an acid fast organism after porcine heart valve replacement. Lancet 1978;i:656
4 Anyanwuh CH, Nassau E, Yacoub M. Millitary tuberculosis following homograft valve replacement. Thorax 1976;31:101-6
5 Centre for Disease Control. Isolation of mycobacteria species from porcine heart valve prostheses. CDC Morbidity and Mortality Report 1977;26(11 February):42-3

Aids virus infection

Sir, Is the Aids virus the only member of the Lentivirus family in addition to maedi-visna of sheep, infectious anaemia virus of horses, and capperine arthritis-encephalitis of goat? Or is bovine viana virus, cultured in leukaemic bone marrow in 1977, another member of the family?

It is the gospel of the United States NIH that the AIDS virus arose spontaneously in monkeys—animals not commonly known to harbour visna-like
viruses or known to be adversely affected by the AIDS virus until they are inoculated.

Most likely the AIDS virus arose by heterodimer recombination of bovine leukemia virus and visna virus in a commonly infected host cell. Furthermore, it seems more probable that the virus expanded its host range and perhaps replicative rate (trivialities to those initiated in reaction rate kinetics of retrovirus recombination) by culture growth in malignant bone marrow tissue.

Where is the sorcerer to banish the flood created by the apprentices of the World Health Organization and United States National Institute of Health?

When the retrovirus strains, oncogenic genes and transacting genes are added to the airborne human DNA viral genomes in combination with host cell information, we all will regret the infinitely culturable HeLa.

ROBERT B STRECKER Preferred Risk Partners Inc Glendale, California, USA

References
1 Seale J. AIDS virus infection: prognosis and transmission. J. R. Soc Med 1986;78:613-4

Haematuria due to urinary bladder metastases from carcinoma of the bronchus

Sir, The correspondence in your columns on this subject (December 1985 JRSM, p 1053 and April 1986, p 250) brings to mind a patient of mine who presented with haematuria early in 1981. He appeared to be a healthy 80-year-old and it was not considered justified to investigate his haematuria.

Some months later he developed a skin ulcer in the lumbar region. This was a secondary deposit from a carcinoma of the bronchus, which grew rapidly. He died two months later in spite of treatment.

It is unusual for carcinoma of the bronchus to present with haematuria.

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Joan of Arc, creative psychopath: is there another explanation?

Sir, I read with interest Dr Ratnasuriya’s intriguing comments about Joan of Arc (April JRSM, p 234) and, during a recent visit to the imposing ruins of Chinon Castle, I could not help considering the amazing fact that she was ever accepted by the Court of Charles VII. At any rate, I would like to offer yet another explanation: the full account is in Joan of Arc and her Secret Missions by Pierre de Sermois. This theory, in brief, is that Joan was the illegitimate daughter of Louis of Orleans and Isabella of Bavaria (brother and wife of Charles VI). This is certainly possible given the personalities of these members of the then French royal family! Such a person would have been accepted, had access to the knowledge of conducting warfare and been able to afford the extremely expensive armour which she wore.

During her trial Joan was never put to physical torture, which would be in keeping with Sermois’s view that that was a put-up job and that Joan was not burnt at the stake but that a witch victim was substituted at the last moment. This was why she was so covered up – again a very unusual occurrence, for the guilty were usually openly paraded as an ‘aut de fa’ so as to discourage the others.

Certainly, I would recommend the ideas of Pierre de Sermois. I think that those people who postulate a physical illness have to explain how Joan could cope with the rigours of mediaeval warfare, and those who postulate a mental illness have to explain how she was accepted as a leader so rapidly.

Perhaps Shakespeare had it right (Henry VI, part 1):

‘Not me begotten of a shepherd swain
But issu’d from the progeny of Kings’.

JOHN B BOWES Department of Anaesthetics Frenchay Hospital, Bristol

Sir, The evidence given by Dr Ratnasuriya (April JRSM, p 234) for Joan of Arc’s behaviour being based upon a tubercular pathogenesis is interesting but rather unconvincing. Although it would be only supporting evidence, I do not think that it is recorded that she had or had had scrofula, which seems always to have been the commonest manifestation of bovine tuberculosis. Further, while I cannot account for her intestines, there is good literary evidence of the presumably normal heart surviving incineration of all other tissues including bone. I quote from Trelawney’s account of the immolation of Shelley’s five-week dead body on the beach near Viareggio: ‘The only portions that were not consumed were some fragments of bone, the jaw, and the skull; but what surprised us all was that the heart remained entire’.

Finally, I do think Joan’s cachexia and amenorrhoea are much more likely to have been due to anorexia nervosa – relatively recently recognized but I am quite sure already existent in the 16th century.

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Dr Ratnasuriya replies below:

Sir, The explanation that Joan of Arc had tuberculosis takes into account that she suffered from the illness during her late childhood and the tuberculosis developed later, as is evidenced by the fact that she first heard voices when she was about 13. She then recovered from the illness, as is possible with tuberculosis1,2, which was why she was able to cope with the rigours of warfare.

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References