LETTERS TO THE EDITOR

Preference is given to letters commenting on contributions published recently in the *JRSM*. They should not exceed 300 words and should be typed double spaced

Multicentre research ethics committees

Although the comments of Miss Larcombe and Professor Mott (October 1999 *JRSM*, pp. 500–1) are valid, their research was a victim of unusual timing and circumstances. I was party to the ethical review of both stages of their proposal and suggest that their complaint is misdirected, mistimed and possibly harmful.

Their study was not 'simple', but complex in terms of confidentiality. Their proposal was reviewed by the local research ethics committee (LREC) in December 1996, the month before the International Conference of Harmonisation Good Clinical Practice (ICHGCP) guidelines came into operation. It was later reviewed by a multicentre research ethics committee (MREC) at its inaugural meeting. At this time, procedures were new and the division of LREC and MREC responsibilities was not universally appreciated. Some LRECs continue to seek changes to the protocol not because of faults in the MREC system but because of a lack of training of members of LRECs, which training is often inadequately supported by trusts or health authorities.

The paper is mistimed because new proposals for the ethical review of epidemiological research are currently being considered to avoid some of the frustrations described. Other frustrations quoted were the failures to give approval by chairman's action, the required nomination of a local contact and the failure of the MREC to include the changes later required by LRECs. LREC-chairman's action is not acceptable for most MREC-approved studies; local contact details are required by ICHGCP with which all ethics committees must now comply; and cogent arguments were advanced for keeping close to the original form of the proposal.

Not all proposals are acceptable and members of ethics committees, especially lay members, spend much effort protecting the interests of the patient for no discernible reward. Repetitious criticism will erode the willingness to perform this necessary function.

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I do hope that Isobel Larcombe and Martin Mott have sent a copy of the paper about their tribulations to each of the 51 LRECs who were approached about their study, with a request for a rapid response.

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Severe parkinsonism secondary to carbon monoxide poisoning

The case report by Dr Gillespie and colleagues (October 1999 *JRSM*, pp. 529–30) is erroneous in regard to the anatomy of the magnetic resonance imaging (MRI) findings. The published image does not show increased signal in the periventricular white matter. The plane of the section can be identified as passing through the upper parts of the orbits, the anterior commissure, and the splenium of the corpus callosum. This section plane passes through the basal ganglia, and it shows increased signal in the globus pallidus on both sides. The radiological demonstration of injury to these structures in carbon monoxide poisoning has been reported on several occasions, on both computed tomography and MRI[1], and is entirely consistent with the patient's neurological presentation. Damage to the periventricular white matter has also been reported, but is not shown in the published image from this case.

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Antioxidants in wine and tea

Professor Cheng (March 1999 *JRSM*, p 157) states that 'the brewing of black tea oxidizes the catechins, destroying any beneficial effects'. He correctly cites Jankun *et al.*[1] for this statement, which unfortunately is a misinterpretation regarding black tea in a review by Yang and Wang, whom Cheng also cites[2]. Yang has corrected the error in these words: 'There is no evidence from the review or elsewhere to support the statement'[3].

The total catechin content of black tea (the fermented tea generally consumed in western countries) is about one-third that of green tea[4]. However, *per caput* consumption of tea in the UK is over three times that in Japan and over eight times that in China[5], so British black tea drinkers obtain at least as much antioxidant from the beverage as do the Japanese and Chinese green tea drinkers. Thus Cheng's suggestion that the low rates of coronary artery disease (CAD) in China and Japan may be attributable to the preference for green tea in these countries is untenable.

Consumption of black tea has been found to be associated with a reduced risk of CAD in men in the Netherlands[6] and in men and women in the USA[7], but not in Britain[6,8] where milk is customarily taken with tea (unlike in these other two western countries). A suggestion

that the milk prevents absorption of the tea antioxidants[6] has been disproven[4].

So why should Britain, with a *per caput* consumption of tea four and eight times greater than in the Netherlands and USA, respectively[5], not benefit from its antioxidants? A possibility that should be addressed is that the lactose in the milk has an atherogenic potential[9] which counteracts the protective effect of the tea antioxidants.

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Transport and temperature effects on blood potassium

Dr Seamark and his colleagues (July 1999 *JRSM*, pp. 339–41) do not refer to our 1998 paper[1] reporting results similar to theirs. We demonstrated that increasing ambient temperatures may produce artefactually lower potassium values if there is an appreciable delay between collection and analysis. We showed that this effect was significant for general practitioner samples and outlying hospitals but considerably reduced when there was a short transit time.

We speculated that the erythrocyte membrane Na/K pump may remain more active at higher temperatures and therefore accumulate potassium in the cell leading to lower measured extracellular values.

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REFERENCE

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Honey: is it worth rubbing it in?

Honey has been a medicine used worldwide over many centuries, previously without any supportive scientific evidence. Recent reports have shown antibacterial activity in manuka and pasture honey against coagulase-positive *Staphylococcus aureus*[1] but Dr McGovern and colleagues (August 1999 *JRSM*, p. 439) found manuka honey ineffective in eradicating *Helicobacter pylori* in patients with positive CLO tests. Studies have indicated the effectiveness of honey in the prevention of experimentally induced gastric lesions in rats[2], and the concentrations achieved may be relevant. Ali *et al.*[3] showed *H. pylori* to be inhibited *in vitro* by 20% natural honey in all 30 cases tested. *Acinetobacter*, *Enterobacter* and *Brucella* species, *Staphylococcus aureus* and β-haemolytic streptococcus were inhibited in the presence of 20% honey but *Proteus* species required 30%. The use of honey should not be dismissed and further investigations of optimal concentrations and dosage frequencies and possible combination therapies need to be undertaken. Effective monotherapy or less expensive combination treatments would lead to vast savings for both general and hospital practitioners, since anti-ulcer treatments account for a large proportion of prescription budgets.

In dermatology, a proposed but unconfirmed link of *H. pylori* with acne rosacea has been reported[4] and a 'patient-friendly' topical application of honey might be a useful adjunct to other antibacterial therapies.

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Royal Medical Benevolent Fund Christmas Appeal

The season of Christmas is upon us. Irrespective of race or creed we look forward to one of the happiest times of the