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

NSAID chemoprevention of colorectal adenomas and oesophageal metaplasia

I read with interest the article by Kune and Vitetta (November 1995 *JRSM*, pp 625–628) which illustrated the role of adenoma control as a key to reducing the incidence of colorectal cancer. I agree with my learned Australian colleagues that 'to eliminate colorectal cancer in the twenty-first century' is indeed an exciting prospect. I was disappointed, however, that no mention was made of the possibility of nonsteroidal antiinflammatory (NSAID) chemoprevention. There is very good evidence that the regular consumption of NSAIDs reduces the risk of fatal colorectal cancer. It appears that part of this protection results from the ability of NSAIDs to reduce the size and number of colorectal adenomas. In the context of primary prevention described in their paper, NSAIDs could be used as a secondary measure in patients who do not modify their diet, reduce smoking or alcohol intake or increase physical activity.

In extension of this, the above risk factors for the colorectal adenoma/carcinoma sequence are also implicated in the aetiology of oesophageal carcinoma. NSAIDs also appear to reduce the risk of oesophageal carcinoma. It is my (as yet unproven) belief that NSAIDs reduce the risk of oesophageal carcinoma by preventing mucosal malignant changes from precancerous Barrett's metaplastic cells[1]. Thus, NSAIDs could be used as a secondary measure in Barrett's patients who are unresponsive to primary prevention. Interestingly, Barrett's patients appear to have increased risks of colorectal cancer and may receive additional benefit from NSAID cancer chemoprevention[2].

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REFERENCES

1 Morgan GP. NSAIDs and the chemoprevention of colorectal and oesophageal cancers [Leading Article]. *Gut* (in press)

2 Morgan GP. Relationship between colorectal and oesophageal cancer [Letter]. *Dis Colon Rectum* (in press)

Analgesia for venous cannulation

Selby and Bowles (May 1995 *JRSM*, pp 264–267) overstate their case by claiming that 'there is, however, no excuse for not using a local anaesthetic before any cannulation'.

They have shown that the use of lignocaine and ethyl chloride anaesthesia triples and doubles the failure rate for cannulation. It is of note that these results were obtained by presumably experienced anaesthetists (both FRCA) in patients who were not acutely ill. This is completely different to an inexperienced house officer or senior house officer attempting to cannulate an ill or non-cooperative patient in the frontline of casualty or the medical admission wards. I also have reservations about their data collection. The authors do not state how many attempts were needed in 'failures' before a cannula was eventually sited or what was the 'total pain' after multiple attempts. In my experience, after a cannulation failure, the patient becomes progressively more distressed by subsequent attempts and would therefore record higher levels. The authors also fail to record the total time taken to obtain intravenous access, including obtaining the necessary equipment, time for analgesia to work and time for repeated cannulations. Again, while this may be practicable in the preoperative setting, it is a luxury we cannot afford in acute medicine. There are many instances in which the priority is to obtain an intravenous line rapidly and any delay in this is detrimental to patient care. The authors' sweeping generalization from the preoperative setting to universal medical practice is untenable and not supported by the evidence they have presented.

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The authors reply below:

We would like to thank Dr Ulahannan for his interest in and comments on our work.

We appreciate that there are great differences between a patient admitted for an elective procedure and one admitted *in extremis* needing emergency resuscitation. We would agree that in such patients the 30 s required to apply local anaesthetic may be detrimental to their well being and therefore should be dispensed with. However, these patients form a minority of those requiring venous access.

As for the experience of the cannulator, we do not accept inexperience as a reason not to strive after the best possible care for patients. Patients tolerate failure of venous cannulation much better if the failed attempt was performed under local anaesthetic and their expectation of a painful experience not already realized.

The total pain of cannulation has been investigated by Langham *et al.*[1] and their results confirm the benefits of local anaesthetic. We would also contend that the extra time taken to collect the equipment necessary to administer the local anaesthetic is vastly reduced with practice and organization, for example the use of ethyl chloride only requires a bottle to be picked up. All the cannulae in our study were inserted on a day surgical ward where, I suspect, far fewer cannulae were inserted than on the average medical ward. We had no problems once a routine had been established.

Patients fear needles[2]. We feel that this is unnecessary, and that if medical staff would put a little extra effort into these routine tasks patient care could be significantly improved.

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REFERENCES

1 Langham BT, Harrison N. Local anaesthetic: does it really reduce the pain of insertion of all sizes of venous cannula? *Anaesthesia* 1992;**47**:890–1

2 Van Wijk MGF, Smallhout B. A post operative analysis of the patient's view of anaesthesia in a teaching hospital. *Anaesthesia* 1990;**45**:679–82

Sam Johnson's lung not in *Baillie's Atlas*

In his letter (December 1995 *JRSM*, p 724) H D Atwood did not quote as he did in his paper[1], from the report[2] by James Wilson FRS, the surgeon and anatomist, of his