letters TO THE EDITOR

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Medical treatment at the end of life

Editor – The position statement *Medical treatment at the end of life* (*Clin Med JRCPL* March/April 2001, pp115–7) is, in the main, a welcome outcome to the Working Party's hard work and deliberations.

However, I find it strange that the Working Party should be at such pains to include the word 'active' in its definition of euthanasia when, two paragraphs later, it seeks to abandon the use of the adjectives 'non-voluntary', 'involuntary' and 'passive'. This is hardly logical!

The most important point they make (p 116) is that 'it is clear that the intention behind a therapeutic decision…is a central issue'. This is indeed ethically fundamental. Consequently it cannot be true to say, without qualification, that 'an intention to withhold or withdraw burdensome or futile treatment is not an intention to kill'. It is not an intention to kill precisely to the extent that the treatment is being withheld or withdrawn for another reason, ie because it is burdensome and being withdrawn specifically to relieve the burden. Treatment can be withheld or withdrawn with the intention of ending the patient's life and this is no less lethal than a positive act. The intention behind such an act (or omission) can, it is true, only be clear to the doer but I find it hard to see why the Working Party failed to recognise the distinction. Let us hope treatment will not be withheld or withdrawn from patients for no better reason than that the BMA/RCP said that this was right.

I also regret the Working Party's support for the BMA guidelines *Withholding and withdrawing life-prolonging medical treatment* which, in several respects, is on much less sure ground, both ethically and even legally, with its recommendations.

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Aspirin against cancer

Editor – Professor Elwood (*Clin Med JRCPL* March/April 2001, pp132–7) meticulously describes the development of aspirin as an antiplatelet drug over the past three decades, and elegantly illustrates that the axiom 'if a little bit works, a lot works better' is not necessarily true. Unfortunately, his allusion to a similarly structured approach to the development of aspirin as an anticancer agent is falsely optimistic.

Although the epidemiological evidence that aspirin reduces the incidence of colorectal cancer is convincing[1], only one truly large-scale randomised Phase III trial is taking place. Unlike the highly focussed clinical development of the selective cyclooxygenase-2 inhibitors such as celecoxib, licensed last year in the USA for the chemoprevention of colorectal cancer in patients with familial adenomatous polyposis[2], the choice of subject population and biomarker (recurrence of sporadic polyps, whose aetiology is known to be more multifactorial than inherited cancer) is weighed against positive results for aspirin.

Unless research funding bodies are willing to support the development of aspirin as an anticancer agent in the way that selective cyclooxygenase-2 inhibitors have been driven by the pharmaceutical giants, this well known, pleiotropic, natural derivative will be superceded by its younger, relatively unknown, selective relatives. Since aspirin inhibits many of the carcinogenic processes that the selective agents do not[3], this is not necessarily progress.

References

1 Sharma RA, Manson MM, Gescher A, Steward WP. Colorectal cancer chemoprevention: Biochemical targets and clinical development of promising agents. *Eur J Cancer* 2001;**37**:12–22.

2 Steinbach G, Lynch PM, Phillips RKS, *et al*. The effect of celecoxib, a cyclooxygenase-2 inhibitor, in familial adenomatous polyposis. *N Engl J Med* 2000;**342**:1946–52.

3 Marnett LJ. Aspirin and the potential role of prostaglandins in colon cancer. *Cancer Res* 1992;**52**:5575–89.

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Temporary cardiac pacing and the physicians of tomorrow

Editor – I read with interest the clinical and scientific letter by Murphy *et al* (*Clin Med JRCPL* March/April 2001, p156). In my three years as a specialist registrar in general internal medicine (GIM) and gastroenterology, I have had to insert a temporary cardiac pacing wire on approximately ten occasions, while on call. As yet there have been no significant complications, suggesting that I have probably not performed enough! My only training consisted of watching the procedure done twice by a registrar during acute medical on calls as an SHO, and one supervised procedure soon after. Only once was I supervised whilst on call as a registrar, and this was during 'office hours'. I once asked my GIM consultant if he would be able to help me, should I have trouble pacing at night, and he admitted to never having performed the procedure himself. I am sure this is not an unusual occurrence and fully sympathise with the authors who suggest that temporary cardiac pacing is inadequately taught, and question whether it should be performed by non-cardiologists.

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Editor – The advent of thrombolysis has greatly reduced the need for emergency temporary pacing and in some district general hospitals so also has the provision of permanent pacing. However, removing training in temporary pacing from general internal medicine (GIM) training with a view to the service being provided entirely by cardiologists would have major implications. There are still significant numbers of district general hospitals in this country with only a single cardiologist, often without a specialist registrar. In many other