Guidelines for MRSA control

Sir,

Dr Wiggins (Vol 61(1), 2000, p. 4) is absolutely right when he says that ward closures became unacceptable in the 1990s. In areas where methicillin-resistant *Staphylococcus aureus* (MRSA) was endemic not just in hospitals, but also in the community, it became difficult, if not impossible, to define when an MRSA outbreak had occurred. Such issue had been straightforward in the 1980s. The increase in numbers of MRSA isolates is complicated by the unrelenting pressure on beds and patient throughput so that the MRSA carrier had probably passed through a number of wards before their MRSA status became known. From my own point of view I have certainly felt like King Canute over the years — whatever energetic measures were employed failed to stem the incoming tide of MRSA.

There is no difference between MRSA and any other self respecting *Staph. aureus* in terms of either pathogenicity or epidemiology. The 'fuss' made about MRSA is because of its resistance pattern. What we need therefore is first to reduce the use of existing antibiotics wherever possible. This will negate the selective advantage which the use of antibiotics confers upon MRSA. Second what we need are new agents which are active against exiting MRSA strains. The welcome arrival of quinupristin/dalfopristin is perhaps the first step in this direction.

In the meantime pragmatism in hospital in dealing with infection control and MRSA must surely rule the day. There is a lack of literature in the medical/scientific press giving a sound foundation to risk assessment analysis. It is not an easy area in which to gather good scientific data. Pragmatism should nevertheless be based on individual risk assessment as well as assessment of the unit. The hunt and kill strategies of the 1980s did not work then and are inappropriate now.

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Bisoprolol in heart failure

Sir,

Davis quite rightly states that in angiotensin-converting enzyme (ACE) inhibitors and β-blockers we now have the tools to combat the increasing neurohormonal activation that underlies the progression of congestive heart failure (CHF) (Vol 61(2), 2000, p. 120). He postulates a scenario of a large increase in CHF incidence consequent to both an aging adult population and improved survival post-myocardial infarction.

The Heart Outcomes Prevention Evaluation (HOPE) study (2000) demonstrated that ACE inhibitors are remarkably effective at preventing not only the development of heart failure but also cardiovascular morbidity and mortality in those at high risk of developing CHF. Importantly, these benefits seemed independent of those of blood pressure reduction. While the corresponding case for β-blockers remains to be formally tested, recent advances suggest analogous benefits (Sabbah, 1999). Bisoprolol, metoprolol and possibly carvedilol are likely equally efficacious. Many are currently on β-blockers other than those tested but there are dangers in assuming a class effect (Drummond and Squire, 1999; Furberg et al, 1999).

Intervening before the onset of overt CHF is the key to reducing its incidence. It is imperative to identify and treat patients at high risk particularly those with hypertension and ischaemic heart disease. Perceived contraindications to β-blockade should be both real and substantial if one is to deny a potentially life-saving therapy. Those at greatest risk such as the elderly or diabetics are often those least likely to be given β-blockers. This mind-set needs to change. HOPE and the β-blocker heart failure trials may revolutionize the treatment of hypertension and ischaemic heart disease as well.

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Photodynamic therapy

Sir,

The article on photodynamic therapy (PDT) in *Hospital Medicine* (Vol 61(3), 2000, p.160) provides a sound overview of how this therapy has evolved and how it may develop in the future. It also highlights the contentious issue of selectivity. Early enthusiasts claimed selective destruction of cancers leaving adjacent normal tissue undamaged, but this could seldom be substantiated and undoubtedly led to some serious complications, as in treatment of the bladder. This was one of the reasons that PDT got a bad name in the 1980s.

Many photosensitizers are retained at slightly higher concentrations in cancers than in adjacent normal tissue. This can be used for diagnostic purposes as most photosensitizers fluoresce, but the selectivity is rarely great enough to achieve selective necrosis when normal and neoplastic tissues are exposed to the same light dose. However, it is possible to get selective mucosal necrosis without damaging underlying muscle which has enormous potential for treating dysplasia in hollow organs. The real attraction of PDT is the nature of the tissue damage as there is essentially no damage to connective tissue, so the mechanical integrity of hollow organs is better preserved and healing is excellent.

In the 1990s, we learned what PDT does to many normal and diseased tissues and so can better identify its potential clinical role. As in so many branches of medicine, it took enthusiasts to get things off the ground, then a long pause for the scientists to work out what was really going on. Now we are undertaking more rational clinical trials on what is likely to become an important new treatment for a range of diseases.

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Sir,

Dr Rhodes provides a crisp overview of photodynamic therapy (PDT), a modality with therapeutic potential in several areas of medicine (Vol 61(3), 2000, p. 160). For any body site to which light can be delivered, PDT offers the potential of cure in early superficial disease and palliation in advanced disease.

The skin has been the ideal organ in which to first assess the efficacy of PDT. Photodynamic therapy using the topically active agent 5-aminolaevulinic acid (ALA) has several dermatological applications, in particular; actinic keratoses, squamous cell carcinoma in situ (Bowen's disease) and superficial basal cell carcinoma. Tumour depth remains a limiting factor in the success of PDT, with poorer clearance rates for the thicker nodular basal cell carcinomas. However, new photosensitizers and different delivery methods, we anticipate, will further extend the therapeutic indications for PDT.

PDT is a tissue sparing modality, useful for large/multiple lesions, or where site presents a therapeutic challenge to conventional surgery. PDT is generally well tolerated by patients with a low potential for scar formation.

There is also a diagnostic application in the photosensitization of neoplastic/dysplastic disease. Fluorescence emissions from the absorbed photosensitizer can be utilized to delineate tumours from surrounding tissue as a consequence of the differential uptake of photosensitizer.

The popular press has dubbed PDT the 'light of life'. While appropriately avoiding such sensational claims, Dr Rhodes provides a balanced review of the present state of PDT. I expect readers will become increasingly aware of PDT in their own specialities in the near future.

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