Methicillin resistant Staphylococcus aureus

Sir,—During the past five years a particular strain of methicillin resistant Staphylococcus aureus has been recognised as being epidemic in the Thames regions. This strain not only can cause serious disease in some groups of patients but has also spread within and between hospitals in spite of antiseptic and other precautions taken against its spread.1 2 3 4

Over the past 12 months we have obtained evidence that this organism is spreading outside the Thames regions, and we know of 10 instances where transfer of a patient infected or colonised with epidemic methicillin resistant S aureus has resulted in the first introduction of this strain into a hospital. Transfer of the organism is often associated with patients who have undergone specialised surgical or medical procedures in the London area and may be returning to hospitals distant from the Thames regions.

The introduction of epidemic methicillin resistant S aureus into a hospital is a serious event; its prevention is not possible and the curtailment of clinical services, increased patient morbidity, and even mortality. Goodfield and Littlewood have recorded some of the consequences of a transfer with subsequent spread to domiciliary patients.3 4

We strongly advise that thought should be given to the undesirability of transferring patients in this way. If such transfers are necessary patients returning from London should be isolated until they are shown to be free of the organism. Recipient hospitals should be warned by hospitals transferring patients if this methicillin resistant strain is circulating.

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2 Marples RR, Richardson JD, de Saxe MJ. Bacteriological characters of strains of Staphylococcus aureus submitted to a reference laboratory 'related to methicillin resistance'. J Hyg (Camb) 1986;97:217-23.


7 FEBRUARY 1987

The unremitting burden on carers

Sir,—Mr Robert Anderson in his concise review of the pressures on people caring for seriously disabled people living at home includes among the services required "planned respite care for their charges and free time" (10 January, p 73).

Both of these forms of help require more thoughtful planning than they have had in the past. Respite care needs to be firmly booked months in advance if the carer is to be able to have a holiday with her or his family. The disabled person will be staying in either a "home" or a hospital. If the former he or she should have the opportunity to visit the place beforehand. The staff should be aware of the different needs of short stay residents and long stay residents, whose only home the place may be. Problems of coping with short stay residents in long stay homes have been studied by Allen, who states, "the idea that short stay care was in any way different from long stay care was clearly one which others and staff found difficult to cope with and it was a recurrent theme in this research."

Sir,—Dr Markku Koskenvuo and colleagues (3 January, p 16) state that snoring is related to obstructive sleep apnoea and that these patients may develop hypoxaemia and hypercapnia, together with increased pulmonary and systemic arterial pressure. They later speculate that low oxygen saturation has an unfavourable effect on the activity of lipoprotein receptors, leading to increased serum low density lipoprotein concentrations; the role of thrombocyte aggregation and coronary artery spasm in sleep apnoea is also unclear. At no point do they mention the effect of hypoxia on the haemoglobin concentration or whether there was any significant difference in the haemoglobin concentration, packed cell volume, or blood viscosity between the various groups.

Snoring is known to cause cardiovascular reactions, and hypoxia during sleep is thought to be an important determinant of the degree of secondary polycythaemia in patients with chronic obstructive airways disease.2 4 It is possible that the packed cell volume through two different mechanisms. Firstly, it causes a release of erythropoietin from the kidney, which then causes an increase in the red cell mass (a true polycythaemia). Secondly, hypoxia, both acute and chronic, can cause a contraction of the plasma volume (pseudopoly- cythaemia) through mechanisms which are poorly understood but possibly related to increased adrenergic activity and venoconstriction.1 In some patients both of these factors, increased red cell mass and reduced plasma volume, are present.

This is the mechanism by which some patients with polycythaemia vera (a disease caused by a hyperactive response to hypoxia, is itself associated with an increased incidence of both ischaemic heart disease and cerebral vascular disease, thought to be due to its effects on the viscosity of blood. Several studies have shown that cerebral blood flow is reduced in patients with a high packed cell volume and can be improved by reducing the packed cell volume.2 3 It is unfortunate that the authors do not mention the packed cell volume or haemoglobin concentration of their patients, even if only to state that there was no significant difference between the habitual snorers and the non-snorers, since they regard the intermittent hypoxia as the key to the increased incidence of arteriovascular disease in these patients. It would also be of interest to know if other risk factors such as enhanced coagulation or decreased fibrinolysis could be shown in the habitual snorers.

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4 Snoring as a risk factor for ischaemic heart disease and stroke in men

Sir,—Dr Markku Koskenvuo and colleagues (3 January, p 16) state that snoring is related to obstructive sleep apnoea and that these patients may develop hypoxaemia and hypercapnia, together with increased pulmonary and systemic arterial pressure. They later speculate that low oxygen saturation has an unfavourable effect on the activity of lipoprotein receptors, leading to increased serum low density lipoprotein concentrations; the role of thrombocyte aggregation and coronary artery spasm in sleep apnoea is also unclear. At no point do they mention the effect of hypoxia on the haemoglobin concentration or whether there was any significant difference in the haemoglobin concentration, packed cell volume, or blood viscosity between the various groups.

The relationship of snoring to polycythaemia in animal studies,1 and hypoxia during sleep is thought to be an important determinant of the degree of secondary polycythaemia in patients with chronic obstructive airways disease.2 4 In these patients, the packed cell volume is increased through two different mechanisms. Firstly, it causes a release of erythropoietin from the kidney, which then causes an increase in the red cell mass (a true polycythaemia). Secondly, hypoxia, both acute and chronic, can cause a contraction of the plasma volume (pseudopoly- cythaemia) through mechanisms which are poorly understood but possibly related to increased adrenergic activity and venoconstriction.1 In some patients both of these factors, increased red cell mass and reduced plasma volume, are present.

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Association between liberalisation of Scotland's liquor licensing laws and admissions for self poisoning

Sir,—In our paper (6 December, p 1466) we did not seek to attribute the sharp and immediate increase in the percentage of patients taking alcohol with their overdose to changes in the Scottish licensing laws alone, as the percentage of women has increased over time. This was supported by data from before 1976,3 Pace DRS P S Lockhart, JH Baron, and Stephen Platt (10 January, p 116), we still believe, however, that the sharp and immediate rise which occurred in the two years after the change in the law justified our concern, although there was likely to be a direct relation between these two facts. Our clinical data on poisoned patients have been under consistent and detailed review over the past 17 years and we