LETTERS

Severe childhood pneumonitis caused by the Queensland strain of community-acquired methicillin-resistant *Staphylococcus aureus*

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To the Editor: We report a case of severe pneumonia in a previously healthy 3-year-old girl of European background. Non-multiresistant methicillin-resistant *Staphylococcus aureus* (MRSA) was isolated from her sputum, and she was treated with intravenous vancomycin, followed by oral rifampicin and fusidic acid.

After antibiotic therapy ceased, symptoms recrudesced, and computed tomography of the chest showed bronchiectasis. Sputum again grew MRSA (now also resistant to rifampicin, fusidic acid and erythromycin), as well as *Pseudomonas aeruginosa*. She was treated with intravenous vancomycin, ceftazidime and tobramycin. As the MRSA persisted, vancomycin was replaced with intravenous linezolid, followed by a course of oral linezolid and trimethoprim–sulfamethoxazole. More than a year after her original illness, she continues to have a productive cough and requires nebulised tobramycin to prevent exacerbations.

The patient's only risk factor was contact with her mother, who had an MRSA buttock abscess incised several months earlier. Phage typing and pulsed-field gel electrophoresis revealed that the mother's and daughter's isolates were identical (Box). They were found to belong to the Queensland strain of community-acquired MRSA (CA-MRSA) and to possess Panton–Valentine leukocidin, a virulence factor which is highly associated with necrotising pneumonitis and invasive primary skin infection.[1]

CA-MRSA is a growing problem in Australia, and severe pneumonia due to this organism has recently been reported in adults.[2,3] The isolation of CA-MRSA before prolonged courses of antibiotics, its characterisation as a virulent strain, the response to appropriate treatment and the recrudescence of symptoms on cessation of therapy demonstrate that it was the causative organism in this case. The case is also noteworthy for the documented intrafamilial spread and the fact that the patient did not belong to the Pacific Islander community, in which CA-MRSA infections in south-western Sydney most commonly occur.[4]

Most *S. aureus* strains in the community are sensitive to flucloxacillin and dicloxacillin, so these remain the empirical treatments of choice, unless CA-MRSA is isolated or strongly suspected. CA-MRSA strains are non-multiresistant, and oral antibiotic options include clindamycin, trimethoprim–sulfamethoxazole or rifampicin and fusidic acid.[5] Intravenous vancomycin is commonly used in severe infections, but recent evidence suggests that linezolid, an oxazolidinone antibiotic with efficacy against multiply resistant bacteria, including MRSA, penicillin-resistant *Streptococcus pneumoniae* and vancomycin-resistant enterococci, may be more effective.[6] Issues of cost, toxicity, local availability, potential development of resistance and sensitivity of isolates *in vitro* need to be considered before determining appropriate treatment.

In conclusion, clinicians should be aware of the growing problem of CA-MRSA and the potentially devastating consequences of infection with this organism, even in otherwise healthy children.

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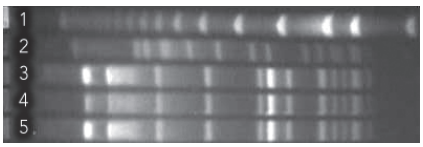
Pulsed-field gel electrophoresis (PFGE) of *Staphylococcus aureus* strains



1. *Low-range PFGE marker.*
2. *Control* S. aureus *strain (NCTC 8325).*
3. S. aureus *isolate from patient.*
4. S. aureus *isolate from mother.*
5. *Queensland strain of community-acquired methicillin-resistant* S. aureus *(CA-MRSA).*

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Impact of a formal removal policy for central venous catheters on duration of catheterisation

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To the Editor: Bloodstream infections are frequent in healthcare settings and cause significant mortality and morbidity.[1,2] Most of these infections are caused by intravenous catheters, particularly central venous catheters (CVCs). Over 250 000 catheter-related bloodstream infections occur annually in the United States,[1] and over 3000 in Australia.[2] Many CVCs are retained when no longer essential. For example, a recent one-day audit in a US teaching hospital found that 15% of CVCs (11/74) were "unjustified"; most of these had been inserted in the intensive care unit but retained unecessarily after discharge from the unit.[3]

The risk of bloodstream infection is much higher with CVCs than with peripheral venous catheters (4.0 versus 0.2 per 1000 line-days).[2,4] Such simple facts are often overlooked or inadequately emphasised in preventive programs, and CVCs may be retained for convenience.

Comparison of patient characteristics and CVC outcomes when removal policy was followed versus when it was breached

	Policy followed			Policy breached	*P* (policy followed v breached)
	CVC removed	CVC retained*	Total	CVC retained	
Patient characteristics					
Number of patients	176	71	247	25	
Age (years) (SD)	60.2 (17.9)	64.9 (15.7)	61.7 (17.5)	69.0 (15.3)	0.02
ICU length of stay (days) (SD)	4.7 (8.2)	3.3 (5.2)	4.3 (7.6)	2.1 (2.0)	0.06
APACHE II score (SD)	14.7 (6.9)	14.8 (7.0)	14.6 (17.5)	14.2 (5.1)	0.31
Ventilation time (h) (SD)	51 (89)	46 (123)	51 (103)	31 (55)	0.19
CVC outcomes					
No. of CVCs (% of all CVCs)	202 (66%)	77 (25%)	279 (91%)	26 (8%)	nt
In-situ time					
Hours (SD)	97 (115)	202 (186)	124 (148)	197 (136)	0.009
Days	4.0	8.4	5.1	8.1	
Tips cultured (% of CVCs)	136 (67%)	51 (66%)	187 (67%)	19 (73%)	nt
Tips infected (% of CVCs)	20 (9%)	11 (14%)	31 (11%)	4 (15%)	0.51
Catheter-related bloodstream infections					
Total no.	2 (1%)	0	2 (1%)	1 (4%)	nt
Per 1000 CVC days	2.5	0	1.4	6.0	0.33
CVC reinsertions (% of CVCs)	15 (7%)	4 (5%)	19 (7%)	0	0.38
Mean no. of ports idle (per day)	na	1.5	na	1.6	nt
Peripheral catheters					
Total no.	260	36	296	50	nt
Mean no. per patient	1.5	0.5	1.2	0.5	nt
Mean in-situ time (h)	63	66	64	81	nt

CVC = central venous catheter. nt = not tested. na = not applicable.
* Reasons for appropriate CVC retention were drug administration (32%), poor peripheral access (34%), transfer to another high dependency unit (25%) and total parenteral nutrition (9%).

Our intensive care unit maintained an informal clinical practice of routinely removing CVCs when patients were discharged from the unit. However, an audit found that many CVCs were retained, often inappropriately, thus exposing patients to needless increased risk.[5] A formal intervention policy aimed at improving CVC removal was implemented. This included a month of staff education, culminating in introduction of a formal written policy in March 2003. CVCs were to be removed when no longer clinically required or at discharge from the intensive care unit, unless the patient met predetermined retention criteria (ie, administration of vasoactive or venotoxic drugs [eg, dopamine or vancomycin] or parenteral nutrition solutions; poor peripheral venous access [after two attempts] with ongoing need for intravenous therapy; or transfer to another intensive care or coronary care unit).

We undertook a prospective observational study of all patients with CVCs in the intensive care unit of our hospital in the period March to August 2003. Patients were grouped according to whether the CVC was removed per policy before or at discharge from the intensive care unit; whether it was retained per policy at discharge from the unit; or whether it was retained in breach of policy. All patients were followed up for 7 days after CVC removal. Those who died within this time were excluded from the analysis.

We studied a total of 305 CVCs in 272 patients (Box). We observed:

- high compliance with the written policy (91%),
- significantly lower CVC in-situ times when policy was followed (5.1 v 8.1 days),
- low CVC reinsertion rates (7%),
- no difference in incidence of bloodstream infections between the groups.

This study demonstrates that a formal policy directed at early CVC removal is effective in lowering CVC in-situ times without incurring clinical cost to the patients (eg, excessive CVC reinsertion rates). Policy breaches were infrequent (8% of all CVCs), but, when they occurred, CVC retention appeared unnecessary, and CVC in-situ times were significantly prolonged. The risk of sepsis with CVCs may be substantially lowered by policy-driven removal of CVCs, without compromising patient care.

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Inhalation-device polypharmacy in asthma

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To the Editor: The delivery of asthma drugs via inhalation offers the best balance between efficacy and safety. However, poor inhalation technique limits the efficacy of this approach. In recent years, there has been a progressive increase in the types of inhalation devices used in asthma management. We questioned whether this would lead to "inhaler-device polypharmacy", a

Number of asthma patients using single or multiple inhalation devices and proportion of those patients with inadequate technique, over two time periods

	One device	Two devices	Three or more devices
Period 1* ($n = 278$)	75 (27%)	150 (54%)	53 (19%)
Period 2† ($n = 233$)	69 (30%)	129 (55%)	35 (15%)
Patients with inadequate inhalation-device technique			
Period 1*	19/75 (25%)	35/150 (23%)	23/53 (43%)
Period 2†	25/69 (36%)	64/129 (50%)	21/35 (60%)

* 1 Jan 2000–1 Jan 2002. † 2 Jan 2002–1 Jan 2004.

situation in which an individual used multiple types of inhalation device to deliver his or her asthma medications. We conducted a novel investigation of this issue in 2004.

We examined the computerised records of adults with asthma who had been enrolled in a standardised, evidence-based asthma management and education program[1] between 2000 and 2004. We noted the number and type of inhaler devices used, as well as competence with each device (a trained asthma educator had observed and scored inhalation technique). We defined "inhaler-device polypharmacy" as the use of two or more different types of inhalation device. The devices assessed in the education program included a pressurised metered-dose inhaler (with and without a spacer), turbuhaler, accuhaler, aeroliser, autohaler, and handihaler. Nebuliser use was not included in the evaluation.

We assessed a total of 511 patients: 278 (107 male; mean age, 37 years) between 1 January 2000 and 1 January 2002 (Period 1), and 233 patients (55 male; mean age, 40 years) between 2 January 2002 and 1 January 2004 (Period 2).

Period 1 patients were distinct from Period 2 patients in that the latter began their treatment after the release of combination asthma therapy in a single inhaler, when polypharmacy may have been expected to diminish.

Inhaler-device polypharmacy was present in 203 (73%; 95% CI, 68%–78%) patients during Period 1 and 164 (70%; 64%–75%) in Period 2 ($P = 0.3$) (Box).

In Period 1, inhalation technique was inadequate with at least one device in 58 (29%) patients using inhaler polypharmacy and in 19 (25%) using only one device (Box). In Period 2, inhalation technique was inadequate with at least one device in 85 (52%) patients using inhaler polypharmacy and in 25 (36%) using only one device. In both Period 1 and Period 2 patients, inadequate inhaler technique with at least one device increased with the number of devices used (P values 0.02 and 0.05, respectively) (Box).

We conclude that inhaler-device polypharmacy is a common problem among adults with asthma. Inadequate inhalation-device technique is also common, especially among patients using three or more delivery devices. Inhaler-device polypharmacy could lead to poor asthma control through inadequate delivery of medication. Patients with poor asthma control should be evaluated for their asthma management skills, including competency in using inhaler devices. These skills should be optimised before a new drug and/or device is added to their treatment regimen. We see no justification for the use of more than two inhalation delivery devices in asthma management.

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Smoking and pregnancy

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TO THE EDITOR: Problems with interpreting odds ratios reported in meta-analyses of smoking-cessation interventions have recently been highlighted.[1] Ford and Dobson[2] have erred in a different way when applying the findings of the Cochrane review on smoking cessation interventions in pregnancy[3] to calculate the public health benefits of delivering such interventions to all pregnant women in Australia.

When all methodologically acceptable randomised controlled trials were considered, the Cochrane review did find the prevalence of smoking at end-of-pregnancy was 6% lower in intervention than control groups.[3] However, this does not equate to a 6% reduction in the population prevalence of smoking among pregnant women, as Ford and Dobson assume. A mean between-group difference reported in a meta-analysis is not equivalent to a difference of exactly the same magnitude in a population prevalence of a risk factor unless 100% of the population exhibit that risk factor. Clearly, as Ford and Dobson have reported, this is not the case with smoking in pregnancy, where they correctly note that about 20% of pregnant women report current smoking at their first antenatal visit.[2] Therefore, smoking-cessation interventions would not reduce the prevalence of smoking by 6% from 20% to 14%. The expected reduction can be calculated as follows: expected reduction in prevalence of smoking in pregnant women = current smoking prevalence in pregnant women (20%) between-group difference in smoking prevalence (0.06) = 1.2%.

This calculation rests on two assumptions: namely, that all pregnant women in Australia currently receive usual smoking-cessation care equivalent to that of control group conditions in the Cochrane review[3] and that, in the short term, antenatal care can be transformed to the point where all future pregnant women receive smoking-cessation care equivalent to that received by those in intervention groups in the Cochrane review.

Therefore, it is obvious that the expected smoking prevalence of 18.8% (20% minus 1.2%) is considerably higher than the 14% calculated by Ford and Dobson.[2] Unfortunately, this means the rates of reduced infant deaths, hospital separations and costs to the healthcare system estimated by Ford and Dobson have also been overstated.

In summary, the gains to be expected by clinical interventions with pregnant smokers are modest. Furthermore, past evaluations of media campaigns directed specifically at pregnant women have not shown significant positive effects.[4] This reinforces the importance of tobacco-control strategies which target the whole population in addition to those which target pregnant women.[5]

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3 Lumley J, Oliver SS, Chamberlain C, Oakley L. Interventions for promoting smoking cessation dur-

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5 Lowe JB, Wakefield M. Smoking and pregnancy: time to implement evidence-based solutions. *Aust N Z J Public Health* 1998; 22: 523. ❑

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In reply: We thank Walsh and Lumley for correcting the error in our letter. The 6% reduction in smoking during pregnancy referred to an absolute difference in prevalence of continued smoking in late pregnancy among women who smoked early in pregnancy, from 91% in the control groups to 85% in the treatment groups.[1] We incorrectly hypothesised a reduction from 20% to 14% in prevalence of any smoking during pregnancy.

In fact, there was a decline in smoking during pregnancy, from 22% in 1994 to 17% in 2001 in New South Wales.[2] These figures illustrate well the final point that Walsh and Lumley make: whole-of-population approaches to smoking reduction can yield much greater benefits (a 5% reduction in 7 years in NSW) than high-risk approaches (from our data, the 1.2% calculated by Walsh and Lumley).

Our estimates of the adverse effects of smoking in pregnancy are, at present, correct. Although we unfortunately overstated the possible reductions resulting from interventions targeted only at pregnant women, such reductions are plausible for whole-of-population approaches.[3]

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Trends in the use of hospital beds by older people in Australia: 1993–2002

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To the Editor: Gray, Yeo and Duckett used the wrong basis of measure for their analysis of bed trends.[1] Bed use per thousand of the population masks the trends in total bed-days or separations and does not address the issue of supply. These issues have important ramifications for policy decision-making.

Using the same sources of data,[2,3] we compared bed-related statistics and population changes for the periods 1993–94 and 2001–02. What should be of most interest to planners is that the number of multi-day bed-days only declined marginally (from 14 434 to 14 231; −1.4%), despite the significant increase (from 1698 to 3343; + 96.8%) in same-day activity.

Although multi-day separations and bed-days did decline (separations, −4.2%; bed-days, −14.9%) for those aged 65–74 years, for those aged 75 years or over bed-days and separations increased significantly (separations, + 41.6%; bed-days, +27.7%). Furthermore, same-day activity increased significantly for those aged 65 or more years.

Furthermore, the authors failed to highlight the implications of changes in the relative age mix of activity. For those aged 75 years or more, the increase in proportion of separations (+ 5.8 percentage points) and bed-days (+ 1.8 percentage points) was greater than the increase in this proportion of the population (+ 1.1 percentage points). For the 65–74-years age group, the proportion of same-day hospital activity increased (+ 1.3 percentage points), unlike the reduction in that proportion of the population (−0.2 percentage points).

Moreover, the question of whether an ageing population has resulted in the need for more beds can not be answered without considering the supply of beds. From our experience, the growth in same-day activity has been achieved, at least in part, by substituting same-day beds for inpatient beds. The need for increased same-day beds has been considerable. Statistics relating to same-day beds do not appear to be reported for Australia as a whole. However, the increasing implied bed occupancy (including same-day) shown in the Box supports this conclusion.

We surmise that the reduction in supply of multi-day beds combined with a marginally altered demand for multi-day beds has led to increasing numbers of bed crises. Given that relative growth in same-day activity can be attributed to people aged 65 years or over, and that the number of multi-day bed-days for those aged 75 years or more has increased, it appears that the ageing of the population, combined with the manner in which the substitution of beds has occurred, has contributed to increasing bed crises.

Competing interests. None identified. The views of Mark Mackay are personal and in no way represent those of his employer.

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2 Australian Bureau of Statistics. Australian historical population statistics. 3. Population age–sex structure. Canberra: ABS, 2002. (ABS Catalogue No. 3105.0.65.001.)
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Changes in implied bed occupancy

	Financial year				Change from 1998–99 to 2001–02
	1998–99	1999–00	2000–01	2001–02	
Total bed-day utilisation (000s)	22 323	22 597	22 467	23 218	+ 4.0%
Total available bed-days (000s)	28 868	28 540	28 675	28 787	−0.3%
Implied occupancy	77%	79%	78%	81%	+ 4.3%

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In reply: Mackay and Millard have raised some important issues in relation to our analysis. Our article was developed to encourage wider reflection and their response is thus welcomed.

The primary criticism levelled by Mackay and Millard was that we underplayed the importance of supply of beds in our interpretation of the trends. We agree that bed supply is an important driver of utilisation patterns. We acknowledged this, in part, in the discussion as a possible explanation for rising separation and declining bed-utilisation rates in the older patient population. We are also sympathetic to the hypothesis that there may be a process of substitution of same-day separations for multi-day separations.

However, data relating to bed availability are not readily available, and thus could not be included in our study. Our article was designed to highlight different trends between age groups, which have not previously been reported. Now that these trends have been identified, further research and analysis is required to fully explain them, with a view to supporting an intelligent strategy to prepare for future population ageing. ❑

Whistleblowing in the Australian public hospital system

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To the Editor: Three recent articles in the *MJA* about complaints by patients attending hospital,[1] attitudes of hospital staff toward incident reporting[2] and whistleblowing[3] show that complaints are common, that cultural change is needed to allow staff to understand that a complaint from a patient or staff member should be viewed as an opportunity for change, and that quality assurance sometimes relies on whistleblowers but does not always appreciate their efforts.

Faunce and Bolsin report on three whistleblower incidents,[3] but fail to mention one at the Royal Children's Hospital, Melbourne, in which I was involved. Attempts to highlight deficiencies in delivery of paediatric surgical care and concerns about the response to adverse events were managed with threatening tactics (of dismissal) by the division of surgery. This was followed by a hospital board investigation that, in my opinion, had neither the skill mix nor the terms of reference to adequately investigate the quality of care or the bullying. The subsequent investigation by the Department of Human Services involved narrowly focused terms of reference and failed to consider outcomes in some circumstances, thereby facilitating the "shooting of the messenger".

Current legislation does not effectively allow for dealing with threatening behaviour in the workplace, particularly when the refusal to look at complaints and adverse

events in a productive manner goes well beyond the confines of the hospital involved. The Community Advisory Committee parliamentary enquiry was held in camera, with evidence being kept from the public. Worksafe legislation on bullying does not deal well with the complex situations that arise in the healthcare industry.[4]

The Colleges and other professional bodies, such as the AMA and the Medical Boards, need to take a proactive rather than a reactive role if further whistleblower incidents are to be avoided.

I concur with the statement of Faunce and Bolsin that: "Even after substantiation of their allegations, the whistleblowers ... received little respect and support from their institutions or professions".[3] From personal experience, I am very aware of the lack of support that stems from an ethos wary of public criticism, and the reactive bullying to which the whistleblower is often subjected.

Until the culture of healthcare focuses on quality and caring, whistleblower sagas will continue to occur.

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3 Faunce TA, Bolsin SNC. Three Australian whistleblowing sagas: lessons for internal and external regulation. *Med J Aust* 2004; 181: 44-47.
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The direct thrombin inhibitor melagatran/ximelagatran

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To the Editor: If a new drug such as ximelagatran is to be considered as a replacement for warfarin in preventing the thromboembolic complications associated with atrial fibrillation (AF), drug cost becomes an important issue. Brighton's recent article in the Journal,[1] while comprehensive, does not discuss the cost-effectiveness of ximelagatran treatment. Ximelagatran was approved in several European countries for the prevention of venous thromboembolism associated with orthopaedic surgery. The cost of the drug for this indication (24 mg given twice daily) is 4.5 euros (A$7.7) per day.[2] This represents the best available estimate of the cost of using ximelagatran for AF, although the dose is higher in AF (36 mg twice daily), and there are limitations in applying the drug cost in one country to another country.

Routine monitoring of the antithrombotic effect of ximelagatran (ie, international normalised ratio [INR] testing) was not conducted in clinical trials. While this is potentially advantageous, frequent testing of alanine aminotransferase (ALT) levels is recommended at baseline and monthly for the first 6 months of therapy, every second month for the remainder of the first year, and every third month thereafter, for safety reasons.[3] This is because some patients taking ximelagatran will develop elevated ALT levels (about 6.1% of patients to greater than threefold normal, and 3.4% to greater than fivefold normal) when ximelagatran therapy is commenced.[3]

The costs of INR and ALT tests are very similar (about $25 and $22, respectively).

Estimated costs of treating 1000 patients with atrial fibrillation (AF) with ximelagatran or warfarin for the first year of therapy.

	Ximelagatran*	Warfarin†
Total cost	$2 803 821.00	$106 800.00
Cost per patient	$2 803.82	$106.80
Monitoring		
Test (frequency/year)	ALT (10)‡	INR (20)§
Total cost	$217 000.00	$507 000.00
Cost per patient	$217.00	$507.00
Major bleeding¶		
Annual incidence**	1.6%	2.2%
No. of expected events	16	22
Total cost	$38 730.00	$53 253.00
Ischaemic stroke††		
Annual incidence‡‡	1.6%	1.6%
No. of expected events	16	16
Total cost	$101 936.00	$101 936.00
Overall cost		
Total	$3 161 487.00	$768 989.00
Per patient	$3 161.49	$768.99
Cost difference compared with warfarin		
Total	$2 392 498.00	—
Per patient	$2 392.50	—

ALT = Alanine aminotransferase. INR = International normalised ratio.
* Cost of giving ximelagatran (24 mg twice-daily) to prevent venous thromboembolism post-surgery (German data; the dose for prevention of thromboembolism in AF is 36 mg twice-daily).[3]
† Cost of warfarin taken from the Australian Pharmaceutical Benefits Scheme, December 2004.
‡ Monitoring cost derived from the cost of conducting ALT testing (Medicare Benefits Schedule, December 2004) according to the manufacturer's directions (tests at baseline, monthly for the first 6 months, 2-monthly for remainder of the first year).
§ Cost derived from Medicare Benefits Schedule (December 2004) based on a frequency of 20 tests per annum.
¶ The cost of a hospital admission caused by a major bleed was $2420.60 in Australia for the years 2002–2003 (estimate based on 2002–2003 public hospital data).[4] Intracranial haemorrhage may be associated with significant ongoing costs, but a smaller proportion of major bleeding incidents.
** No significant difference between warfarin and ximelagatran in either SPORTIF III[5] or V;[6] statistically significant when data from both trials were combined at $P < 0.05$.
†† Cost ($6371) taken from the NEMESIS study[7] and covers acute admission to an Australian hospital with ischaemic stroke only; this is an underestimate of the ongoing costs associated with ischaemic stroke.
‡‡ No significant difference between warfarin and ximelagatran in SPORTIF III[5] and V;[6] no significant difference when data from both trials were combined at $P < 0.05$.

Although INR monitoring may be more frequent with warfarin than ALT testing with ximelagatran, the cost difference associated with therapeutic monitoring would remain far less than the likely cost of ximelagatran. We estimate the cost associated with treating 1000 patients with AF with ximelagatran instead of warfarin for 1 year, taking into account drug costs, monitoring costs and the slight difference in major bleeding rates, to be about $2.4 million (Box).

A United States Food and Drug Administration advisory committee has recently raised concerns about the safety of ximelagatran (after episodes of severe liver damage), and has recommended that it not be granted any indication for use without further safety data. In particular, ALT monitoring did not prevent 3 deaths attributable to ximelagatran-associated hepatocellular necrosis.[3] In light of the recent withdrawal of rofecoxib (Vioxx; Merck Sharp & Dohme), warfarin carries the intangible benefits of a long and proven track record. It certainly requires careful management and ongoing monitoring, but healthcare resources might be better spent on improving the use of warfarin rather than paying substantially increased costs for a drug with similar efficacy and an uncertain safety profile.

1 Brighton TA. The direct thrombin inhibitor melagatran/ximelagatran. *Med J Aust* 2004; 181: 432-437.
2 Controversial: oral thrombin inhibitor ximelagatran (exanta). Available at: www.arznei-telegramm.de/journal/j_0407_a.html (accessed Dec 2004).
3 US Food and Drug Administration: Statistical review and evaluation-clinical studies (Exanta 36 mg bid oral formulation). Available at: www.fda.gov/ohrms/dockets/ac/04/briefing/2004-4069b1.htm (accessed Dec 2004).
4 Australian Institute of Health and Welfare. Australian hospital statistics 2002–03. Available at: www.aihw.gov.au/publications/index.cfm/title/10015 (accessed Dec 2004).
5 SPORTIF III Investigators. Stroke prevention with the oral direct thrombin inhibitor Ximelagatran compared with warfarin in patients with nonvalvular atrial fibrillation (SPORTIF III): randomised controlled trial. *Lancet* 2003; 362: 1691-1698.
6 Stroke prevention using the oral direct thrombin inhibitor Ximelagatran in patients with nonvalvular atrial fibrillation (SPORTIF V). Late-breaking clinical trial abstracts [abstract]. *Circulation* 2003; 108: 2723. Available at: http://circ.ahajournals.org/cgi/content/full/108/21/2723 (accessed Feb 2005)
7 Dewey HM, Thrift AG, Mihalopoulos C, et al. Lifetime cost of stroke subtypes in Australia: findings from the North East Melbourne Stroke Incidence Study (NEMESIS). *Stroke* 2003; 34: 2502-2507. ❑

Australasian Association of Doctors' Health Advisory Services

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To the Editor: Thank you for your in-depth look at some of the concerns in doctors' health in the October 2004 issue of the *MJA*.[1]

I believe it would have been useful to include in the issue some practical information for doctors wanting to seek help, either for themselves or for a colleague.

The attached table of contact phone numbers does not cover every state and territory, but it is a starting point. The services offered are confidential, and can be anonymous if desired. In Western Australia, the contact can include access to a list of doctors willing and able to be GPs for their colleagues. Further information is available on the Doctors' Health Advisory Service website <www.doctorshealth.org.au>.

Australasian Association of Doctors' Health Advisory Services (DHASs): helplines for doctors

Victorian Doctors' Health Programme (VIC)	(03) 9495 6011
DHAS (SA)	(08) 8273 4111
Colleague of First Contact (WA)	(08) 9321 3098
DHAS (NSW)	(02) 9437 6552
DHAS (New Zealand)	(04) 471 2654
DHAS (QLD)	(07) 3833 4352
DHAS (TAS)	(03) 6223 2047 (in hours) (03) 6235 4165 (after hours)

Editor's note: The AMA website also has a very useful "Doctors' Health Database" at <www.ama.com.au/web.nsf/tag/doctors-health-database>.

1 Doctors' health. *Med J Aust* 2004; 181 (7). ❑

Prescription shoppers line

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To the Editor: Over the past two years, the Journal has pointed out the health hazards and the lack of logic in the Health Insurance Commission's closure of its previously cost-effective and successful "Doctor Shopping Hotline".[1-4] But it has taken the death of a 25-year-old "prescription shopper" in Cairns, his crusading mother, a scathing report by the Queensland Coroner[5] and public exposure of these problems by Mark Bannerman on ABC TV (*The 7.30 Report*, 22 Dec 2004) for discernible action to occur.

On that program, the Federal Minister of Health and Ageing promised that a "Prescription Shopper Line" would be up and running by the end of January 2005, and indeed it was activated on 31 January.

This leaves two outstanding issues. The first is for the Health Insurance Commission to engage in an open exercise of mutual education by clearly reviewing its process of thinking in closing the previously successful Doctor Shopping Hotline and its lack of urgency in reinstituting its proposed better and broader successor.[6]

The second, and more important, issue is in understanding the underlying factors and thought processes of those doctors whom prescription shoppers describe as an "easy touch".[7,8]

1 Kamien M. Doctor shoppers' rights: privacy or lunacy? [letter]. *Med J Aust* 2003; 178: 248.
2 Kamien M. "Doctor shoppers": at risk by any other name [editorial]. *Med J Aust* 2004; 180: 204-205.
3 Martyres RF, Clode D, Burns JM. Seeking drugs or seeking help? Escalating "doctor shopping" by young heroin users before fatal overdose. *Med J Aust* 2004; 180: 211-214.
4 Hart JM. "Doctor shoppers": at risk by any other name [letter]. *Med J Aust* 2004; 181: 342-343.
5 Inquest into the cause and circumstances surrounding the death of George Shoobridge. 28102004 D9 T4/GRB m/T CAIR03/298 (Previtera, Coroner).
6 Whalan I. "Doctor shoppers": at risk by any other name [letter]. *Med J Aust* 2004; 181: 343.
7 MacQueen AR. "Doctor shoppers": at risk by any other name [letter]. *Med J Aust* 2004; 181: 342.
8 Breen CL, Degenhardt LJ, Bruno RB, et al. The effects of restricting publicly subsidized temazepam capsules on benzodiazepine use among injecting drug users in Australia. *Med J Aust* 2004; 181: 300-304. ❑

Free Auslan interpreter service for healthcare practitioners

31 January, 2005, saw the commencement of a new initiative through the Commonwealth Department of Family and Community Services. The new service — the National Auslan Interpreter Booking and Payment Service (NABS; <www.nabs.org.au>) — is available to private medical and healthcare practitioners and Auslan users **free of charge**.

Until now the cost of having an Auslan (Australian Sign Language) interpreter at private medical or healthcare appointments has been borne by either the patient, or by state deaf societies. This has been recognised as an access and equity issue for the Australian deaf community and other users of Auslan. As a result, the Australian Government has allocated $18.4 million over the next 3.5 years to provide this essential service.

Wesley Mission Brisbane has been selected as the organisation to establish and operate the service, which operates from a national call centre located at the corporate office of Wesley Mission on the north side of Brisbane.

The national call centre will operate between 8 am and 8 pm local time in all Australian states and territories.

An interpreter to attend appointments with users of Auslan can be booked by:

- Phone (voice):1800 24 69 45
- Phone (telephone typewriter [TTY]): 1800 24 69 48
- Fax: 1800 24 69 14
- Email: bookings@nabs.org.au
- SMS: 0427 671 261
- Post: National Auslan Interpreter Booking and Payment Service, 930 Gympie Road, Chermside, QLD 4032

NABS provides free interpreting services for private medical appointments with:

- general practitioners and specialists
- Aboriginal health workers
- audiologists
- dietitians
- mental health workers
- occupational therapists
- physiotherapists
- podiatrists and chiropodists
- chiropractors
- osteopaths
- psychologists
- speech pathologists
- dentists
- optometrists

Correspondents

We prefer to receive letters by email (medjaust@ampco.com.au). Letters must be no longer than 400 words and must include a word count. All letters are subject to editing. Proofs will not normally be supplied. There should be no more than 4 authors per letter. An "Article Submission Form" (www.mja.com.au/public/information/instruc.html) must be completed and attached to every letter.

There should be no more than 5 references. The reference list should not include anything that has not been published or accepted for publication. Reference details must be complete, including: names and initials for up to 4 authors, or 3 authors et al if there are more than 4 (see mja.com.au/public/information/uniform.html#refs for how to cite references other than journal articles).

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