LETTERS

Stroke among Indigenous Australians at Royal Darwin Hospital, 2001–02

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To the Editor: Although the age-standardised stroke mortality rates among Australia's Indigenous people is more than twice that of the non-Indigenous population,[1] the medical literature contains only one audit of Indigenous stroke patients in Perth metropolitan hospitals.[2] No review of hospital care has been reported.

Royal Darwin Hospital (RDH) is the referral centre for Australia's "Top End", where 8.7% of Indigenous Australians reside; 40% of RDH inpatients are Indigenous. In 2002, while planning for the RDH stroke service, we audited stroke admissions from the previous year.

Among 121 eligible patients admitted between 1 July 2001 and 31 June 2002 with *International classification of diseases, 10th revision, Australian modification* (ICD-10-AM) codes 160–164 (haemorrhages [subarachnoid, intracerebral, other non-traumatic intracranial] and cerebral infarction), records for 116 (96%) were available, but six patients were excluded because of incorrect coding.

Box 1 outlines patient characteristics, while Box 2 examines risk factors and medication use for ischaemic stroke (because haemorrhages were few). Despite the observed differences between subgroups, there were no significant differences in mortality (4/36 for Indigenous v 7/42 for non-Indigenous; $P = 0.204$) or stroke severity at admission or discharge.

Box 3 highlights differences in risk factors between Indigenous males and females.

Retrospective data, particularly from a sample identified by medical record coding, should be interpreted with caution. In addition, the potential for random error due to small numbers, and the referral bias inherent in tertiary hospital admissions, mean our results may not truly represent the "Top End" Indigenous population. However, our data corroborate findings that Indigenous Australians suffer premature cerebrovascular disease, and have higher rates of vascular risk factors than other Australians,[1] with some risk factor differences between males and females. Further, recent evidence suggests differences in standards of stroke care in regional (Queensland) hospitals.[3] We found disparity in hospital care of Indigenous patients, and this requires further detailed investigation. A prospective, community-based study is urgently needed.

1 Baseline characteristics for 110 patients admitted to Royal Darwin Hospital with subarachnoid, intracerebral, and other non-traumatic intracranial haemorrhages and cerebral infarction in 2001–02

Baseline characteristics	Indigenous	Other	*P*
Number of patients	45	65	
Female sex	22 (49%)	19 (29%)	0.018
Mean age (years)	54	61	0.005
Rural dwelling	41 (91%)	22 (34%)	< 0.001
Ischaemic stroke	36 (80%)	42 (65%)	0.081

2 Risk factors and medication use for the 78 patients who had ischaemic stroke

Risk factors and medications	Indigenous	Other	*P*
All patients	36	42	
Smoking	23 (64%)	11 (26%)	0.001
Diabetes mellitus	16 (44%)	10 (24%)	0.030
Rheumatic heart disease	8 (22%)	1 (2%)	< 0.001
Males	19 (53%)	30 (71%)	0.089
Smoking	14 (74%)	12 (40%)	0.017
Diabetes mellitus	10 (53%)	7 (23%)	0.030
Females	17 (47%)	12 (29%)	0.089
Smoking	9 (53%)	0	0.002
Rheumatic heart disease	6 (35%)	0	0.026
Antiplatelet therapy			
Before admission	11 (31%)	19 (45%)	0.078
Admission	20 (56%)	38 (91%)	< 0.001
Discharge	18/32 (56%)	29/35 (83%)	0.013
Anticoagulant therapy			
Before admission	4 (11%)	1 (2%)	< 0.001
Discharge	4/32 (13%)	6/35 (17%)	0.235

3 Risk factor differences between Indigenous males and females who had ischaemic stroke

	Males	Females	*P*
Number of patients	19	17	
Hypertension	16 (84%)	6 (35%)	0.003
Non cerebral vascular disease	6 (32%)	1 (6%)	< 0.001
Excessive alcohol intake	7 (37%)	1 (6%)	< 0.001

1 Australian Institute of Health and Welfare. Heart, stroke and vascular diseases — Australian facts 2004. Canberra: AIHW, 2004: 140.
2 Crowley P, Hankey GJ. Stroke among Australian Aboriginals in Perth WA, 1988–1992 [letter]. *Aust N Z J Med* 1995; 25: 55.
3 Read SJ, Levy J. Differences in stroke care practices between regional and metropolitan hospitals. *Intern Med J* 2005; 35: 447-450. ❑

Community-acquired methicillin-resistant *Staphylococcus aureus* in bone and joint infections: development of rifampicin resistance

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To the Editor: Community-acquired methicillin-resistant *Staphylococcus aureus* (CA-MRSA) is usually susceptible to a wider range of antibiotics than nosocomial or multiresistant MRSA, but evidence is lacking to guide antibiotic choices.

Rifampicin plus fusidic acid is commonly used in multiresistant MRSA infection, and familiarity makes this combination attractive for CA-MRSA. We report two cases of CA-MRSA infection in which rifampicin resistance developed during treatment. Non-compliance may have been a significant factor, but high bacterial load and persistent foci of infection were also likely contributors. We caution against using regimens containing rifampicin in such cases.

Patient 1: A previously well 16-year-old girl presented with a 4-day history of left knee pain. She had fever and a moderate joint effusion. Plain x-rays were unremarkable, and blood cultures showed no growth. An aspirate of the knee joint had a white blood cell count of 1100×10^6 cells/L, but showed no growth on culture. Non-multiresistant MRSA (sensitive to all non-β-lactams) was subsequently grown from a synovial biopsy specimen. She was treated with 7 weeks of intravenous vancomycin, followed by 6 months of oral rifampicin (450 mg daily) plus fusidic acid (500 mg twice daily). Her compliance was uncertain, and she did not return for follow-up appointments.

The patient presented again 12 months later with increasing pain in the left knee and thigh. X-ray and magnetic resonance imaging revealed extensive osteomyelitis of the distal femur. Culture of purulent material removed at sequestrectomy grew MRSA with the same sensitivity profile as previously, but now resistant to rifampicin. She was treated with clindamycin, but required further sequestrectomy. She continues to take clindamycin long term.

Patient 2: A 74-year-old man developed severe cellulitis and an abscess of his left hand after a golfing injury. He had bacteraemia with non-multiresistant MRSA (sensitive to all non-β-lactams). He also had significant pain in his left prosthetic hip. After 7 weeks of intravenous vancomycin and oral moxifloxacin, levels of inflammatory markers remained high. He was treated with oral rifampicin (600 mg daily) plus fusidic acid (500 mg twice daily) for several weeks until lost to follow-up.

Four months later, the patient still had pain in the hip, and rifampicin plus fusidic acid treatment was begun again. After 3 months with no improvement, he underwent surgical exploration, and the loose femoral prosthesis was replaced. Culture of debrided material showed MRSA with the same sensitivity pattern as previously, but now resistant to rifampicin. He was treated with intravenous vancomycin for 6 weeks, followed by trimethoprim–sulfamethoxazole, which was later changed to clindamycin because of intolerance. He remains taking indefinite clindamycin suppression therapy.

Traditional oral therapy for MRSA infection is rifampicin plus fusidic acid, but alternative oral agents for non-multiresistant CA-MRSA include clindamycin,[1] tetracyclines[2] and trimethoprim–sulfamethoxazole.[3] There is wider experience with clindamycin, which penetrates well into skin and soft tissues, has good oral bioavailability, and has been used successfully.[1] However, a concern is erythromycin-inducible clindamycin resistance, which may lead to clindamycin failure, particularly in severe infections.[4] Its prevalence varies.

Studies in vitro have shown that rifampicin resistance develops as readily in CA-MRSA as in nosocomial MRSA,[5] through a single-step mutation. Resistance did not develop in vitro to clindamycin,[5] suggesting it may be a more reliable alternative in situations where compliance is uncertain, the bacterial load is high, or the source (eg, an infected prosthesis) cannot be removed. Further in-vitro and in-vivo investigations are urgently required to determine the optimal drug therapy for CA-MRSA infections.

1 Martinez-Aguilar G, Avalos-Mishaan A, Hulten K, et al. Community-acquired, methicillin-resistant and methicillin-susceptible *Staphylococcus aureus* musculoskeletal infections in children. *Pediatr Infect Dis J* 2004; 23: 701-706.

2 Ruhe JJ, Monson T, Bradsher RW, Menon A. Use of long-acting tetracyclines for methicillin-resistant *Staphylococcus aureus* infections: case series and review of the literature. *Clin Infect Dis* 2005; 40: 1429-1434.

3 Stein A, Bataille JF, Drancourt M, et al. Ambulatory treatment of multidrug-resistant Staphylococcus-infected orthopedic implants with high-dose oral cotrimoxazole (trimethoprim-sulfamethoxazole). *Antimicrob Agents Chemother* 1998; 42: 3086-3091.

4 Drinkovic D, Fuller ER, Shore KP, et al. Clindamycin treatment of *Staphylococcus aureus* expressing inducible clindamycin resistance. *J Antimicrob Chemother* 2001; 48: 315-316.

5 Munckhof WJ, Kleinschmidt SL, Turnidge JD. Resistance development in community-acquired strains of methicillin-resistant *Staphylococcus aureus*: an in vitro study. *Int J Antimicrob Agents* 2004; 24: 605-608. ❑

Evidence in palliative care research: how should it be gathered?

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To the Editor: Aoun and Kristjanson's article highlighted some of the difficulties facing researchers in palliative care[1] and questioned the role of randomised controlled trials (RCTs). These difficulties do not exempt palliative care from seeking to improve care through thoughtful research but, rather, highlight areas that need to be specifically designed to cope with these difficulties.

In our recently completed large RCT of palliative care in southern Adelaide, for which we recruited 461 patients,[2] we were able to overcome many of the obstacles cited by these authors.

As they rightly stated, many trials in palliative care are pragmatic. This should not be seen as a bad thing, as pragmatic studies are designed to test clinically relevant interventions[3] within diverse populations across different settings and to report on a broad range of health outcomes. Such studies are more in keeping with the realities of palliative care. We would argue that traditional RCTs that examine a specific intervention within a specific patient type are less applicable to a palliative care population. In such a population, unlike many other areas of health, the diagnosis and prognosis do not dictate care needs.

Among the methodological issues, recruitment and retention are perhaps the biggest hurdles to be overcome. We found that developing systematic, evidence-based protocols, which were pilot tested before initiating the trial, significantly enhanced recruitment.[4] To minimise burden, we ensured that all data collection was kept to a minimum and, where possible, data were recorded by study staff or collected from other sources as part of routine clinical encounters. Sample size calculations need to allow for attrition caused by increasing severity of disease. This will, however, inflate the numbers required for a study. Use of multiple sites for recruitment is a strategy that we have used successfully to reach sample size goals.

The article states that "RCTs are seldom acceptable to patients and their families".[1] This has not been our experience. Patients are willing to participate in research and find it a meaningful way to give back to the community.[5] The challenge is to ensure that this willingness to participate in research is not exploited by palliative care researchers. The other ethical concern raised by Aoun and Kristjanson is the use of a control arm in which patients "deliberately have support services withheld". We would agree that, if that were to happen, it would be a serious ethical breach. For RCTs in palliative care, the control arm should consist of the standard care provided — the rationale for doing a trial is that there is equipoise regarding the benefit of the intervention.

We feel that RCTs are feasible and appropriate for palliative care research.

1 Aoun SM, Kristjanson LJ. Evidence in palliative care research: how should it be gathered? *Med J Aust* 2005; 183: 264-266.

2 Abernethy AP, Currow DC, Hunt R, et al. A pragmatic 2x2x2 factorial cluster randomized controlled trial of educational outreach visiting and case conferencing in palliative care-methodology of the Palliative Care Trial (ISRCTN 81117481). *Contemp Clin Trials* 2006; 27: 83-100.

3 Tunis SR, Stryer DB, Clancy CM. Practical clinical trials: increasing the value of clinical research for decision making in clinical and health policy. *JAMA* 2003; 290: 1624-1632.

4 Abernethy AP, Currow DC, Frith P, Fazekas BS. Prescribing palliative oxygen: a clinican survey of expected benefit and patterns of use [letter]. *Palliat Med* 2005; 19: 168-170.

5 Bruera E. Ethical issues in palliative care research. *J Palliat Care* 1994; 10: 7-9. ❑

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TO THE EDITOR: Aoun and Kristjanson's viewpoint on palliative care research reminds us of the possible limitations of an evidence schema that is built on efficacy of intervention studies.[1] For emerging fields and for care areas that cross disciplines, the possible sources of useful knowledge to guide practice — particularly in advance of the establishment of a significant evidence base — need to be recognised and valued.

To further complicate the gathering of evidence for such fields are the difficulties of accessing useful knowledge. Preliminary research on search strategies suggests that even good quality searches may recover fewer than half the articles relevant to palliative care in the general biomedical literature.[2] More disturbing is the possibility that much of the research and thought in this field is not published, and therefore cannot be easily and actively searched.

In a systematic review of publication rates associated with conference presentation, the usual publication rate was seen to be around 45%.[3] A recent investigation into publication rates associated with conference presentation in palliative care in Australia suggests a "conversion" rate of less than 20%.[4] Publication represents an important step in the spectrum of knowledge dissemination. Such a low rate of publication of conference abstracts therefore represents a significant loss of information, opinion and evidence for the discipline of palliative care.

Evidence issues for complex and emerging areas are complicated not only by the restrictions of an evidence hierarchy that is intervention based, but also by the difficulties in searching and retrieving existing knowledge and evidence in such fields.

1 Aoun SM, Kristjanson LJ. Evidence in palliative care research: how should it be gathered? *Med J Aust* 2005; 183: 264-266.

2 Sladek R, Tieman J, Fazekas B, et al. Developing and validating a palliative care subject search filter. Oral presentation at the 3rd International Evidence Based Librarianship Conference. Evolution of evidence. Brisbane, Oct 2005. Available at: http://conferences.alia.org.au/ebl2005/program.html (accessed Oct 2005).

3 von Elm E, Costanza MC, Walder B, et al. More insight into the fate of biomedical meeting abstracts: a systematic review. *BMC Med Res Methodol* 2003; 3: 12. Epub 2003 Jul 10.

4 Tieman J, Abernethy A, Fazekas B, et al. CareSearch: finding and evaluating Australia's missing palliative care literature. *BMC Palliat Care* 2005; 4: 4. ❑

The use of therapeutic medications for soft-tissue injuries in sports medicine

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To the Editor: Paoloni and Orchard provided a concise summary of the evidence for injections for soft-tissue injuries,[1] but omitted some important references on the mechanism of action of corticosteroids and on prolotherapy.

An important action of corticosteroids is blocking of transmission in nociceptive C-fibres.[2] Given the lack of evidence of inflammation in chronically painful tendinopathies,[3] this is a more probable mechanism of action than the suppression of inflammation. Paoloni and Orchard correctly report that steroids have only a temporary effect in suppressing soft tissue pain. However, in low back pain, if their use is preceded by manual therapy and exercises they have the potential to give more prolonged relief of pain and disability.[4]

A recent Swedish randomised controlled trial (RCT) of polidocanol prolotherapy injections for chronic Achilles tendinopathy showed reduced pain and normalisation of ultrasound abnormalities.[5] Similarly, a New Zealand case series of glucose prolotherapy injections showed very positive results for the same condition.[6] An Australian RCT into prolotherapy for chronic low back pain (average duration, 14 years) showed sustained reductions in pain and disability with glucose prolotherapy injections, although similar results were obtained with saline injections.[7] A pilot study of glucose prolotherapy in 24 elite male kicking-sport athletes with chronic groin pain (mean duration, 15.5 months) who had failed physical therapy reported a pain-free state and return to sports in 82% at an average follow-up of 17.2 months.[8] This evidence would suggest there is a role for this glucose prolotherapy in managing soft-tissue pain, especially as musculoskeletal pain is one of the major presentations to primary practice in Australia. Training primary care physicians in prolotherapy injection techniques should be a priority in medical education.

1 Paoloni JA, Orchard JW. The use of therapeutic medications for soft-tissue injuries in sports medicine. *Med J Aust* 2005; 183: 384-388.

2 Johansson A, Hao J, Sjolund B. Local corticosteroid application blocks transmission in normal nociceptive C-fibres. *Acta Anaesthesiol Scand* 1990; 34: 335-338.

3 Khan KM, Cook JL, Kannus P, et al. Time to abandon the "tendinitis" myth. *BMJ* 2002; 324: 626-627.

4 Blomberg S, Svardsudd K, Tibblin G. A randomized study of manual therapy with steroid injections in low-back. *Eur Spine J* 1994; 3: 246-254.

5 Alfredson H, Ohberg L. Sclerosing injections to areas of neo-vascularisation reduce pain in chronic Achilles tendinopathy: a double-blind randomized controlled trial. *Knee Surg Sports Traumatol Arthrosc* 2005; 13: 338-344.

6 Lyftogt J. Prolotherapy and Achilles tendinopathy: a prospective pilot study of an old treatment. *Australas Musculoskel Med* 2005; 10: 16-19.

7 Yelland MJ, Glasziou PP, Bogduk N, et al. Prolotherapy injections, saline injections, and exercises for chronic low-back pain: a randomized trial. *Spine* 2004; 29: 9-16.

8 Topol GA, Reeves KD, Hassanein KM. Efficacy of dextrose prolotherapy in elite male kicking-sport athletes with chronic groin pain. *Arch Phys Med Rehabil* 2005; 86: 697-702. ❑

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In reply: We thank Masters and Yelland for their interest in this topic and their notification of additional references, some of which were published after our article was written.

We stated in our article that "the mechanism of any effect of corticosteroid injections in reducing symptoms in purely degenerative tendinopathies is unknown", and that only where bursitis or tenosynovitis is present would the implication of an anti-inflammatory effect be appropriate.[1] While blocking nociceptive C-fibres in normal tendon is demonstrated in the study quoted by Masters and Yelland,[2] we still believe that corticosteroids should be used with caution for any tendinopathy where tendon weakening would be potentially harmful. We agree that corticosteroids have a much greater potential role in low back pain, which is a broad entity involving both soft-tissue and joint disorder.

At the time of writing our article there was a pilot study on polidocanol in painful tendons displaying neovascularisation;[3] we thank the authors for advising that a randomised controlled trial has since been published.[4] While undoubtedly an exciting new therapy, proponents of polidocanol do not consider its mechanism to be simply a "prolotherapy" effect; they also consider sclerosing the neovessels to be critical, and therefore, that hypertonic glucose (the most commonly recommended prolotherapy agent) may not work as well.

We still maintain that prolotherapy currently lacks evidence of efficacy for the treatment of soft-tissue injury in general, although it is relatively cheap and generally free of side effects. Both chronic low back pain, and chronic groin pain, are multifactorial conditions involving joint/bone abnormality which we considered slightly beyond the scope of an article on soft-tissue injuries. We await further publications on the efficacy of prolotherapy with interest.

1 Paoloni JA, Orchard JW. The use of therapeutic medications for soft-tissue injuries in sports medicine. *Med J Aust* 2005; 183: 384-388.

2 Johansson A, Hao J, Sjolund B. Local corticosteroid application blocks transmission in normal nociceptive C-fibres. *Acta Anaesthesiol Scand* 1990; 34: 335-338.

3 Alfredson H, Ohberg L. Neovascularisation in chronic painful patellar tendinosis — promising results after sclerosing neovessels outside the ten-

don challenge the need for surgery. *Knee Surg Sports Traumatolo Arthrosc* 2005; 13: 74-80.
4 Alfredson H, Ohberg L. Sclerosing injections to areas of neo-vascularisation reduce pain in chronic Achilles tendinopathy: a double-blind randomized controlled trial. *Knee Surg Sports Traumatol Arthrosc* 2005; 13: 338-344. ❑

Mandatory fortification of flour with folic acid: an overdue public health opportunity

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To the Editor: The editorial on mandatory fortification of flour with folic acid by Maberly and Stanley is subtitled: "The scientific benefit is clear, but translating this into practice requires advocacy".[1]

The only benefit that is scientifically clear is the reduction in the incidence of neural tube defects. All the other "benefits" listed by the authors are observational and have occurred in a setting where myriad environmental changes have occurred concurrent with folic acid fortification. To imply that the reduction in the rate of heart attacks and stroke is the result of folic acid fortification is, at best, anecdotal, because it is not supported by any randomised controlled studies, and is an extrapolation from the relationship between reduced homocysteine levels and the incidence of stroke and heart disease.

The authors also did not mention the increased incidence of multiple pregnancies that has been observed with folate supplementation (relative risk, 1.02; 95% CI, 0.97–1.07).[2] While this represents only a slight increase in the risks associated with the birth process, it should not be ignored when considering perceived risks. It is also possible that in a planned pregnancy where the mother is prescribed folic acid before conception, the additional folate intake from fortified flour may further increase the risk of multiple pregnancy.

It seems to me that the editorial was in fact an item of advocacy rather than a dispassionate scientific assessment of the arguments for and against mandatory folic acid fortification. At the very least, if mandatory folic acid fortification is implemented, prospective mothers will have to be made aware of the increased risk of multiple pregnancy and the as yet unknown risk of combining the fortified diet with medically prescribed folic acid.

1 Maberly GF, Stanley FJ. Mandatory fortification of flour with folic acid: an overdue public health opportunity [editorial]. *Med J Aust* 2005; 183: 342-343.
2 Lumley J, Watson L, Watson M, Bower C. Periconceptual supplementation with folate and/or multivitamins to prevent neural tube defects. *Cochrane Database Syst Rev* 2001; (3): CD001056. ❑

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In reply: Ekert suggests that in our article advocating for mandatory fortification with folate to reduce neural tube defects,[1] we omitted to mention the "increased risk of multiple pregnancies". He then misquotes the Lumley meta-analysis "(relative risk, 1.02; 95% CI, 0.97–1.07)" — the real relative risk was 1.40 (95% CI, 0.93–2.11). This Cochrane systematic review shows that folate supplementation does not carry a statistically significant risk for multiple births, but confirms the dramatic reduction in neural tube defects.[2] Another study, which did suggest an increased risk, did not control for the known increased risk of multiple births following infertility treatments, which could explain the increase observed.[3]

Ekert suggests that we inform women about this unsubstantiated risk and "the as yet unknown risk" which mandatory fortification might add to "medically prescribed folic acid". Folic acid is found in leafy green vegetables and in many fruits, nuts and other components of a healthy diet. Tablets are available over the counter. What advice would he give to women about these "risks"? Our evidence is that we are not reaching many women in our society by education and voluntary fortification, and that countries that have fortified their flour have achieved much better reductions in these major defects than we have in Australia. Hence our advocacy.

We acknowledge that the evidence for stroke and heart disease reduction is not as solid as that for neural tube defects. However, there is an increasing literature on the protective effects of folate on cardiovascular risk and possible mechanisms.[4-8]

Hence, with consideration of the proven benefits and the unsubstantiated risks, we will continue to advocate for the mandatory fortification of flour with folate.

1 Maberly GF, Stanley FJ. Mandatory fortification of flour with folic acid: an overdue public health opportunity [editorial]. *Med J Aust* 2005; 183: 342-343.
2 Lumley J, Watson L, Watson M, Bower C. Periconceptual supplementation with folate and/or multivitamins to prevent neural tube defects. *Cochrane Database Syst Rev* 2001; (3): CD001056.
3 Berry RJ, Kihlberg R. Folic acid supplementation is associated with an increase in dizygotic twinning. *Early Human Dev* 2005; 81: 465-467.
4 Schnyder G, Roffi M, Pin R. Decreased rate of coronary restenosis after lowering of plasma homocysteine levels. *N Engl J Med* 2001; 345: 1593-1600.
5 Baker F, Picton D, Blackwood S, et al. Blinded comparison of folic acid and placebo in patients with ischaemic heart disease: an outcome trial [abstract]. *Circulation* 2002; 106 Suppl II: S741.
6 Lange H, Suryapranata H, De Luca G, et al. Folate therapy and in-stent restenosis after coronary stenting. *N Engl J Med* 2004; 350: 2673-2681.
7 Toole JF, Malinow MR, Chambless LE, et al. Lowering homocysteine in patients with ischemic stroke to prevent recurrent stroke, myocardial infarction and death. The Vitamin Intervention for Stroke Prevention Randomized Controlled Trial. *JAMA* 2004; 291: 565-575.
8 Cronin S, Furie KL, Kelly PJ. Dose-Related association of MTHFR 677T with risk of ischemic stroke. Evidence from a cumulative meta-analysis. *Stroke* 2005; 36: 1581-1587. ❑

The Bundaberg hospital scandal: the need for reform in Queensland and beyond

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To the Editor: It is heartening to see a positive professional response to clinical quality systems in the wake of Bundaberg. However, these are secondary responses, and overlook the primary, preventive solution.

Clinical monitoring systems presuppose that some damage is done before a problem becomes apparent. Hospitals already have mechanisms to examine clinical competence, and their appointments credentialing and privileging procedures.

The Queensland Health Systems Review Final Report (the Forster report)[1] comments repeatedly on the apparent failure of these procedures in the Patel case at Bundaberg Hospital:

> It appears that the process of checking credentials did not involve the College of Surgeons and no written clinical privileges appeared to have been granted on appointment. (p 169)

The report draws specific attention to special purpose registration for areas of need, pointing out the inherent conflict of interest:

> As an employer under pressure to fill medical vacancies, Queensland Health faces a conflict of interesting making . . . determinations of area of need to allow

special purpose registration of overseas trained doctors. (p 175)

It adds that these doctors are not subject to the same requirements as locally trained doctors, and recommends:

> No overseas trained doctor should commence employment in a senior position intended to be filled by a specialist before ... [assessment via the established Australian Medical Council/ Specialist College pathway]. (p 174)

Furthermore, deemed specialists should participate in the usual clinical performance management processes applicable to all doctors. (p 175) And, finally:

> ... local clinical leaders and managers have a conflict between credentialing someone about whom they are uncertain and having no one to deliver the service. It appears that these issues may have been relevant at Bundaberg. (p 176)

More recently, the Queensland Public Hospitals Commission of Inquiry report (the Davies report)[2] confirms a practice of appointing overseas trained doctors as senior medical officers in specialist roles in hospitals in designated areas of need. Such appointments in Bundaberg, Hervey Bay, Townsville and Charters Towers not only bypassed procedures for recognition as a "deemed specialist", but were also not considered by hospital credentialing and privileging committees. The Davies report states that, in some hospitals, these committees did not exist.

It may be premature for the proponents of clinical quality systems to dance on the ashes of Bundaberg. These recent reports outline a chain of existing systems problems including the Medical Board, the Department of Health, successive Health Ministers and Cabinets, and appointments, credentialing, privileging and complaints procedures in a sample of Queensland public hospitals.

According to the Davies report (section 6.173), around half of the doctors in Queensland public hospitals were appointed under area-of-need arrangements by 2002. How are other states balancing the politically sensitive area of need registration with long-standing appointments, credentialing and privileging procedures?

1 Queensland Government. Health Systems Review. Final Report. Forster report. September 2005. Available at: http://www.thepremier.qld.gov.au/news/media_matters/2005/30_09_05.shtm (accessed Dec 2005).
2 Queensland Public Hospitals Commission of Inquiry. Final report. Davies report. November 2005. Available at: http://www.qphci.qld.gov.au/Final_Report.htm (accessed Dec 2005). ❑

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