## LETTERS

## Prevalence of trachoma in Aboriginal communities in the Katherine Region of the Northern Territory in 2007

Katrina Roper, Claude-Edouard C Michel, Paul M Kelly and Hugh R Taylor

**To the Editor:** Trachoma, caused by the bacterium *Chlamydia trachomatis*, is the leading cause of infectious blindness worldwide.[1] In Australia, the burden of disease falls almost exclusively on the Aboriginal population.[2] However, there has been little consistent data collection on the prevalence of trachoma in recent years in Australia.[3,4] Furthermore, despite Australian Government recommendations for biennial screening of people aged 40–54 years and annual screening of people aged ⩾ 55 years in areas where trachoma is or has been endemic,[5] very little screening of older people for trachomatous trichiasis has been conducted.[2,4]

We report on the first large-scale population study in 30 years of the current prevalence of active and cicatricial trachoma in the Northern Territory Aboriginal population. We conducted a standardised clinical screening study of five Aboriginal communities in the Katherine Region of the NT over a 5-week period in 2007.

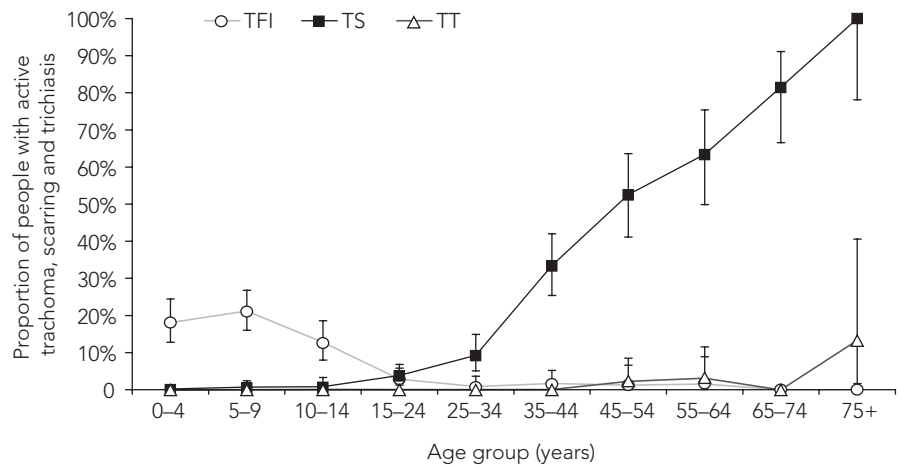
A representative sampling frame of those believed to be currently living in each community was constructed using the medical clinic patient list, the council housing list and the local knowledge of Aboriginal Health Workers seconded from the clinics to assist with the project. All people in each community were invited to undergo a clinical eye examination for trachoma. The parameters of the World Health Organization simplified grading scheme[6] were used to determine prevalence of the five signs of trachoma: tarsal conjunctival follicles, intense inflammation, tarsal scarring, trichiasis and corneal opacity.

A total of 1316 people (85.2% of the total estimated population), including 415 children aged under 10 years, were screened for trachoma. Across the five communities, active trachoma (assessed as the presence of either follicles or inflammation in one or both eyes) was at an endemic level (> 10%). The prevalences of active trachoma, scarring and trichiasis in different age groups are summarised in the Box. The overall rate of active trachoma in children under 10 years of age was 19.8% (95% CI, 16.0%–23.9%) ($n = 82$), and two communities had hyperendemic prevalence of trachoma (> 20%) in this age group. The youngest child observed with active trachoma was just over 1 year old.

The prevalence of scarring in people aged 20 years and over was 32% (95% CI, 28.3%–35.9%) ($n = 193$). The youngest person identified with scarring was 7 years old. Six people (2.3% of all people aged 40 years and over) were identified with trichiasis requiring urgent ophthalmological attention. Across the population, this placed the prevalence of unoperated trichiasis at more than four times the acceptable threshold set by the WHO. A seventh person had had trichiasis surgery.

That trachoma is still hyperendemic in Aboriginal communities more than 30 years after the National Trachoma and Eye Health Program first identified the extent of trachoma is unconscionable. Urgent and sustained public health and clinical interventions are required, with greater commitment from politicians and health policymakers, if Australia is to join the ranks of other developed nations in eradicating endemic trachoma. The guidelines for trachoma control developed by the Communicable Diseases Network Australia[5] need to be resourced appropriately and implemented.

**Prevalence of active trachoma, scarring and trichiasis in five Aboriginal communities in the Northern Territory in 2007, by age group***




TFI = active trachoma (follicles [TF] and/or inflammation [TI]). TS = trachomatous scarring. TT = trachomatous trichiasis. * Vertical bars indicate 95% CIs. ◆

*Acknowledgements:* We wish to acknowledge the funding and assistance provided by the Fred Hollows Foundation, in particular Alison Edwards, Nick Di Candilo and Bino Toby. We thank the health clinic staff in each community, in particular the Aboriginal Health Workers, and also the Katherine region health services, without whose cooperation this project would not have been possible: Sunrise Health Service, Wurli Wurlijang and Katherine West Health Board. We are grateful for the help of our field assistants Cath Kelaher, Bianca Webb-Pullman, Tomer Shemesh, Anu Mathew, Janet Taylor and Robyn Lilienthal. Katrina Roper conducted this study as part of her scholarship in the Master of Applied Epidemiology (MAE) degree at the Australian National University. The MAE program is funded by the Australian Government Department of Health and Ageing. Paul Kelly's salary is partly supported by the National Health and Medical Research Council.

**Katrina Roper,** Epidemiologist[1,2]
**Claude-Edouard C Michel,** Research Scientist[3]
**Paul M Kelly,** Associate Professor and Director[1]
**Hugh R Taylor,** Harold Mitchell Chair of Indigenous Eye Health[4]
1 National Centre for Epidemiology and Population Health, Australian National University, Canberra, ACT.
2 Centre for Disease Control, Department of Health and Community Services, Northern Territory Government, Darwin, NT.
3 Department of Haematology, University of Cambridge, Cambridge, UK.
4 School of Population Health, University of Melbourne, Melbourne, VIC.
h.taylor@unimelb.edu.au

1 Resnikoff S, Pascolini D, Etya'ale D, et al. Global data on visual impairment in the year 2002. *Bull World Health Organ* 2004; 82: 844-851.
2 Taylor HR. Eye health in Aboriginal and Torres Strait Islander communities. Report of a review commissioned by the Commonwealth Minister for Health and Family Services, the Hon Dr Michael Wooldridge. Canberra: Commonwealth of Australia, 1997.
3 Mak DB, O'Neill LM, Herceg A, McFarlane H. Prevalence and control of trachoma in Australia, 1997–2004. *Commun Dis Intell* 2006; 30: 236-247.
4 Tellis B, Keeffe JE, Taylor HR. Surveillance report for active trachoma, 2006: National Trachoma Surveillance and Reporting Unit. *Commun Dis Intell* 2007; 31: 366-374.
5 Communicable Diseases Network Australia. Guidelines for the public health management of trachoma in Australia. Canberra: Australian Department of Health and Ageing, 2006. http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-cdna-pubs-trachoma.htm (accessed Jan 2008).
6 Thylefors B, Dawson CR, Jones BR, et al. A simple system for the assessment of trachoma and its complications. *Bull World Health Organ* 1987; 65: 477-483. ❑

## A heart-stopping orbital injury

Vivek B Pandya, R Max Conway and Richard Conway

**To the Editor:** The oculocardiac reflex is a potentially life-threatening phenomenon requiring prompt recognition and management. It is defined as a 20% or greater reduction in heart rate and/or the presence of arrhythmias during stimulation of the orbital contents.[1] It is most commonly encountered in the context of paediatric squint surgery.

We report its occurrence in a young healthy adult man after a traumatic facial injury. While being transported to hospital by ambulance, he had intermittent bradycardia, with a heart rate as low as 38 beats/min, and was administered a 1 g dose of intravenous atropine. His medical and ocular histories were unremarkable, and he had no history of unexplained syncope.

In the emergency department, his heart rate remained low (40 beats/min) and his blood pressure was 122/52 mmHg. There was diffuse periorbital lid swelling and bony tenderness along the inferolateral orbital margin. He had restricted upgaze of his left eye; during this manoeuvre, his heart rate dropped to 20 beats/min and he developed hypotension, with a blood pressure of 92/48 mmHg. He was given three intravenous 500 µg boluses of atropine to improve his haemodynamic condition.

An electrocardiogram showed sinus bradycardia, and subsequent recordings showed intermittent Mobitz II second-degree atrioventricular block. A computed tomography scan of the left orbit showed a moderately displaced fracture of the orbital floor involving the maxillary bone, with entrapment of orbital fat and the inferior rectus muscle (Box).

Given the presence of the oculocardiac reflex with haemodynamic compromise, the fracture was immediately repaired surgically. Postoperatively, the patient's blood pressure was 134/90 mmHg and his heart rate was 86 beats/min, with normal sinus rhythm. He recovered full eye movement, and no further oculocardiac reflex was recorded. He remained well 1 year after discharge.

The oculocardiac reflex was first described by Aschner as a slowing of the radial pulse when pressure was applied to the eye.[2] It is a rare but recognised occurrence among young healthy adults with orbital fractures. Clinical manifestations of the reflex may include bradycardia, hypotension, nausea, vomiting and syncope.[3] The reflex is acknowledged as an important indication for immediate surgical repair of the orbit.[4] In addition to reducing morbidity from the reflex, urgent repair is beneficial as it releases incarcerated soft tissue, leading to a more favourable outcome with less likelihood of squint.[5]

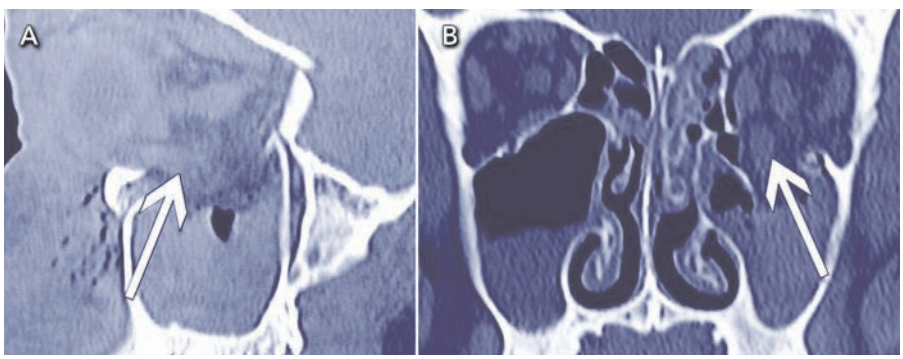
Cardiac decompensation due to traumatic facial injuries should alert clinicians to the possibility of the oculocardiac reflex and the need for urgent surgical intervention.

***Acknowledgement:*** We thank Mr Douglas Nash for his assistance in image preparation.

**Vivek B Pandya,** Ophthalmology Resident[1]
**R Max Conway,** Ophthalmologist[1]
**Richard Conway,** Maxillofacial Surgeon[2]
1 Sydney Eye Hospital, Sydney, NSW.
2 Westmead Hospital, Sydney, NSW.
**vivekpandya@hotmail.com**

1 Ziccardi VB, Russavage J, Sotereanos GC, Patterson GT. Oculocardiac reflex: pathophysiology and case report. *Oral Surg Oral Med Oral Pathol* 1991; 71: 137-138.
2 Aschner B. Über einen bisher noch nicht beschriebenen Reflex, vom Auge auf Kreislauf und Atmung. Verschwinden des Radialispulses bei Druck auf das Auge [Concerning a hitherto not yet described reflex from the eye on circulation and respiration. Disappearance of the radial pulse with pressure on the eye] [German]. *Wien Klin Wochenschr* 1908; 21: 1529-1590.
3 Sires BS, Stanley RB, Levine LM. Oculocardiac reflex caused by orbital floor trapdoor fracture: an indication for urgent surgical repair. *Arch Ophthalmol* 1998; 116: 955-956.
4 Burnstine MA. Clinical recommendations for repair of orbital facial fractures. *Curr Opin Ophthalmol* 2003; 14: 236-240.
5 Sires BS. Orbital trapdoor fracture and the oculocardiac reflex [letter]. *Ophthal Plast Reconstr Surg* 1999; 15: 301. ❑

### Computed tomography scans of the patient's left orbit




*Sagittal (**A**) and coronal (**B**) computed tomography scans showing left orbital floor fracture with entrapment of the inferior rectus muscle (arrows).* ◆

## Devastating outcome after only 6 hours of contact lens wear

Vivek B Pandya, Alessandra Martins and Shanel Sharma

**To the Editor:** Microbial keratitis associated with soft contact lens wear is a well recognised, not uncommon, clinical entity and a preventable cause of ocular morbidity.[1,2]

A 31-year-old woman who occasionally wore soft contact lenses presented to a general practitioner with a 2-day history of bilateral red eye associated with ocular discomfort, photophobia and purulent discharge. She was diagnosed with bilateral conjunctivitis, commenced on chloramphenicol drops and told to return in 5 days. Her symptoms initially improved, but then worsened. Seven days after symptom onset, she presented to hospital for assessment.

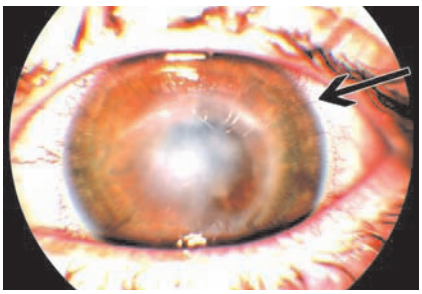
History revealed that she had worn soft monthly disposable contact lenses twice in the previous month to correct her mild myopia. On examination, the patient's visual acuity was equivalent to being legally "blind", being hand movement in the right eye and light perception in the left. On inspection, both eyes appeared grossly abnormal. The right eye had a large central corneal abscess and the left eye had a complete corneal abscess with 360° peripheral corneal thinning (Box, A) — a significant risk for globe perforation. The conjunctiva was markedly injected bilaterally.

Corneal scrapes, the contact lenses and the case containing turbid solution were sent for urgent gram stains and microbiological culture. The patient was admitted to hospital and received intensive topical treatment with gentamicin 0.9%, cephalothin 5% and tobramycin ointment. The corneal scrapes revealed *Pseudomonas aeruginosa* as the causative organism. The patient showed slow improvement with antibiotic therapy. Although the infection cleared, the residual corneal scarring resulted in permanent loss of corneal clarity and hence vision.

Four months after treatment was commenced, the patient's visual acuity had improved to 6/24 in the right eye and 6/36 in

**Left eye at presentation and after 4 months of treatment**

*A: At presentation, there was marked conjunctival injection, 100% epithelial defect (stained with fluorescein [green]) and severe circumferential peripheral corneal thinning (arrow).*



*B: Four months after presentation, there was dense central scarring and peripheral corneal neovascularisation (arrow).* ◆

the left (Box, B). An Australian review of outcomes after keratitis found that 52% of patients had a final visual acuity of worse than 6/12, the legal visual acuity for driving.[3] In this patient, despite saving both eyes and the improvement in her vision, she still has significant functional impairment, being unable to work as a teacher or drive. Corneal transplantation is now her only option for potentially regaining the loss in her functional vision, with a minimum expected recovery time of 2 years.

GPs have a difficult job distinguishing between red eye requiring immediate referral and red eye that is not vision-threatening. All contact lens wearers who present with red eye need to be examined for yellow/white corneal infiltrates and, if present, or if the patient cannot be assessed adequately, immediate referral is mandatory. *P. aeruginosa* is the most common pathogen and one of the most aggressive organisms isolated in contact lens-related microbial keratitis.[4] As it is invariably resistant to the bacteriostatic chloramphenicol, the appropriate empirical treatment is either fluoroquinolones or cephalosporins, which ideally should be commenced after corneal scrapes have been performed.[5]

**Vivek B Pandya,** Ophthalmology Resident[1]
**Alessandra Martins,** Ophthalmology Registrar[1,2]
**Shanel Sharma,** Ophthalmologist[1,2]
1 Department of Ophthalmology, Sydney Eye Hospital, Sydney, NSW.
2 Department of Ophthalmology, Royal Prince Alfred Hospital, Sydney.
**shanel@unsw.edu.au**

1 Green M, Apel A, Stapleton F. A longitudinal study of trends in keratitis in Australia. *Cornea* 2008; 27: 33-39.
2 Keay L, Edwards K, Naduvilath T, et al. Microbial keratitis predisposing factors and morbidity. *Ophthalmology* 2006; 113: 109-116.
3 Green MD, Apel AJ, Naduvilath T, Stapleton FJ. Clinical outcomes of keratitis. *Clin Experiment Ophthalmol* 2007; 35: 421-426.
4 Stapleton F, Keay L, Sanfilippo PG, et al. Relationship between climate, disease severity, and causative organism for contact lens-associated microbial keratitis in Australia. *Am J Ophthalmol* 2007; 144: 690-698.
5 Constantinou M, Daniell M, Snibson GR, et al. Clinical efficacy of moxifloxacin in the treatment of bacterial keratitis: a randomized clinical trial. *Ophthalmology* 2007; 114: 1622-1629. ❑

## Ocular syphilis: are we seeing all there is to see?

Matthew T Kitson, Michelle K Yong and Jennifer F Hoy

**To the Editor:** A 37-year-old man infected with HIV through exposure to men who have sex with men (MSM) presented with a 6-week history of intermittent fevers, patchy alopecia and a widespread brown/grey macular rash involving his face, trunk, abdomen and all limbs, with scaling of the face, palms and soles. He had no symptoms of meningism or visual disturbance, and had a chronic mild intermittent headache with no recent change in frequency or intensity. His most recent CD4 cell count was 70 cells/μL and his HIV viral load was > 100 000 copies/mL.

On admission, a serological test for syphilis was reactive and showed a rapid plasma reagin (RPR) titre of 1:64 and a positive enzyme immunoassay for antibody (EIA-Ab), whereas at the onset of the rash 6 weeks previously, a serological test for syphilis was negative. A punch biopsy of the skin lesions showed a perivascular infiltrate with lichenoid inflammation consistent with secondary syphilis. An ophthalmological review showed a bilateral anterior uveitis. Examination of cerebrospinal fluid (CSF) showed a white cell count of $1 \times 10^6$/L (100% lymphocytes), a normal glucose level, a mildly elevated protein level of 0.45 g/L and a weakly positive result for a fluorescent treponemal antibody absorbed (FTA-ABS) test. CSF RPR, EIA-Ab and *Treponema pallidum* particle agglutination (TPPA) test results were all non-reactive.

The patient was diagnosed with asymptomatic ocular and neurosyphilis, and treated with intravenous benzylpenicillin (1.8 g 4-hourly for 15 days) and prednisolone eye drops (four times daily for 14 days). The rash faded and the anterior uveitis subsided in the first week of treatment.

There is currently a syphilis epidemic among MSM in Victoria, with the number of syphilis notifications increasing 25-fold between 2000 and 2006.[1] A strong association between HIV infection and infectious syphilis has been demonstrated in recent years, including in Victoria.[2] HIV-infected MSM with early syphilis have a 1.7% risk of having symptomatic early neurosyphilis[3] and a 13% risk of having symptomatic ocular syphilis regardless of CD4 cell count.[4] There is no pathognomonic finding in ocular syphilis, and the disease may manifest as uveitis, retinitis, optic neuritis, perineuritis, retinal detachment and papillitis.

It is recommended that all patients with ocular syphilis undergo CSF examination and be managed as if they had neurosyphilis.[5] Current guidelines also recommend CSF examination in HIV-infected patients who have late-latent syphilis, syphilis of unknown duration, any neurological signs or symptoms, or suspected treatment failure.[6]

The finding of asymptomatic ocular syphilis in our patient suggests there should be a low threshold for eye examination in HIV-infected people with early syphilis, even in the absence of any ocular symptoms, as the diagnosis of ocular syphilis dramatically alters the management of these patients.

**Matthew T Kitson,** Registrar
**Michelle K Yong,** Registrar
**Jennifer F Hoy,** Director, Victorian HIV Service
Department of Infectious Diseases, Alfred Hospital, Melbourne, VIC.
**mattkitson@hotmail.com**

1 Higgins N. Sexually transmissible infections. Chlamydia, gonorrhoea and infectious syphilis. *Vic Infect Dis Bull* 2007; 10: 16-22. http://www.health.vic.gov.au/ideas/downloads/bulletin/vidbv10i1.pdf (accessed Aug 2008).
2 Leslie DE, Higgins N, Fairley CF. Dangerous liaisons — syphilis and HIV in Victoria. *Med J Aust* 2008; 188: 676-677.
3 Centers for Disease Control and Prevention. Symptomatic early neurosyphilis among HIV-positive men who have sex with men — four cities, United States, January 2002–June 2004. *MMWR Morb Mortal Wkly Rep* 2007; 56: 625-628.
4 Balba GP, Kumar PN, James AN, et al. Ocular syphilis in HIV-positive patients receiving highly active antiretroviral therapy. *Am J Med* 2006; 119: 448-449.
5 Workowski K, Berman S. Centers for Disease Control and Prevention. Sexually transmitted diseases treatment guidelines. *MMWR Recomm Rep* 2006; 55: 1-94.
6 Zetola N, Klausner J. Syphilis and HIV infection: an update. *Clin Infect Dis* 2007; 44: 1222-1228. ❑

## Wrist guards and wrist and elbow injury in snowboarders

Graham M Slaney, Judith C Finn, Angus Cook and Philip Weinstein

**To the Editor:** Snowboarding is increasing in popularity, but Australian snowboarders have been shown to have 2.4 times as many fractures as skiers, with 35% of upper limb injuries being fractures.[1] The most common site of injury is the wrist, accounting for 21.6% of all snowboarding injuries.[2] It has been suggested that the use of wrist guards could reduce the risk of injury, particularly as snowboarding injuries tend to be impactive rather than torsional.[3,4] However, concern has been raised that use of wrist guards will redistribute the impact of the force to more proximal areas of the arm, causing elbow injuries.[5]

We conducted a case–control study at the Mount Buller Medical Centre, Victoria, during the 2004 and 2005 ski seasons to assess:

- the association between wrist guard use and wrist fracture in snowboarders in Australia; and
- the association between wrist guard use and the severity of wrist and elbow injury.

Cases were defined as any snowboarder seen at the clinic with a fractured wrist ($n = 119$). Controls ($n = 375$) were snowboarders — identified by their boots — who attended the clinic, either as companions to case participants or other patients, or as patients presenting for a reason other than wrist fracture.

Study participants completed a questionnaire about wrist guard use and snow-sport behaviour. The site and severity of fractures were recorded by clinic staff. Logistic regression was used to determine adjusted odds ratios for risk factors against the main outcome measure of wrist fracture and injury in snowboarders with and without wrist guards.

Characteristics strongly associated with wrist fracture were being of school age (odds ratio [OR], 2.37; $P < 0.001$) and being a novice at snowboarding (OR, 3.41; $P < 0.001$) (Box). After adjustment for all significant variables — sex, age, days of snowboarding and snowboarder ability — the odds of having worn wrist guards were lower in snowboarders with a wrist fracture (cases) than in those without such a fracture, but the difference did not reach significance (adjusted OR, 0.58; 95% CI, 0.32–1.04; $P = 0.07$). Full analysis of all factors considered is available from the authors.

Among the sample of 494 snowboarders, 15 had elbow injuries, comprising:

- five with soft tissue injuries (4/86 wearing wrist guards v 1/391 not wearing wrist guards; adjusted OR, 17.6; 95% CI, 1.93–160.2; $P = 0.01$); and
- 10 with elbow fractures or dislocations (3/86 wearing wrist guards v 77/391 not wearing wrist guards; adjusted OR, 1.84; 95% CI, 0.46–7.30; $P = 0.39$).

The association between wrist guard use and increased soft tissue elbow injuries, but not elbow fractures and dislocations, supports the value of wearing wrist guards to reduce overall injury severity.

Despite a lack of overall statistical significance, the clinical context and consistency in direction of the findings suggest that snowboarders who wear wrist guards in Australian snow conditions could benefit from a reduction in wrist fracture injury of approximately 42%. This is consistent with reports from other countries that show a protective effect of wrist guards of 52% to 87%, with the greatest benefit in novice snowboarders.[4] We suggest that wrist guard use should be strongly recommended for novices, and should be mandatory for school-aged snowboarders. Local schools in the Mansfield district, near Mt Buller, have adopted a policy of "no wrist guard = no snowboard", and we hope that other schools visiting Mt Buller, and indeed other ski resorts in Australia, will follow this lead.

**Graham M Slaney,** Rural Medical Practitioner[1]
**Judith C Finn,** Professor of Nursing Research[2]
**Angus Cook,** Senior Lecturer in Public Health[2]
**Philip Weinstein,** Professor of Public Health[2]
1 Mansfield Medical Clinic, Mansfield, VIC.
2 School of Population Health, University of Western Australia, Perth, WA.
gslaney@swiftdsl.com.au

1 Bladin C, Giddings P, Robinson M. Australian snowboard injury data base study. A four-year prospective study. *Am J Sports Med* 1993; 21: 701-704.
2 Idzikowski JR, Janes PC, Abbott PJ. Upper extremity snowboarding injuries. Ten-year results from the Colorado snowboard injury survey. *Am J Sports Med* 2000; 28: 825-832.
3 Pino EC, Colville MR. Snowboard injuries. *Am J Sports Med* 1989; 17: 778-781.
4 Russell K, Hagel B, Francescutti LH. The effect of wrist guards on wrist and arm injuries among snowboarders: a systematic review. *Clin J Sport Med* 2007; 17: 145-150.
5 Hagel B, Pless IB, Goulet C. The effect of wrist guard use on upper-extremity injuries in snowboarders. *Am J Epidemiol* 2005; 162: 149-156. ❑

### Characteristics associated with wrist fracture among snowboarders

| Characteristic | Cases ($n = 119$) | Controls ($n = 375$) | Odds ratio (95% CI) | $P$ for difference |
|---|---|---|---|---|
| **Wearing wrist guards today** | | | | |
| No | 100 (84%) | 299 (80%) | 1.00* | |
| Yes | 18 (15%) | 75 (20%) | 0.72 (0.41–1.26) | 0.25 |
| Missing data | 1 (0.8%) | 1 (0.3%) | | |
| **Age (years)** | | | | |
| 0–19 | 72 (61%) | 149 (40%) | 2.37 (1.55–3.63) | < 0.001 |
| > 19 | 46 (39%) | 226 (60%) | 1.00* | |
| Missing data | 1 (0.8%) | | | |
| **Ability of snowboarder** | | | | |
| Novice | 60 (50%) | 112 (30%) | 3.41 (1.79–6.49) | < 0.001 |
| Intermediate | 43 (36%) | 173 (46%) | 1.58 (0.82–3.04) | 0.17 |
| Advanced | 14 (12%) | 89 (24%) | 1.00* | |
| Missing data | 2 (2%) | 1 (0.3%) | | |

* Reference category. ◆

## Two cases of *Streptococcus suis* endocarditis in Australian piggery workers

Karina J Kennedy, Assad A Jadeer, Chong W Ong, Sanjaya N Senanayake and Peter J Collignon

**TO THE EDITOR:** *Streptococcus suis* is an emerging zoonosis in humans and a common pathogen in Australian pigs.[1] However, only two cases of human infection have previously been reported in Australia.[2,3] We recently treated two Australian piggery workers, from the same town in New South Wales, for *S. suis* endocarditis.

The first patient, a previously healthy 46-year-old woman, presented in October 2006 with a 3-month history of fatigue, anorexia, night sweats and weight loss of 20 kg. Her job involved hands-on work at a piggery. She was afebrile, with blood pressure of 145/50 mmHg, bilateral clubbing, splenomegaly, and a long diastolic murmur without evidence of heart failure. Transthoracic echocardiography confirmed severe aortic regurgitation associated with a vegetation. Blood cultures grew *S. suis*. The patient was treated with benzylpenicillin (1.8 g, 4-hourly for 6 weeks) and gentamicin (60 mg, 8-hourly for 2 weeks). Severity of aortic regurgitation necessitated aortic valve replacement.

The second patient, a 58-year-old man, presented in January 2008 with headache, fever (38.7° C), neck stiffness and confusion. He had been unwell for 1 month with fevers, sweating, fatigue and weight loss of 6 kg. His job involved transporting pigs from local piggeries, including the one at which the first patient worked, to an abattoir, and involved direct contact with pigs. On admission, he was treated with ceftriaxone (2 g) and benzylpenicillin (2.4 g). Cerebrospinal fluid (CSF) examination showed leukocytosis ($10\,300 \times 10^6$/L; reference range, $< 5 \times 10^6$/L) with 95% polymorphs. Scanty gram-positive cocci were visible, but there was no growth on culture of the CSF. Blood cultures grew *S. suis*. Transthoracic echocardiography revealed an aortic valve vegetation with trivial regurgitation. The patient was then treated with benzylpenicillin (1.8 g, 4-hourly for 4 weeks) and gentamicin (80 mg, 8-hourly for 2 weeks), and made a full recovery.

Both cases were investigated by the NSW Department of Health, including assessment of workplace practices, staff education and rates of porcine infection at the piggeries and abattoir at which the patients had worked, but no significant factors for infection were identified.

Since the first reported human infection with *S. suis* in Denmark in 1968,[4] more than 400 cases have been reported, including an outbreak in China in 2005 that affected 215 people.[5] The most common manifestations are meningitis (affecting 72.5% of patients) and bacteraemia (24.2%); endocarditis is relatively uncommon (1.1%).[5] In a series of 16 cases of *S. suis* endocarditis, the mean period between onset of symptoms and diagnosis was 23 days, and seven patients required valve replacement.[6] Similarly, our patients had subacute presentations: severe aortic regurgitation and secondary meningitis. In Australia, it is likely that there have been other *S. suis* infections in humans that have gone unrecognised because of mild presentations, difficulties with laboratory diagnosis, and empirical treatment of unrecognised infection. While it is unlikely that *S. suis* is a common zoonosis in Australia, these cases show that it is an occupational hazard in Australian piggeries, with potential public health, animal health and medicolegal implications.

**Karina J Kennedy,** Infectious Diseases Physician and Microbiology Registrar
**Assad A Jadeer,** Basic Physician Trainee
**Chong W Ong,** Infectious Diseases Advanced Trainee
**Sanjaya N Senanayake,** Infectious Diseases Physician
**Peter J Collignon,** Director of Infectious Diseases and Microbiology
Canberra Hospital, Canberra, ACT.
karina.kennedy@act.gov.au

1 Robertson ID, Blackmore DK. Prevalence of *Streptococcus suis* types 1 and 2 in domestic pigs in Australia and New Zealand. *Vet Rec* 1989; 124: 391-394.
2 Hampson DJ, Trott DJ, Clarke IL, et al. Population structure of Australian isolates of *Streptococcus suis*. *J Clin Microbiol* 1993; 31: 2895-2900.
3 Tramontana AR, Graham M, Sinickas V, Bak N. An Australian case of *Streptococcus suis* toxic shock syndrome associated with occupational exposure to animal carcasses. *Med J Aust* 2008; 188: 538-539.
4 Perch B, Kristjansen P, Skadhauge KN. Group R streptococci pathogenic for man. Two cases of meningitis and one fatal case of sepsis. *Acta Pathol Microbiol Scand* 1968; 74: 69-76.
5 Lun ZR, Wang QP, Chen XG, et al. *Streptococcus suis*: an emerging zoonotic pathogen. *Lancet Infect Dis* 2007; 7: 201-209.
6 Wangkaew S, Chaiwarith R, Tharavichitkul P, Supparatpinyo K. *Streptococcus suis* infection: a series of 41 cases from Chiang Mai University Hospital. *J Infect* 2006; 52: 455-460. ❑

## The Safer Patients Initiative: the UK experience of attempting to improve safe clinical care

Peter J Shirley

**To the Editor:** The study by Nichols and colleagues[1] and the associated editorial by Hughes[2] struck a chord of familiarity for me, as patient safety issues are currently high on the political agenda in the United Kingdom.

An estimated 850 000 incidents of harm or near harm affect National Health Service (NHS) hospital patients in the UK each year.[3] In April 2004, the Health Foundation (an independent charity that aims to improve the quality of UK health care), together with the Institute for Healthcare Improvement, launched the Safer Patients Initiative (SPI). The four hospitals initially chosen to participate conducted hospital-wide programs to radically improve patient safety, with the aim of reducing adverse events by 50% by October 2006. In November 2006, Phase 2 was launched, adding 20 more sites.[3] These hospitals meet regularly to report on progress and exchange ideas. Discrete projects focus on medicines reconciliation, ward-based care, critical care, and perioperative care.

The overall aim of the SPI is to improve the patient safety culture within each organisation. Specific targets for all participating hospitals include a 15% reduction in mortality of in-hospital patients; 300 days between central line bloodstream infections in critical care units; 80% of blood sugar levels in diabetic patients falling within their target treatment range; a 30% reduction in cardiac arrest calls; and 50% reductions in methicillin-resistant *Staphylococcus aureus* bloodstream infections, harm from anticoagulation, and surgical site infections.

In my intensive care unit, hand hygiene compliance among medical staff has been regularly audited and has improved from a range of 20%–90% per day to 60%–100%. This compares favourably with a recent Australian hand hygiene initiative.[4]

When practice improvements are shown in one location, the project team takes on the responsibility of spreading these across the hospital. Initial scepticism from senior clinicians and nurses in my hospital has generally been replaced by cooperation and, in many cases, ideas for other ways to improve delivery of safer clinical care.

Large multisite evidence-based trials in intensive care units in the United States showed that, with focused effort on sterile technique and catheter care and by rectifying lapses in standard procedures, a reduction of 66% in catheter-related bloodstream infections was possible.[5] The SPI is trying to replicate results such as these within a relatively short period. My intensive care unit has now gone 190 days without a line-related bacteraemia; previously, we had infections almost every month.

The SPI Phase 2 completion date is November 2008, with full national reporting due at that time. It is intended that practice improvements will then be spread across the NHS. The experience in my hospital has been that sharing experiences both within the hospital and externally has led to tangible progress in this area.

**Peter J Shirley,** Intensive Care Physician
Royal London Hospital, London, UK.
**Peter.Shirley@bartsandthelondon.nhs.uk**

1 Nichols P, Copeland T-S, Craib IA, et al. Learning from error: identifying contributory causes of medication errors in an Australian hospital. *Med J Aust* 2008; 188: 276-279.
2 Hughes CF. Medication errors in hospitals: what can be done [editorial]? *Med J Aust* 2008; 188: 267-268.
3 Institute for Healthcare Improvement. The Health Foundation's Safer Patients Initiative. http://www.ihi.org/IHI/Programs/StrategicInitiatives/SaferPatientsInitiative.htm (accessed Aug 2008).
4 Grayson ML, Jarvie LJ, Martin R, et al. Significant reductions in methicillin-resistant *Staphylococcus aureus* bacteraemia and clinical isolates associated with a multisite, hand hygiene culture-change program and subsequent successful statewide roll-out. *Med J Aust* 2008; 188: 633-640.
5 Pronovost P, Needham D, Berenholtz S, et al. An intervention to decrease catheter-related bloodstream infections in the ICU. *N Engl J Med* 2006; 355: 2725-2732. ❑

## Changes to the University of Sydney medical curriculum

Thomas KF Taylor

**To the Editor:** The recent article on the review of the University of Sydney Medical Program (USydMP)[1] by Goulston and Oates included a lengthy list of "changes made or planned in accordance with key recommendations".[2] Regrettably, and to the despair of many, the most needed change will not take place.

It is reported in the review that the average age of future USydMP graduates will be 29–30 years, as it has been for the past decade.[1] From 2008, those who aspire to be surgeons will competitively enter the new Surgical Education and Training (SET) program of the Royal Australasian College of Surgeons (RACS) after completing "at least their second postgraduate year".[3] The SET program is for 5–6 years, depending on the specialty. Most trainees subsequently take a subspecialty fellowship for 1–2 years, either in Australia or overseas, by which time our hapless graduates will be 40 years old. A woman may have to have leave of absence for a pregnancy. A postgraduate degree is now a prerequisite for an academic career and this involves an additional 2–3 years of full-time research. Finally, it takes in the order of 5 years to establish a referral specialist practice in most branches of medicine.

In the review document, Goulston and Oates nihilistically state "There is no opportunity for streaming within the USydMP".[1] There is no mention of undergraduate education in Australia, let alone the streaming of such education, in the article from the RACS outlining the SET program.[4] However, strangely enough, it is noted therein: "North American students must make their long-term career choice in the final year of their medical school and are streamed accordingly". One wonders if there is any purposeful communication between the RACS and our universities. There certainly should be.

It is imperative in this day and age that undergraduate and postgraduate medical education be considered as a continuum so that we can begin to rid ourselves of the absurdly long and manifestly inefficient process outlined above. The historian and journalist Paul Johnson put his astute finger on the problem in writing a column about universities generally: "...a visitor from another planet, unfamiliar with the history of the institution, would think it odd that our ablest boys and girls, at a time when their mental and physical powers are at their highest, are withdrawn from the service of society and kept in comparative idleness..."[5]

One can but conclude that, at least for future surgeons, the wrong people are at the helm at the RACS and at the University of Sydney's Faculty of Medicine.

**Thomas KF Taylor,** Emeritus Professor of Orthopaedic and Traumatic Surgery
University of Sydney, Royal North Shore Hospital, Sydney, NSW.
**tktaylor@med.usyd.edu.au**

1 University of Sydney. Faculty of Medicine. Curriculum review — USydMP (University of Sydney MedicalProgram). http://www.medfac.usyd.edu.au/forstaff/usydmp-review (accessed Aug 2008).
2 Goulston KJ, Oates RK. Changes to the University of Sydney medical curriculum. *Med J Aust* 2008; 188: 461-463.
3 Waxman BP. Ready, SET, go for academic surgery [editorial]? *Med J Aust* 2008; 188: 67-68.
4 Collins JP, Gough IR, Civil ID, Stitz RW. A new surgical education and training programme. *ANZ J Surg* 2007; 77: 497-501.
5 Johnson P. Is your journey really necessary, professor? *The Spectator* 1991; 7 Sep. ❑

### Ian R Gough

***In reply:*** Taylor raises several issues that the Royal Australasian College of Surgeons (RACS) has carefully considered. The new Surgical Education and Training (SET) program commenced this year.[1] The acronym SET could equally mean Shorter Efficient Training.

In the previous program, 2–5 years of basic surgical training was followed by 4–6 years of advanced training, resulting in surgeons entering specialist practice an average of 10 years after graduating with their medical degrees. We responded to societal and regulatory factors, including the older age of medical (compared with other) graduates, sex, work–life balance, safe-hours requirements, workforce pressures and competition from other specialties. Our new system aims to streamline training by early selection directly into one of nine surgical specialties and completion of training by Postgraduate Year 7 or 8 in most cases.

It is not only shorter, but more comprehensive than previously. It is much more than an apprenticeship, where training occurs by random clinical exposure. We are covering defined curriculum objectives for every trainee by offering training in metropolitan and regional hospitals, synthetic laboratories and the private sector. We are moving away from reliance on the number of years of training and numbers of operations as measures of experience. We are focusing on the development of a range of surgical competencies encompassing professionalism, communication, collaboration, clinical decision making, scholarship, leadership and health advocacy, as well as essential medical and technical expertise. These competencies are regularly monitored by performance assessment throughout training.

At the completion of training and the RACS fellowship examination, a surgeon is competent to commence practice as a specialist. Additional formal training experience is optional. Of course, the RACS promotes the concept of lifelong learning.

The RACS and universities are communicating. Common concerns are the compromised state of basic science education and the limited exposure of medical students to a range of surgical specialties. The College has had discussions with many universities and health authorities about the possibility of "streaming" in the later years of medical school and in Postgraduate Years 1 and 2. It is worth noting that the University of Sydney has plans for the final 2 years of its course to be integrated, with increased emphasis on critical care and surgery.[2]

**Ian R Gough,** President
Royal Australasian College of Surgeons, Melbourne, VIC.
ian.gough@surgeons.org

1 Collins JP, Gough IR, Civil ID, Stitz RW. A new surgical education and training programme. *ANZ J Surg* 2007; 77: 497-501.
2 Goulston KJ, Oates RK. Changes to the University of Sydney medical curriculum. *Med J Aust* 2008; 188: 461-463. ❑

### Kerry J Goulston and R Kim Oates

***In reply:*** We are in sympathy with Taylor's concerns about the length of medical training and point out that a more careful reading of our review of the University of Sydney Medical Program[1] contained the following comments (on page 203):

> Discussion with the Royal Australasian College of Physicians canvassed the possibility that students might master some educational modules during their medical degree which would be credited by the College. This could well apply to other Colleges. Such a process would be more easily achieved if the Colleges moved to an overall "point system" i.e. students (especially in nonclinical areas such as Ethics, Quality and Safety etc) could study postgraduate modules pari passu with the USydMP, gaining some points towards their chosen college specialty qualification.

This is followed by three recommendations on page 204:

> Students with an early interest in a specialty could gain some speciality experience or credit towards their specialty by either working towards an MPhil and by making use of the electives, options and Honours research project.
>
> The future situation with regard to medical training in some of the specialties is fluid eg, the Royal Australasian College of Surgeons is introducing a method of streaming for surgical training (SET) and IMET [Institute of Medical Education and Training] is looking at criteria for competency in the residency years. In view of this the Dean should explore further opportunities for streaming when the options for future specialty training become clearer.
>
> Early streaming should be re-examined by Faculty when the curriculum and educational changes being considered by some of the Colleges become clearer.

**Kerry J Goulston,** Emeritus Professor of Medicine
**R Kim Oates,** Emeritus Professor of Paediatrics
University of Sydney, Sydney, NSW.
kerry@med.usyd.edu.au

1 University of Sydney. Review of the University of Sydney Medical Program. October 2007. http://www.medfac.usyd.edu.au/forstaff/usydmp-review/USYDMP_Report_online.pdf (accessed Aug 2008).❑

## KFC sponsorship of cricket

### Stephen Colagiuri and Ian D Caterson

**To the Editor:** During the recent international cricket series between Australia and India, we were alarmed by the sight of our Australian cricketers prominently badged with the logo of the fast food giant KFC.

Australia is experiencing an epidemic of overweight and obesity, a problem that is especially affecting children, adolescents and young adults.[1] This epidemic is worsening as a direct result of unhealthy eating habits and low levels of physical activity. Obesity is associated with chronic and costly diseases that lead to premature death and ill health. These include diabetes, cardiovascular disease, respiratory problems, sleep apnoea, certain cancers, mental illness and osteoarthritis, which can begin in adolescence.[1]

Cricket in Australia enjoys considerable popularity and a strong national following. Our cricketers are national sporting heroes who enjoy widespread support and respect throughout the community, particularly among younger members of the community, who aspire to emulate them.

Against this background, we are increasingly concerned and disappointed that Cricket Australia has a sponsorship agreement with, and consequently promotes, KFC — going as far as publicly declaring the company the "official fast food restaurant of Cricket Australia".[2] This advertising uses the standing of cricket and its players to endorse and promote unhealthy eating habits, one of the major root causes of obesity in Australia.

KFC products have caloric and fat contents well above the national dietary guidelines, which recommend < 30% energy from total fat and < 10% energy from saturated fat.[3] For example, a standard serve of original-recipe chicken contains about 58% total fat and 24% saturated fat.[3] Furthermore, we have shown that even one common KFC

meal per week can adversely affect recommended healthy diets.[4] It is ironic and regrettable that Cricket Australia, while having done so much for the sport, encourages the promotion of unhealthy, high-fat, high-calorie KFC products that negate the benefit of increased physical activity associated with playing cricket.

With the explosion of obesity-related illness, we need champions to encourage health-promoting behaviours, particularly healthy eating and increased physical activity. The enthusiastic encouragement of unhealthy and undesirable eating habits should have no place in sporting sponsorship. Not so long ago, similar sentiments were being expressed about tobacco sponsorship of sport, which fortunately has been eradicated.

Cricket Australia should consider its responsibilities to Australia's children and youth and review its sponsorship by KFC. This would benefit the health of the community and demonstrate leadership and social responsibility by Cricket Australia and Australia's elite cricketers.

**Stephen Colagiuri,** Professor of Metabolic Health

**Ian D Caterson,** Boden Professor of Human Nutrition

Institute of Obesity, Nutrition and Exercise, University of Sydney, Sydney, NSW.
**scolagiuri@med.usyd.edu.au**

1 Booth M, Okely AD, Denney-Wilson E, et al. NSW Schools Physical Activity and Nutrition Survey (SPANS) 2004. Full report. Sydney: NSW Health, 2006.
2 KFC nutrition information. KFC, 2006. http://www.kfc.com.au/files/061220NutritionInformationKFC.pdf (accessed Mar 2008).
3 National Health and Medical Research Council. Dietary guidelines for Australian adults. Canberra: Commonwealth of Australia, 2003. http://www.nhmrc.gov.au/publications/synopses/_files/n33.pdf (accessed May 2008).
4 Malouf NM, Colagiuri S. The effects of McDonalds, Kentucky Fried Chicken and Pizza Hut meals on recommended diets. *Asia Pac J Clin Nutr* 1995; 4: 265-269. ❑

## Peter Young

***In reply:*** Cricket Australia (CA) actively supports physical activity, healthy eating and healthy lifestyles and continues to invest considerable effort in encouraging Australians to play cricket, in everything from formal, organised competitions to social games in the backyard, in parks and schoolyards and at the beach. In a time of declining community physical activity, we are heartened to see that active participation in cricket is growing strongly, and note that female cricket is the fastest growing female sport in Australia.

As a community-based, not-for-profit organisation, we are heavily dependent on the support of all of our sponsors, including KFC, to be able to implement the activities we undertake. These range from community-based programs that get kids running around outside to programs in schools, clubs and Indigenous communities, and further activity needed to develop and put elite international cricketers onto the field.

In relation to KFC, we believe in a little of everything and everything in moderation.

Setting aside the truly elite athletes, the formula that is going to work best for most cricketers and cricket fans is reasonable, not extreme, training and physical activity, together with a balanced diet, not one that features total abstinence from high-energy foods. Our view on alcohol is the same. Our CA advertising featuring Merv Hughes encourages fans to enjoy a beer, but not at the rate of one per over.

The overall issue is about balance. Consumer research — commercially confidential, so it can't be referenced here — shows that KFC consumption in Australia is an occasional treat, not a dietary staple.

More broadly, CA is comfortable that Australian cricket's collective activity has a net positive impact in encouraging healthy and active lifestyles.

**Peter Young,** General Manager Public Affairs
Cricket Australia, Melbourne, VIC.
**Peter.Young@cricket.com.au** ❑

## Correction

*Re:* "**Does practice make perfect? The effect of coaching and retesting on selection tests used for admission to an Australian medical school**", by Barbara Griffin, David W Harding, Ian G Wilson and Neville D Yeomans, in the 1 September 2008 issue of the Journal (*Med J Aust* 2008; 189: 270-273). In the Results section, under "Prevalence of coaching", the words "were more likely to be male (57.6% v 44.4%; $\chi^2 = 4.88$; $P = 0.03$)" should read "were more likely to be male (57.6% v 42.4%; $\chi^2 = 4.88$; $P = 0.03$)". ❑