LETTERS

Drugs, sport and the Olympics 2000–2004

Michael C Kennedy
Research Associate, Department of Clinical Pharmacology and Toxicology, St Vincent's Hospital, Darlinghurst, NSW 2010.
drmkenn@ozemail.com.au

To the Editor: Since the Sydney Olympics in 2000, many developments have occurred in drug use and the rules regulating drugs in sport.

The most significant regulatory development is the acceptance by the Olympic Federation, and many other sports bodies, of the World Anti-Drug Agency's World Anti-Doping Code.[1]

Caffeine and pseudoephedrine have been removed from the Prohibited List, and an in-competition monitoring program is under way to detect any changes in the patterns of use of caffeine, pseudoephedrine and other drugs not on the banned list. Had this code been used in 2000, the Romanian gymnast Andreea Raducan would have retained her gold medal, lost after she inadvertently used a cold preparation containing pseudoephedrine.

Precise in-competition limits on blood and breath alcohol have been introduced in sports such as archery and modern pentathlon. β-Blocking agents and diuretics are completely banned in specific sports.

A new category of "specified substances" now exists:

> …the prohibited list may identify specified substances which are particularly susceptible to unintentional anti-doping rule violations because of their general availability in medicinal products or which are less likely to be successfully abused as doping agents.

These substances include cannabinoids, probenecid, glucocorticosteroids and ephedrine.

Doctors treating athletes should advise them to inform their relevant sporting authority of drugs prescribed. If necessary, athletes can apply to the Australian Sports Drug Advisory Committee for a therapeutic use exemption for a banned substance. Notifiable substances can be documented on an Abbreviated Therapeutic Use Exemption form held by the national sporting body.

There can be no doubt of the need for drug testing to ensure a level playing field. Drug use to enhance performance is unabated since the Sydney games, with scandals occurring around the world. The Bay Area Laboratory Corporation scandal, involving the anabolic steroid tetrahydrogestrinone, is the most prominent. This has ruined several sporting careers and led to criminal charges against company directors.[2] Other anabolic steroids continue to be widely used, including nandrolone, which causes problems because of contamination of dietary supplements and some foods.[3]

One of the "holy grails" for drug cheats over the past 4 years has been to enhance oxygen transport and delivery. RSR13 (efaproxiral), an allosteric modifier of haemoglobin, is in clinical trial as a radiosensitising agent. It has been shown to increase $\dot{V}O_2max$ in dogs and hence has been of interest to endurance athletes. The manufacturer's collaboration with the Olympic Analytical Laboratory of the University of California (Los Angeles) resulted in an analytical method now being available for detection of the drug in sport.[4]

Haemoglobin- and non-haemoglobin-based oxygen carriers are now available commercially. There are few scientific data about their use in sport, but it is likely they are misused by some athletes.[5] Recombinant human erythropoietin is widely used in cycling and other endurance sports. A detection method developed from Australian research will limit its use, at least at the Olympic venue.[6]

Genetic manipulation is unlikely in 2004, but its potential is foreseen. This technology is also prohibited in the new code.[1]

Unfortunately, drugs will continue to be misused. The opportunity for Olympic winners to gain huge financial rewards will fuel their use.

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Emergence of heteroresistant vancomycin-intermediate *Staphylococcus aureus* (hVISA) infection in Western Australia

Ronan J Murray,* Kishore Sieunarine,† Peter B Ward,‡ John W Pearman§
* Senior Microbiology Registrar, § Clinical Microbiologist, † Vascular Surgeon, Royal Perth Hospital, Perth, WA; ‡ Senior Scientist, Department of Microbiology, Austin Repatriation Medical Centre, Heidelberg, VIC. ronan.murray@health.wa.gov.au

To the Editor: Previous articles in the Journal have described the emergence of *Staphylococcus aureus* with reduced susceptibility to vancomycin (also known as heteroresistant vancomycin-intermediate *Staphylococcus aureus*, or hVISA) in populations where methicillin-resistant *S. aureus* (MRSA) is endemic in healthcare settings.[1,2] We describe a case of infection caused by hVISA from a region where healthcare-associated MRSA infection is relatively uncommon.[3]

A 79-year-old woman with an extensive medical history, including type 2 diabetes mellitus and multiple bypass procedures for lower-limb ischaemia, presented with critical ischaemia of the right lower leg. After above-knee amputation, she developed a discharge from the stump wound from which multiresistant MRSA was cultured. Despite receiving several courses of intravenous vancomycin (a total of 25 days of therapy over 5 months), the infection did not resolve.

Correspondents

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Extensive debridement surgery, with removal of multiple grossly infected vascular grafts, was performed, and MRSA was cultured from the graft material. Subsequently, the patient developed a discharging sinus from which MRSA with reduced susceptibility to glycopeptide antibiotics was cultured (vancomycin minimal inhibitory concentration [MIC], 8 mg/L; teicoplanin MIC, 24 mg/L). This isolate was shown to be hVISA by population analysis profiling (PAP). When the original MRSA isolate was subsequently tested by PAP, heterogenous subpopulations of bacteria with reduced susceptibility to vancomycin were present which had *not* been detected by routine susceptibility testing (ie, the isolate was already hVISA).

Review of the patient's medical records from other Perth healthcare institutions revealed no evidence of vancomycin administration before the initial isolation of MRSA, or contact with known MRSA-colonised patients or healthcare workers. Multilocus sequence typing and staphylococcal cassette chromosome *mec* allotyping identified the MRSA strain as ST239-MRSA-III, a multiresistant "international" MRSA clone frequently isolated in Australia, mainly on the east coast.[4]

Despite further surgery and institution of alternative antimicrobial therapy (initially rifampicin and fusidic acid and subsequently linezolid), the patient died of ongoing ischaemia and uncontrolled infection.

Prolonged or repeated use of vancomycin in patients with implanted prostheses that are infected with MRSA should be discouraged, not only because it is commonly futile, but also because it may promote the emergence of subpopulations of *S. aureus* with reduced susceptibility to vancomycin, as occurred in this case. The fact that these resistant subpopulations were detected in an isolate before the commencement of vancomycin therapy (and then only with specialised testing) reinforces our recommendation.

Acknowledgements: We thank Geoff Coombs, Frances O'Brien, Mary Malkowski and Julie Pearson, from the Department of Microbiology and Infectious Diseases and the Gram Positive Typing and Research Unit, Royal Perth Hospital and Curtin University of Technology, for performing MRSA typing of the isolates.

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Fatal necrotising pneumonia due to community-acquired methicillin-resistant *Staphylococcus aureus* (MRSA)

Anton Y Peleg,* Wendy J Munckhof†

* Infectious Diseases Registrar, Alfred Hospital, Prahran, VIC 3181; † Specialist in Infectious Diseases and Microbiology, Infection Management Service, Princess Alexandra Hospital, Brisbane, QLD.
A.Peleg@alfred.org.au

To the Editor: Infection with community-acquired methicillin-resistant *Staphylococcus aureus* (CA-MRSA) is emerging in many countries, including Australia.[1] We report the first case of fatal necrotising pneumonia caused by CA-MRSA in Australia.

A previously well 21-year-old Aboriginal man presented to the emergency department with fever and a productive cough. He had no known risk factors for sepsis (such as immunosuppression, diabetes, HIV infection, alcoholism, asplenia or recent influenza) and no history of hospitalisation in the previous 12 months. Chest x-ray revealed left midzone consolidation. He was prescribed amoxycillin–clavulanate and discharged.

Two days later, the patient re-presented, with rigors, haemoptysis and agitation. Examination revealed a respiratory rate of 38 breaths per minute, oxygen saturation of 79% breathing room air, temperature of 38.7°C, sinus tachycardia (135 beats per minute), and a systolic blood pressure of 80 mmHg. Respiratory examination revealed diffuse coarse crepitations. No other source of infection was identified.

The patient required intubation, mechanical ventilation and inotropic support. Sputum and blood samples were taken for culture, and empirical treatment was begun with intravenous ceftriaxone, erythromycin and a single dose of gentamicin and rifampicin. Initial investigations showed leukopenia (2.3×10^9/L; reference range [RR], $3.9–12.7 \times 10^9$/L), acute renal failure with a serum creatinine level of 0.18 mmol/L (RR, 0.06–0.11 mmol/L), and severe metabolic and respiratory acidosis (pH, 7.19; RR, 7.38–7.43).

The following day, blood and sputum cultures showed gram-positive cocci resembling staphylococci, and intravenous flucloxacillin was added to the antibiotic regimen. Repeat chest x-ray revealed bilateral necrotising pneumonia. Despite resuscitation efforts, the patient died 48 hours after admission.

Susceptibility testing of blood and sputum isolates subsequently confirmed MRSA. The isolate was sensitive to erythromycin, clindamycin, gentamicin, ciprofloxacin, tetracycline, vancomycin, rifampicin and fusidic acid. Methicillin resistance was confirmed by detection of the *mecA* gene by polymerase chain reaction (PCR).

Further PCR testing of the isolate revealed the Panton–Valentine leukocidin (*pvl*) gene, an important virulence factor that has been associated with necrotising pneumonia[2] and death,[3] and is rarely found in methicillin-susceptible *S. aureus* or hospital-acquired MRSA isolates.[1,2,5] Typing of the isolate by pulsed-field gel electrophoresis showed that it was the recently described "R" pulsotype of CA-MRSA, or "Queensland clone".[4] This clone was first noted in the white population in south-east Queensland in 2000, and is uncommon in Aboriginal people.[4] Most CA-MRSA infection in Aboriginal people is caused by WA-MRSA, which may be less virulent than the Queensland clone of CAMRSA as it lacks the Panton–Valentine leukocidin.[5]

This is the first reported case of fatal necrotising pneumonia caused by CA-MRSA in Australia and illustrates the invasive nature of this infection. Thus far, CA-MRSA has predominantly caused skin and soft tissue infections, but the incidence of life-threatening sepsis is increasing. The first case of severe pneumonia caused by CA-MRSA in Australia was reported in early 2003.[6] Fatal cases of necrotising pneumonia caused by CA-MRSA have also been described in the United States[3] and France,[7] and this presentation is becoming a particular feature of this organism.

Clinicians should consider the possibility of CA-MRSA in any patient presenting to hospital with severe staphylococcal sepsis or pneumonia and should consider including parenteral vancomycin in the initial empirical therapy, particularly in geographic locations where CA-MRSA has been reported and in ethnic groups at increased risk.

Acknowledgements: We thank the staff of the Microbiology Department at the Royal Brisbane and Women's Hospital for the isolate. We also thank Sharon Kleinschmidt for performing the pulsed field gel electrophoresis and Alex Stephens for performing the polymerase chain reaction test for the *pvl* gene.

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Fatal leptospirosis presenting as musculoskeletal pain

Lloyd K Morgan

General practitioner (retired), PO Box 150, Lorne, VIC 3232. lloydmorgan@iprimus.com.au

To the Editor: O'Leary et al[1] are pessimistic about the value of antibiotic treatment for leptospirosis, based on an inconclusive Cochrane review[2] and no proven mortality decrease. Yet we can be more optimistic, given the efficacy of prophylactic doxycycline (soldiers in Panama were 95% protected by 200 mg once-weekly[3]), the susceptibility of leptospira to various antibiotics *in vitro* (especially penicillin, but not erythromycin[4]), and the existence of a Herxheimer reaction with penicillin,[5] which indicates in-vivo activity.

The Cochrane review[2] was of randomised controlled trials of confirmed cases. It included 75 patients given antibiotics and 75 given placebo. Penicillin (61 patients) and doxycycline were not compared. The time from symptom onset to starting antibiotics was stated in only two trials (9 and 2 days). Nevertheless, the review concluded that penicillin or doxycycline may do more good than harm.

In the spirochetaemic phase of leptospirosis (Days 4–7), vasculitis causes multiorgan failure. Successful treatment with antibiotics seems likely if commenced *within 2 days* of symptom onset, before generalised vasculitis is irreversible. Unfortunately, early diagnosis and efficacy assessment is difficult because symptoms are protean and nonspecific, no rapid laboratory test exists, the condition is mild and self-limited in most cases, and mortality varies from zero to 7%. A high index of suspicion is essential so that antibiotics can be commenced empirically.

The consensus is that antibiotics should be commenced within 4 days. In one study, starting antibiotics within 7 days was associated with shorter illness, and if antibiotics were started within 2 days the shorter duration was highly significant ($P = 0.006$).[6] Another trial showed penicillin commenced on Day 9 was beneficial, but antibiotics had been used before entry to the trial.[2]

Various penicillins and tetracyclines have seemed useful, but only oral doxycycline and intravenous benzylpenicillin are recommended. These are the first and second preferences, respectively, in *Therapeutic guidelines antibiotic*.[7]

In the case described by O'Leary et al,[1] antibiotics were commenced on Day 3, but the penicillin dose was only half the recommended daily dose for leptospirosis. The vasculitis was probably terminal before the patient was transferred to Concord Hospital.

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Australia was indeed the "lucky country" in the recent worldwide SARS epidemic

Marianne E Jauncey,* Paul K Armstrong,† Emily L Morgan,‡ Jeremy M McAnulty§

NSW Public Health Officer, Public Health Training and Development Branch; † Medical Epidemiologist, § Director, Communicable Diseases Branch; NSW Health, North Sydney, NSW. ‡ General Practitioner, Ballina West Medical Centre, Ballina, NSW.
Marianne.jauncey@yahoo.com.au

To the Editor: In 2003, severe acute respiratory syndrome (SARS) became the first pandemic of the 21st century. Despite spreading to 29 countries, a rapid and coordinated international effort led to its containment. Here, we examine Australia's only laboratory-confirmed case, and the investigation of possible subsequent transmission.

In June 2003, the World Health Organization (WHO) notified Australian health authorities of a 26-year-old tourist in whom SARS-coronavirus-specific antibodies had recently been detected. She was part of a retrospective serological survey of people who stayed at the Hotel Metropole, Hong Kong, on 21 February,[1] the same time a SARS source case infected at least 14 other hotel guests.[2] On 22 February, the 26-year-old tourist travelled to Australia and 4 days later developed myalgia, lethargy and cough. On 6 March, 6 days before the first WHO global alert on SARS, she saw a general practitioner (GP) in northern New South Wales, to whom she also reported nausea, vomiting, nocturnal fever and pronounced lethargy. On examination she was afebrile, pale, unwell, with a cough and clear chest on auscultation. She declined further investigations and hospital admission; her condition gradually improved, and she left Australia 6 days later. She reported close contact with only three people during her Australian visit — her partner, the GP, and the GP's surgery nurse. None reported subsequent illness and all tested negative for SARS-coronavirus antibody by direct immunofluorescence, a highly sensitive and specific method.[3] Australia was fortunate that the tourist was not particularly infectious. The Hotel Metropole case was identified as the source case for four national and international clusters of SARS.[2] The resulting human and economic cost was substantial.[4]

Without specific treatments, basic public health measures proved the only effective means to contain SARS. These included rapid case detection and isolation, contact tracing, handwashing and the correct use of personal protective equipment.[5] Many GP practices and some hospitals in Australia do not have isolation facilities or infection control resources to effectively contain diseases like SARS. In the event of local transmission of SARS, infection may well have occurred in Australian healthcare workers.

In the wake of SARS and, more recently, avian influenza, GPs must develop infection control plans to protect their own health as well as that of their patients. These should include obtaining a history of travel to outbreak-affected areas, reserving an area for patient isolation, and using appropriate

infection control precautions during such outbreaks. Clinicians in other healthcare settings also need to review current infection control practices. If Australia is to remain the "lucky country" with regard to communicable diseases, basic public health measures aimed at preventing transmission of infection in healthcare settings is essential.

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Teaching on the run tips: doctors as teachers

Jennifer W Majoor,* Joseph E Ibrahim†

* Associate Professor, Department of Psychiatry, Alfred Hospital, Prahran, VIC; † Monash University Professor of Aged Care Medicine, Peninsula Health, Frankston, VIC. **J.Majoor@alfred.org.au**

To the Editor: We applaud the Journal's new series to improve the standard of teaching within the medical profession.[1,2] However, it seems ironic that a profession that relies so heavily on an experiential, apprentice-based model of learning should be running such a series.

Since the early nineties, we have seen a paradigm shift with regard to improving the quality of healthcare. However, this recent managerial preoccupation with systems, processes and outcomes has largely ignored the relationship between effective teaching and patient care. Clinical service work is given priority over training and education activities, and it is likely that, if it weren't for the clauses in our employment contracts, *all* training, conferences and educational activities would occur out of work hours.

Although we have seen a number of structural interventions to promote ongoing education, such as the introduction of Continuing Medical Education programs, the idea that "any" education will do, and that "anyone" can teach, remains pervasive. The danger of promoting "teaching on the run" is to reinforce the view that teaching is not a specialised discipline that requires specific skills and training. It is astounding that no formal qualifications in education are required for teaching at the most senior level, whereas to be taken seriously as a researcher requires an MD or PhD.

It is a rare gifted teacher who instinctively performs well without formal training. High-quality teaching requires formal training, just as high-quality research does. Do we allow anyone in medicine to simply do "research on the run"? Would we ever consider a series called "Research on the run"? In medicine, we recognise that people are

drawn to particular specialties because of their different knowledge, skills, interests and temperaments. This is not always the case in teaching, and names on a tutorial roster are too often allocated without regard for the style or ability of the teacher.

Lake points out that the majority of problems with teaching are related to the traditional culture of medical practice and health service delivery.[2] Similarly, Quadrio has observed that "career advancement in medicine … depends primarily upon research productivity, less upon clinical work and teaching …".[3]

In our view, medicine requires another paradigm shift towards competency assessment and promotion of our teachers in academic settings. Furthermore, the allocation of appropriate resources is paramount and is justified because of the likely spin-offs for improved quality of patient care. We look forward to the day when medical education is rewarded as a highly valued endeavour, rather than a burden for busy clinicians and academics. We eagerly await further instalments of this well intentioned series on teaching, and hope that it goes some way towards effecting a "Kuhnian revolution".[4] (US scientist Thomas Kuhn proposed that scientific knowledge proceeds according to popular paradigms that, every now and then, undergo "intellectually violent revolutions … in each of which one conceptual world view is replaced by another …".)

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Fiona R Lake

Associate Professor in Medicine and Medical Education, Faculty of Medicine and Dentistry, University of Western Australia, Nedlands, WA.
flake@cyllene.uwa.edu.au

In reply: As noted by Majoor and Ibrahim, teaching and learning, as a mission, are not well regarded when compared with research. Not only that, but changes in healthcare are making it harder to teach. Shorter patient stays and more complex patients result in "survival" learning by junior staff, rather than in-depth learning.[1,2]

Experts in medical education will be increasingly important in teaching, guiding curricula, and assessing trainees.[2] However, professional learning occurs while doctors immerse themselves in clinical practice. "On the run" teaching doesn't mean substandard teaching, but relates to doing it while delivering patient care.[3] Although there is room for improvement, many clinicians teach well. Most are keen to teach and would like to have formal training,[4] and evidence suggests that, with support, they can improve.[2,3] How much support? Short workshops have been shown to have an impact, as has the provision of a few simple educational ideas.[5] By not supporting our clinicians/teachers in ways that could be very simply put into practice, we risk losing in-context learning and wasting an enormous resource.

Along with focusing on the teacher, I believe we need an important shift in the way health services recognise (provide time for) and reward (see as important) the mission of

teaching and supervision alongside their mission of excellence in delivery of care.

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Suboptimal management of subclinical hypothyroidism

Chin-Pin Yeo,* Melissa J Gillett,† Samuel D Vasikaran‡

* Chemical Pathologist, † Biochemistry Registrar, ‡ Head, Core Clinical Pathology and Biochemistry, Royal Perth Hospital, GPO Box X2213, Perth, WA 6847.
gptycp@sgh.com.sg

To the Editor: In about 2% to 5% of patients with subclinical hypothyroidism, the condition progresses to overt hypothyroidism each year.[1] It is currently recommended that thyroid function tests should be repeated at 6- to 12-month intervals to monitor improvement or worsening in level of thyroid-stimulating hormone (TSH).[1] Testing for thyroid peroxidase antibody (TPOAb) is also common practice in subclinical hypothyroidism, as it has been shown that individuals with raised TSH and TPOAb levels have a 40-fold increased risk of developing overt hypothyroidism.[2,3]

We audited the management of patients who had a result indicating subclinical hypothyroidism from our hospital laboratory, focusing on follow-up thyroid function and TPOAb tests. In December 2003, we retrospectively inspected clinical case notes of patients who had been reported in November 2002 with a TSH level of 4.1–9.9 mIU/L (normal reference interval, 0.4–4.0 mIU/L) and a free thyroxine (FT_4) level within the reference range of 10–23 pmol/L (subclinical hypothyroidism). We also contacted the patients' general practitioners (GPs) for further information when necessary.

There were 72 patients with results suggesting subclinical hypothyroidism. Of these, we excluded 29 from other hospitals and three whose GPs were not able to be contacted. Of the remaining 43 patients, 18 had no previous history of thyroid disease (8 men and 10 women; age range, 24–90 years). Six patients were seen in the emergency department, four in the outpatient clinics, and eight in the wards. All the laboratory reports of subclinical hypothyroid results were accompanied by a comment advising repeat thyroid studies at a later date and thyroid antibody tests. However, only three of the 18 patients (17%) had TPOAb tested. Only seven of the 18 patients (39%) were followed up with repeat thyroid function tests, at intervals ranging from 3 days to 7 months. One patient, with a TSH level of 9.8 mIU/L, was started on thyroid replacement therapy. The GPs of 10 of the 11 patients who did not have follow-up testing were not informed of the initial TSH results.

The low rate of follow-up of hospital patients with a first-time diagnosis of subclinical hypothyroidism is of concern. While the increase in TSH level in some of these patients may have been related to sick euthyroidism, this can only be confirmed by normalisation of TSH level on repeat testing. TPOAb testing can be deferred until confirmation of persistently raised TSH level. Better strategies, such as a computerised system for selective copying of results to GPs whenever relevant, and inclusion of treatment advice dependent on TPOAb status in the reports,[4] may be needed to improve follow-up.

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