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Tungiasis in recently arrived African refugees

Ashwin Swaminathan,* Iain B Gosbell,†
Nicholas A Zwar,‡ Mark W Douglas§

* Infectious Diseases Registrar, † Director and Associate Professor, § Infectious Diseases Physician, Department of Microbiology and Infectious Diseases, Liverpool Hospital, South Western Area Pathology Service, Locked Bag 7090, Liverpool, NSW 1871; ‡ Director and Professor of General Practice, Sydney South West Area Health Service General Practice Unit, Fairfield Hospital, Sydney, NSW.
Iain.Gosbell@swsahs.nsw.gov.au

TO THE EDITOR: Infestation with the sandflea *Tunga penetrans*, or “chigoe flea”, is rarely encountered in Australia, but has been noted in children recently arrived from Central–East Africa. There have been only two previous Australian case reports of this parasitic infection, both in adult travellers returning from Africa.^{1,2}

Several families who had been living in crowded refugee camps in Tanzania underwent routine screening for infection within 2 weeks of arrival in Australia. Four of 14 children examined had cutaneous lesions on their feet — mainly on the toes, nail beds and interdigital spaces (Box). These lesions were papular, less than 1 cm in diameter, pale yellow with dark centres, and were variably painful and/or itchy. Chronic, adjacent skin and nail bed changes were evident, as were small, loosely attached seed-like objects. Papules could be lifted with a sterile needle, leaving a small, non-bleeding cavity. *Tunga penetrans*, with numerous attached eggs, was identified by microscopy.

Tungiasis occurs when an impregnated female sandflea burrows into the unprotected skin of a warm-blooded host. There is a predilection for the feet, although the perineum, buttocks and arms may also be infected.³ The head of the sandflea breaches the upper dermis to feed on blood vessels, while the abdomen traverses the epidermis, with its posterior components (anus, genital opening and respiratory spiracle) reaching the surface, forming a papule. Over several weeks, the flea releases hundreds of eggs before dying. After hatching, the larvae thrive in dust, soil and sand; they are found on beaches and in animal stockyards of tropical countries.^{3,4} Infection of pigs and other livestock, the usual host reservoirs, has led to significant problems in the livestock industry.^{1,4}

Apart from pruritis and pain caused by local inflammation, morbidity results from ulceration and secondary bacterial infec-

Lesions caused by *Tunga penetrans*, the “chigoe” sandflea



A characteristic *Tunga penetrans* lesion (thick arrow), with pale-yellow papule and dark centre, and a less obvious lesion (thin arrow) with surrounding chronic skin changes and multiple, loosely attached eggs.

tion, including tetanus and gas gangrene.^{1,2,4}

Fleas can be removed using a sterile needle and forceps, and secondary complications prevented with tetanus prophylaxis, and antibiotics as appropriate. Successful outcomes with antiparasitic agents, such as ivermectin and thiabendazole, have also been recently reported.⁵

Tungiasis is indigenous to Latin America and the Caribbean, but was introduced to Africa, where it is now endemic, and to parts of South Asia.⁴ Given the current influx of African refugees into Australia, including the tropical north, the obvious concern is whether *Tunga penetrans* could establish itself here.

We encourage medical practitioners dealing with newly arrived African refugees to examine for tungiasis in their screening evaluation. If, as suspected, this condition is prevalent, national infection control guidelines aimed at preventing establishment of the disease in Australia may be needed.

1 Ott MB, Charters AD, Bowman RA. Tungiasis: imported disease. *Med J Aust* 1980; 2: 623-624.

2 Spradbery JP, Bromley J, Dixon R, et al. Tungiasis in Australia: an exotic disease threat [letter]. *Med J Aust* 1994; 161: 173.

3 Eisele M, Heukelbach J, Van Marck E, et al. Investigations on the biology, epidemiology, pathology and control of *Tunga penetrans* in Brazil: 1. Natural history of tungiasis in man. *Parasitol Res* 2003; 90: 87-99.

4 Heukelbach J, Araujo F, Oliveira de S, et al. Tungiasis: a neglected health problem of poor communities. *Trop Med Int Health* 2001; 6: 267-272.

5 Heukelbach J, Eisele M, Jackson A, Feldmeier H. Topical treatment of tungiasis: a randomized, controlled trial. *Ann Trop Med Parasitol* 2003; 97: 743-749. □

Incidental finding of *Dracunculus medinensis* in Australia

Tulsi Menon

Resident Medical Officer, Department of Orthopaedic Surgery, Royal Perth Hospital, 19 Morgan Road, Redcliffe, WA 6104
kaltul@optusnet.com.au

TO THE EDITOR: I report an infection with *Dracunculus medinensis* diagnosed incidentally on x-ray. The patient was a Sudanese immigrant, who had sustained a left knee joint effusion after falling. During management at Royal Perth Hospital, a calcified lesion with a serpentine appearance was seen on x-ray within soft tissues near the left knee joint. Its characteristic appearance, combined with the patient's background, led to a diagnosis of *D. medinensis* (known as guinea-worm), a nematode parasite which causes dracunculiasis.

The patient's knee effusion was managed conservatively and improved within a few weeks. The effusion was secondary to the fall and believed to be unrelated to the calcified *D. medinensis*.

Transmission of dracunculiasis is through consumption of contaminated water. The guinea-worm larvae mature and migrate towards the skin surface over 1 year (during which the patient remains asymptomatic), with over 90% of the worms appearing from the lower limbs. When in contact with water, the exposed guinea-worm releases larvae, and the lifecycle is completed when people ingest contaminated water.¹ If the guinea-worms die before maturation, they usually calcify.

The Global Dracunculiasis Eradication Campaign was established in 1981 with the aim of eliminating dracunculiasis through water sanitation efforts.¹ With no vaccine or treatment, prevention is the only method of eliminating dracunculiasis. Since the campaign was established, the number of people affected by dracunculiasis has decreased by 98%. Currently, Sudan alone accounts for 73% of cases.¹

Our patient migrated to Australia as a refugee from Sudan during the civil war. Her village in Sudan had only one source of water used for daily activities, including drinking. The patient knew many people with dracunculiasis, but did not know she had been infected.

When calcified guinea-worms are discovered during routine radiological examination, they usually do not need treatment. Many people are not aware they have been infected.

Lateral view of left knee joint and distal left thigh



A calcified lesion, representing *Dracunculus medinensis* within the soft tissues, is visible posterior and lateral to the distal femur near the knee joint. The calcification has a serpentine appearance: the proximal part coiled in appearance, the middle having a string-like linear appearance, and the distal part having dense curvilinear opacity.

Muller reported 89% of patients with calcified guinea-worms were asymptomatic.²

No known previous case of a radiologically diagnosed calcified guinea-worm has been reported in Australia. The consequences of war and famine, with a resultant increase in refugees and immigrants from affected nations, is likely to increase the number of incidental calcified guinea-worms found in non-endemic countries. This disease may have a significant impact if affected immigrants arrive during the incubation period (when asymptomatic) and the parasite emerges from the skin after immigration (rather than calcifying), similar to the patient described by Spring.³ Thus, it is important for health personnel to be aware of dracunculiasis, including its radiological manifestations.

Acknowledgements: I thank Mr Alan Prosser, Dr Mike Ledger, Dr Vera Kinzel, and Dr Song for encouragement and assistance in preparing this letter.

1 Greenaway C. Dracunculiasis (guinea worm disease). *CMAJ* 2004; 170: 495-500.

2 Muller R. *Dracunculus* and dracunculiasis. In: Dawes B, editor. *Advances in parasitology*. New York, NY: Academic Press, 1971; 73-140.

3 Spring M, Spearman P. Dracunculiasis: report of an imported case in the United States. *Clin Infect Dis* 1997; 25: 749-750. □

Vitamin D and adult bone health in Australia and New Zealand: a position statement

Simon J Vanlint

Lecturer, Department of General Practice, University of Adelaide, SA 5005.
simon.vanlint@adelaide.edu.au

TO THE EDITOR: The Working Group of the Australian and New Zealand Bone and Mineral Society, Endocrine Society of Australia and Osteoporosis Australia are to be commended for their clear and succinct position statement on vitamin D and bone health.¹ This statement highlights an important public health issue which is under-recognised in this country. Of particular value is the box of recommendations on high-risk groups, testing and treatment.

However, I believe an important high-risk group has been omitted, a group which too often escapes the notice of the broader medical community. People with intellectual disability have been shown to be at particularly high risk of low vitamin D levels, reduced bone density and fractures.²⁻⁵ The reasons for this are multifactorial and include poor mobility, insufficient sun exposure, reduced muscle mass and strength, problems with dietary intake, and medications which interfere with vitamin D metabolism.^{3,4} There is also some evidence that people with intellectual disability are prone to premature ageing, together with the health problems associated with older age in the general population.³ Some conditions which cause or are associated with intellectual disability are also linked with hypogonadism and reduced peak bone mass.⁴

To add to all of this, several of the above risk factors, vitamin D deficiency itself, and the increased incidence of epilepsy in the population with intellectual disability, also result in an increased incidence of falls and trauma.⁵ This unfortunate combination of poor bone health and increased risk of falls and trauma results in a markedly increased incidence of fracture when compared with age- and sex-matched controls from the general population.²⁻⁵

In conclusion, people with intellectual disability, particularly those with poor mobility or who are also being treated for epilepsy, should be added to the list of high risk groups. It is likely that the relatively simple steps set out in the position statement (screening for vitamin D deficiency and supplementation) will result in substantial health benefits for this small but particularly vulnerable group of people.

- 1 Working Group of the Australian and New Zealand Bone and Mineral Society, Endocrine Society of Australia and Osteoporosis Australia. Vitamin D and adult bone health in Australia and New Zealand: a position statement. *Med J Aust* 2005; 182: 281-285.
- 2 Wagemans A, Fiolet J, van der Linden E, Menheere P. Osteoporosis and intellectual disability: is there any relation? *J Intellect Disabil Res* 1998; 42: 370-374.
- 3 Foster B, Walkley J, Temple V. Bone mineral density status of women with intellectual disability. *Adapted Phys Activity Q* 2001; 18: 48-59.
- 4 Schrage S. Osteoporosis in women with disabilities. *J Womens Health* 2004; 13: 431-437.
- 5 Jancar J, Jancar M. Age-related fractures in people with intellectual disability and epilepsy. *J Intellect Disabil Res* 1998; 42: 429-433. □

Alvin L K Chia,* Stephen Shumack,† Peter Foley‡

* Research Fellow, † Dermatologist, St George Dermatology and Skin Cancer Centre, Level 3, 22 Belgrave St, Kogarah, NSW 2217;

‡ Dermatologist, St Vincent's Hospital, Melbourne, VIC. sshumack@bigpond.com

TO THE EDITOR: We read with alarm the extraordinary statements in the position statement on vitamin D and adult bone health published recently in the *Journal*.¹ The suggestion that "it is a fallacy that Australians receive adequate vitamin D from casual exposure to sunlight" is not true.

The suggested basis for this statement is an extraordinary extrapolation from a single study in which a small number of volunteers had whole body exposure on one occasion for 10–15 minutes to midday summer sun in Boston. It is not possible to extrapolate in such a way from this single demonstration, as the effect of shorter exposure times or repeated daily exposures were not examined. In fact, a study in Australia showed that the adult population (including those aged over 70 years) received sufficient sunlight while using sunscreen to ensure that no-one was found to have vitamin D deficiency during the study period.²

While it is well accepted that ultraviolet B (UVB) radiation is essential for the formation of vitamin D₃ in the skin, it is equally well established that continued exposure of vitamin D to UVB radiation results in its degradation. Hence, the importance of knowing the effect of lower sun exposures on vitamin D production.

It is intriguing that the authors of the position statement recommended a daily sun exposure dose that they calculate will produce 1000 IU of vitamin D, but, if sun exposure is not possible, a vitamin D supplement of at least 400 IU per day.

The high prevalence of vitamin D deficiency among institutionalised older Australians is a tragedy, but this cannot be used

as the basis of advice for the general population who do receive daily sun exposure and appear to be the target of the statement. Nor can the mild vitamin D deficiency found in a single study in southern Victoria be used to recommend sun exposure in more northern Australian climes. Finally, while vitamin D supplementation has been shown to reduce the risk of fractures in the elderly, the proposed beneficial effect of deliberate sun exposure has not been demonstrated.

Recently, a joint position statement was approved by the Australian and New Zealand Bone and Mineral Society, Osteoporosis Australia, the Australasian College of Dermatologists and the Cancer Council Australia. This included the statement that "The majority of Australians generally have sufficient ultraviolet radiation exposure to enable adequate vitamin D production . . . to form and maintain healthy, strong bones".³ This statement, endorsed only a few weeks ago, is in obvious conflict with the position statement from the same organisations that was published in this Journal. The latter puts the vast majority of Australians at further risk of skin cancers, which are already epidemic in our country.⁴

- 1 Working Group of the Australian and New Zealand Bone and Mineral Society, Endocrine Society of Australia and Osteoporosis Australia. Vitamin D and adult bone health in Australia and New Zealand: a position statement. *Med J Aust* 2005; 182: 281-285.
- 2 Marks R, Foley PA, Jolley D, et al. The effect of regular sunscreen use on vitamin D levels in an Australian population. Results of a randomized controlled trial. *Arch Dermatol* 1995; 131: 415-421.
- 3 Risks and benefits of sun exposure position statement. Approved by the Australian and New Zealand Bone and Mineral Society, Osteoporosis Australia, Australasian College of Dermatologists and the Cancer Council Australia. Sydney: Osteoporosis Australia, 2005. Available at: <http://www.osteoporosis.org.au/files/crksandbenefitsMarch8.pdf> (accessed May 2005).
- 4 Marks R. Epidemiology of non-melanoma skin cancer and solar keratoses in Australia: a tale of self-immolation in Elysian fields. *Australas J Dermatol* 1997; 38 Suppl 1: S26-S29. □

Terrence H Diamond,* John A Eisman,[†]
Rebecca S Mason,[‡] Caryl A Nowson,[§]
Julie A Pasco,[¶] Philip N Sambrook,**
John D Wark^{††}

* Associate Professor, Endocrinology, University of NSW, Sydney, NSW; † Professor and Director, Bone and Mineral Research Program, Garvan Institute of Medical Research, Sydney, NSW; ‡ Associate Professor of Physiology, ** Professor of Rheumatology, University of Sydney, NSW; § Associate Professor, School of Health Sciences, Deakin University, Melbourne, VIC; ¶ Senior Research Fellow, Clinical and Biomedical Sciences: Barwon Health, The University of Melbourne, PO Box 281, Geelong, VIC 3220; †† Professor of Medicine, The University of Melbourne, VIC. juliep@barwonhealth.org.au

IN REPLY: We agree with Vanlint that any individual who has limited mobility, or is housebound or institutionalised, is at risk of vitamin D deficiency, as highlighted in Box 3 of the position statement.¹ Disability in general is likely to be a risk, with motor disability as well as intellectual disability liable to limit sun exposure. The problem may be further exaggerated by any increased risk of falls or convulsions.

Chia and colleagues have raised important issues. After a number of meetings, the Cancer Council of Australia, the Australasian College of Dermatologists and the Australian and New Zealand Bone and Mineral Society developed a considered consensus statement on vitamin D deficiency, risk of skin cancers and sunlight exposure, which was published at <http://www.cancer.org.au/documents/Risks_Benefits_Sun_Exposure_MAR05.pdf>. This document refers to the position statement published in this Journal in relation to sun-exposure guidelines for vitamin D.

Chia and colleagues' objection to the abstract of this position statement appears ill-founded. If "a significant number of Australians are deficient in vitamin D", then it follows logically that "it is a fallacy

that Australians receive adequate vitamin D from casual exposure to sunlight", as sunlight is the main source of vitamin D in Australia. The significant number of Australians deficient in vitamin D are not the majority, as was clearly shown in the article, so this statement does not conflict with the complementary statement in the risks and benefits statement.

We stand by our original claim that a number of groups in the Australian community have a high prevalence of vitamin D deficiency, including elderly men with hip fracture (63%), Muslim women (68%), elderly ambulant men with prostate cancer (34%), "healthy" elderly men living in Southern Sydney (16%), healthy community-dwelling, ambulatory women in Geelong (20% in the age group 20–39 years, increasing to 53% in older age groups), men and women (some with psychiatric disorders) in south-east Queensland (23%), and even pregnant women in south-eastern Australia (7%) (references are available from the authors on request).

As noted in the position statement, and by Chia and colleagues, continued exposure to ultraviolet (UV) radiation may lead to degradation of pre-vitamin D, so that short exposures are likely to be more efficient. This degradation is marked only at relatively high UV doses.² Studies that used lower UV doses^{2,3} produced indirect UV equivalence data similar to those quoted in the position statement.¹ As the relationship between UV exposure and vitamin D dosage varies from person to person, and as sun exposure is also likely to be variable and on most days, rather than every day, the recommendation that vitamin D supplementation be at least 400 IU/day in people likely to be at risk of inadequate skin-derived vitamin D is entirely appropriate.

We strongly agree with Chia and colleagues about the need to avoid sun damage while still obtaining the small amount of sun exposure needed to make adequate vitamin D, which is why the position statement advocates short exposures, easily achieved by casual exposure, and reiterates other Sun-Safe messages, such as avoidance of exposure in peak UV periods and the use of sunscreens where appropriate.

1 Working Group of the Australian and New Zealand Bone and Mineral Society, Endocrine Society of Australia and Osteoporosis Australia. Vitamin D and adult bone health in Australia and New Zealand: a position statement. *Med J Aust* 2005; 182: 281-285.

2 Davie M, Lawson DE. Assessment of plasma 25-hydroxyvitamin D response to ultraviolet irradiation over a controlled area in young and elderly subjects. *Clin Sci (Lond)* 1980; 58: 235-242.

3 Chel VGM, Ooms ME, Popp-Snijders C, et al. Ultraviolet irradiation corrects vitamin D deficiency and suppresses secondary hyperparathyroidism in the elderly. *J Bone Miner Res* 1998; 13: 1238-1242. □

Outcome of overseas commercial kidney transplantation: an Australian perspective

Deborah J Verran

Senior Transplant Surgeon, Royal Prince Alfred Hospital, Missenden Road, Camperdown, NSW 2050.
deborah@email.cs.nsw.gov.au

TO THE EDITOR: A recent editorial by Mathew et al¹ and an article by Kennedy et al² tackle the issue of commercial kidney transplantation.

In their editorial, Mathew and colleagues conclude that if the nationwide Australian deceased donor organ donation rate approached South Australian levels, dialysis patients would not travel overseas to purchase renal allografts.¹ I believe that this statement oversimplifies the situation with respect to organ trafficking and the motives behind patients' acceptance of this option.

Organ trafficking is illegal under all state and territory laws within Australia and hence cannot occur. In a number of other countries around the world this is not the case. Although laws have been introduced in India, this has not necessarily led to cessation of trafficking in kidneys.³ Compounding this, the option of purchasing an organ is becoming more readily accessible by means of an increasing number of Internet sites.^{4,5}

What motivates patients to go through with purchasing an organ overseas is not explored either in the editorial by Mathew et al¹ or the article by Kennedy et al.² Kennedy et al do not state whether any of the patients who travelled overseas for a kidney had been deemed not fit to be on the renal transplant waiting list in New South Wales. They also do not give the waiting time on dialysis for each patient before he or she decided to travel overseas. What is clear is that only patients who can afford to pay the US\$70 000 or more currently quoted for a renal allograft will be the ones who end up travelling overseas.

It would be nice to think that, with an increased organ donor rate in Australia, patients will no longer travel overseas to purchase organs. However, the ready availability of the commodity in not-too-distant countries and the increasing ease of access to this organ trade, combined with sufficient cash, will mean that there is no major barrier to some individuals.

1 Mathew TM, Faull RJ, Snelling P. The shortage of kidneys for transplantation in Australia. *Med J Aust* 2005; 182: 204-205.

2 Kennedy SE, Shen Y, Charlesworth JA, et al. Outcome of overseas commercial kidney transplantation: an Australian perspective. *Med J Aust* 2005; 182: 224-227.

3 Goyal M, Mehta RL, Schneiderman LJ, Sehgal AR. Economic and health consequences of selling a kidney in India. *JAMA* 2002; 288: 1589-1593.

4 Yeson Transplant Network — China Transplant Information. Available at: <http://www.yeson.com.tw> (accessed Mar 2005).

5 Magnifica Libertad Incorporated, The Philippines. Available at: <http://www.liver4you.org> (accessed Mar 2005). □

Sean E Kennedy,* Yvonne Shen,*
John A Charlesworth,†
James D Mackie,‡ John D Mahony,§
John J P Kelly,¶ Bruce A Pussell**

* Renal Registrar, † Professor of Renal Medicine, Prince of Wales Hospital, Sydney, NSW.

‡ Renal Physician, Prince of Wales Hospital, Sydney, and Illawarra Regional Hospital, Wollongong, NSW.

§ Renal Physician, Royal North Shore Hospital, Sydney, NSW. ¶ Renal Physician, St George Hospital, Sydney, NSW.

** Professor of Medicine, Department of Nephrology, Prince of Wales Hospital, Sydney NSW 2031. b.pussell@unsw.edu.au

IN REPLY: We agree that the motives for organ trafficking are complex. The reasons that people choose to travel overseas to obtain an organ was not addressed in our article on this occasion, but some possible reasons may be self evident given the long waiting time, especially in New South Wales. Our aim was to alert our colleagues to the increased risks associated with overseas commercial transplant procedures so that they could ensure that their patients were fully informed when making a decision.

Most of our patients were on the waiting list, and the time on dialysis was detailed in Box 2 in our article.¹ However, two were not on the waiting list — one had a pre-emptive transplant and another was not on the list because of age-related medical problems.

1 Kennedy SE, Shen Y, Charlesworth JA, et al. Outcome of overseas commercial kidney transplantation: an Australian perspective. *Med J Aust* 2005; 182: 224-227. □

The shortage of kidneys for transplantation in Australia

Raymond F Raper,* Elizabeth Fugaccia,†
Yahya Shehabi‡

* Board Member, † Member, ‡ Chairman, NSW Regional Committee, Joint Faculty of Intensive Care Medicine, 117 Alexander Street, Crows Nest, NSW 2065.
yshehabi@ozemail.com.au

TO THE EDITOR: We are writing in response to the pejorative, unhelpful and somewhat misleading editorial “*The shortage of kidneys for transplantation in Australia*”.¹

There are many possible reasons for lower organ donation rates in Australia. Several Australian initiatives have led the way in reducing the incidence of severe, traumatic brain injuries. These include the compulsory wearing of seat belts and helmets, random breath testing, and a zero blood alcohol limit for inexperienced drivers. Intensive care medicine is better structured and organised in Australia and New Zealand than in most of the countries cited by Mathew et al, with higher organ donation rates. Intensive care outcomes in Australia are world-leading. So the donor rate may be lower because patient outcomes are better. A comprehensive chart audit of donor potential in New South Wales carried out under the supervision of the Organ Donation Network NSW/ACT, identified very few missed donors (T Wills, Manager, Organ Donation Network NSW/ACT, personal communication), and a Victorian audit has suggested that the donor pool may be much lower in Australia than previously estimated.²

Similarly, organ donation rates will appropriately vary considerably among hospitals. To improve outcomes, critically ill patients are transported to centres with specific experience and expertise, resulting in a preponderance of potential donors in hospitals with trauma and neurosurgical services compared with hospitals lacking these. The intensive care community supports these life-saving initiatives, notwithstanding the effect they may have on organ donation potential.

In fact, the principal “barrier” to organ donation in Australia appears to be the consent rate. From 2000 to 2004, 44% of families declined organ donation when faced with an actual rather than a hypothetical request.³

The intensive care community represents the interests of critically ill patients and their families. We will continue our best endeavours to improve both the survival and quality of life of patients suffering devastating brain injuries (our performance standard). When all brain function ceases, despite our best efforts, we will continue to facilitate organ donation, in discussion with the family, and in consideration of the patient’s known or projected wish. The inten-

sive care community has led the way in developing organ donation-related practice guidelines⁴ and in related education. Australian Donor Awareness Program — Training (ADAPT) workshops are now a compulsory component of Fellowship of the Joint Faculty of Intensive Care Medicine training. The editorial implication of poor performance and lack of commitment is inaccurate and offensive.

- 1 Mathew T, Faull R, Snelling P. The shortage of kidneys for transplantation in Australia. *Med J Aust* 2005; 182: 204-205.
- 2 Opdam HI, Silvester W. Identifying the potential organ donor: an audit of hospital deaths. *Intensive Care Med* 2004; 30: 1390-1397.
- 3 Alvaro C. Identification and review of potential organ donors — 2004 summary report. Sydney: Organ Donation Network NSW/ACT, 2004.
- 4 Australian and New Zealand Intensive Care Society. Recommendations concerning brain death and organ donation. 2nd ed. Melbourne: ANZICS, 1998. Available at: http://www.anzics.com.au/files/brain_death_organ_donation.pdf (accessed Jun 2005). □

Timothy H Mathew,* Randall J Faull,†
Paul L Snelling‡

* Medical Director, Kidney Health Australia, GPO Box 9993, Adelaide, SA 5001; † Nephrologist, Royal Adelaide Hospital, Adelaide, SA; ‡ Nephrologist, Royal Prince Alfred Hospital, Camperdown, NSW.
tim.mathew@kidney.org.au

IN REPLY: We regret that Raper et al have misinterpreted our editorial on the shortage of donor kidneys in Australia.¹ We are particularly concerned and indeed mystified by their last statement, where they state that we implied that “poor performance and . . . lack of commitment” were to blame. This was in no manner our message. Rather, we sought to emphasise that all possibilities to optimise local donation rates should be explored, so desperate patients seeking grafts from potentially dangerous overseas sources need not expose themselves to serious potential morbidity and mortality.

We consider that our intensive care colleagues perform superbly under the most difficult of circumstances when managing potential organ donation. We understand they are often unsupported with managing potential donations while they must at the same time deal with the grieving family, and the immediate demands of treating other seriously ill patients. We simply suggest that problems within the system (for example, differences between states in the number of intensive care beds per head of population or in the provision of specifically funded donor coordinators) that might hinder increasing organ donation should be carefully examined.

We believe it is unhelpful to suggest that the South Australian experience should simply remain unexplained. In the article by

Opdam and Silvester (cited by Raper et al), of 112 potential donors, 46 were considered medically suitable unrealised potential donors, and their estimated maximal potential donor rate was 30 per million,² remarkably similar to the rates seen in Spain (the country with the highest organ donor procurement rate) and South Australia. We agree with the conclusions of Opdam et al that “an increase in the organ donation rate may be possible through increasing consent and the identification and support of potential donors”. As they also state, this would require substantial changes in clinical practice, with resource and ethical complications

We did not mean to offend our intensivists colleagues. We merely suggest that we all need to assess the systems in which donation occurs and attempt to improve donation rates, for the sake of the many desperate people awaiting organ transplants in this country.

- 1 Mathew T, Faull R, Snelling P. The shortage of kidneys for transplantation in Australia. *Med J Aust* 2005; 182: 204-205.
- 2 Opdam HI, Silvester W. Identifying the potential organ donor: an audit of hospital deaths. *Intensive Care Med* 2004; 30: 1390-1397. □

Detention for tuberculosis: public health and the law

A Medical Registrar and a
Respiratory Physician

A New South Wales Teaching Hospital
Names withheld to protect patient privacy

TO THE EDITOR: The article by Senanayake and Person¹ on detention for tuberculosis included two case histories. In the case of “Patient 2”, we believe the details published were misleading and unnecessarily disclosed potentially identifying information. This case was presented in sufficient detail to allow identification of “Patient 2” by including date of admission, personal details, city of admission and details regarding his past history of alcohol addiction and attempts to self-discharge. All this information was not necessary and did not take into account all the relevant medical complicating factors.

We were surprised that, as the primary treating team involved in this patient’s care over the inpatient stay of 3 months and the following 6-month outpatient follow-up, we were not informed of the authors’ plans for publication, nor requested to comment on the facts of the report. We were also surprised to read details of the article in *The Sydney Morning Herald* on the day of the article’s publication in the Journal.²

An individual's right to privacy is a fundamental human right. It is unfortunate that the careful consideration that had been given to his detention was not extended to publishing his case details. As Senanayake and Ferson point out, "Patient 2" recognised that his human rights were being "infringed" because he was being held in a "jail cell", which was a temporarily modified isolation room in a public hospital under 24-hour guard.

In law, information provided to a medical practitioner by a patient becomes subject to a statutory duty to protect the patient's privacy and a common-law duty of confidence is owed by the treating medical practitioner to the patient. The NHMRC *Guidelines approved under Section 95A of the Privacy Act 1998* indicate that, when a patient history is published, an important principle is that a patient may not be identified or held up to ridicule.³ Furthermore, public access to medical journals on the Internet has allowed increased availability of such reports to the general public and increases the chance of family members and others identifying individuals.

The principle of protecting patient privacy has previously been respected by the Journal. An article published in 1994 reported a 1979 outbreak of tuberculosis in medical students who attended an autopsy of an immunosuppressed patient with unsuspected active tuberculosis.⁴ A report of the incident was not published contemporaneously, mainly to protect the privacy of the students involved.

As the report by Senanayake and Ferson suggests, our patient was socially disadvantaged, and several aspects of his behaviour were probably attributable to a Jarisch-Herxheimer reaction in conjunction with acute alcohol withdrawal. Although we recognise the need to serve the public interest in health service management activities, this must be balanced against the requirement for patient privacy regardless of social class. We suggest that the publisher has an obligation to ensure that patient consent is obtained, and that the primary treating team has been involved in the review of case details so that misleading and potentially identifying information is not released inappropriately.

1 Senanayake SN, Ferson MJ. Detention for tuberculosis: public health and the law. *Med J Aust* 2004; 180: 573-576.

2 Locking up TB cases defended. *The Sydney Morning Herald* 2004; 7 Jun: 5.

3 National Health and Medical Research Council. Guidelines approved under Section 95A of the Privacy Act 1988. Canberra: NHMRC, 2001: 35-46. Available at: <http://www.health.gov.au/nhmrc/publications/pdf/e43.pdf> (accessed Oct 2004).

4 Wilkins D, Woolcock AJ, Cossart YE. Tuberculosis: medical students at risk. *Med J Aust* 1994; 160: 395-397. □

Ruth M Armstrong,*
Martin B Van Der Weyden†

* Deputy Editor, † Editor, *The Medical Journal of Australia*, Locked Bag 3030, Strawberry Hills, NSW 2012.
medjaust@ampco.com.au

IN REPLY: We concur with the principle that patient privacy should be protected in case reports. As recommended by the International Committee of Medical Journal Editors,¹ measures in place at the Journal include asking authors to obtain patient permission for publication where possible, and directing authors to remove potentially identifying patient information.

The article in question was not a case report. Case details were given to exemplify the circumstances that might lead to detention of a patient for public health reasons in New South Wales, and dates and some details were retained to illustrate the temporal flow of the story. We agree that this may have made the patient identifiable, if not to the general public, to himself or his family. We regret this editorial lapse, and have reworded the patient details in the electronic version of the article.²

Although chastened by our anonymous colleagues' observations, we are somewhat puzzled as to why, given their concern about their patient's privacy, they are determined to draw further attention to the exposing details. We assume that, in this case (as in the cases of detaining patients for treatment of tuberculosis), public interest prevails.

We also agree that case reports of detailed clinical histories require the input of the primary treating team, but the question of authorship should be determined by the involved parties, not the Journal. Be that as it may, as the article was a public health report (and thus not meant to be a detailed clinical exposition), we do not believe that the input of the treating team was required.

- 1 International Committee of Medical Journal Editors. Uniform requirements for manuscripts submitted to biomedical journals: writing and editing for biomedical publication, 2004. Available at: <http://www.icmje.org/> (accessed Jun 2005).
- 2 Senanayake SN, Ferson MJ. Detention for tuberculosis: public health and the law. *Med J Aust* [Internet version]. Available at: http://www.mja.com.au/public/issues/180_11_070604/sen10776_fm.html (accessed Jun 2005). □

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The Medical Journal of Australia (MJA) is published on the 1st and 3rd Monday of each month by the Australasian Medical Publishing Company Proprietary Limited, Level 2, 26-32 Pyrmont Bridge Rd, Pyrmont, NSW 2009. ABN 20 000 005 854. Telephone: (02) 9562 6666. Fax: (02) 9562 6699. E-mail: medjaust@ampco.com.au. The Journal is printed by Offset Alpine Printing Ltd, 42 Boorea St, Lidcombe, NSW 2141.

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28,010 circulation as at
29 April, 2005



ISSN 0025-729X