LETTERS

Lymphoedema in breast cancer patients

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TO THE EDITOR: It has been known for a long time that washing soda (crystalline sodium carbonate) is effective for removing fluid from joint effusions. The crystals are simply wrapped in a tea towel, crushed with a rolling pin and wrapped around the joint overnight. In the morning the sodium carbonate is rock solid and the joint effusion has markedly improved.

I was discussing with one my patients her problem of gross upper-limb lymphoedema after surgery and radiotherapy for breast cancer to the axilla. She went home and made an appropriate pack of sodium carbonate, which she wears overnight. She can now use her arm throughout the day. Obviously, the lymph fluid reaccumulates because her lymphatic system is well and truly obstructed.

This patient spoke to several other patients in the Day Centre at the Monash Medical Centre, who tried this in addition to other exercises for lymphoedema, and they have found it to be extraordinarily effective. One woman had gross oedema of her hand, which rendered it useless: she simply immersed her hand in a solution of sodium carbonate and thus dialysed the fluid from her hand. Thereafter she could use her hand for 12 hours before significant amounts of fluid reaccumulated.

While this is obviously not the perfect solution to lymphoedema in patients with breast cancer, anything that may help them is worth noting. Perhaps a suitable linen device could be made which would cover the whole arm at night.

Evidence for the use of this simple dialysis treatment is currently anecdotal. It might be appropriate to design a clinical trial to determine whether this method has potential in treating this extremely troublesome form of lymphoedema. ❑

Human fasciolosis acquired in an Australian urban setting

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TO THE EDITOR: Although liver flukes (genus, *Fasciola*) are parasites of livestock, human infection is a significant global health problem,[1] albeit seldom seen in Australia.[2] Infected livestock contaminate waterways with parasite eggs, leading to infection of snails that shed metacercariae on to vegetation, such as watercress.[1,3,4] Adult parasites reside in and damage the bile ducts.[1,3] Liver flukes could cause disease if introduced into the food chain.[1]

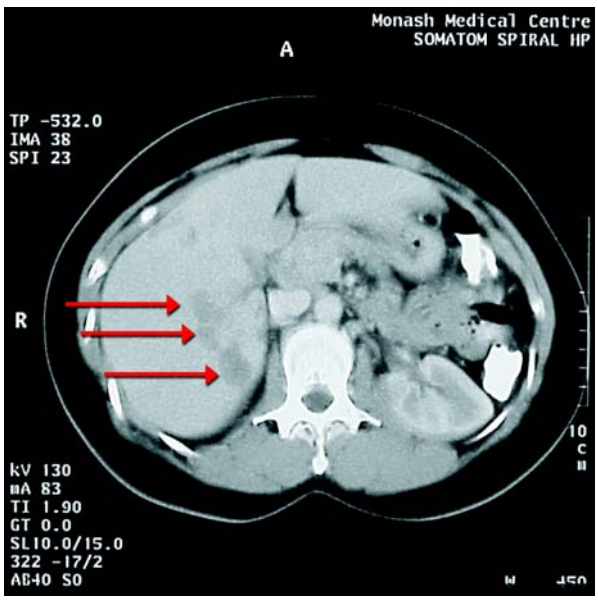
We report the first case in Australia of liver fluke infection (fasciolosis) in a patient with no history of farm or livestock contact. She probably acquired the disease from eating watercress purchased at a Melbourne market four to five months before symptom onset.

A 35-year-old woman presented in August 1998 with fever and right upper quadrant abdominal pain. Blood examination showed eosinophilia (2.5×10^9/L; reference range [RR], $<0.6 \times 10^9$/L]). Liver function tests gave normal results apart from elevated serum aspartate aminotransferase levels of 48 U/L (RR < 41 U/L). Abdominal computed tomography showed multiple low density lesions in the right lobe of the liver, with diameter up to 3 cm (Box). A fine needle aspirate showed no evidence of malignancy. Blood tests four weeks later revealed increasing eosinophilia (3.5×10^9/L) and worsening liver function (serum levels: alanine aminotransferase, 163 U/L [RR, 7–56 U/L]; alkaline phosphatase, 126 U/L [RR, 30–120 U/L]; γ-glutamyl transferase, 98 U/L [RR, 5–45 U/L]).

A parasitic infection was suspected, but four faecal samples and serological tests for hydatids, *Schistosoma*, *Strongyloides* and *Entamoeba* spp. were negative. Coprological diagnosis of fasciolosis can be problematic, as eggs may be released intermittently and in small numbers, especially in low-intensity infection.[1] Enzyme-linked immunosorbent assay (ELISA) using *Fasciola hepatica* antigen, performed at Westmead Hospital, Sydney, was borderline positive. However, ELISA for IgG4 antibodies against recombinant *F. hepatica* cathepsin L5 antigen, performed at Monash University, Melbourne, was strongly positive.

The patient was treated with two doses of triclabendazole (12 mg/kg body weight per dose) on successive days in October 1998. Abdominal pain subsided within two weeks, her appetite was restored, and eosinophil count and liver function normalised within four weeks. Computed tomography two months after treatment showed

Computed tomography of the liver in a woman with fasciolosis



Low density lesions (arrowed) following intrahepatic ductal branches in the right lobe of the liver were consistent with Fasciola *parasites causing biliary obstruction.*

a reduction in size of the liver lesions. The patient remained well six months later.

This case demonstrates that fasciolosis may present to urban medical practitioners in Australia. Ingestion of watercress is an important clue to the aetiology.[2] Serological diagnosis is possible before eggs appear in faeces using a new specific ELISA test that detects the IgG4 response to cathepsin L antigen.[5]

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Community-acquired MRSA bacteraemia: four additional cases including one associated with severe pneumonia

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TO THE EDITOR: Collins and colleagues[1] reported a case of bacteraemic community-acquired MRSA (CAMRSA) infection that they believed to be the first reported in Australia. One of us (GN) published a reference to a case of septicaemia and osteomyelitis in Brisbane caused by CAMRSA in 2000.[2] This severe case occurred in a previously healthy 16-year-old boy with no risk factors for MRSA infection who, after prolonged ventilatory and inotropic support and vancomycin therapy, required a long period of rehabilitation. A further two cases of septicaemia occurred in Ipswich and will soon be published as part of a study of CAMRSA conducted in 2000–2001.[3]

We recently encountered another case involving a previously well 23-year-old man who presented to the emergency department with a large abscess on his upper lip and extensive cellulitis of the surrounding face and neck, and with left-sided pleuritic chest pain and associated fevers and rigors. The patient denied previous antibiotic use or contact with healthcare facilities at any time in the past. There was no history of injecting drug use or trauma. *Staphylococcus aureus* was isolated from blood cultures, and resistance to oxacillin and susceptibility to erythromycin, clindamycin, tetracycline, gentamicin, ciprofloxacin, fusidic acid, rifampicin, and vancomycin was shown. Specimens from operative debridement of the facial abscess yielded *S. aureus* with the same susceptibility pattern. Chest x-rays showed extensive consolidation of the left lower lobe and an associated loculated pleural effusion. Clinical, radiological, and echocardiographic evaluations did not reveal another focus of infection. The patient was treated with intravenous vancomycin for three weeks followed by oral clindamycin, with complete clinical resolution.

It is now clear that CAMRSA infection may result in severe, life-threatening sepsis. The possibility of pneumonia associated with CAMRSA is of particular concern. A 1999 report from Minnesota and North Dakota documented four deaths in children from CAMRSA, including two with necrotising pneumonia.[4] A further two fatal cases of necrotising pneumonia caused by CAMRSA were recently reported from France.[5] The strains involved in all of these cases carry the gene for Panton-Valentine (P-V) leukocidin, a staphylococcal toxin that has been shown to be strongly associated with cases of severe superficial abscesses and necrotising pneumonia.[6]

As the strain of CAMRSA most commonly encountered in Eastern Australia also carries the P-V leukocidin gene (Professor J Etienne, Faculty of Medicine, Claude Bernard Lyon 1 University, personal communication), doctors should be aware of the possibility of severe community-acquired pneumonia caused by this organism.

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Ventricular tachycardia following ingestion of a commonly used antihistamine

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TO THE EDITOR: Kuchar et al[1] describe a patient who received an implantable defibrillator discharge after a single ingestion of loratadine. We are concerned that their conclusion — that this patient "probably" had drug-induced *torsade de pointes* — is incorrect.

Review of the intracardiac electrograms from this patient (shown in Box 2 of their article) with known monomorphic ventricular tachycardia (VT; shown in their Box 1) shows a relatively fixed rate of the VT without the large variations in cycle length consistent with *torsade de pointes*.

While there are no established guidelines for determining *torsade de pointes* based on intracardiac electrograms, it is clear that during monomorphic VT, electrocardiograms can show variability in amplitude and orientation. Consistent with the early stages of monomorphic VT,[2] the first three electrograms have a different orientation compared with the remaining electrograms, which are largely similar. Unfortunately, as the transition from supraventricular rhythm to tachycardia was not shown, it cannot be ascertained whether the tachycardia began with a pause-dependent mechanism, an important criterion to help diagnose *torsade de pointes*.[3] Given that this patient's implantable defibrillator intracardiac electrograms do not show a continually changing electrogram pattern, that the cycle length is relatively constant, and that there is a lack of documented QT prolongation, there is

no evidence of the patient's arrhythmia being *torsade de pointes*.

Incidentally, it is unclear whether these electrograms were recorded before (as specified in the discussion) or after defibrillator discharge (title of Box 2). It is well documented that a defibrillator discharge can have significant effects on the recording of intraventricular electrograms. Most likely, this patient, with documented pre-existing monomorphic VT (their Box 1[b]), had an episode of VT (not *torsade de pointes*) appropriately treated by the implanted defibrillator, probably having no direct relationship with loratadine. Notably, their Box 3 shows *torsade de pointes* in another patient, not receiving loratadine.

In summary, Kuchar et al[1] correctly state that there have been no documented episodes of torsade de pointes after ingestion of loratadine. Similarly, their report does not appear to document an episode of *torsade de pointes*.

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IN REPLY: We agree that there are no guidelines defining *torsade de pointes* based on intracardiac electrograms, but there are several reasons why the likelihood of *torsade de pointes* (as opposed to any other arrhythmia) in our patient is high.

The electrogram shows the arrhythmia just before delivery of direct current shock, this being about 30 minutes after the patient took her first ever dose of loratadine. There are marked variations in electrogram morphology, despite minimal variation in RR interval, in a short strip of recording in this patient with documented QT prolongation. Further, she had no history of monomorphic ventricular tachycardia, no inducible monomorphic ventricular tachycardia at electrophysiologic examination, and no evidence of structural heart disease. Neither was a mechanism for supraventricular arrhythmia identified.

The absence of initiating beats showing pause-dependence is unfortunate, but this is not provided by the generation of device implanted in this patient. Hence, we believe the word "probable" is an apt description for the observation made. ❑

Indigenous health: chronically inadequate responses to damning statistics

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TO THE EDITOR: We welcome Ring and Brown's editorial comment[1] on the Public Report Card 2002 *No More Excuses*,[2] produced by the Australian Medical Association's Task Force on Indigenous Health. We hope that drawing attention to the poor outcomes of Indigenous Australians will catalyse Federal and State governments to take action, particularly as international comparisons demonstrate the likelihood of success.

Australia's poor performance in relation to its Indigenous people is a complex phenomenon, involving political, sociocultural and historical factors, as well as health factors. Levels of ill health among Indigenous communities in post-colonial Australia, Canada and New Zealand are particularly disturbing from a global health perspective, as they persist despite the relative affluence and excellent health status enjoyed by the general population in these nations. One of the difficulties in assessing progress is the lack of high-quality data for comparative purposes.

The types of indicators of Indigenous health in common use in Australia, Canada and New Zealand range from central indicators (such as the age-standardised rate ratios for Aboriginal people) to secondary indicators (such as change in the prevalence and incidence of chronic diseases, like diabetes, in Aboriginal communities). It would be useful to develop additional indicators that more closely reflect Aboriginal community knowledge models and values.[3] Existing indicators emphasise outcomes rather than opportunities for early intervention, such as early childhood development and youth resilience. Finally, there need to be greater attempts to explore how to use and compare international experiences to help Indigenous people most effectively. The Memorandum of Understanding between the Canadian Institutes of Health Research, the Medical Research Council of Australia, and the Health Research Council of New Zealand may provide a framework for international collaboration.[4]

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4. Memorandum of Understanding between the Canadian Institute of Health Research the National Health and Medical Research Council of Australia and the Health Research

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Council of New Zealand on Cooperation on Health Research for Indigenous Populations. Available at http://www.dfait-maeci.gc.ca/aboriginalplanet/resource/canada/mou/mou_aus_new_can-en.asp?prn=1 (accessed December 2002). ❑

Inhaled steroids — too much of a good thing?

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TO THE EDITOR: Our recent study of patients' priorities for asthma care[1,2] provides additional evidence supporting the concerns of Wilson and Robertson in their editorial questioning the possible overuse of inhaled corticosteroids.[3]

We have reported a qualitative study of 62 individuals who presented to an emergency department at either a central city, suburban or rural hospital, in which we explored individuals' perceptions about their asthma, its care and the impact of asthma on their lives.[1,2] We also asked participants to complete a questionnaire on the use of medications and sought to amplify this information by further probing the use of medications in our qualitative data collection.

Of the 82% of participants in our study currently using inhaled corticosteroid medication (51), 30% (16) were taking 1000 μg of fluticasone or equivalent daily and another 19% (10) were taking more than 1500 μg or equivalent. Current product information for fluticasone suggests a maximum dose of 1000 μg twice daily, whereas National Asthma Council (NACA) guidelines recommend that 500 μg fluticasone or equivalent daily may be the upper limit of useful effect.[4,5]

We also asked patients how long their medication lasted. Eleven (18%) stated that inhaled corticosteroid devices lasted three weeks or less. Use above recommended doses did not only occur for inhaled corticosteroids, but also for symptom controller medications. Twenty-four (35%) of the 31 (50%) patients receiving this medication reported that a device lasted three weeks or less, indicating use above usual recommended doses.

Most patients in our study voiced concerns about the cost of asthma and drug side effects, some adjusted their medication use to manage these issues.[1] In such individuals, high use or overuse of preventive and controller medication would increase both costs and side effects, partly explaining these patients' concerns.

Doctors may be overprescribing inhaled corticosteroid medication because there is a discrepancy between dosages recorded in published drug information and newer recommendations for optimal inhaled corticosteroid dose.[4,5] Our findings show that, in some patients, the risks associated with the use of inhaled corticosteroids are likely to be compounded by using them at higher doses than those recommended. Doctors need to be aware of this in managing patients with asthma who have severe symptoms, in whom overuse, rather than underuse, is likely to be a problem.

1. Goeman D, Aroni R, Stewart K, et al. Patients' views of the burden of asthma: a qualitative study. *Med J Aust* 2002; 177: 295-299.
2. Douglass J, Aroni R, Goeman D, et al. A qualitative study of action plans for asthma. *BMJ* 2002; 324: 1003-1007.
3. Wilson J, Robertson C. Inhaled steroids — too much of a good thing? The goal is to achieve optimal asthma control with the lowest effective dose. *Med J Aust* 2002; 177: 288-289.
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Attention-deficit hyperactivity disorder: divergent perspectives

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TO THE EDITOR: Halasz and Vance[1] are correct to point out that there is a diversity of causes that can contribute to a child exhibiting symptoms of attention-deficit hyperactivity disorder (ADHD), as defined in the *Diagnostic and statistical manual of mental disorders* (DSM-IV).[2] In their article, they describe a child who meets the DSM-IV criteria for diagnosis of ADHD and in addition has been affected by environmental factors including poor bonding (due to maternal depression), domestic violence and parental separation. The child also exhibits developmental disability, as exemplified by delayed language development. The message is that, by explaining his symptoms in terms of his early experiences and his developmental disability, a diagnosis of ADHD can be excluded.

Children with ADHD frequently come from families with disharmonious parental relationships. This may be associated with ADHD in one of the parents, perhaps the violent father in the case described.

As clinicians our aim is to ameliorate symptoms as promptly and effectively as possible, and I am frequently impressed by the dramatic improvement that stimulant medication can make to a child's functioning both at school and within the family, with follow-on improvements in mood and self-esteem. Behavioural interventions and family therapy are important adjuncts to medication, but families such as the one described can be difficult to work with and this can limit the effectiveness of such interventions.

A carefully monitored one-month trial of stimulant medication, with behavioural rating scales completed by the class teacher, may be appropriate in cases such as the one described. On the other hand, to deny a child a trial of stimulant medication on the basis of adverse early experiences and developmental disability may be to keep from the child the treatment that would help most.

1. Halasz G, Vance ALA. Attention deficit hyperactivity disorder in children: moving forward with divergent perspectives. *Med J Aust* 2002; 177: 554-557.
2. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4th ed (DSM-IV). Washington, DC: American Psychiatric Association, 1994. ❑

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IN REPLY: We believe the core symptoms of attention-deficit hyperactivity disorder (ADHD) in children reflect a behavioural "final common pathway"

of developmental risk factors,[1] which can include transgenerational associations of core symptoms, as Poulton notes. Current scientific evidence suggests both genetic and environmental contributions, such as verbal and visuospatial executive dysfunction[2] and/or early patterns of attachment deficits.[3] Increased levels of parental psychopathology, associated with (in the child) deficiencies in problem solving, affect regulation, emotional communication and secure attachment, may contribute to the child's symptoms. For this reason, we advocate that medical management be based on a thorough assessment, to ensure that appropriate psychological interventions (eg, parent and teacher management training) are offered alongside psychostimulant medication.

In a recent speech at a scientific meeting of the Faculty of Child and Adolescent Psychiatry, Dr A Mawdsley, a distinguished child psychiatrist, expressed his belief that "prescribing medication in the absence of a careful emotional state assessment is inferior medical practice". He went even further to state that "prescribing medication in the absence of a behavioural modification program should be considered medical negligence".

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2. Vance ALA, Luk ESL. Attention deficit hyperactivity disorder: progress and controversies. *Aust N Z J Psychiatry* 2000; 34: 719-730.
3. Halasz G, Anaf G, Ellingsen P, et al. Cries unheard. A new look at attention deficit hyperactivity disorder. Altona: Common Ground, 2002: 75-91. ❑

Doctor shoppers' rights: privacy or lunacy?

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TO THE EDITOR: I wish to draw attention to a draconian anomaly in the *National Health Act 1953* (Cwlth).

All GPs encounter patients "shopping" for narcotics and/or tranquillisers. The Doctor Shopper phone line (which enabled GPs to rapidly obtain information from the Health Insurance Commission to identify non-genuine patients) was a boon in guiding GPs' management of such situations. Concern over the new private sector amendments to the *Privacy Act 1988* (Cwlth) led to an examination of the legal standing of the Doctor Shopper phone line, and it has now been cancelled.

Concerned GPs are now limited to requesting that a "patient" sign a voluntary release of their Pharmaceutical Benefits Scheme record. This tells the "patient" that they have been rumbled, and they move on to the next practice on their list.

If they have signed the Privacy Release Form, then the GP will receive a printout of the drugs they have received under the Pharmaceutical Benefits Scheme in the previous six months. This is accompanied by a letter informing the doctor that he or she "cannot make a record of, divulge or communicate to any person, any information with respect to the affairs of the person whose information has been released. To do so attracts a penalty of $5000 and/or imprisonment for a period not exceeding two years".

So, under the provisions of the National Health Act (subsection 135A), even putting this information in the medical records of a multidoctor practice would appear to be illegal. It is clearly illegal to warn other doctors outside the practice. There is no corresponding legislation which affects doctor shoppers. So the "right-doers" can finish up in jail, while the "wrong-doers" can, with impunity, continue to play their dissembling, time-consuming, and sometimes harassing, games. ❑

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