LETTERS

Sustained increase in infectious syphilis notifications in Victoria

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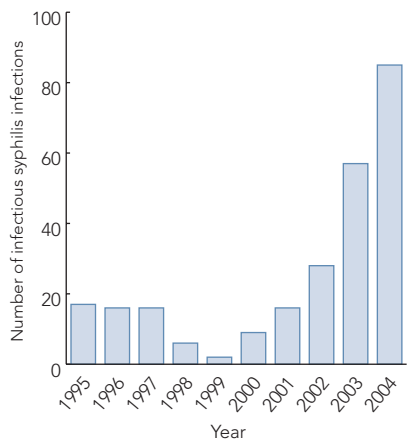
To the Editor: In Victoria, notifications of infectious syphilis infection (primary, secondary and early latent [< 2 years' duration]), reported by the Department of Human Services, have increased more than fivefold in the past decade, from 16 in 1995 to 85 in 2004 (Box). An increase in notifications has also been observed in Sydney.[1] Whereas previously in Victoria, very few infectious syphilis notifications were reported among men who have sex with men (1 of 16 cases in 1995), in 2004, 63 of a total of 85 cases (74%) were in this group (68% of these were acquired in Victoria).

The Victorian Infectious Diseases Reference Laboratory (VIDRL) conducts testing for sexually transmitted infections (STI) and HIV for three Melbourne sexual health clinics with a high proportion of patients who are men who have sex with men. In 2004, 62 male patients tested positive for infectious syphilis, and 40% of these were HIV-positive. This is similar to the situation in Sydney, where in 2003, 54% of infectious syphilis cases were reported among HIV-positive men who have sex with men.[1]

Syphilis outbreaks among men who have sex with men have been reported elsewhere in recent years. There was an outbreak of syphilis in this group in Greater Manchester; between 1999 and 2002, and 37% of cases were in HIV-positive men.[2] In this population, syphilis infection was associated with unprotected oral sex with high numbers of partners, seeking sexual partners at venues (darkrooms, cruising areas and saunas) and use of drugs (GHB [gamma hydroxybutyrate] and poppers [amyl nitrate]).[3] A San Fransisco study in 2000, performed in response to a syphilis outbreak among men who have sex with men, reported that meeting sexual partners through use of the Internet was a factor significantly associated with syphilis infection.[4]

It is likely that some or all of the factors reported in these outbreaks overseas are contributing to the sustained increased in infectious syphilis notifications in Victoria, but it is important to have local data to ensure interventions are targeted appropriately and cost effectively. In Victoria, responses to the increase in syphilis notifications have already included an alert to general practitioners to encourage men who have sex with men to have syphilis testing and individual counselling, and syphilis testing of men who have sex with men at a popular sex-on-premises venue over a 4-week period. Depending on further studies in this population in Victoria, other responses could include enhancing outreach at Internet chat rooms, intensive counselling of HIV-positive men who have sex with men, and education interventions such as peer-led community-based strategies for countering unsafe sex and substance-use behaviours. Finally, it is vital that interventions are multidisciplinary, collaborative and evidence-based.

Infectious syphilis notifications by year, Victoria



Data from the Notifiable Infectious Diseases Surveillance System Database, Communicable Diseases Section, Victorian Department of Human Services.

Competing interests: None identified.

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Locally acquired lymphogranuloma venereum in a bisexual man

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To the Editor: Lymphogranuloma venereum (LGV) is an uncommon sexually transmitted infection caused by *Chlamydia trachomatis* serovars L1–3. LGV is not endemic in Australia, and rare Australian cases of LGV have been seen in patients who have either acquired the infection while travelling overseas in an endemic area, or have had local contact with an imported case. Currently, there is an outbreak of LGV in western Europe (in particular, The Netherlands) and the United States.[1-4] A case of LGV in an Australian man with no history of overseas travel was managed recently.

A 42-year-old bisexual man with previously treated early syphilis and hepatitis C infection presented to a Melbourne hospital in August 2004 complaining of 3 months of tender right inguinal lymphadenopathy. An excisional biopsy showed the formation of necrotising granuloma indicative of LGV. He had no history of penile ulceration, urethritis or proctitis. The surgical wound healed normally. The patient gave a history of attending sex-on-venue premises ("gay saunas") and "beats". He reported having oral sex with men, and recently having non-insertive sex involving masturbation with an unknown casual male contact who was apparently an overseas visitor. The patient had a female sexual partner with whom he had irregular, unprotected vaginal intercourse.

The diagnosis of LGV was confirmed by polymerase chain reaction (PCR), which detected *C. trachomatis*, identified as serovar L2 by nucleotide sequencing, from the excised lymph gland. IgG and IgA antibodies to *C. trachomatis* were demonstrated by enzyme-immunoassay. Tests for other active sexually transmitted infections were negative. The patient was treated with doxycycline (100 mg twice daily for 3 weeks). His asymptomatic female partner was also treated.

LGV is endemic in developing countries in our region, but occurs only sporadically in industrialised countries. The first stage of disease consists of a papule or ulcer that may occur on the penis, urethra or cervix. Proctocolitis may also be present, mimick-

Histological section of the lymph node showing the thickened node capsule and necrotising granuloma

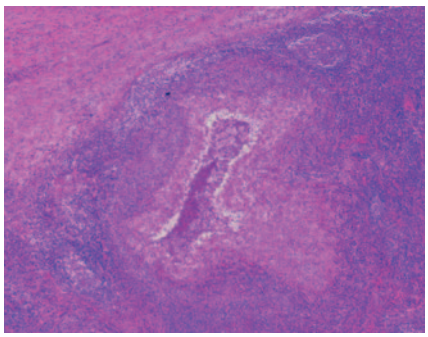

Image courtesy of Dr Malcolm Buchanan, Department of Anatomical Pathology, Royal Melbourne Hospital.

ing inflammatory bowel disease. Regional lymphadenopathy develops in the secondary stage of disease when there may be systemic symptoms. Fistula formation at these sites can be prevented by early recognition and treatment. Late, severe genital ulceration is rarely seen.

Confirmation of a diagnosis of LGV requires showing *C. trachomatis* serovars L1–3 by serological tests or PCR on genitourinary specimens. Lymph node resection is not favoured because of the possibility of sinus formation. Prolonged treatment with doxycycline or roxithromycin for 3 weeks is required for affected patients. Asymptomatic contacts are treated with doxycycline for 1 week or a single dose of azithromycin.

This case of locally acquired LGV highlights the features of this progressive disease that may now be recognised more frequently in Australian men who have sex with European or North American men.

Competing interests: Damon Eisen receives support from the Clinical Centre for Research Excellence in Infectious Diseases, Victorian Infectious Diseases Service, Royal Melbourne Hospital.

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Spontaneous bruising, haematomata and prolonged APTT with meloxicam

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To the Editor: A 47-year-old woman presented with a 1-week history of spontaneous prominent and painful bruising and haematomata, 5–6 weeks after commencing meloxicam 15 mg daily for osteoarthritis and plantar fasciitis. The bruises varied in size from 2 cm × 2 cm to 3 cm × 4 cm. She had a past history of acne rosacea, for which she was taking clonidine 100 μg daily, and reflux oesophagitis, for which she was taking pantoprazole 40 mg daily.

Meloxicam, being the only new drug taken by the patient, was suspected as the causative agent and was therefore discontinued. Activated partial thromboplastin time (APTT) at presentation was 58 s (reference range, 22 s–38 s). A week after stopping meloxicam, it had fallen to 37 s. All other haematological parameters, including platelet count and international normalised ratio, were normal, as were renal and liver function. New bruises and haematomata stopped appearing 2 days after the patient stopped taking meloxicam. She continued taking clonidine and pantoprazole.

Meloxicam is a selective nonsteroidal anti-inflammatory drug (NSAID) (a cyclo-oxygenase 2 [COX-2] inhibitor). It was chosen for this patient in view of her reflux oesophagitis and because she had already been unresponsive clinically to one of the other COX-2 inhibitors. Unlike non-selective NSAIDs, meloxicam has been shown to have negligible effect on platelet function as measured by bleeding time.[1-3] Rinder et al[4] reported no prolongation of APTT or prothrombin time after 8 days of regular administration of meloxicam at 7.5 mg, 15 mg or 30 mg.

In spite of these contrary findings, I believe the prolongation of APTT and spontaneous bruising and haematomata in this patient were directly attributable to meloxicam, as the bruises disappeared 2–3 days after stopping the drug (with no other changes in the patient's existing medication) and a repeat APTT a week after cessation of the drug was normal.

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A potential link between magnesium intake and diabetes in Indigenous Australians

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To the Editor: Diabetes in Indigenous Australians occurs at a younger age and at almost four times the rate in non-Indigenous Australians. The age-adjusted prevalence of diabetes among Indigenous people is 16% in remote areas and 9% in non-remote areas, with the actual prevalence estimated to be between 20% and 25%, and possibly higher than 30% in some remote areas.[1] The cause for this disparity in diabetes incidence is multifactorial, and recent evidence suggests that nutrition — particularly magnesium intake — may play a role.

Although central obesity remains a major risk factor, magnesium deficit has been posited to be an underlying common mechanism for the insulin resistance found in type 2 diabetes, as well as in metabolic syndrome, hypertension, and impaired glucose tolerance.[2] The clinical correlations between low magnesium and diabetes have been well documented,[3] with serum magnesium deficits being reported in 25%–39% of diabetic

outpatients in the United States and Switzerland, and up to 73% of diabetic outpatients in Mexico.

With magnesium deficits being observed in diabetes, studies examining the effects of magnesium-rich foods on diabetes risk become relevant. The Nurses' Health Study and the Health Professionals' Follow-up Study, which included 85 060 women (18 years follow-up) and 42 872 men (12 years follow-up), demonstrated that, after adjusting for confounding variables, a magnesium-rich diet reduced the relative risk of developing diabetes by 34% in women and 33% in men.[4] A similar inverse correlation between magnesium intake and diabetes risk was shown in the Iowa Women's Health Study with a cohort of 35 988 older women,[5] and in the Honolulu Heart Program and the Women's Health Study with cohorts of 8006 men and 39 345 women, respectively.[6,7]

Despite this growing body of evidence supporting the involvement of magnesium in diabetes, consideration of magnesium status has not been integrated into Australian medical care for diabetes, and more specifically, for Indigenous Australians. It is known that the traditional diet of hunter-gathers such as Indigenous Australians was much more nutrient- and magnesium-rich than the current estimated Australian intake.[8] Nonetheless, there remains a lack of information about current magnesium status, including dietary intake, in Indigenous Australians.

It is possible that dietary magnesium intake may be too low to maintain normal serum magnesium homoeostasis, and that this might contribute to the development of type 2 diabetes. Further research into this issue may provide this information.

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Venous thromboembolism: diagnosis and management of pulmonary embolism

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To the Editor: The clinical update on venous thromboembolism by Lee and colleagues advises that "Ventilation perfusion (V/Q) isotope scanning reliably establishes the diagnosis of PE [pulmonary embolism] if the V/Q features suggest a high probability of PE…".[1] Although this is probably true for patients with intermediate or high pretest probability, a discordant result (low pretest probability and high probability V/Q) should be regarded with suspicion.

From the original PIOPED data, high probability V/Q was predictive of angiographically confirmed PE in 80% of patients,[2] which drops to 56% by Bayesian analysis if the pretest probability is low. False positive results may be due to previous PE or unrelated parenchymal lung disease. There is significant potential morbidity associated with a false positive result for PE, both from the acute anticoagulation and for future presentations with PE-type symptoms, where PE will be accorded a higher probability because of the previous documented diagnosis.

As Lee and colleagues also state, D-dimer testing must be combined with an estimate of pretest probability to be useful. They advocate excluding PE on the basis of low pretest probability and negative D-dimer result. However, a negative D-dimer result (rapid enzyme-linked immunosorbent assay [ELISA] type) may be used to exclude PE in intermediate as well as low probability patients.[3] This is dependent on the type of assay available as well as the local PE prevalence, and local guidelines should therefore be developed.

1 Lee CH, Hankey GJ, Ho WK, Eikelboom JW. Venous thromboembolism: diagnosis and management of pulmonary embolism. *Med J Aust* 2005; 182: 569-574.

2 PIOPED Investigators. Value of the ventilation/perfusion scan in acute pulmonary embolism: results of the prospective investigation of pulmonary embolism diagnosis (PIOPED). *JAMA* 1990; 263: 2753-2759.

3 British Thoracic Society Standards of Care Committee Pulmonary Embolism Guideline Development Group. British Thoracic Society guidelines for the management of suspected acute pulmonary embolism. *Thorax* 2003; 58: 470-484. ❑

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To the Editor: I read with interest the article by Lee et al regarding the investigation and treatment of pulmonary embolism (PE).[1] The investigation of patients presenting with PE as a diagnostic possibility is of great interest to emergency physicians, and such presentations are a daily occurrence in emergency departments around the country.

Unfortunately, only a small amount of text is devoted to describing the relative merits of ventilation perfusion (V/Q) scanning and computed tomography pulmonary angiography (CTPA), and no guidance is provided as to which is the test of choice when both are available. The British Thoracic Society has recommended CTPA as the lung imaging modality of first choice for patients presenting with non-massive PE.[2] There is a large and increasing body of evidence that CTPA provides superior specificity to V/Q scanning in the detection of PE. CTPA also provides the opportunity of establishing diagnoses other than PE and, in addition, a negative multi-slice CTPA is of sufficient sensitivity to enable the withholding of anticoagulation.[3] It is also my experience that CTPA is easier to obtain out of hours, compared with V/Q scanning.

The authors state that V/Q scanning "reliably establishes the diagnosis of PE if the V/Q scan features suggest a high probability of PE…".[1] Unfortunately, this statement is incorrect. It is essential that V/Q scan results be interpreted in the light of the patient's clinical probability for PE. In the PIOPED study, only 56% of patients with high probability V/Q scan reports had pulmonary embolism if the pretest probability was low.[4]

No mention is made of the special situation of pregnant women presenting with pleuritic pain, or which lung imaging test is considered "safest" for both mother and baby. Although the risks of PE are generally agreed to be increased in pregnancy, it is my experience that pregnant women are extremely reluctant to undergo any form of diagnostic investigation that exposes the fetus to radiation.

The Wells criteria have been validated for the assessment of PE in emergency department patients only, and provide a means for clinicians with little experience to make an accurate assessment of an individual patient's clinical probability of PE.[5] Once initiated, clinical assessment of the patient with possible PE is straightforward. The key question facing emergency physicians is this: is there a group of patients that have such low probability for PE that no investigation at all is required?

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In reply: Pulmonary embolism (PE) remains a complex diagnosis despite the availability of validated prediction models and D-dimer testing to direct the need for diagnostic imaging.

We agree with Bailey that the ability to exclude the diagnosis of PE on clinical grounds in patients with a low pretest probability is highly desirable. Unfortunately, clinical features lack sensitivity and specificity for the diagnosis of PE, and clinical prediction models, laboratory investigations, and diagnostic imaging are likely to remain an integral part of the clinical work-up.

As suggested by Bragg, it may be possible to simplify the diagnostic approach by using a highly sensitive D-dimer assay, and simplified pretest probability models have been proposed. However, this may come at a cost of reduced specificity,[1] which leads to unnecessary diagnostic imaging studies and thus limits the clinical utility of these approaches. Further improvements in the diagnostic approach to PE are clearly needed.

There are emerging data demonstrating the accuracy of computed tomography pulmonary angiography (CTPA) for the diagnosis of PE. However, CTPA has limitations (a large contrast load, high radiation dose, and lack of sensitivity of first generation scanners for small thrombi[2]), some of which are evident in the recently published validation study referred to by Bailey:[3] 25% of screened patients with suspected PE were not eligible for this study because of renal impairment, a contraindication to CT, or other reasons.

The diagnostic algorithm that we provided in our review suggests that either ventilation perfusion (V/Q) scanning or CTPA can be used for patients with suspected PE who require diagnostic imaging,[2] with the choice determined by patient factors and availability.

The diagnosis of PE during pregnancy is challenging because of concerns about radiation exposure and uncertainty about whether CTPA or V/Q delivers more radiation to the fetus.[4] Furthermore, clinical decision rules have not been validated in pregnancy. However, recommendations from experts and professional bodies suggest that V/Q scanning can be used in combination with compression ultrasound to establish or exclude the diagnosis of PE during pregnancy in most cases with minimal fetal radiation exposure.[5,6]

The comments by Bailey and Bragg concerning the interpretation of high probability V/Q scan results highlight the pitfalls of performing diagnostic imaging without considering the patient's pretest probability of PE. Although a high probability V/Q scan is diagnostic in patients with a moderate or high pretest probability of PE (prevalence of disease $\geq 90\%$), the prevalence of disease is only about 50% in those with a low pretest probability.[7,8] Therefore, V/Q scanning should not be performed in patients with a low pretest probability *unless* the D-dimer test is positive. In this situation the algorithm for moderate or high pretest probability should be followed,[2] and a high probability scan reliably establishes the diagnosis.

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Screening for venous thrombosis by ultrasonography before hospital discharge after major joint surgery

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To the Editor: In a recent editorial, Gallus estimates the cost of doing ultrasonography in all patients after unilateral hip or knee replacement, with further testing in the 9% or 26% of patients, respectively, found to have deep vein thrombosis (DVT), to be about $200 000 per 1000 patients.[1] We agree.

He then states, "Many would argue that extended prophylaxis is likely to be the simplest, cheapest and perhaps safest solution".

However, prophylaxis is also expensive. Subcutaneous enoxaparin 40 mg administered daily for 30 days costs $170, or $170 000 per 1000 patients.[2]

In our study, we found DVTs in 1086 of 5999 patients (18.1%) before discharge,[3] so that extended prophylaxis would involve 81.9% of patients receiving prophylactic doses of anticoagulants, with the risk of unwanted bleeding, despite the absence of DVT on ultrasound at Day 7 postoperatively.

In addition, if an ultrasound scan was not done before discharge, the 18.1% of patients

with a DVT would receive only prophylactic (not therapeutic) doses of anticoagulant for their DVT.

We plan a further study to check the prevalence of post-discharge DVT by repeating ultrasonography at 90 days postoperatively in patients without DVT on ultrasound at Day 7. We suspect the prevalence is lower than suggested in the literature, as the data on late presentation of DVTs have been obtained by retrospective study of the number of patients re-admitted to hospital with DVT.

Finally, on the question of whether performing ultrasonography on all patients has clinical benefit, we concur with Gallus when he writes that "Logic suggests it should…".

Competing interests: Richard O'Reilly has received speaker fees and travel assistance to attend meetings from AstraZeneca.

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In reply: O'Reilly and colleagues belatedly address the need to consider bleeding risk and costs when choosing between management routines designed to prevent venous thrombosis and pulmonary embolism. Their otherwise valuable article[1] failed to record bleeding rates when patients (almost 17%) with subclinical calf-vein thrombosis were exposed to therapeutic (not prophylactic) anticoagulant dosages. Nor did they evaluate the dollar and manpower costs of their complex management routines.

Present evidence-based international guidelines from the Seventh ACCP (American College of Chest Physicians) Conference on Antithrombotic and Thrombolytic Therapy recommend effective prophylaxis for at least 10 days in all patients having hip or knee replacement, extending to 28–35 days after hip replacement.[2] The ACCP guidelines also recommend against routine use of ultrasound screening because it is "neither clinically effective nor cost effective".[2] This is a Grade 1A recommendation from the ACCP ("Grade 1" implies certainty "that the benefits do, or do not, outweigh the risks, burdens, and costs"; "Grade A" refers to recommendations based on "randomized clinical trials with consistent results [that] provide evidence with a low likelihood of bias").[3] To reverse this recommendation would require randomised comparisons between routine prophylaxis alone or routine prophylaxis supplemented by screening ultrasonography — powered to permit meaningful measures, in both groups, of thromboembolism rates, bleeding rates and costs. Routinely screening for subclinical thrombosis after major joint surgery should not be done outside suitably designed clinical trials until such evidence is available. The role of logic in medicine is to generate hypotheses, which must then be tested by clinical trial. Unfortunately, evidence derived from uncontrolled cohort studies remains limited to Grade C (based on "observational studies or [on] generalization from one group of patients included in randomized trials to a different, but somewhat similar, group of patients").[3]

Competing interests: The author has received consulting fees for participating in clinical trial steering committees (from Sanofi, Bristol-Myers Squibb, Bayer and Organon) and expert committees (from AstraZeneca, Bayer and CSL).

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Familial hypercholesterolaemia: a look back, a look ahead

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To the Editor: In their editorial, Burnett and colleagues correctly emphasise the importance and cost-effectiveness of cascade family screening in the early diagnosis of familial hypercholesterolaemia (FH) among relatives of known cases. They point out that Australia does not have "a national program for detecting the vast majority of patients with FH in our community…".[1]

Such an approach has been advocated since the 1980s by the international MEDPED-FH project, initiated in Utah to raise public and professional awareness of the need to detect and treat FH at an early age (MEDPED-FH stands for Make Early Diagnosis to Prevent Early Deaths in Familial Hypercholesterolaemia).[2] Since then, the project has spread to over 30 countries, including Australia, and has over 25 000 patients with FH registered worldwide.[3]

In Australia, the MEDPED-FH program has registered about 700 patients with FH, about 2% of the estimated 33 000 patients overall.[4] This proportion is similar to registrations in many other countries (including the US). Only the Netherlands, Denmark and Finland, where government-financed screening programs are in place, have higher proportions of registrations, with 10%–50% of patients with FH registered with MEDPED-FH. Also, in these countries, DNA detection of low-density lipoprotein cholesterol receptor gene mutations is used routinely for the diagnosis of FH.

At present in Australia, nurse practitioners are performing FH cascade screening in collaboration with MEDPED-FH physicians in each capital city, supported by Pfizer Australia. Further work on FH is being carried out by Associate Professor David Sullivan and colleagues in Sydney, supported by the Western and Central Sydney Area Health Services.

In addition, the Cardiac Society of Australia and New Zealand has established a working party to investigate cardiac genetic disorders, including FH, and is involved in further education among cardiologists regarding screening, diagnosis and treatment of FH.

But these efforts are not enough. I support Burnett and colleagues in recommending the establishment of a nationwide screening program for FH, and also recommend that DNA diagnosis be incorporated as an essential component. There is a definite cost benefit of early detection and treatment with statins of patients with FH.

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In reply: The international MEDPED-FH project has made a major contribution towards introducing family-based cascade screening into the Australian health care system. Although these achievements are important, we agree with Hamilton-Craig's view that they are dwarfed by the magnitude of what now has to be done to deliver to all at-risk relatives the health gains that can be achieved by a proactive program of population screening.

The risk of familial hypercholesterolaemia (FH) for a first-degree relative of an affected index case is 250 times greater than the risk for a member of the general population. The relative cost-efficiency of family-based cascade genetic screening is high, compared with population-wide screening for a dominantly inherited disorder.[1,2]

We disagree with Hamilton-Craig that DNA diagnosis is an essential component of an effective screening program. Among FH-affected families, biochemical testing has a sensitivity of 95%, and a specificity of 96%.[3] The additional diagnostic gain from DNA testing in the context of screening relatives at 50% prior risk is marginal, although of use in determining with certainty whether or not a relative has inherited the family-specific trait.

FH offers a paradigm of best clinical practice for improving health care outcomes for a widening range of "monogenic" disorders with complications that can be avoided or reduced by focused health care provision. The time has come when physicians, cardiologists, paediatricians, biochemists, geneticists, public health physicians, general practitioners and those administering the funding of health care delivery in Australia come together and formulate the changes necessary to allow cascade genetic screening for FH to become part of routine health care.

1 Krawczak M, Cooper DN, Schmidtke J. Estimating the efficacy and efficiency of cascade genetic screening. *Am J Hum Genet* 2001; 69: 361-370.
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A picture of Australia's children

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To the Editor: I am prompted to write to you in response to a recent *MJA* editorial.[1] It amazes me that the health sector in Australia, as I think the editorial did, continues to largely ignore the magnitude of the problem of injury in our children. This is despite clear evidence of the excess ill-health burden that injury places on our children, according to the Australian Institute of Health and Welfare (AIHW) report (the subject of the editorial)[2] and other reports.[3-5]

Having said this, the editorial did highlight a very pleasing trend — there has been a steady decline in injury deaths in later childhood. Unfortunately, however, this was the only mention of injury in the editorial, and readers could be forgiven for thinking that this is the end of the story: the injury death rate is declining; therefore, we are doing all we can, and injuries are not a major issue. Nothing could be further from the truth. Our children continue to die from road and drowning accidents and will do so until injury prevention is recognised as paramount.

The AIHW report clearly states that the single highest cause of death in children remains injury and poisoning.[2] Accordingly, trauma is the single highest contributor to premature mortality and years of potential life lost of any health condition in Australia. If we don't develop new approaches to reducing the incidence of drownings and road deaths, in particular, we will not see further declines in injury-related death rates, and injury will continue to rate highly as a killer of young people.

Importantly, injuries do not only kill young people — they also hospitalise and maim them. The second most common reason for hospitalisation in Australian children is injury.[2] Unlike injury deaths, there has been no trend in the rate of hospitalisation for injury. Across age groups, there appears to be a shift from fatalities to an increasing number of people with a high lifetime burden of significant disability, including brain and spinal cord damage. Imagine what this does to the quality of life and life expectancy of a child. How many of these children will be able to lead physically active lives?

It is time for the health sector, particularly public health agencies, to properly recognise injury as a critical issue for the ongoing health of Australian children and to formally commit to appropriate preventive actions, commensurate with the priority ranking of childhood injuries.

1 Patton GC, Goldfeld SR, Pieris-Caldwell I, et al. A picture of Australia's children [editorial]. *Med J Aust* 2005; 182: 437-438.
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In reply: There is little to disagree with in this excellent summary of injury morbidity and mortality in Australian children. However, the principal point of our editorial[1] was to highlight important problems where adequate data are currently unavailable.

The Australian Institute of Health and Welfare report was able to give extensive coverage to injuries and accidents in children.[2] Indeed, seven indicators specifically addressed aspects of childhood injury, with a range of others (eg, child abuse and neglect, neighbourhood safety) addressing relevant aspects of the family and social context. This emphasis reflected not only the importance of childhood injury, but the extent to which reasonably good data are available.

We agree that, despite some favourable mortality trends, the burden of childhood injury remains high, as are associated health care costs. However, childhood injury is an area where advocacy has translated into action.[3] One of the reasons for the success of that advocacy has been the availability of

sound data, both to make the case and to ensure an appropriate focus in policy responses.[4] While there is undeniably much more to do, we can learn much from injury prevention about how to tackle the newly emerging problems of childhood.

1 Patton GC, Goldfeld SR, Pieris-Caldwell I, et al. A picture of Australia's children [editorial]. *Med J Aust* 2005; 182: 437-438.
2 Australian Institute of Health and Welfare. A picture of Australia's children. Canberra: AIHW; 2005. Available at: http://www.aihw.gov.au/publications/index.cfm/title/10127/ (accessed Jul 2005).
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Physical examination: bewitched, bothered and bewildered

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To the Editor: Reilly et al draw attention to the lack of research on the impact of the findings of physical examination on patient care, and state that, in the United States, many doctors "do not know how to do it, and do not see why they should".[1] Should we continue to teach these skills in Australia?

Experience tells us, even if research does not, that the presence or absence of one or more physical findings may make a crucial difference to patient care. The important thing for teachers is that, while students learn the rituals, we should encourage them to think critically about what they are doing. They focus on passing the next objective structured clinical examination (OSCE), which often requires them to carry out a ritual. They are vaguely aware they will be required to think selectively about real patients and the necessity to make a diagnosis, but have little practice in selecting appropriate physical examination. We who teach them should formally recognise the distinction between ability to perform and ability to choose and interpret physical signs.

I learned an inductive process: take a history and perform a complete examination, and then engage the brain. Research would not tolerate such a "fishing trip"! The inductive method has been replaced by a hypothetico-deductive approach. This is criticised, but all doctors take intelligently selected short cuts. We should acknowledge this and critically examine the skill.

Students must learn systematic examination. This is the repertoire from which they choose when faced with a clinical problem. OSCEs early in the undergraduate course should assess their competence in this, and students should know exactly what they are to do. Our bedside clinical teaching and later OSCEs should explicitly challenge students to consider the selection of relevant clinical examination required in the light of the history, just as they should consider the value of all other tests, rather than using a blunderbuss approach.

Together, history and physical examination have two objectives. One is diagnosis; the other defines or excludes comorbidity. Without them, medical care cannot begin to function effectively or economically.

Neither I nor your authors are bewitched; their anecdotes make this clear, and they define the reasons for the lack of research. A focus on considering what can, and cannot, be gained by selective physical examination should reduce our own and our students' bother and bewilderment.

1 Reilly BM, Smith CA, Lucas BP. Physical examination: bewitched, bothered and bewildered [editorial]. *Med J Aust* 2005; 182: 375-376. ❑

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