LETTERS

The switch to new conjugated vaccines may compromise immunisation coverage for refugees

Christine B Phillips and Mahomed Patel

To the Editor: On 1 November 2005, the Australian states and territories introduced quadrivalent, pentavalent or hexavalent vaccines for childhood immunisations. This simplifies vaccination for young children, but may impair the ability of health services to provide primary immunisation for refugees over the age of 8 years.

The Australian refugee and humanitarian program targets refugees from many countries that have poor primary health infrastructure. In the 2004–05 financial year intake, at least 75% of the 12 096 entrants under the offshore resettlement program came from countries that had immunisation coverage rates below 50% in the 1990s.[1,2] Adolescents and adults from these countries generally have patchy vaccination histories and no records. According to Australian guidelines, they warrant full catch-up vaccination, often involving a primary vaccination course.[3]

Primary vaccination against tetanus, diphtheria and pertussis requires three doses of vaccine. The dose of diphtheria toxoid in vaccines for children or adults over 8 years of age is significantly lower than in early childhood preparations because of potential adverse effects. In 2004, the conjugated pertussis–adult diphtheria–tetanus vaccine for adolescents (Boostrix, GlaxoSmithKline) was introduced to the immunisation schedule to provide boosters against pertussis, diphtheria and tetanus. However, Boostrix has no proven efficacy for primary vaccination against pertussis and is not recommended for adolescents and adults who have no primary cover against pertussis.[3,4] As monovalent pertussis vaccine is not available, refugees over the age of 8 years cannot be provided with a primary vaccination course against pertussis.

Adult diphtheria–tetanus vaccination (ADT) is the most-used primary vaccine for refugees over the age of 8 years. After the introduction of Boostrix, many state and territory health departments reduced their supply of ADT to immunisation providers. Some refugee health services have attempted to meet demand for ADT by collating individual doctors' stocks provided under the Emergency Drug (Doctors Bag) supplies section of the federally-funded Pharmaceutical Benefits Scheme, which provides for up to 15 doses of ADT per month. But this is a cumbersome and unsustainable strategy. Some jurisdictions, such as the Australian Capital Territory, supply ADT directly to refugee health service providers.

All the new polyvalent childhood vaccines include inactivated polio vaccine. Unless states and territories procure monovalent polio vaccine, primary vaccination against polio for people over 8 years will remain inadequate.

People from refugee backgrounds warrant the same level of protection against vaccine-preventable diseases as other Australians. The level of protection may be reduced by failure to provide suitable vaccines. We encourage state and territory health departments to stock sufficient vaccines for adult and adolescent refugees, including ADT and monovalent inactivated polio vaccine. We also recommend that the Australian Technical Advisory Group on Immunisation provide detailed advice on the needs of refugees when crafting immunisation guidelines.

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Leprosy: an uncommon infection with varied presentations

Sebastiaan J van Hal and Bernard J Hudson

To the Editor: Leprosy rates in Australia are low (less than one case per million population)[1] and predominantly occur in Indigenous Australians and immigrants from leprosy-endemic areas.[2]

A 21-year-old pregnant Burundian woman had migrated to Australia in 2005 from a refugee camp in Tanzania. In the year before her arrival, she had received intermittent courses of steroids for an undefined illness characterised by fever, nightsweats and painful symmetrical peripheral polyarthritis. Three months after arriving in Australia, the patient presented to a rural hospital with a recurrence of the previous symptoms.

The symptoms improved on recommencement of prednisolone treatment. The patient was transferred to the Royal North Shore Hospital, where examination revealed bilateral peripheral sensory neuropathy (confirmed by nerve conduction studies); bilateral, enlarged, tender ulnar nerves; and tender hyperpigmented 2–3 cm nodules on the upper arms, but no other skin lesions or infiltrations. Skin biopsy revealed features consistent with erythema nodosum leprosum (ENL), but no acid-fast bacilli (AFB) were detected. Slit-skin smears were also negative for AFB. Leprosy was confirmed by histopathological examination of a sural nerve biopsy, which showed AFB and granulomatous changes of leprosy. The patient commenced multidrug therapy for multibacillary leprosy, with prednisolone for ENL.

Leprosy is a chronic granulomatous infection of skin and peripheral nerves with *Mycobacterium leprae*. Host immune responses determine the spectrum of clinical presentations. Leprosy is classified into either multibacillary disease (≥ 6 skin lesions and/or skin smears positive for AFB) or paucibacillary disease (<6 skin lesions, with no bacilli on skin smears).[3] Type 1 (reversal) reactions are delayed-type hypersensitivity reactions and manifest as neuritis and increased inflammation of pre-existing skin lesions. Type 2 reactions (ENL) are a systemic response to immune complex deposition and manifest with multiple tender nodules, fevers, neuritis, arthritis and iritis.[4,5] ENL occurs exclusively in multibacillary disease in 10%–20% of patients, and

negative slit-skin smears (as in our patient) are unusual. Possible explanations include undisclosed diagnosis and treatment of leprosy in Tanzania or the combination of steroid therapy and immune changes that occur during pregnancy.[6] Multidrug therapy is well established and regarded as safe for pregnant women.

Diagnosis of infections that are uncommon in Western countries, especially leprosy, is often delayed.[7] For refugees living in remote areas, access to expertise and support may be limited. Therefore, doctors, especially those involved in refugee health, should be aware of "exotic" infections and their varied presentations. Furthermore, effective referral networks should be encouraged, as this resulted in a swift positive outcome in our case.

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Vigilance is required for Australia to remain polio free

Bruce R Thorley, Kerri Anne Brussen, Elizabeth J Elliott and Heath A Kelly

To the Editor: Australia and the other member nations of the World Health Organization's Western Pacific Region were declared free of circulating endemic poliovirus in 2000, although the last case of endemic polio in Australia occurred in the 1970s.[1] Nevertheless, the low risk of vaccine-associated paralytic poliomyelitis (VAPP) persisted through the continued use of the Sabin live attenuated oral polio vaccine (OPV) until it was replaced by the Salk inactivated polio vaccine in the National Immunisation Program from 1 November 2005.[2]

Despite the eradication of indigenous wild poliovirus and the removal of the risk of VAPP, Australia cannot afford to be complacent with surveillance for cases of poliomyelitis. Polio is a highly infectious disease and is quickly spread through international travel. All countries risk importation of wild poliovirus from the four remaining endemic countries (Afghanistan, India, Nigeria and Pakistan) — as occurred in Indonesia and 11 other countries during 2005.[3] Until the latest outbreak, involving over 300 cases, Indonesia had not reported a single case of poliomyelitis since 1995. Genetic sequencing of the wild polioviruses from Indonesia determined that they originated in Nigeria and were related to strains isolated in Sudan, Saudi Arabia and Yemen.

Australia is also at risk from imported vaccine-related strains of poliovirus, as indicated by two reports from the United States in 2005. The first was a case of imported VAPP in an unimmunised adult, who had been in close contact with an infant recently immunised with OPV, while in Costa Rica.[4] The second report described isolation of OPV poliovirus type 1 with a significant number of mutations (referred to as vaccine-derived poliovirus [VDPV]) from unvaccinated members of a religious community.[5] Given that OPV has not been used in the USA since 2000, the source of the virus is unknown. VDPVs have been associated with paralytic polio worldwide.

It is imperative that the Australian community maintains the current high rate of polio vaccination coverage, especially for travellers, which remains the best defence against all forms of imported polio. A surveillance scheme for investigation of children with acute flaccid paralysis, the major clinical presentation of poliomyelitis, was established in Australia in 1995. It is coordinated by the National Poliovirus Reference Laboratory and the Australian Paediatric Surveillance Unit (Box). While the scheme focuses on children, specimens from patients of all ages are tested. Notification of all cases with a clinical suspicion of poliomyelitis is essential for the detection of imported polio.

Surveillance for acute flaccid paralysis (AFP) within Australia

Paediatricians notify cases of AFP via a monthly report card to the Australian Paediatric Surveillance Unit and submit a clinical questionnaire to the National Poliovirus Reference Laboratory (NPRL). Stool specimens from AFP cases are tested at the NPRL for isolation of poliovirus. The Australian Polio Expert Committee reviews the clinical and laboratory data to determine whether the case is compatible with poliomyelitis. The Committee reports to the Australian Government Department of Health and Ageing and the World Health Organization.

Protocol for investigation of suspected polio cases

Clinicians should phone the NPRL to notify the case and arrange for two stool specimens to be collected 24 hours apart (due to intermittent virus shedding) and within 14 days of onset of symptoms, for testing at the NPRL. Polio antibody testing requires acute and convalescent serum, and is only performed when there is a clinical suspicion of poliomyelitis.

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Murine typhus mimicking acute cholecystitis in a traveller

Vidyut P Suttor and Robert B Feller

To the Editor: *Rickettsia typhi* is an endemic cause of atypical pyrexial illness worldwide.[1] Its non-specific presentation can lead to misdiagnoses, with overseas reports of unwarranted laparotomies in affected patients.[2,3] We describe a patient with *R. typhi* infection presenting as cholecystitis, in whom a cholecystectomy was avoided by vigilance for *R. typhi*.

A 51-year-old businessman presented to a general practitioner with a 5-day history of fever, sore throat, headaches and myalgia. The illness had begun a week after his return to Sydney from a business trip to major cities in Asia, his last stop being Hong Kong. Investigations revealed mild lymphopenia and thrombocytopenia, negative results on screening for malaria, and normal results on chest x-ray. A non-specific viral illness was provisionally diagnosed.

A week later, the patient presented again to a GP with fever, abdominal pain, cough, dehydration and confusion. Investigations revealed lymphopenia (0.7×10^9/L; reference range [RR], 1.5–4 $\times 10^9$/L], thrombocytopenia (93×10^9/L; RR, 150–400 $\times 10^9$/L), and raised serum levels of bilirubin (25 μmol/L; RR, 0–17 μmol/L) and hepatic enzymes (alanine aminotransferase, 307 U/L [RR, 5–40 U/L]; alkaline phosphatase, 381 U/L [RR, 30–115 U/L]; aspartate aminotransferase, 389 U/L [RR, 5–40 U/L]; and γ-glutamyl transferase, 364 U/L [RR, < 66 U/L]. Serological tests were negative for hepatitis viruses A, B and C, and dengue and Epstein–Barr viruses. The patient was referred to an emergency department.

On presentation to the hospital, the patient was febrile, with severe right upper quadrant abdominal tenderness, and a slight truncal macular rash. Abdominal computed tomography and ultrasound examination indicated cholecystitis (Box). Acute cholecystitis was diagnosed, and treatment begun with intravenous fluids, ampicillin, gentamicin and metronidazole. Following clinical improvement, the patient was discharged on Day 8 with plans for an elective cholecystectomy.

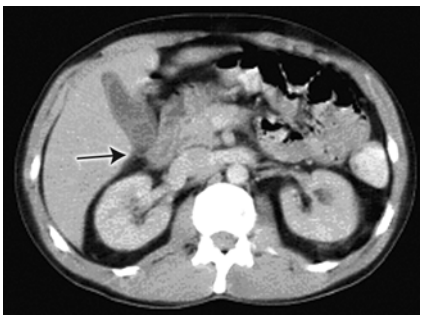
In view of the atypical symptom complex, serological testing for leptospirosis, syphilis, and rickettsial, amoebic and HIV infection had been requested during his admission. Results received after discharge indicated a *R. typhi* antibody titre of 1:1024 (RR, < 1:128). Results of the remaining serological tests were negative. The patient was contacted, and the cholecystectomy cancelled. He has remained well.

Murine typhus is a zoonosis caused by *R. typhi*, and is acquired from rodent flea faeces, either by bite inoculation or inhalation. Hepatobiliary involvement occurs in up to 34% of cases. Histopathology specimens show neutrophilic sinusoidal infiltrates and cloudy swelling of hepatocytes,[2] but hepatocyte injury and cholestasis are transient, resolving over 1–3 weeks.

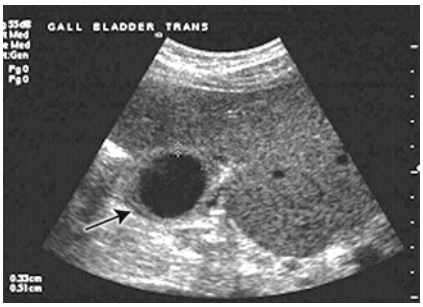
Diagnosis is by serological testing: a single indirect immunofluorescent antibody (IFA) titre against *R. typhi* of at least 1:400; or a fourfold rise in IFA titre from the acute to the convalescent phase (2 weeks apart). The treatment of choice is doxycycline. Although the clinical course is usually benign, the mortality rate can reach 4%.[1]

Rickettsial diseases remain an underreported cause of febrile illness.[4] As *R. typhi* has now been described throughout Australasia,[1,5] it is important that murine typhus is excluded in patients with atypical pyrexial illnesses and abnormal liver function results.

Computed tomography and ultrasound examination in a patient with murine typhus



Computed tomography on admission showed pericholecystic inflammation, suggesting acute cholecystitis, and a possible gallstone at the lower pole (arrow), which was later noted to be a fibrous septum on ultrasound examination.



Ultrasound examination also showed a thickened gall bladder wall and pericholecystic fluid. ◆

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Is it time to review the screening guidelines for younger diabetic children?

Catherine Dunlop

To the Editor: Routine school vision screening has been discontinued in many regions.[1]

Since 2000, children around Newcastle and Lake Macquarie in New South Wales only have their vision checked at school if their parents request it. I am particularly concerned that this may disadvantage young children with diabetes, who may also have undetected amblyopia. These children are already at risk of diabetes-related vision impairment, and simple screening could prevent further disability related to amblyopia.

Amblyopia, commonly known as "lazy eye", is an asymptomatic, potentially treatable condition of poor vision in a "normal" eye. It is caused by the brain suppressing an unclear image from the affected eye. Amblyopia occurs in 2.5%–3.2% of the population.[2] The condition needs to be detected early, and treatment needs to be instituted before the end of practical vision development at about 7–8 years of age, otherwise even intensive treatment is unlikely to restore normal vision.[3] This is especially important because people with untreated amblyopia have an increased lifetime risk of loss or impairment of vision in their good eye,[4] as well as the poor vision in their amblyopic eye.

The current Australian screening guidelines for children with diabetes recommend screening for retinopathy after 5 years of diabetes in those who are prepubertal, and annually in adolescents after 2 years of diabetes.[5] The International Society for Pediatric and Adolescent Diabetes recommends retinopathy screening in children with diabetes of prepubertal onset at 5 years after the onset of diabetes, or 11 years of age, or at puberty, whichever is earlier.[6] Neither document specifies other visual screening (although the National Health and Medical Research Council guidelines do recommend a clinical examination of the eyes for cataract soon after diagnosis). Thus, a 6-year-old child with diabetes would not have his or her vision screened until 11 years of age. An eye with significant amblyopia detected at this age will not achieve normal vision, and the child would be reliant on only one eye for his or her lifetime. Even a 3-year-old child with diabetes would not have visual screening until 8 years of age, the end of practical vision development.

The case has been made recently for biennial retinopathy screening for children with diabetes.[7] I propose that diabetic children under 9 years of age have their vision fully assessed soon after the diagnosis of diabetes. Should amblyopia be detected then, treatment could commence before the end of active vision development.

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Chronic kidney disease and automatic reporting of estimated glomerular filtration rate

Alan McNeil

To the Editor: I agree with Jones[1] that the body surface area (BSA) formula printed in the position statement on reporting of estimated glomerular filtration rate (eGFR)[2] is wrong, even though the authors say that he is mistaken.[3]

As stated by Jones, the correct formula[4] for BSA in m^2, for a body weight W kg and height H cm is:

(i) $BSA = W^{0.425} \times H^{0.725} \times 0.007184$.

However, the position statement[2] gave the following formulas:

(ii) $BSA = W^{0.425} \times H^{0.725} \times 0.007184/1.73$; and

(iii) Uncorrected eGFR = GFR estimate ($mL/min/1.73m^2$) × BSA.

It appears that the denominator "1.73" has migrated from formula (iii) to formula (ii), so in fact both of these formulas are incorrect. This is potentially misleading for doctors and others who may want to calculate the eGFR for someone who is unusually big or small.

Formula (iii) should in fact be:

(iii) Uncorrected eGFR = GFR estimate ($mL/min/1.73m^2$) × BSA/1.73.

It is interesting that the same two errors in BSA calculations are present on the US National Kidney Disease Education Program website,[5] which was presumably the source of the formulas used by the Australian Creatinine Consensus Working Group.

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Timothy H Mathew, Graham Jones and David Johnson

In reply: McNeil draws attention to the detail in the correction factor we published in an attempt to assist users to "uncorrect" the eGFR derived from the MDRD (Modification of Diet in Renal Disease) equation used in calculating GFR from a serum creatinine concentration. Recalculating the eGFR

to remove the adjustment for body surface area (BSA) in an individual is unnecessary except at extremes of body size.[1]

Readers can be reassured that the formulas published in the position statement,[2] if used as directed, will not lead to any error. However, it would have been clearer if we had labelled the "BSA" equation as "correction factor" instead of "BSA". In the position statement[2] it can be misinterpreted that the BSA formula has a denominator, whereas, when used primarily to calculate BSA, it of course does not. Both versions of the formulas (ours in the position statement and McNeil's) therefore lead to identical answers. The reader can choose which one to use.

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Screening couples for cystic fibrosis carrier status: why are we waiting?

Louise M Christie, Elvira M Zilliacus, Angela J Ingrey and Gillian Turner

To the Editor: This question was recently posed by Massie and colleagues in an editorial in the Journal.[1] We have experience in providing cystic fibrosis (CF) carrier testing in pregnancy and in those planning pregnancy. In 1998, we ran a 12-month pilot program, the Double Testing Program, offering ΔF508 carrier testing to couples attending the John Hunter Hospital, Newcastle, NSW, for nuchal translucency screening. Of 491 participants, 84% chose CF carrier testing; 23 carrier–non-carrier couples were identified, and no carrier–carrier couples. A postnatal questionnaire compared knowledge of CF, anxiety levels and perceptions of the service between non-carrier couples and couples in which one partner was a carrier.[2] Both groups were very satisfied with the service, with no increased levels of anxiety.

The second initiative has been to offer CF carrier testing to clients attending the "drop-in" clinic at Hunter Genetics, Newcastle. This clinic has been available for 10 years and provides genetic counselling for clients referred by their general practitioners for pre-pregnancy or pregnancy counselling. Over the past 3 years, there have been 560 occasions of service with CF carrier testing in 499 individuals. Indications for testing include pregnancy screening or a family history of CF. With couples, both are tested for ΔF508, and, if one partner is identified as a carrier, the other partner is tested for 28 other mutations. The area health service, Hunter New England Health, meets the cost of the service and testing — $100 per couple for the ΔF508 test, and $250 for the full mutation screen. Of the 499 individuals, 65 (13%) were found to be CF carriers; after excluding those with a family history of CF, the carrier rate was 5.8%. Twenty carrier–non-carrier couples and one carrier–carrier couple with a family history of CF were identified.

Benefits included enabling the carrier–carrier couple to know their 1 in 4 risk of having a child with CF. Most women who have a child with CF want to avoid having further affected children, and most who have a subsequent pregnancy choose prenatal diagnosis.[3] Cascade testing can be offered to carrier families. Non-carrier couples can be reassured that they have a low risk.

The uptake of CF couple screening is low, given that there are over 3500 births annually at the John Hunter Hospital. The main obstacles are lack of awareness and costs. CF tests are not covered by public health funding or by private health insurance, and this issue needs urgent attention. We believe that GPs are best placed to offer CF couple testing. Testing could be incorporated into routine first trimester pregnancy care. We can provide a distance CF learning package, pamphlets and informed consent material to those interested.

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Medical handover

Francis J Bowden, Christian Lueck, Mark Hurwitz and Karina Kennedy

To the Editor: We were interested to note that the evolution of morning handover at Launceston General Hospital, as described by Fassett and Bollipo,[1] closely parallels our own experience at the Canberra Hospital, and we endorse their points about running a successful meeting. Our hospital has a large Geriatric Unit and all subspecialties are covered, but we do not have a general medicine unit.

In 2002, we began a formal morning handover meeting from 08:00 to 08:30 for junior medical officers (JMOs) in the Department of Medicine, with the initial intention of providing an opportunity for Royal Australasian College of Physicians (RACP) basic trainees to present cases they had seen overnight. Scrutiny of the individual's clinical approach by consultants, in preparation for the RACP examination, was the main emphasis, and "interesting" cases were chosen. The meeting was also used for case presentations by specialty units. Attendance was variable, and many junior staff reported feeling somewhat threatened by having their patient management approach examined in a public forum.

Handover of most newly admitted patients did not occur during this meeting. The format was incrementally modified over the following 3 years so that, by 2005, the meeting had become a formal handover of all patients admitted during the previous evening and overnight.

Attendance is now compulsory (except for staff attending medical emergencies), and breakfast of brewed coffee and tea with

fruit and muffins is provided (funded by the Canberra Hospital). We have over 40 daily attendees (comprising registrars, residents, interns, medical students and 5–10 consultants). We have minimised the number of specialty presentations: these now usually take the form of "red flag" sessions, in which a specialist unit highlights areas of common and/or life-threatening importance (eg, a patient with unstable angina needs admission, regardless of their troponin level; recurrent rigors in a middle-aged person usually signal a bacterial infection).

A survey of 57 of the attendees this year revealed that over 90% thought the format and duration of meetings and attendance by consultants was appropriate; 54% and 39%, respectively, said they learned new information every day or every week. Over the past 4 years, the handover has become embedded in the clinical culture of the hospital. The long-term commitment of a small group of consultants has demonstrated that this is a safe and encouraging environment for clinical teaching, and the level of discomfort of the JMOs appears to have receded. The morning handover has been an important means of ensuring that young doctors are exposed to a broad perspective on patient care and that their after-hours patient care can be supervised.

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Impact of multiple impairments on quality of life, hospitalisations and use of aged-care services

Ee-Munn Chia, Jie Jin Wang, Elena Rochtchina and Paul Mitchell

To the Editor: Healthy ageing is listed as a National Research Priority by the Australian Government. The higher prevalence of sensory, cognitive and mobility impairments in older people presents a major challenge in achieving this goal. The effects of single impairments are recognised,[1,2] but the cumulative effects of multiple impairments have not been reported from population-based samples.

We aimed to assess the impact of multiple impairments (vision, hearing, cognitive, mobility) on health-related quality of life (HRQOL), hospitalisation, and aged-care service use in an older Australian population. In the second cross-sectional Blue Mountains Eye Study,[3] HRQOL was measured by means of the self-administered Short Form 36-item Health Survey (SF-36)[4] ($n = 3509$; mean age, 66.7 years; 57% women). Visual impairment was defined as best-corrected visual acuity (after refraction) of less than 6/12 (better eye). Hearing impairment was defined as average hearing threshold (pure-tone air conduction, frequencies 500–4000 Hz) over 25 decibels (better ear). Possible cognitive impairment was defined as Mini Mental State Examination scores less than 24/30. Mobility impairment was recorded. General linear regression was used to calculate age-adjusted SF-36 mean scores,[5] and logistic regression was used to estimate likelihood ratios for use of health and aged-care services. Models were age-adjusted to eliminate confounding.

For 2873 participants who had completed the SF-36 (90.9%), the mean physical component score (PCS) was 44.9 (95% CI, 44.5–45.3) and the mean mental component score (MCS) was 51.9 (95% CI, 51.5–52.2). Age was significantly associated with the preva-

1 Prevalence, mean SF-36 physical and mental component scores, and use of services by impairment

		Age-adjusted mean SF-36 scores (95% CI)		Use of services: %, age-adjusted and sex-adjusted odds ratio (95% CI)	
Impairments	Prevalence (%)	Physical component score	Mental component score	Hospitalisation in past 12 months	Regular use of community services
Visual impairment	2.7	42.8 (39.9–45.7)	47.6 (44.8–50.3)*	34.9%, 1.3 (0.8–2.2)	24.2%, 2.9 (1.4–6.0)
Hearing impairment	33.4	43.8 (43.0–44.7)*	51.1 (50.3–51.9)*	27.5%, 1.1 (0.9–1.3)	7.4%, 2.7 (1.4–5.0)
Cognitive impairment	2.2	42.2 (39.5–44.8)*	46.0 (43.4–48.5)*	28.2%, 1.0 (0.6–1.6)	14.1%, 1.7 (0.8–3.7)
Mobility impairment	7.6	32.3 (30.8–33.7)*	48.1 (46.7–49.5)*	41.0%, 2.0 (1.5–2.7)	21.3%, 6.8 (4.2–11.0)

All mean values adjusted to 66.7 years, the overall sample mean age. SF-36 = Short Form 36-item Health Survey.[4] * Significantly lower than without disability. ◆

2 Mean physical and mental component scores and use of services by increasing number of impairments

	Age-adjusted mean SF-36 scores (95% CI)		Use of services: %, age-adjusted and sex-adjusted odds ratio (95% CI)	
No. of impairments*	Physical component score	Mental component score	Hospitalisation in past 12 months	Regular use of community services
0 ($n = 1031$)	46.6 (45.9–47.2)	52.8 (52.6–53.8)	22.0%, 1.0	0.4%,1.0
1 ($n = 616$)	42.6 (41.8–43.3)	51.0 (50.3–51.7)	25.3%, 1.1 (0.8–1.3)	4.2%, 7.4 (2.7–19.8)
2 ($n = 121$)	38.6 (37.1–40.0)	48.8 (47.4–50.2)	35.5%, 1.5 (1.0–2.3)	19.0%, 24.9 (8.5–73.2)
⩾ 3 ($n = 31$)	34.5 (32.2–36.8)	46.6 (44.5–48.8)	45.2%, 2.0 (0.9–4.1)	41.9%, 47.4 (13.1–171.4)

SF-36 = Short Form 36-item Health Survey.[4] * Includes vision, hearing, cognitive and mobility impairments. ◆

lence of these impairments ($P < 0.001$). After adjusting for age, people with any of the impairments had poorer mean PCS and MCS than those without the impairment (Box 1). Hospitalisation within the last year was reported by 743 participants (23.5%; 58.3% women), and 97 (3.1%; 65.0% women) reported regular use of community support services. Use of community support services was reported more frequently by people with any impairment, except possible cognitive impairment (Box 1).

The presence of two or more impairments was associated with a cumulative, linear decline in HRQOL (Box 2). The successive addition of each impairment was associated with a decrease of 4.0 in mean PCS and 2.1 in mean MCS, and with greatly increased reporting of regular community support service use.

The likelihood of participating in or completing the SF-36 decreased with increasing number of impairments. Hence, the prevalence of impairments and the extent of detrimental impacts on HRQOL may be underestimated. Nevertheless, our data highlight a linear increasing pattern of cumulative effects from multiple impairments on HRQOL, hospitalisation, and use of aged-care services. Preventing and reducing these impairments is crucial in maximising healthy ageing.

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Australia's media reporting of health and medical matters: a question of quality

Julie Robotham

To the Editor: Despite a new survey with some interesting results, the *MJA*'s *Medicine and the Media* special failed overall to advance the topic.

Van der Weyden and Armstrong enthusiastically cite Schwartz and Woloshin: "don't report preliminary findings".[1] (In fact, they wrote, "In general, don't report preliminary findings",[2] but let's allow this journalistic context tweak.) In their article, Schwartz and Woloshin contend that this is because "what is new may turn out to be wrong".

Many things reported in newspapers are subject to subsequent change. So, to be on the safe side, let's exclude them all. No more Cabinet leaks: let's wait till everything is resolved and perfect-bound by the government stationery office. An end to covering murder trials: what if the accused is found not guilty? Forget about half-time match scores, and definitely no celebrity weddings because they'll be divorced within the year.

A moratorium on discussing preliminary findings would put off-limits highly respected annual meetings such as those of the American Society of Clinical Oncology and the American Society for Reproductive Medicine, which set the treatment agenda annually for clinicians and patients in the fastest-paced medical specialties. It is nonsense to suggest the media should censor

early results, a point that has been made previously.[3]

The world has moved on. News is no longer a series of monolithic reports, each entirely true and complete. Like life, news is a work in progress — a rolling tide of updates, each modifying the last.

Everyone loves a winner, and it is gratifying to note the *Sydney Morning Herald* is currently top of the *Media Doctor* league table.[4] This website is a useful focal point, and Smith and colleagues' suggestion that researchers take some responsibility for how their results are presented to the public is helpful.[5]

But of the three solicited commentaries,[6-8] not one was from a newsroom health reporter, or — better still — a daily news editor or producer who decides, amid the controlled chaos of breaking and evolving stories, which reports should run and how prominently.

Was this because they were not approached?

Imagine a five-article package on immunisation practice without a view from a general practitioner, or one on appendicectomy without the insight of a surgeon.

It is a serious omission that undermines the credibility of the *MJA*'s package and calls its motivation into question. Genuine rapprochement might threaten the sport of media sniping that has become a lively sideline for some medical journals.

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Ruth M Armstrong and Martin B Van Der Weyden

In reply: Would that we could convince newspaper editors not to publish inaccurate stories, cabinet leaks, sensational and one-sided details of trials in progress, hope-raising interim football scores or the details of celebrity weddings. However, our media package was not that ambitious!

In the context of medical reporting, we stand by our advice against publishing interim results. The reason that clinical trials have protocols, power calculations and stopping rules is that (just as football is a game of two halves) the results are really not relevant or reliable until all the data have been collected and analysed.

Our media package included contributions from two highly respected medical writers[1,2] and a representative of Australian journalism's peak body.[3]

Robotham is correct in surmising that we did not approach a newsroom health reporter or a news editor, but the immunisation analogy seems spurious. If we published a research paper that identified, for instance, deficiencies in general practitioners' delivery of vaccinations, we would not necessarily accompany it with a commentary from a GP. Whatever their discipline, we would assign the task to someone who could point the way towards best practice.

Nevertheless, we are grateful for Robotham's interest and acknowledge the responsibilities shared by both journalists and medical journal editors, albeit with differing emphasis — the dissemination of accurate and timely information.

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