LETTERS

Medical workforce issues in Australia: "tomorrow's doctors — too few, too far"

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TO THE EDITOR: The workforce article by Brooks et al[1] identifies key factors causing the medical workforce shortage and notes, correctly in our view, that: "The full impact of these factors is yet to be felt, but might occur very rapidly".

However, the authors fail to address why this has occurred and what should be done.

The answer as to why is quite simple. In the 1990s, the Labor and Coalition federal governments introduced a series of measures to ration the supply of doctors and the provision of services in order to restrain the health budget. Measures such as restrictions on medical student places, reduced training places, restricted provider numbers, failure to properly index the Medicare Benefits Schedule or introduce the Relative Value Study, and the move away from fee-for-service with the rapid expansion of red tape, were all designed to restrict services that cost the government money. The current doctor shortage, falling participation rates (the trend to doctors retiring early or working part-time) and demoralisation of significant sections of general practice are a tribute to the success of these policies.

As the recent Australian Medical Workforce Advisory Committee careers study shows, the much-discussed feminisation of the GP workforce is as much a consequence of a declining number of young male doctors considering general practice to be a rewarding career as it is the result of a need by both male and female doctors for an occupation that allows a flexible work and family lifestyle.[2]

Nevertheless, the outcome — the falling participation rate among current and future general practitioners — is at the heart of the problem.

The solution will require a total shift in policy direction from sticks to carrots. It will need to cover Medicare, training, working conditions, and the removal of red tape and all forms of restrictions not required to ensure good clinical practice. Attempts to use regulations or commercial levers to enforce bulk-billing in an already depleted workforce will only serve to exacerbate the current situation.

1. Brooks PM, Lapsley HM, Butt DB. Medical workforce issues in Australia: "tomorrow's doctors — too few, too far". *Med J Aust* 2003; 179: 206-208.
2. Australian Medical Workforce Advisory Committee. Career decision making by doctors in vocational training. AMWAC Medical Careers Survey, 2002. AMWAC report 2003.2, May 2003. Available at: www.healthworkforce.health.nsw.gov.au/amwac/amwac/pdf/career_decision_making_report_2003.2.pdf (accessed Oct 2003). ❑

Could it be sarcoid arthritis?

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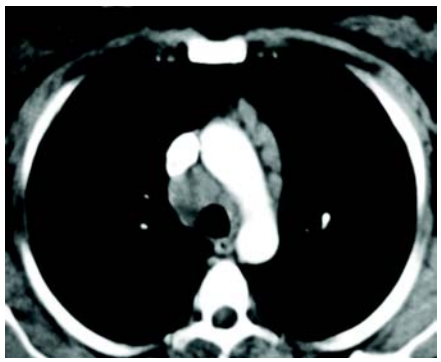
TO THE EDITOR: Sarcoid arthritis is often underdiagnosed because it may mimic reactive or rheumatoid arthritis. We report a case which was initially misdiagnosed.

A 38-year-old white woman was admitted to hospital because of pain and swelling of her hands and feet. Two years earlier, she had been admitted because of joint pain and erythematous, painful round lesions on her shins. A chest x-ray at that time was normal. She was diagnosed with reactive polyarthritis based on positive serological tests for *Rickettsia conorii* and *Coxiella burnettii*. Doxycycline and indomethacin were given and her condition improved.

Three days before the current admission her fingers, wrists and ankles had become painful and swollen. There was tenderness and swelling of the metacarpophalangeal, wrist and ankle joints. She was afebrile. Chest x-rays showed an enlarged left hilum. Computed tomography (CT) of the chest (Box) showed lymphadenopathy in the mediastinum and both hila. Her erythrocyte sedimentation rate was 104 mm/h (normal, 3–12 mm/h). Laboratory test results were normal, except for an elevated serum level of angiotensin-converting-enzyme (ACE). A mediastinoscopy was performed, and specimens obtained for biopsy revealed sarcoidosis. Prednisone (30 mg/day) was prescribed and she was discharged 7 days later with no symptoms.

Computed tomography scan showing mediastinal and hilar lymphadenopathy



"Sarcoid arthritis" is a sarcoid process whose main or unique manifestation is joint disease. Some of its characteristics are seasonal clustering (typically in spring), higher incidence among non-smoking patients, and the presence of the human leukocyte antigen DQ2 (DQB1*0201) and DR3 (DRB1*0301) haplotypes. It occurs slightly more frequently in women. The median age of affected patients is 40 years. The process affects mainly ankle and knee joints symmetrically. Acute sarcoid arthritis is a self-limiting joint disease with a benign prognosis, but some patients can develop chronic sarcoidosis of the lungs, specially those who suffer recurrent episodes of arthritis.[1,2]

In our patient, the first episode, with associated erythema nodosum, was misdiagnosed as a reactive arthritis as there were false positive serological test results for Rickettsia and Coxiella secondary to an immune polyclonal response. In the second episode, the patient had mediastinal and hilar lymphadenopathy and an elevated ACE level. Although sarcoid arthritis is very often associated with lymphadenopathy and erythema nodosum (Löfgren syndrome), we should keep in mind other forms of joint involvement in sarcoidosis.[3]

Doctors should consider sarcoid arthritis in the differential diagnosis of seronegative arthritis. Chest x-ray and ACE assay are useful in identifying sarcoidosis.

1. Visser H, Vos K, Zanelli E, et al. Sarcoid arthritis: clinical characteristics, diagnostic aspects, and risk factors. *Ann Rheum Dis* 2002; 61: 499-504.
2. Gran GT, Bohmer E. Acute sarcoid arthritis: a favourable outcome? A retrospective survey of 49 patients with review of the literature. *Scand J Rheumatol* 1996; 25: 70-73.
3. Horusitzky A, Dumont D, Valeyre D, et al. *Localisations ostéoarticulaires de la sarcoïdose. Encycl Méd Chir* (Elsevier, Paris-France). Appareil locomoteur. 14-027-C-10, 1998. ❑

Cardiovascular risk among urban Aboriginal people

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TO THE EDITOR: In a recent article, Thompson and colleagues provided useful information on the prevalence of cardiovascular risk factors in urban Aboriginal people.[1] Using the Sheffield table of absolute risk,[2] the authors estimated that "15% men and 6% women had an absolute risk > 15% of a cardiovascular event within 10 years".

The Sheffield risk table was developed for assessing the risk of coronary *deaths* rather than the risk of cardiovascular events.[2] Moreover, the validity of applying the Sheffield table and other risk assessment tools based on the Framingham risk functions to Aboriginal people is yet to be assessed. The lower risk estimate in women reported by Thompson and colleagues may simply reflect the higher cholesterol concentration cut-offs for women in the Sheffield table.

The true risk difference between sexes in Aboriginal people may not be as dramatic as Thompson and colleagues suggest.

Firstly, data in Box 1 of their article show that there was little difference between men and women as regards past history of cardiovascular disease.

Secondly, Aboriginal women experience a higher prevalence than men of some cardiovascular risk factors such as diabetes,[1,3] abnormal HDL cholesterol level and overweight.[3]

Thirdly, our own research suggests that there may be a substantial difference between estimated and observed risks. Using data from a cross-sectional study of 681 Australian Aboriginal people in a remote community,[3] we performed a similar analysis to that of Thompson et al. Based on the Framingham functions,[4] we estimated that 10-year risks of coronary heart disease for women were much lower than those for men in all age groups (a finding similar to that of Thompson and colleagues). However, in a related study of the same Aboriginal community (as yet unpublished), when we analysed cohort data from 838 participants with 13 years of follow-up, the observed coronary disease rates for women were as high as those for men (Box).

Incidence rates per 1000 person-years of coronary heart disease (95% CI), by age and sex (based on a cohort study of 838 Aboriginal people in a remote community)

Age (years)	Women	Men
20–34	4.1 (1.8–9.1)	3.2 (1.4–7.0)
35–44	15.6 (9.4–25.9)	8.6 (4.5–16.5)
45–54	19.3 (10.9–33.9)	26.5 (15.0–46.7)
⩾55	50.2 (32.4–77.9)	31.9 (16.6–61.2)

The discrepancy we found between estimated and observed risks is a warning that researchers and clinicians need to be cautious when applying existing risk assessment tools to Aboriginal people.

1. Thompson PL, Bradshaw PJ, Veroni M, Wilkes ET. Cardiovascular risk among urban Aboriginal people. *Med J Aust* 2003; 179: 143-146.
2. Haq IU, Jackson PR, Yeo WW, Ramsay LE. Sheffield risk and treatment table for cholesterol lowering for primary prevention of coronary heart disease. *Lancet* 1995; 346: 1467-1471.
3. Wang Z, Hoy W. Hypertension, dyslipidemia, body mass index, diabetes and smoking status in Aboriginal Australians in a remote community. *Ethn Dis* 2003; 13: 324-330.
4. Anderson KM, Odell PM, Wilson PW, Kannel WB. Cardiovascular disease risk profiles. *Am Heart J* 1991; 121(1 Pt 2): 293-298. ❑

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IN REPLY: We appreciate the commentary by Wang and Hoy on the problems of the use of risk scores for assessing cardiovascular risk in Aboriginal people.

In general, we agree that caution is essential in using tables that predict absolute risk of cardiovascular events. However, despite their limitations, absolute risk estimates are being encouraged by Australian, European, New Zealand and US authorities as a practical aid to targeting coronary disease preventive measures.[1] An estimated risk of > 15% of a fatal cardiovascular event within 10 years, based on the Sheffield or Framingham scores, is now recommended as an indication for active treatment. Our prime purpose in providing an estimate of absolute risk in the Perth urban Aboriginal population was to demonstrate that a program of cardiovascular risk assessment with strong Aboriginal community support is capable of detecting high-risk people who will benefit from intensive risk-lowering strategies.

Wang and Hoy's caution about applying absolute risk estimates based on the Framingham population to unrelated populations is of particular importance in the case of Australian Indigenous people, in whom diabetes and the related metabolic syndrome may be the predominant risk factors.

We have recently completed an analysis of the determinants of carotid atherosclerosis in the same population described in our earlier study.[2] Our results confirm that, while the Framingham estimates (based on sex, age, LDL cholesterol and blood pressure) are indeed predictors of carotid atherosclerosis, their predictive value is significantly enhanced by the addition of markers of diabetes status and obesity.

The 13-year follow-up study of the Aboriginal cohort referred to by Wang and Hoy will provide unique data to help identify reliable risk predictors specific to Aboriginal people, and we look forward to its publication.

1. Joint British recommendations on prevention of coronary heart disease in clinical practice. British Cardiac Society, British Hyperlipidaemia Association, British Hypertension Society, endorsed by the British Diabetic Association. *Heart* 1998; 80 (Suppl 2): S1-S29.
2. Thompson PL, Bradshaw PJ, Veroni M, Wilkes ET. Cardiovascular risk among urban Aboriginal people. *Med J Aust* 2003; 179: 143-146. ❑

A comparison of buprenorphine treatment in clinic and primary care settings: a randomised trial

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TO THE EDITOR: The trial of buprenorphine-assisted heroin detoxification in primary care and a specialist clinic by Gibson et al[1] was intended to compare the effectiveness and cost-effectiveness of buprenorphine-assisted withdrawal in a specialist clinic with treatment by general practitioners. However, of the average $191 for primary care staff costs, $69 was incurred at the clinic. As

at least a third of interactions between patients and staff actually took place in the clinic, the primary care arm of the trial was really a combination of specialist clinic and primary care.

Another design problem was the study's lack of statistical power. A study would need 550 participants to have an 80% chance of identifying (at the 0.05 level of statistical significance) a difference of 50% in self-reported abstinence during the 8-day detoxification (ie, improving the percentage reporting abstinence from 22% to 33%). The trial by Gibson and colleagues had only 115 participants.

As expected, the trial produced statistically non-significant results. Yet, the authors highlight the finding that 23% of primary care patients reported being abstinent during the 8-day detoxification, compared with 22% of the clinic patients, (95% CI risk difference, –14.1% to 16.5%; $P = 0.9$ [χ^2]). Moreover, the clinic group performed better on an objective and more reliable measure of abstinence: 20% of clinic patients versus 14% of primary care patients gave morphine-free 8th day urine specimens, (95% CI risk difference, –7.7% to 19.8%; $P = 0.4$ [χ^2]).

As the confidence intervals for these risk differences include zero, the confidence interval for any estimate of incremental cost-effectiveness includes infinity. It is quite misleading for Gibson and colleagues to claim that "it costs $20 to achieve a 1% improvement in outcome in primary care", as this ignores both the conflict and the variability in their clinical outcomes.[1] Moreover, the statement ignores the variability in the estimated costs of treatment (eg, mean cost per clinic patient, $332; SD, $70).

Surprisingly, Gibson and colleagues did not collect any information on continuing abstinence at the 13-week follow-up. Rather, they collected information on patients' current treatment. While patients in whom detoxification therapy fails should be offered other treatment, post-withdrawal engagement in maintenance treatment is not a meaningful measure of the effectiveness of detoxification. If anything, it is a measure of failure.

The trial needed sufficient statistical power to identify clinically meaningful differences in abstinence at the end of the 8-day detoxification and at 13 weeks. Staff working in the specialist clinic should not have been extensively involved in the delivery of primary care. Gibson and colleagues should have summarised their findings using appropriate estimates of clinical effect and cost-effectiveness with 95% confidence intervals.[2]

1. Gibson AE, Doran CM, Bell JR, et al. A comparison of buprenorphine treatment in clinic and primary care settings: a randomised trial. *Med J Aust* 2003; 179: 38-42.
2. Moher D, Schulz KF, Altman DG. The CONSORT statement: revised recommendations for improving the quality of reports of parallel-group randomised trials. *Lancet* 2001; 357: 1191-1194. ❑

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IN REPLY: The primary focus of our study[1] was retention in treatment, and not differences in abstinence. Caplehorn has previously argued compellingly that an orientation to abstinence can have an adverse impact on treatment outcomes in opioid dependence.[2] We were using buprenorphine to redefine detoxification, not as a treatment producing lasting abstinence but as a way of promoting engagement in ongoing treatment. The power of our study was calculated on the basis of the proportion of subjects entering post-detoxification treatment, not on their self-reported abstinence levels.

During the detoxification stage in the primary care setting, we used a shared-care dosing arrangement. This was primarily because of the need to give an initial research assessment to all participants before they were randomly allocated to treatment arms — something that would only occur in the context of a research study, and noted in the discussion. Further details of the health economic analysis are soon to be published.[3]

Ours was a study of the setting for buprenorphine treatment. Its critical finding was that patients were equally as likely to be engaged in maintenance treatment with practitioners in primary care as in specialist clinics.

1. Gibson AE, Doran CM, Bell JR, et al. A comparison of buprenorphine treatment in clinic and primary care settings: a randomised trial. *Med J Aust* 2003; 179: 38-42.
2. Caplehorn J, Bell J. Methadone dosage and retention in maintenance treatment. *Med J Aust* 1991; 154: 195-199.
3. Doran CM, Shanahan M, Bell J, Gibson A. A cost-effectiveness analysis of buprenorphine assisted heroin withdrawal. *Drug Alcohol Rev*. In press. ❑

Troponin testing: an audit in three metropolitan hospitals

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TO THE EDITOR: In the article by Davey[1] no evidence other than deviation from a protocol published months before the study is produced to document the implied inappropriateness of single troponin assays.

Emergency physicians are experienced in assessing undifferentiated chest pain. Acute coronary syndromes are but one cause of presentation to emergency departments (EDs) of patients with chest pain, and indeed are but one cause of elevated serum troponin levels.

Many reasons may justify the "appropriate" ordering of single troponin assays. Some patients present to EDs many hours after their episode of chest pain. A single troponin test may be a very useful and sensitive test for a patient whose chest pain occurred yesterday. How many patients in the study group had their single troponin test done more than 12 hours after their episode of pain? How many patients discharged themselves against medical advice as they were unwilling to wait 6–8 hours for a second blood test to triage their risk for an acute coronary syndrome? How many patients died or were transferred to another hospital? How many patients had their single troponin test ordered in the investigation of a primarily non-cardiac illness, such as sepsis or pulmonary embolism?

I have no doubt that many troponin assays ordered in the study population were inappropriate. But, by failing to conduct an explicit medical record review of those patients whose tests were deemed inappropriate, the author has failed to answer his stated aim of determining if the troponin assay is used appropriately when chest pain is encountered. We are left with no knowledge of whether this problem is small or large.

Finally, does it matter? Are two consecutive negative troponin assays required to triage patients with chest pain? Recently, the Journal published a clinical outcome study that examined the implementation of a chest pain assessment protocol at a metropolitan university teaching hospital in Banks-

town, Sydney.[2] Patients presenting to the ED with "possibly cardiac" non-traumatic chest pain who were deemed to be low risk did not receive a second, late troponin assay, and yet this approach appeared to be safe.

Those of us who have an interest in the rational use of diagnostic testing for patients with acute coronary syndromes eagerly await the publication of further evidence on this important matter.

1. Davey RX. Troponin testing: an audit in three metropolitan hospitals. *Med J Aust* 2003; 179: 81-83.
2. Boufous S, Kelleher PW, Pain CH, et al. Impact of a chest-pain guideline on clinical decision-making. *Med J Aust* 2003; 178: 375-380. ❑

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IN REPLY: In acute myocardial infarction (AMI) diagnosis, the sensitivity and specificity of troponin rise with time after symptom onset. The sensitivity of troponin-I testing was shown to increase from 35% at 0–4 hours to 97% at 12–24 hours after an infarct.[1] Similarly, a meta-analysis found that "multiple testing of individual biomarkers over time substantially improves sensitivity, while retaining high specificity" for AMI diagnosis.[2] This position is taken by the National Academy of Clinical Biochemistry[3] and European and American cardiologists.[4]

Furthermore, the diagnostic clock starts from a patient's emergency department presentation if there is any unreliability suspected in the patient's assessment of pain onset. Pain onset may have been stuttering, indeterminate, simply forgotten, some combination of these, or even absent. Bailey describes several situations such as these, thought to justify, or to explain, singlicate troponin testing, and suggests that, as I did not audit records, I was not able to quantify the true extent of inappropriate ordering. I acknowledged this shortcoming, but believe it does not detract from the endpoint found.

The Bankstown low-risk patients[5] are only a confounder here. In our protocol they probably would not have been thought to have cardiac pain, and all the remaining Bankstown patients had serial biomarker testing.

In my audit,[6] 93% of singlicate troponin test orders did not diagnose an AMI, and if AMI were still considered, then the tests contravened the protocols.[3,4] This is why they were called "inappropriate" — no arbitrary whim.

I assumed, moreover, that no clinician ordered a troponin test unless seeking a cause for chest pain. Our protocol begins with chest pain, *recognises uncertainty*, and leads through to treating an AMI or reconsidering the diagnosis.

Obliquely invoking Ockham's principle is also dangerous here. Illnesses such as sepsis may "provoke" an AMI, but this must then be investigated independently, and according to its own rules of engagement, which do not alter solely because of the primary (co-)morbidity.

In short, we did know what is appropriate troponin use, and surveyed it. Like Bailey, we look forward to seeing further evidence.

1. Chiu A, Chan W-K, Cheng S-H, et al. Troponin-I, myoglobin, and mass concentration of creatine kinase-MB in acute myocardial infarction. *Q J Med* 1999; 92: 711-718.
2. Balk EM, Ioannidis JP, Salem D, et al. Accuracy of biomarkers to diagnose acute cardiac ischemia in the emergency department: a meta-analysis. *Ann Emerg Med* 2001; 37: 478-494.
3. Wu AHB, Apple FS, Warshaw MM, editors. Recommendations for the use of cardiac markers in coronary artery disease. Washington: AACC Press, 1999.
4. The Joint European Society of Cardiology/American College of Cardiology Committee. Myocardial infarction redefined — a consensus document of the joint European Society of Cardiology/American College of Cardiology committee for the redefinition of myocardial infarction. *JACC* 2000; 36: 959-969.
5. Boufous S, Kelleher PW, Pain CH, et al. Impact of a chest-pain guideline on clinical decision-making. *Med J Aust* 2003; 178: 375-380.
6. Davey RX. Troponin testing: an audit in three metropolitan hospitals. *Med J Aust* 2003; 179: 81-83. ❑

Pneumococcal meningitis masquerading as subarachnoid haemorrhage

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TO THE EDITOR: New imaging and pathology investigations continually improve diagnostic accuracy. But tests must be used because they supplement clinical deduction, not because they are available, and the constellation of clinical features should not be ignored.

The case report by Chatterjee and colleagues is valuable for describing delayed diagnosis of meningitis, based on imaging which suggested subarachnoid haemorrhage and aspiration pneumonia.[1] The 4-day history, examination (raised respiratory and heart rates, high fever) and results of initial investigations (neutrophilia, raised C-reactive protein level, lung consolidation) suggested a primary respiratory infection. The absence of a typical history of onset of subarachnoid haemorrhage is excused by the 5.5-hour hiatus before the patient was found semicomatose.

Subarachnoid haemorrhage was diagnosed because of density in the subarachnoid space on computed tomography (CT). The authors noted a 1980 report of this appearance in a patient with bacterial meningitis.[2] They also noted only one previous report of purulent meningitis mimicking subarachnoid haemorrhage on CT scan (in 1994),[3] but there is reluctance to publish "negative" outcomes. Aspiration as the cause of upper-lobe consolidation was unlikely. Bacterial pneumonia and chemical pneumonitis affect the lower lobe.[4]

Clinical findings were not consistent with subarachnoid haemorrhage, and meningitis was *the* differential diagnosis, so only the overweighted CT results prevented lumbar puncture on Day 0, which would have resulted in earlier, broader antibiotic therapy and possibly resumption of warfarin. By Day 1, it was too late to prevent permanent blindness (it was possibly too late on Day 0, but pupils were reactive at that time).

Even on Day 1, repeat cranial CT showed infarction but less evidence of bleeding; "a disparity between the amount of [alleged] subarachnoid blood and the patient's clinical condition" was followed by magnetic resonance imaging then angiography and venography, instead of lumbar puncture as suggested by hindsight in the last sentence of the report. Shadows do not always equate with pathology.

Compare an article on the clinical diagnosis of meningococcaemia.[5]

Holistic care of Chatterjee et al's patient included anticoagulation therapy. "It probably could have recommenced earlier" than after "a large pulmonary embolus" on Day 13 — perhaps, given the presence of long-term indications (lupus inhibitor, anti-cardiolipin antibody and previous thrombosis) and cerebral vessel inflammation causing infarction, on Day 1.

The main lesson, which we were all taught as students but needs career-long reinforcement, is in the penultimate sentence of the case report: "Investigations should not be interpreted in isolation from the clinical picture".

1. Chatterjee T, Gowardman JR, Goh TD. Pneumococcal meningitis masquerading as subarachnoid haemorrhage. *Med J Aust* 2003; 178: 505-507.
2. Stovring J, Snyder RD. Computed tomography in childhood bacterial meningitis. *J Pediatr* 1980; 96: 820-823.
3. Mendelsohn DB, Moss ML, Chason DP, et al. Acute purulent leptomeningitis mimicking subarachnoid haemorrhage on CT. *J Comput Assist Tomogr* 1994; 18: 126-128.
4. O'Connor S. Aspiration pneumonia and pneumonitis. *Aust Prescriber* 2003; 26: 14-17.
5. Yung AP, McDonald MI. Early clues to meningococcaemia. *Med J Aust* 2003; 178: 134-137. ❑

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IN REPLY: We agree with Morgan that the symptoms, signs and laboratory investigations in our case report, although non-specific, supported a diagnosis of infection.[1] The C-reactive protein level was not available for 24 hours. The unwitnessed drop in level of consciousness that occurred between 09:00 and 14:30 could have been secondary to meningitis or an acute cerebral event, and, while it is true that the lower lobes are predominantly involved in aspiration, they are not solely involved. Consolidation in other gravity-dependent segments, including the posterior segments of the upper lobes, can occur.[2]

The suggestion that "permanent blindness" could have been prevented is not supported. Fortuitously, an appropriate antibiotic to which the organism was fully sensitive was given from Day 0 (ceftriaxone). Adjunctive supportive care was quickly provided. In retrospect, anti-coagulation therapy could have recommenced earlier, but this remained a difficult decision in the context of the computed tomography findings. It remains unclear how this would have modulated the meningeal process, but it possibly contributed to the complication of pulmonary embolism.

We agree that there is a reluctance to publish what may be perceived as "negative" outcomes, but, educationally, these may be the most rewarding. This case was an uncommon presentation of a common disease, and we considered it sufficiently important to notify other practitioners. Of most importance in this era when technology in medicine advances exponentially, any investigation must be considered only an adjunct to, and not a replacement for, thorough clinical evaluation.

1. Chatterjee T, Gowardman JR, Goh TD. Pneumococcal meningitis masquerading as subarachnoid haemorrhage. *Med J Aust* 2003; 178: 505-507.
2. Goodman LR. The postoperative and critically ill patient — aspiration pneumonitis. In: Grainger RG, Allison DJ, Andrews A, Dixon AK, editors. Grainger and Allison's diagnostic radiology; a textbook of medical imaging. London: Churchill Livingstone, 2001: 548-549. ❑

Dosing information for paediatric patients: are they really "therapeutic orphans"?

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TO THE EDITOR: Tan et al outline deficiencies in product information documents (PIs) as published in MIMS.[1] It needs to be clarified that MIMS Australia is not responsible for the content of PIs — this is specified and approved by the Therapeutic Goods Administration in consultation with the sponsoring company. The conclusion by the authors that the "PIs for many prescription products listed ... do not adequately detail paediatric doses" should not be specifically attributed to *MIMS*, as all published medicines information that relies on approved PIs will suffer from the same deficiencies.

1. Tan E, Cranswick NE, Rayner CR, Chapman CB. Dosing information for paediatric patients: are they really "therapeutic orphans"? *Med J Aust* 2003; 179: 195-198. ❑

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