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Calcium supplementation does not increase mortality

Benjamin M P Tang and
BE Christopher Nordin

TO THE EDITOR: Calcium and vitamin D play a central role in preventing osteoporosis and fractures,¹ so a recent study published in the *BMJ* claiming that calcium supplements increased the risk of heart attacks and strokes in postmenopausal women² naturally received widespread media attention — so much so that many patients are already stopping calcium treatment.

The study, based on a previously published randomised controlled trial of calcium supplementation in 1471 healthy women,³ showed that self- or family-reported heart attack, stroke or sudden death was significantly more common in those taking calcium than in the placebo group ($P=0.008$). This conflicted with the findings of a much larger study.⁴ Further, the difference became non-significant when the analysis was corrected for covariables ($P=0.08$), or when the analysis was repeated using data on cardiovascular events obtained from medical records ($P=0.08$). Yet, it still gained a place in a leading medical journal.

The small excess of cardiovascular events in the women taking calcium could be due to chance and needs to be tested further; one way of doing this is to examine available data for evidence of mortality in patients taking calcium. We have done this. In the 29 randomised trials in a recent meta-analysis of the effect of calcium and vitamin D in fracture risk,¹ five trials comprising 12 609 subjects provided crude mortality data.⁵⁻⁹ When these mortality data were pooled using a random effects model, there was no evidence that calcium supplementation increased mortality (Box).

We find it hard to believe that calcium can have a significant adverse effect on cardiovascular disease without increasing mortality. Our reservations about this study are further strengthened by the weak theoretical basis of the case against calcium. Metastatic calcification in renal failure, which the authors quote as an analogy,² is due to the high serum calcium–phosphorus (CaxP) product levels caused by hyperphosphataemia, which may be aggravated by calcium supplementation. In women without this condition, this degree of oversaturation cannot be reached by the 5% rise in plasma calcium¹⁰ resulting from the recommended dose of calcium citrate used for supplementation.

Moreover, coronary blockage is not due to calcification of atheromatous vessels, which is a dystrophic calcification secondary to tissue damage, but rather to ruptured atheromatous plaques and the thrombi which form upon them.

Thus, it is premature to conclude that calcium supplementation should not be given to older women.

Benjamin M P Tang, Associate Researcher¹
Christopher Nordin, Professor²

1 School of Public Health, University of Sydney, Sydney, NSW.

2 School of Medicine, University of Adelaide, Adelaide, SA.

benjamin@clubsalsa.com.au

1 Tang B, Eslick G, Nowson C, et al. Use of calcium or calcium in combination with vitamin D supplementation to prevent fractures and bone loss in people aged 50 years and older: a meta-analysis. *Lancet* 2007; 370: 657-666.

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A case of primary cerebral vasculitis

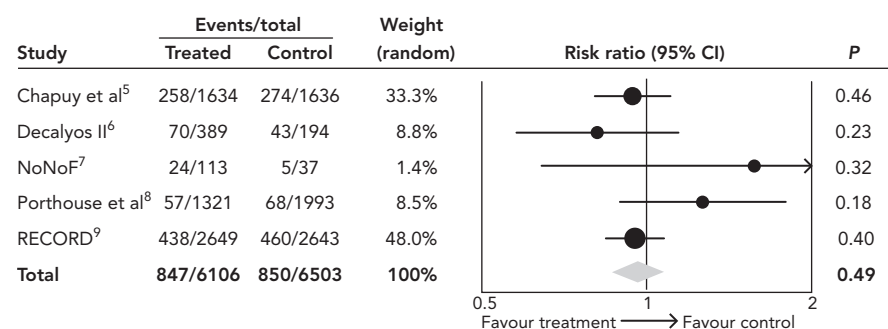
Sanjaya S Herath, Dayna B Law,
Peter J O Stride, Vernon J Heazlewood
and Luke S Gaffney

TO THE EDITOR: Primary cerebral vasculitis (PCV) is a potentially fatal disease. Early diagnosis and therapy are vital. We describe a case where confounding factors delayed diagnosis.

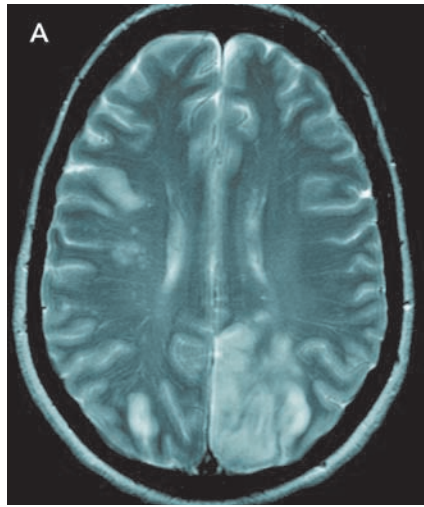
A 42-year-old woman presented with headache, nausea, vomiting, malaise and binocular blindness for 3 days. Two weeks previously, she had presented to the emergency department with headache and vomiting, but investigations, including computed tomography (CT) of the brain and lumbar puncture, gave normal results. She had a history of depression, was a smoker (20 pack-year history), and used cannabis regularly and alcohol occasionally, but denied other recreational drug use.

Her mood appeared depressed. Vital signs and findings from a general examination were normal. Eye movements were full, direct and indirect pupillary reflexes were intact, and optic fundi were normal. Results of a CT angiogram were reported as normal by a consultant radiologist. Results of blood tests, including inflammatory markers, and a repeat lumbar puncture, were unremarkable. A toxicology screen was not performed. Depression with conversion disorder was diagnosed, and admission with analgesia was advised. A neurologist's review

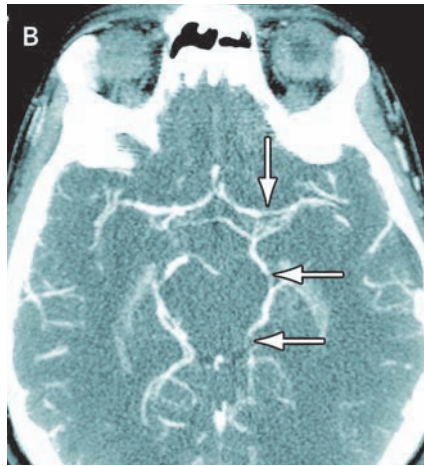
Effect of calcium supplementation on mortality, data pooled by a random effects model



NoNoF = Nottingham Neck of Femur. RECORD = Randomised Evaluation of Calcium Or vitamin D. ◆



A: Magnetic resonance image showing multiple bilateral infarcts.



B: Computed tomography angiogram showing irregular cerebral vessels ("beading", arrows). ◆

on Day 2 did not detect organic disease. The mental health team diagnosed severe depression and prescribed antidepressants.

On Day 4, the patient's condition deteriorated and she became non-communicative with signs of right hemiplegia. An electroencephalogram showed polyrhythmic generalised slow waves consistent with encephalopathy. She was transferred to a tertiary centre where magnetic resonance imaging (MRI) and CT angiography of the brain showed multiple bilateral infarcts (Figure, A) with beaded arteries, the classic appearance of vasculitis. She was given high-dose prednisolone and cyclophosphamide. Investigations were negative for causes of secondary vasculitis. Her condition continued to deteriorate and she died 8 days after admission. Autopsy was refused. Subsequent review of the second CT scan detected irregular cerebral vessels (Figure, B).

PCV is an uncommon disorder of the central nervous system, with unknown aetiology and no specific characteristic features, affecting small cerebral arteries but not extracranial vessels. Symptoms and signs vary but include headache, encephalopathy, seizures, personality change, weakness, and altered level of consciousness, as well as superimposed focal cranial neuropathy or hemiplegia. Recognition is difficult, but differentiation from reversible cerebral vasoconstriction syndrome is important.^{1,2}

Brain biopsy is seen as the "gold standard" for diagnosing PCV. CT angiography may show diffuse or localised changes, with vessel beading, aneurysms, and luminal narrowing. MRI may show areas of white and grey matter infarction, or haem-

orrhage. MRI is more sensitive than CT, but less sensitive than CT angiography. Up to 100% of biopsy-positive cases appear abnormal on MRI. Suspected cases require careful clinical appraisal and either CT angiography or MRI, probably followed by an image-guided brain biopsy.³

Initial reported cases of PCV had a poor prognosis; most patients died within a few weeks.² Immunosuppressive therapy with glucocorticoids and cyclophosphamide (as used in secondary severe vasculitis) may be beneficial, although there are no clinical trials.⁴ A future therapeutic alternative may be infliximab, which has been used successfully for one patient with cerebral vasculitis secondary to Behçet's disease who had known elevated levels of tumour necrosis factor α .⁵

Despite increasing awareness and advances in angiography, PCV remains an uncommon diagnostic and therapeutic problem which should be considered in cases of severe, non-febrile neurological illness with stroke-like features.

Acknowledgements: We thank Dr Chris Staples, Neurologist, Redcliffe Hospital, for reviewing both the case and this manuscript.

Sanjaya S Herath, Registrar

Dayna B Law, Intern

Peter JO Stride, Physician

Vernon J Heazlewood, Physician

Luke S Gaffney, Registrar

Department of Medicine, Redcliffe Hospital, Redcliffe, QLD.

sanjaya_herath@health.qld.gov.au

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Hepatic encephalopathy precipitated by sodium valproate therapy

H S Subhash, Robert J Heddle, David W Schultz, John Ring and Campbell H Thompson

TO THE EDITOR: We report the case of a 71-year-old woman who presented with a 3-week history of lethargy, subacute confusion and drowsiness. She was known to have a seizure disorder for which she had been taking lamotrigine 100 mg and sodium valproate 500 mg twice a day for 2 years.

On examination, the woman was disoriented with regard to person and time, and had constructional apraxia and asterixis. The rest of the physical examination was unremarkable.

A full blood count, electrolyte levels, coagulation parameters, arterial blood gas measurements and hepatitis serology were normal. Tests for immunological markers of autoimmune liver disease were negative. Liver function tests showed longstanding raised levels of alkaline phosphatase (158 U/L [reference range (RR), 30–110 U/L]) and γ -glutamyl transferase (434 U/L [RR, <40 U/L]). Serum drug levels were sodium valproate 51.0 mg/L (therapeutic range, 50–100 mg/L) and lamotrigine 9.5 mg/L (therapeutic range, 3–14 mg/L). The venous blood ammonia level was 109 μ mol/L (RR, <50 μ mol/L).

A liver ultrasound scan was normal. Computed tomography of the brain showed microvascular changes and an old cortical infarct. An electroencephalogram (EEG) showed diffuse slowing, with a predominance of rhythmical theta activity and some delta activity, suggestive of encephalopathy.

As hyperammonaemic encephalopathy secondary to sodium valproate therapy (VHE) was considered a possible diagnosis, sodium valproate treatment was discontin-

ued. The patient's confusion resolved completely and the asterixis disappeared within a week. At the same time, her blood ammonia level fell to 19 μ mol/L and her EEG normalised.

Eight months after discontinuing sodium valproate treatment, the woman was still asymptomatic. A subsequent percutaneous liver biopsy, to investigate her persistently abnormal liver function, showed features consistent with primary biliary cirrhosis.

Sodium valproate is used not only for management of epileptic disorders but also for migraine prophylaxis and treatment of several psychiatric conditions. Although a generally well tolerated drug, it has a few well known side effects, including hyperammonaemia and, rarely, VHE.¹⁻³ The possible pathophysiology of VHE has been described elsewhere.² Gerstner et al reported on a series of 19 patients with VHE between 1994 and 2003.⁴ Review of the literature suggests that VHE is under-recognised, leading to considerable delay in the diagnosis of this potentially reversible condition.^{3,5}

In our patient, it is reasonable to presume that sodium valproate precipitated the encephalopathy on a background of evolving unrecognised liver disease. The marked improvement in her clinical manifestations after discontinuation of valproate further supports this presumption. We have drawn attention to this case to highlight that VHE should be considered in patients presenting with confusion. Prompt measurement of the ammonia level and cessation of valproate treatment should be considered if clinically appropriate. Patients with previously unrecognised liver disease may be at particular risk.

Acknowledgement: We thank Professor Peter Roberts-Thomson, Director of the Department of Immunology at Flinders Medical Centre, for his expert opinion and advice.

H S Subhash, Registrar¹

Robert J Heddle, Director of Allergy, Department of Respiratory Medicine,¹ and Associate Professor²

David W Schultz, Senior Lecturer, Division of Neurology³

John Ring, Senior Consultant, Division of Gastroenterology and Hepatology³

Campbell H Thompson, Professor, Division of Medicine³

1 Division of Medicine, Flinders Medical Centre, Adelaide, SA.

2 Flinders University, Adelaide, SA.

3 Flinders Medical Centre and Flinders University, Adelaide, SA.

hssubhashcmc@hotmail.com

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A national medical register: balancing public transparency and professional privacy

Peter C Arnold

TO THE EDITOR: The timely article by Healy and colleagues¹ should provoke debate within the profession. The authors' decision to not consider "the relative merits of national versus regional registration boards" should not stifle discussion in the Journal.

In particular, Australian doctors and the public should be aware that the medical board system to which we are accustomed is not operational in most countries. In much of the English-speaking world, boards comprise mainly doctors and have considerable independence from government; medical boards in non-English-speaking countries are generally part of the health bureaucracy.²

The fundamental danger of having a Council of Australian Governments-inspired national registration "body" lies in the potential for a switch from the "English" system of self-regulation under common law to the "European" model of bureaucratic rule under administrative law.

Public and professional suspicion of self-regulation lay behind the 1987 amendments to the *Medical Practitioners Act 1938* (NSW), which removed the power of deregistration from the New South Wales Medical Board, handing it instead to the Medical Tribunal chaired by a District Court Judge. This move avoided both the "Scylla" of public distrust of the profession and the "Charybdis" of criticism, such as have afflicted the boards in Victoria and elsewhere, where boards retained that power.

No less important problems with a national board lie in the assessment of local problems and surveillance of doctors whose registration is conditional. This is already difficult in the larger states, such as Queensland, NSW and Western Australia. The con-

tinued failure of the centralised Health Insurance Commission to prevent and prosecute the abuse of Medicare by doctors, despite repeated ineffectual changes to the legislation,³ does not encourage optimism that a national medical board could effectively manage impaired doctors or those performing below standard.

Having served on Commonwealth working parties on both mutual recognition of medical qualifications and Medicare “over-servicing” and “inappropriate practice”, I would opt for an independent, publicly accessible national database containing a “uniform set of items that are allowable under existing privacy legislation”.¹ The elements of such a database are already operational in the safe hands of the Australian Medical Council (AMC). It should not be too difficult, and certainly less cumbersome, for state and territory governments and medical boards to agree on that uniform set, on the foundations already laid by the AMC, and to continue the AMC’s ownership of the database.

Competing interests: I am a former Deputy President of the NSW Medical Board, and was organiser of a professional protest leading to the 1987 amendments to the NSW Medical Practitioners Act.

Peter C Arnold, Retired General Practitioner
Sydney, NSW.
parnold@ozemail.com.au

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Judith M Healy and Paul Dugdale

IN REPLY: Arnold makes some interesting points about the balance of state and professional involvement in medical registration arrangements. Wherever the balance is struck, politicians, bureaucrats and medical professionals all derive their power in some measure from the public whom they serve.

Our point is that variations in registration information and public access to multiple registers make it difficult for members of the public to access and use the information, especially given the mobility of the medical workforce between jurisdictions. A public national medical register should be seen as a practical measure to improve public accountability, rather than as a battleground between the profession and the bureaucrats.

We did not express a view in our article¹ on how national access to medical registra-

tion details should be arranged. As Arnold suggests, the Australian Medical Council is one candidate for maintaining a national database. It is well placed to publicly call on the existing state medical registration boards to cooperate speedily to make national access a reality, pending the negotiations underway regarding a national medical board.

Judith M Healy, Senior Fellow, Regulatory Institutions Network (RegNet)

Paul Dugdale, Director, Center for Health Stewardship

Australian National University, Canberra, ACT.
judith.healy@anu.edu.au

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The real costs of lifetime tobacco usage

C Ross Philpot

TO THE EDITOR: With the World Health Organization’s annual World No Tobacco Day to be held on 31 May, it is timely to encourage all patients who smoke to reconsider their actions.

During my registrar training in the 1970s, I developed a simple and effective method of helping smokers consider some consequences of their tobacco habit. I offer this in the hope that others may find it useful too, as a more meaningful exercise than the concept of “pack-years”.¹⁻³

First, enquire when the patient began to smoke regularly. I call this the “tobacco-arche” (analogous to menarche and coit-arche).

Next, determine how many years the patient has smoked regularly, remembering to subtract any years he or she may have suspended the habit.

Then, have the patient estimate overall daily usage, relying, if possible, on prompting from an accompanying person to determine a realistic rather than idealised figure.

Finally, multiply the number of years by the daily usage and by the number of days in

a year. A reasonable approximation is to multiply by 400 rather than the more cumbersome 365.25.

For example: a 65-year-old person who has smoked 20 cigarettes per day since his or her mid teens (ie, for 50 years) yields $20 \times 50 \times 400 = 400\,000$ — approaching half a million cigarettes lifelong.

In my three decades of experience as a general physician, I have noted that an accumulated intake of a quarter of a million cigarettes usually results in at least some cough, breathlessness and end-expiratory wheeze on forced expiration, and decreased exercise tolerance; half a million cigarettes generally causes chronic smoker’s bronchitis, with or without some degree of emphysema, and other harmful effects on the body; while three-quarters of a million cigarettes makes cancer a distinct possibility.⁴

A further inducement for patients to confront the effects of their harmful habit is to calculate the amount of their lifetime tobacco intake in terms of the dollar cost. In the case of cigarettes, 500 000 at 60 cents each yields the impressive figure of \$300 000. This usually comes as a sobering revelation to the smoker and their “significant others”.

The above method can thus contribute to the desirable effect of reducing or eliminating tobacco consumption, with flow-on benefits to patients’ health, finances, and personal and occupational relationships.

C Ross Philpot, Physician

South Australian Infectious Diseases Services, Adelaide, SA.

ross.philpot@nwahs.sa.gov.au

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Medical specialist education and training in Australia

Rufus M Clarke and Michael K Morgan

TO THE EDITOR: In his article on medical specialist education and training in Australia, Phelan¹ raises two major issues: financial and educational.

On the financial front, Phelan asks about the advantages and disadvantages of the new funding model for specialist (pre-Fellowship) training. One of the advantages of the model is that it makes the funding process more explicit. We believe that all who benefit from this educational exercise — trainees, supervisors, private hospitals and society — should contribute financially in some way.

On the educational front, Phelan is disappointed that we did not provide evidence that university education will enhance the educational experience of trainees. However, undertaking educational studies that meet the standards required of reductionist experiments has proven to be difficult, and we have to make do with a more ecological approach.

We have no doubt that our Macquarie University scholars will derive lasting benefit from working in an environment in which learning is one of the primary goals of their existence, rather than an add-on, after-hours, activity. Learning arises not from watching, but from doing. Modern concepts of neurobiology and learning suggest that learning results in structural changes in the brain, and these will be enhanced for the learner by full participation in all processes of care. We shall ensure that learning is maximised by an appropriate balance between scholars' clinical experience and the educational opportunities that their clinical experience will provide.

The Canadian contracting model, which Phelan mentions, is not dissimilar to the arrangement that will flow from the Memorandum of Understanding between Macquarie University and the Royal Australasian College of Surgeons and the Neurosurgical Society of Australasia, in that College trainees will substitute experience at Macquarie University for time

spent in public hospitals in the College's Surgical Education and Training Program.

In Australia, we do not share the Canadians' advantage of having only two postgraduate colleges. Given the current fragmented state of postgraduate medical education in Australia, we believe that it is better to experiment with and to evaluate new models than slavishly to copy what appears to work in a different setting. One of the flavours of the decade is competition, and we believe that competing models should be set up and should be rigorously evaluated from both educational and financial viewpoints.

If history shows that the Macquarie lighthouse has illuminated the way to improved health for the Australian people, we shall be well satisfied.

Rufus M Clarke, Professor of Medical Education
Michael K Morgan, Professor and Dean
 Australian School of Advanced Medicine,
 Macquarie University, Sydney, NSW.
rufus.clarke@bigpond.com

¹ Phelan PD. Medical specialist education and training in Australia. *Med J Aust* 2007; 187: 687-688. □

Records of the Australian Mesothelioma Surveillance Program have been lost!

Marc Hendrickx

TO THE EDITOR: I recently received written advice from the Australian Safety and Compensation Council (a division of the Department of Employment and Workplace Relations) that the records of the Australian Mesothelioma Surveillance Program (AMSP) have been lost.

As some of your readers would be aware, the AMSP, which ran between 1980 and 1985, was one of the most comprehensive medical surveys of mesothelioma undertaken anywhere in the world.¹ The records of the program contain full occupational and environmental histories of about 1000 mesothelioma cases reported in the early 1980s. The program has played a significant role in helping to understand the epidemiology of mesothelioma in Australia. The level of detail of data in the AMSP has not been repeated by the Australian Mesothelioma Register, which succeeded the AMSP in 1985. This less detailed reporting scheme is the current basis for mesothelioma reporting to cancer registries in the country.

I am a geologist with an interest in medical geology currently studying to obtain a doctorate on naturally occurring asbestos and mesothelioma risk in Australia. I had hoped to use the detailed environmental and occupational data of the AMSP to help determine the possible influence of naturally occurring asbestos on mesothelioma in Australia, in particular in the eastern states and South Australia, but without the records this is no longer possible. Data from the Australian Mesothelioma Register are not sufficiently detailed for this purpose.

My intention in writing this letter is not to embarrass staff from the Australian Safety and Compensation Council, who have done their best to find the records and have been supportive of the project, but to create awareness of the loss, in the hope that the publicity may jog someone's memory and result in the records being located. The potential permanent loss of these records would be a great loss to mesothelioma research in Australia and raises questions about the federal government's policies surrounding long-term storage and archiving of nationally significant scientific research datasets that may be of benefit to future researchers.

Marc Hendrickx, PhD Candidate
Graduate School of the Environment,
Macquarie University, Sydney, NSW.
mhendrickx@internode.on.net

¹ Leigh J, Robinson BWS. The history of mesothelioma in Australia 1945–2001. In: Robinson BWS, Chahinian AP, editors. Mesothelioma. London: Taylor and Francis, 2002: 55–86. □

Julie Hill

COMMENT: The Australian Mesothelioma Surveillance Program (AMSP) operated between 1980 and 1985 and was maintained by the Commonwealth School of Public Health and Tropical Medicine at the University of Sydney. These files were transferred to the National Occupational Health and Safety Commission (NOHSC) on its establishment in 1985. The NOHSC was relocated from Sydney to Canberra in 2001, and AMSP records went into storage at that time.

In February 2005, the NOHSC was succeeded by the Australian Safety and Compensation Council. We attempted to locate the records over several months in 2007. This involved manually searching through all files and boxes held by our contracted storage company marked as relating to either the AMSP or the Australian Mesothelioma Register. In addition, we had a staff member of the storage facility manually search the warehouse for these records in case they were in unmarked boxes or filing cabinets.

In November 2007, having been unable to locate the records, we informed Mr Hendrickx that we would be unable to assist him with access to the AMSP records for his doctoral studies.

It is certainly not our policy to discard records such as these and we were disappointed when they could not be easily located. We regret the potential loss of these important records to the research community and are still attempting to locate them.

Julie Hill, Director
Data Analysis Section, Office of the Australian Safety and Compensation Council, Department of Education, Employment and Workplace Relations, Canberra, ACT.
julie.hill@deewr.gov.au □

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The Medical Journal of Australia (MJA) is published on the 1st and 3rd Monday of each month by the Australasian Medical Publishing Company Proprietary Limited, 227 Clarence Street, Sydney, NSW 2000. ABN 20 000 005 854. Telephone: (02) 9562 6666. Fax: (02) 9562 6699.

E-mail: medj@ampco.com.au. The Journal is printed by Webstar Australia, 83 Derby Street, Silverwater, NSW 2128.

MJA on the Internet: <http://www.mja.com.au/>

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Published in 2 volumes per year.

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Individual Subscriptions (includes 10% GST)

Australia: \$A368.50, Medical students (Australia only): \$A60.00

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Indexes are published online every 6 months.

Single or back issues contact: AMPCo (02) 9562 6666.

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28264 circulation as at

29 October 2007



ISSN 0025-729X