LETTERS

Mis-deca-n identity?

Elizabeth A S Giugni, Rachel S Boddy and Natalie G Limet

To the Editor: We report two cases of previously well male bodybuilders who presented with severe extrapyramidal reactions after intramuscular injection of the antipsychotic fluphenazine decanoate, in the mistaken belief that it was an anabolic steroid.

The first patient, aged 31 years, obtained fluphenazine decanoate from a gym contact. He injected 50 mg intramuscularly on alternate days (Days 1, 3 and 5) to a total of 150 mg, then presented to two local hospitals on Days 7 and 11 with difficulty swallowing, generalised muscle stiffness and lethargy. He withheld the history of fluphenazine use, and was diagnosed with tonsillitis. On Day 14, he presented to our emergency department (ED) with marked dystonia, immobility, and inability to speak or swallow food. On examination, he was afebrile and haemodynamically stable. He was given a trial dose of benztropine 2 mg, but improvement was slight and, given the absence of relevant history, benztropine was not repeated. The neurology team raised the possibility of a conversion disorder, but the psychiatry team, noting the patient's attempts to speak and an absence of recent stressors, believed that further investigation into an organic cause was required. When the patient's wife learned that he had used fluphenazine and alerted the neurology team to this use, he was started on regular benztropine and his condition improved over the next 3 days. The dose of benztropine was reduced on discharge, but his dystonia recurred and required readmission to hospital for further treatment.

The second, unrelated patient, also aged 31 years, openly admitted purchasing fluphenazine decanoate from "a friend of a friend". After injecting two 50 mg depots, he had multiple presentations to three EDs, where he was treated for dystonia with immediate doses and then regular low doses of benztropine. On admission to our hospital 19 days after injection, he was afebrile and haemodynamically stable, with marked dystonia. His initial creatine kinase level was elevated (553 U/L; normal, < 204 U/L), but subsequently normalised and was not accompanied by autonomic dysfunction. His condition improved with regular oral diazepam and benztropine, but symptoms recurred when he inappropriately reduced his benztropine dose after discharge.

On subsequent review, both patients' dystonia was resolving, but they had significant akathisia.

Inadvertent and inappropriate use of a long-acting phenothiazine not only required prolonged anticholinergic therapy for these men, but we believe placed them at risk of neuroleptic malignant syndrome. We have found no previous similar reports in the medical literature, but are aware anecdotally of at least one other case of a patient treated recently at a district hospital.

The anabolic steroid nandrolone decanoate is referred to colloquially on numerous websites and by our patients as "deca" (from the Organon brand name Deca-Durabolin). We believe our patients and their supplier(s) have mistaken the "decanoate" in fluphenazine decanoate for the pharmacologically active component of the drug.

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Non-compliance with Western Australian smoke-free legislation: a complete ban on smoking in hospitality settings is needed

William J Patterson, Michael M Daube, Stephen L G Hall and Denitza Moronova

To the Editor: The introduction of indoor smoking restrictions in Western Australian pubs, clubs and nightclubs[1] has been championed as a public health success and an advance in tobacco control. But how well is it being observed, and is the current legislation adequate?

Although smoking in Australia is declining, a report showed that one in seven 16–17-year-olds had smoked cigarettes in the previous month.[2] Peer pressure has an important influence on smoking uptake,[3] and alfresco hospitality settings, being exempt from the smoke-free restrictions, constitute important risk environments for young people. Moreover, 85% of adults in WA do not smoke, but continue to be affected by second-hand smoke drifting from alfresco balconies, beer gardens and street cafes. Indeed, the legislation risks turning these popular and highly visible entertainment areas into nicotine classrooms[4] for the young, and "no-go" zones for health-conscious non-smokers.

We set out to evaluate compliance with the smoke-free legislation, and its impact on smoking in licensed premises. Twenty medical students were recruited to monitor smoking in 93 Perth hotels and nightclubs on two Friday evenings during November 2006. Entertainment areas were classified as *indoor* (smoke-free), *semi-outdoor* (smoke-free, failing to meet exemption criteria) and *alfresco* (smoking allowed), in accordance with the new legislation. Average observation times were 20 minutes, 17 minutes and 15 minutes respectively.

Indoor compliance with the legislation was high, with smoking noted in only five (5%) of the 93 premises. Whether by default or design, 58 premises (62%) had alfresco areas and there was smoking activity in those areas in 56 (97%) of these premises. Forty premises (43%) also had semi-outdoor areas where, contrary to the legislation, smoking remained prevalent (23 premises; 57.5%). Overall, smoking was observed in 69 of the 93 premises (74%). In 35 of these premises (51%), smoking was visible to the passing public.

These results demonstrate the inadequacy of the new smoke-free legislation in restricting smoking, protecting the health of non-smokers and strengthening tobacco control. The legislation has been well accepted, but smoking (legal and illegal) remains prevalent in licensed premises. This presents a serious challenge to tobacco control and efforts to reduce smoking by young people. A key lesson from the history of tobacco control is that partial bans achieve partial results.[5] A complete ban on smoking, both inside and outside licensed premises and restaurants, is urgently required.

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2 Government of Western Australia Drug and Alcohol Office. ASSAD smoking report 2005. A research report. April 2007. TNS Social Research Job No.

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5 Koh HK, Joossens LX, Connolly GN. Making smoking history worldwide. *N Engl J Med* 2007; 356: 1496-1498. ❑

Management of warfarin in atrial fibrillation

Peter W Ford and Angela Close

To the Editor: Bajorek et al[1] did not address two aspects of compliance that may be an issue in community care of patients with atrial fibrillation taking warfarin: time of dose, and point-of-care testing.

There is no pharmacological reason requiring warfarin administration in the evening. This practice arose to facilitate dose adjustment on the day of testing, initially in hospitals, and subsequently flowed on to community care. We know that compliance is better with once-daily administration regimens, and this patient group invariably needs other medications, such as diuretics, that require morning doses. Concomitant morning administration of warfarin would be logical. In addition, it would reduce attendances by domiciliary nurses to cognitively impaired patients, who may otherwise require twice-daily visits for administration of medications. This would alleviate a significant burden on this stretched resource.

The implementation of point-of-care testing at the general practitioner's surgery by a registered nurse can be of great benefit in the liaison required to manage therapy, and facilitates instant dose adjustments by the doctor, who has comprehensive knowledge of the patient's pharmaceutical and health circumstances.[2]

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1 Bajorek BV, Ogle SJ, Duguid MJ, et al. Management of warfarin in atrial fibrillation: views of health professionals, older patients and their carers. *Med J Aust* 2007; 186: 175-180.
2 Shiach C, Campbell B, Poller L, et al. Reliability of point-of-care prothrombin time testing in a community clinic: a randomized crossover comparison with hospital laboratory testing. *Br J Haematol* 2002; 119: 370-375. ❑

Beata V Bajorek

In reply: I thank Ford and Close for highlighting additional points regarding the optimal management of anticoagulants in general practice. Indeed, neither of these points was raised by our study participants.

Regarding the timing of doses, for medication safety reasons, in many hospitals the warfarin dose is listed for mid-evening administration; the recently introduced National Inpatient Medication Chart (NIMC), which incorporates a designated "warfarin section", nominates 16:00 as the time.[1] In the hospital setting, this timing is necessary to enable the treating medical team (rather than after-hours staff) to review the day's blood test results, and subsequently prescribe the appropriate dose. The process ensures that treatment is optimally managed by those most knowledgeable about the patient's regimen, prevents dose omissions, and reduces the time to dose stabilisation (and potentially, time to discharge). Ford and Close appropriately point out that this timing may not always be convenient for patients once they are discharged to the community setting. The optimal regimen should facilitate the patient's adherence to treatment, and therefore should coordinate with the rational use of existing support services. This needs to be more carefully considered in discharge planning when warfarin therapy is involved.

Point-of-care testing is an efficient mode of monitoring anticoagulation therapy, but we were unable to expand on this in our previous discussion (due to word limits). Internationally, point-of-care testing underpins many comprehensive monitoring services, whereby allied health professionals (eg, trained nurses or pharmacists) perform the blood tests, monitor results, adjust doses, and/or prescribe therapy, as well as educate patients, under the guidance of a medical officer. Such services are conventionally offered on an outpatient basis (eg, the Antithrombosis Center, University of Illinois Medical Center, Chicago, Ill, USA) or within the general practice setting,[2] and are effective and safe models of care. Patient self-management using point-of-care testing devices has also been studied overseas,[3] with reports of good control of international normalised ratio (INR) and high patient satisfaction. Locally, point-of-care testing has been trialled within community pharmacies. In a Sydney-based study, trained community pharmacists monitored INRs using point-of-care testing, reviewed doses according to standardised nomograms, and subsequently liaised with GPs regarding dose adjustments. The results showed that collaborative management effectively maintained INRs within the therapeutic range.[4] There is scope to develop such models further, and we are currently investigating GPs' preferences for models of care, as well as opportunities for mobile anticoagulation services.

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1 New South Wales Health Department. Guidelines for prescribing, dispensing and administering warfarin. Safety Notice SN: 006/07. 12 April 2007. http://www5.health.nsw.gov.au/quality/sabs/pdf/SN00607_warfarin.pdf (accessed Aug 2007).
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Town or country: the ARIA index is not an accurate indicator of access to health services

Alan E Dugdale

To the Editor: In a recent article,[1] Scrimgeour showed the differences between death rates in Aboriginal people and in the general Australian population. He used the Accessibility/Remoteness Index of Australia (ARIA)[2] to show that Aboriginal people in remote areas generally had higher death rates than those near major health facilities. However, this widely used tool is inaccurate when applied to some health facilities.

Cherbourg Aboriginal community (population about 2600) is located in the South Burnett district of south-eastern Queensland, 260 km by road from Brisbane. It has a 10-bed hospital with two resident doctors. There are several small towns (population 1000–3000) in the district. Two have hospitals with no full-time medical staff. The main centre is Kingaroy (population about 12 000), 50 km from Cherbourg. It has a 60-bed hospital with resident staff, but no specialists or consultants. There are general practitioners

but no medical specialists in the district, although some specialists visit for a day or so per month. Most patients needing specialised services go to public hospitals in Brisbane (220 km away [from Kingaroy]), Toowoomba (170 km away) or Nambour (140 km away), where waiting lists are long. Children go to children's hospitals in Brisbane, either by road (a 3-hour journey) or by helicopter.

The ARIA index ranges from 0.0 (major city) to 12.0 (very remote area). Cherbourg (classed with Murgon, 5 km from Cherbourg) has an index of 2.9 and Kingaroy, 2.6. The cities of Darwin, Cairns and Townsville, all with major hospital and health facilities, have an ARIA index of 3.0, while Alice Springs, which also has a major hospital and specialists, has an ARIA index of 6.0. Clearly, the remoteness index is not a good indicator of the availability of local specialist health services.

The ARIA classification is widely used. The anomaly of the South Burnett and Cherbourg Community may (or may not) be the only problem with the index. Until such anomalies are corrected and ARIA is validated, any results derived using the index should be treated with caution.

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1 Scrimgeour D. Town or country: which is best for Australia's Indigenous peoples? *Med J Aust* 2007; 186: 532-533.
2 Measuring remoteness: Accessibility/Remoteness Index of Australia (ARIA). Revised edition. Canberra: Commonwealth Department of Health and Aged Care, 2001. (Occasional papers: New Series No. 14.) http://www.health.gov.au/internet/wcms/Publishing.nsf/Content/9E3A2AAA51E74D2ACA256F1800575941/$File/ocpanew14.pdf (accessed Aug 2007). ❑

Guidelines for the management of acute coronary syndromes 2006

Mark Little and Chris Johnstone

To the Editor: The *Guidelines for the management of acute coronary syndromes 2006*[1] state: "Enoxaparin may be used in conjunction with fibrin-specific fibrinolytic agents in patients under the age of 75 years, provided they do not have significant renal dysfunction. An intravenous bolus dose of 30 mg followed by a 1 mg/kg subcutaneous injection every 12 hours in combination with tenecteplase is the most comprehensively studied therapy."[1]

In Australia, enoxaparin is not licensed for intravenous use (Tony Hall, Team Leader, High Risk Medications and Systems, and Christine Maclean, Associate Director, Safe Medication Practice Unit, Queensland Health, personal communication) and there is no recommendation for the intravenous use of enoxaparin in the drug product information.[2] Are the authors recommending "off-label" use of intravenous enoxaparin, or do they wish to modify the guidelines to reflect what the management should be if clinicians are unable to use intravenous enoxaparin?

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1 Acute Coronary Syndrome Guidelines Working Group. Guidelines for the management of acute coronary syndromes 2006. *Med J Aust* 2006; 184 (8 Suppl): S1-S32.
2 Enxoaparin. Enoxaparin (Clexane) product information. Sydney: Sanofi-Aventis Australia, 22 June 2005. ❑

Constantine N Aroney and Philip Aylward, for the National Heart Foundation of Australia and the Cardiac Society of Australia and New Zealand

In reply: The guidelines were published to provide clinicians with the most contemporary information on the management of acute coronary syndromes based on the international literature, and may include treatments which are not currently available, officially licensed or available through the Pharmaceutical Benefits Scheme in Australia.

In the context of adjuvant therapy for patients with ST-segment-elevation myocardial infarction (STEMI), the guidelines recommend that antithrombin therapy should be used with fibrin-specific fibrinolytic agents.[1] Based on the best evidence available at the time, the guidelines mention two antithrombins, unfractionated heparin and enoxaparin, to be considered for use in this setting. The recommendation for enoxaparin is based on comprehensive evidence of clinical benefit with the regimen of an initial intravenous (IV) bolus dose followed by subcutaneous injections every 12 hours. It is up to individual practitioners to determine whether the IV dose should be provided "off-label" or omitted, based on the evidence and the circumstances of the individual patient and setting.

The issue of superiority of enoxaparin over unfractionated heparin as adjuvant therapy for patients with STEMI is currently being evaluated in light of recent evidence,[2] and will be included in a future update of the guidelines.

Competing interests: We are consultants for, advisory committee members of, or receive honoraria, fees for service, or travel assistance (independent of research-related meetings) from, or have research or other associations with the organisations listed: Constantine Aroney — CSL, Merck Sharpe & Dohme, Sanofi-Aventis; Phil Aylward — Sanofi-Aventis, Pfizer, Merck Sharpe & Dohme, Bristol-Myers Squibb, Boehringer Ingelheim, AstraZeneca, Procter & Gamble, Eli Lilly, The Medicines Co, Servier, CSL, Schering Plough.

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Amoebiasis: current status in Australia

Chien-Ching Hung

To the Editor: I read with great interest the recent updated review of amoebiasis by van Hal and colleagues[1] and their previous letter[2] describing three cases of locally acquired amoebiasis due to *Entamoeba histolytica* in Australian men who have sex with men (MSM). These articles should alert clinicians to the emergence of invasive amoebiasis and the possibility of person-to-person transmission of *E. histolytica* through oral–anal or oral–genital sex among MSM in developed countries. The same phenomenon has been reported in Taiwan[3] and Japan.[4]

The prevalence or incidence of intestinal amoebiasis among people at risk may have been underestimated in the past, as microscopy of stool specimens has lower sensitivity and specificity than *E. histolytica* antigen

detection methods for diagnosing the disease.[1,5] Cases of amoebiasis may evade detection using the diagnostic algorithm proposed by van Hal and colleagues,[1] which suggests using microscopy of stool specimens to detect *E. histolytica* complex followed by confirmation with specific antigen detection methods or molecular methods. To increase diagnostic sensitivity and specificity, I suggest revising the diagnostic algorithm for intestinal amoebiasis in developed countries to include more accurate first-line detection methods. For example, specific antigen detection methods or polymerase chain reactions, as proposed by Tanyuksel and Petri,[5] could be incorporated.

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1 van Hal SJ, Stark DJ, Fotedar R, et al. Amoebiasis: current status in Australia. *Med J Aust* 2007; 186: 412-416.
2 Stark DJ, Fotedar R, Ellis JT, Harkness JL. Locally acquired infection with *Entamoeba histolytica* in men who have sex with men in Australia [letter]. *Med J Aust* 2006; 185: 417.
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5 Tanyuksel M, Petri WA Jr. Laboratory diagnosis of amebiasis. *Clin Microbiol Rev* 2003; 16: 713-729. ❑

Paul Prociv

TO THE EDITOR: van Hal and colleagues deserve congratulations for their lucid, concise and timely review of the complex problem of human amoebic infection and its diagnosis.[1] Not surprisingly, however, their article raises more questions than it answers.

To me, the gist of their message was as follows: what was in the past diagnosed as *Entamoeba histolytica* infection, based on the microscopic identification of organisms from faeces, culture or histological sections, actually may have been caused by other species, viz. *E. dispar* (a recently described non-pathogen) or *E. moshkovskii* (known for a long time from sewage samples, but only recently found to infect humans). Because these species are all identical morphologically, they can be distinguished reliably only by sophisticated molecular techniques.

To complicate matters further, despite *E. dispar* having been virtually defined as the "non-invasive form" of *Entamoeba*, most true *E. histolytica* infections are still asymptomatic.[2] Not only the parasite, but also individual host factors (perhaps including genetics), determine pathogenicity. Thus, not all people infected with the same pathogenic strain will manifest symptoms or signs of invasive disease. Given their biology and evolution, it is conceivable that, eventually, invasive strains of even *E. dispar* will be discovered! Furthermore — and this seems not to have been investigated yet — mixed infections involving different species and strains of these parasites almost certainly occur (not to mention the "traditional" non-pathogenic amoebae, which frequently do occur in mixed infections).

The authors advocate treatment of even asymptomatic *E. histolytica* infections, but how would these be detected outside epidemiological surveys or healthy population screening programs? Given the difficulty and expense of specifically identifying the infective organism even in symptomatic cases, and the relative cheapness of treatment, surely it would be sufficient simply to treat on the basis of clinical presentation

plus the identification of *E. histolytica*-like parasites, with or without objective evidence of histopathology. Anything more could be justified only within the context of a well funded and carefully designed research program and/or epidemiological study.

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2 Stanley SL Jr. Amoebiasis. *Lancet* 2003; 361: 1025-1034. ❑

Sebastiaan J van Hal, Damien J Stark, Debbie Marriott and Jock L Harkness

In reply: We agree with Hung that molecular and antigen testing methods are more sensitive for *Entamoeba histolytica* detection than microscopy and that reliance on microscopy alone would result in under-detection. Our algorithm[1] was presented the way it was for several reasons. Firstly, both molecular and antigen testing are significantly more expensive than microscopy. Secondly, as these tests can currently only detect a single pathogen, they would not replace microscopy. Most patients, especially men who have sex with men (MSM), have multiple intestinal parasites, so the more specific methods would remain an adjunct in parasite detection.[2] Thirdly, the positive predictive value of any test is dependent on the prevalence of the disease. The prevalence of *E. histolytica* in Australia, based on current data, is less than 1% in high-risk populations, including MSM. Thus, at present, molecular and antigen tests would be more likely to give false positive than true positive results. However, we agree that our algorithm could be modified as suggested if prevalence rates were between 5% and 10%. Finally, as seen in the MSM population in Taiwan, this is not a static situation, and ongoing local surveillance is required.[3]

We agree with Prociv that, before the introduction of molecular techniques, *E. histolytica* prevalence would have been overestimated. We also agree that specific host factors and/or undefined parasitic virulence factors can lead to invasive disease. However, given the extensive molecular work that has been undertaken, we believe it unlikely that invasive strains of *E. dispar* will be discovered.[4] Furthermore, recent studies show that mixed infections are common.[2,5]

In symptomatic patients, empirical amoebicidal therapy is warranted. However, to ensure that alternative diagnoses (eg, inflammatory bowel disease) that require different treatment are not overlooked, all attempts to accurately speciate *Entamoeba* complex should be undertaken. We acknowledge that speciation using the polymerase chain reaction is beyond the means of most laboratories, but this is not the case for enzyme immunoassay testing of stool samples, which is rapid, sensitive and relatively cheap.

For asymptomatic patients who are carriers of *E. histolytica* cysts, the World Health Organization recommends treatment.[5] However, in areas of low prevalence such as Australia, *Entamoeba* cysts are more likely to be non-pathogenic *E. dispar* or *E. moshkovskii* species than *E. histolytica*.[3] Thus, in Australia, treatment would be unnecessary in a high proportion of patients. Furthermore, therapy requires a luminal agent (paramomycin), which is difficult to obtain. The most practical solution is to either give no treatment or to treat only those patients who have tested positive for *E. histolytica*.

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2 Stark D, Fotedar R, van Hal S, et al. Prevalence of enteric protozoa in human immunodeficiency virus (HIV)-positive and HIV-negative men who have sex with men from Sydney, Australia. *Am J Trop Med Hyg* 2007; 76: 549-552.
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4 Ackers JP, Mirelman D. Progress in research on *Entamoeba histolytica* pathogenesis. *Curr Opin Microbiol* 2006; 9: 367-373.
5 World Health Organization/Pan American Health Organization/UNESCO report of a consultation of experts on amebiasis. *Wkly Epidemiol Rec* 1997; 72: 97-99. ❑

CORRECTION

Re: "**Influenza outbreak related to air travel**", by Andrew G Marsden, in the 4 August 2003 issue of the Journal (*Med J Aust* 2003; 179: 172-173). The title should have been "Outbreak of influenza-like illness related to air travel", consistent with the text. ❑

Why would anyone be an academic?

Ajay Rane and Caroline de Costa

To the Editor: Two recent articles in the Journal touch on the current plight of medical academics. Hays emphasises the need to reassert the role of teaching in academic medicine — otherwise, "our aim to produce safer, more efficient doctors will be under threat".[1] Joyce and colleagues outline the projected increase in the number of graduates from Australian medical schools, although their concern is more for the postgraduate careers of these young doctors than for their initial clinical training.[2]

In 2006, Van Der Weyden warned that not only do new ways of teaching medical students need to be rapidly explored but also that more skilled teachers must be found and trained.[3] Medical students themselves believe that more clinical teachers are required to ensure that the increasing numbers of students are taught effectively.[4]

From personal experience we know that, while many full-time clinicians are both willing and inspiring teachers at undergraduate level, a core of permanent academics is needed to direct teaching during these clinical years. But why would anyone choose to be a medical academic today?

Certainly not for the money — a recently qualified obstetrician/gynaecologist, starting at senior lecturer level after about 15 years of training, is looking at an income of less than half that of a staff specialist at the same level, and a quarter of that possible in private practice. One day of private practice per week does not help — obstetrics is a full-time commitment, and even in gynaecology the need to pay practice and indemnity costs outweighs any financial benefits.

Is it the kudos? Adjunct academic titles are easily gained by non-academics: hospitals are awash with adjunct associate professors and lecturers. The adjunct appointment system often lacks regular and critical appraisal, and in some cases titles are used to the professional or financial gain of the recipient, with little reciprocal input into teaching at the institution concerned. (However, there are indications of attempts to crack down on such practices.)[5]

The lifestyle then? Academics may have a less frenetic clinical schedule, but the continuing pressure to produce quality research and to jump increasingly higher hurdles to obtain grants can mean that the limits of the working week are much less defined than for our staff specialist colleagues. We have

seen many colleagues depart academia in the past few years for the more verdant pastures of full-time clinical practice.

While we are in agreement with Hays about the need for more research into how best to design medical education, we believe that, unless there are urgent improvements in the remuneration, career structure and professional regard for clinical academics, the core workforce of skilled teachers so clearly needed for incoming students will just not be there to deliver that education.

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2 Joyce CM, Stoelwinder JU, McNeil JJ, Piterman L. Riding the wave: current and emerging trends in graduates from Australian university medical schools. *Med J Aust* 2007; 186: 309-312.
3 Van Der Weyden MB. Increased medical school places: a crisis in the making? *Med J Aust* 2006; 185: 129.
4 Blackham RE, Rogers IR, Jacobs IG. Medical student input to workforce planning [letter]. *Med J Aust* 2006; 185: 55-56.
5 Medical Board of Queensland. Notice to all medical specialists in Queensland — information regarding clinical teachers and academic titles with medical schools [leaflet]. Brisbane: MBQ, 2007. ❑

Patient privacy and Latin: my father's story

Peter Piazza

To the Editor: Like Haley's father,[1] I too lament the fact that Latin terms have fallen out of use in medical terminology. I also think it retrograde that Latin is being taught to fewer and fewer of our secondary students, as they are missing out on an opportunity to learn so much more about our own language, let alone terms that they might use later in their clinical practice.

However, I do not think that it was Latin that helped the young teacher out of a difficult predicament in the 1950s. I can assure Haley that "pseudocyesis" is all Greek to me.

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1 Haley KA. Patient privacy and Latin: my father's story [letter]. *Med J Aust* 2007; 186: 328. ❑

James Mitchell

To the Editor: Thank you for the charming letter about "non-pseudocyesis" published in the 19 March issue.[1] It reminded me of the Brander Matthews quote: "A gentleman need not know Latin, but at least he should have forgotten it." This is particularly apt, as the term "pseudocyesis" is from Greek.

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1 Haley KA. Patient privacy and Latin: my father's story [letter]. *Med J Aust* 2007; 186: 328. ❑

William E M Renton-Power

To the Editor: The tale told by Katherine Haley's father about his clever advice on how to protect the privacy of a young pregnant woman makes a good story.[1] However, while "non" is a Latin word, "pseudocyesis" is very good Ancient Greek (*ψευδοκύησις*). Queen Mary I ("Bloody Mary"), who was believed for many months to be with child but ultimately returned to court childless, may have suffered from pseudocyesis.

I agree with Dr Haley's opinion about the lamentable decline in the use of both Latin and Greek terms in medical practice. I take the time to teach my students, residents and registrars enough Latin and Greek to render classical plurals correctly (eg, fistulae, diverticula, carcinomata). Whether they take any notice of this classical teaching is another matter.

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Roger K A Allen

To the Editor: I was both delighted and a little perturbed to read the recent letter by Katherine Haley about her general practitioner father who outsmarted the Department of Education by a medical sleight of hand, stating that his patient had "non-pseudocyesis".[1] However, it was not Latin, but Greek, that did the trick. Despite Dr Haley's background in Latin, he, like all physicians, had unwittingly used Greek during his medical course.

I was fortunate to do a year of Ancient Greek during my undergraduate medical course and, like my late GP father, also did Latin for matriculation. It is unfortunate that Greek is no longer taught in Queensland schools, and Latin only in a few. I believe that we are the poorer for this, and it shows in all sorts of ways, including a general decline in literacy even at the tertiary level. A classical grounding demands scholarship and precision. I have found that knowledge of both Greek and Latin has enriched my knowledge of English literature and Western philosophy, as well as French. Such a grounding also trains the mind in analytical and ordered thought and provides greater insight into the workings of syntax and grammar and a greater facility with words — the building blocks of our language, many of which are derived from Greek and Latin.

At the risk of stating the obvious, I will remind readers that anatomical words in medicine are predominately Latin or Greek and that the names of nearly all symptoms and diseases are Greek (with some exceptions, such as "angina pectoris"). In Dr Haley's time, it was common for potential doctors to receive an arguably more "polished" education that embraced one or two languages, including Latin and, in some private schools, Greek. Alas, the current system of medical student selection favours those "*idiots savants*" who excel in mathematics and science and not the arts. With respect, I would like to point out that in the term "non-pseudocyesis", the only Latin component is "non", the rest being Greek (*ψευδής*, false; *κύησις*, pregnancy).

For both the enthusiast and the non-classicist, may I recommend my father's 5th edition of *Gould's medical dictionary* (1943), which contains the etymology of every medical word.

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