LETTERS

A case for altruistic surrogacy

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To the Editor: One of the great privileges in practising obstetric medicine is to support a couple through a successful confinement when they have previously been advised against attempting pregnancy because of pre-existing maternal disease. However, in some cases, pregnancy carries a substantial risk of morbidity and mortality to both the mother and infant. Indeed, many maternal deaths in Australia are still preventable,[1] and underlying cardiac disease is an important cause.[2]

Recently, I was consulted for preconception counselling by a young woman with dilated cardiomyopathy. Based on the limited evidence in the literature, her risk of dying as a result of pregnancy would be greater than 25%.[3] Similarly, I was recently involved in the care of a young woman with Eisenmenger syndrome who elected to terminate her pregnancy due to the 50% mortality associated with pregnancy with this condition.[4] Pregnancy in young women with moderate renal failure carries a significant risk of permanent decline in renal function, along with a high risk of intrauterine growth retardation and prematurity for the baby.[5] Organ transplantation offers the best hope for women in this situation, as pregnancy outcomes are excellent after solid organ transplantation (with the exception of lung transplantation). However, many women have organ dysfunction severe enough to compromise pregnancy outcome, but not to warrant transplantation.[6]

Pregnancy in the presence of maternal disease may also pose a substantial cost to the community. A 2001 study in the United Kingdom estimated the mean cost of pregnancy care for five mothers with severe cardiac disease to be £23 000, not including the cost of neonatal care.[7] One mother and one baby died.

Options for couples with pre-existing maternal disease are limited. In Queensland, they are excluded from adopting a child because they are not infertile and because of the mother's medical condition. Altruistic surrogacy would allow them to have a child that is genetically their own without risking the mother's and infant's health. However, legislation on surrogacy varies significantly between Australian jurisdictions (Box),[8] and, in Queensland, all surrogacy arrangements — both commercial and altruistic — are illegal.

Thus, my patient with dilated cardiomyopathy faces prosecution if she were to attempt surrogacy anywhere in Australia while a Queensland resident, whereas it would be freely available to her if she moved 80 km south and became a New South Wales resident.

I believe altruistic surrogacy should be available for women in whom underlying medical conditions result in a significant risk of morbidity or mortality associated with pregnancy.

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4 Expert consensus document on management of cardiovascular diseases during pregnancy. *Eur Heart J* 2003; 24: 761-781.
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7 Smith M, Cooper GM, Clutton-Brock TH, et al. Five cases of severe cardiac disease in pregnancy: outcomes and costs. *Int J Obstet Anesth* 2001; 10: 58-63.
8 Seymour J. ART, surrogacy and legal parentage: a comparative legislative review. Melbourne: Victorian Law Reform Commission, 2004. ❑

Legislation on surrogacy arrangements in Australia*

Queensland: The *Surrogate Parenthood Act 1988* (Qld) makes all arrangements relating to surrogacy illegal in Queensland, imposing criminal penalties on all parties involved in both altruistic and commercial surrogacy arrangements.

Tasmania: The *Surrogacy Contracts Act 1993* (Tas) makes it an offence to make or receive a payment or to publish any advertisement in relation to a surrogacy contract. All surrogacy contracts are void and unenforceable.

South Australia: The *Family Relationships Act 1975* (SA) makes it an offence to enter into a surrogacy contract for valuable consideration, and contracts are illegal and void.

Australian Capital Territory: The *Parentage Act 2004* (ACT) does not prohibit non-commercial surrogacy, provided no advertising or intermediaries are involved, and payments to cover expenses are allowed.

Victoria: The *Infertility Treatment Act 1995* (Vic) prohibits commercial surrogacy, and has complex criteria regarding eligibility of surrogate mothers.

* New South Wales, Western Australia and the Northern Territory do not have surrogacy legislation.

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Comment: Morton feels that women for whom pregnancy poses a substantial risk should be offered altruistic surrogacy, so that they can still have a child that is genetically their own. The suggestion is commendable but opens a hornets' nest.

First, "genetic ownership" is a bit of a fiction at best, given that the only item genetically owned by the mother is an egg with 23 chromosomes and some cytoplasm. Admittedly, Morton refers to couples rather than to women, but few are the men who have incontrovertible evidence of any genetic stake in their alleged offspring,[1] and, given the rate with which partnerships change, thousands willingly care for children in whom they know they have no genetic stake at all. After implantation of the fertilised embryo, the carrier of the pregnancy owns whatever there is to be owned, irrespective of where some of the genes came from. At birth, genetic ownership changes again, and the child becomes its own "genetic owner". So, how much "genetic ownership" of a child can there be?

Second, who would qualify for altruistic surrogacy? It seems reasonable that women with Eisenmenger syndrome should not embark on a pregnancy given the high mortality associated with it. But how do we know that collecting ova and all it entails, and the subsequent years caring for a baby/toddler/child/teenager, would not be an even greater challenge to the woman's health than pregnancy?

Third, where will these surrogates come from (especially for women without sisters or other suitable family volunteers), and how do we ensure that they will be happy

to hand back the child to its "genetic owners"? How will we protect these altruistic women in subsequent years against potential law suits for alleged failures in duty of care to the child that they carried (for example, by exposure to toxins during the pregnancy)?

Fourth, is there not a far easier and more logical solution to this problem, provided that egg collection does not endanger the woman's health? Why not preserve the woman and her partner's frozen embryos until the woman's medical condition is sufficiently stable to both sustain a pregnancy and care for the child that hopefully results from it? If the woman's health cannot be restored sufficiently to achieve this, these couples could then show some altruism of their own by donating the embryos to infertile couples who desperately want a child irrespective of whether they can claim "genetic ownership". Thus far, there is little evidence that altruistic donation and genetic ownership are even half way to meeting each other.[2]

However, Morton should be commended for drawing attention to a national problem in women's health. The disparities and discrepancies between the Australian states and territories in almost anything that relates to reproduction[2-5] is an utter disgrace. Reproductive health should be equitable among all Australians.

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3 Pratt A, Biggs A, Buckmaster L. How many abortions are there in Australia? A discussion of abortion statistics, their limitations, and options for improved statistical collection. Parliament of Australia research brief. Canberra: Parliament of Australia, 2005. Available at: http://www.aph.gov.au/library/pubs/rb/2004-05/05rb09.pdf (accessed May 2005).
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5 de Crespigny LJ, Savulescu J. Abortion: time to clarify Australia's confusing laws. *Med J Aust* 2004; 181: 201-203. ❑

Bisphosphonates and osteonecrosis: analogy to phossy jaw

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To the Editor: Osteonecrosis of the jaw, recently reported in patients treated with bisphosphonates, may be analogous to the historic occupational disease "phossy jaw".[1,2]

Phossy jaw was osteonecrosis of the jaw caused by exposure to white phosphorus during the manufacture of matches. "Lucifer" strike-anywhere matches were first produced in 1833. They were made by dipping the match ends into a mixture containing white phosphorus.[3] Workers were exposed to fumes from the white phosphorus during mixing and spreading of the dip material, and dipping, drying and boxing of the matches.[3,4]

The first case series, comprising 22 cases, was reported in Vienna in 1845.[5] About 11% of those exposed developed the disease.[5] The average period from first exposure to diagnosis was 5 years.[4,5] Occasionally, this period was as short as a few months.[5] The mandible and maxilla could be affected, the mandible in 60% of cases (Box).[3] Dental decay was considered a prerequisite, and preventive measures included dental surveillance and treatment within the factories.[4] In that pre-antibiotic era, phossy jaw was fatal in about 20% of cases, usually because of septicaemia or meningitis.[5]

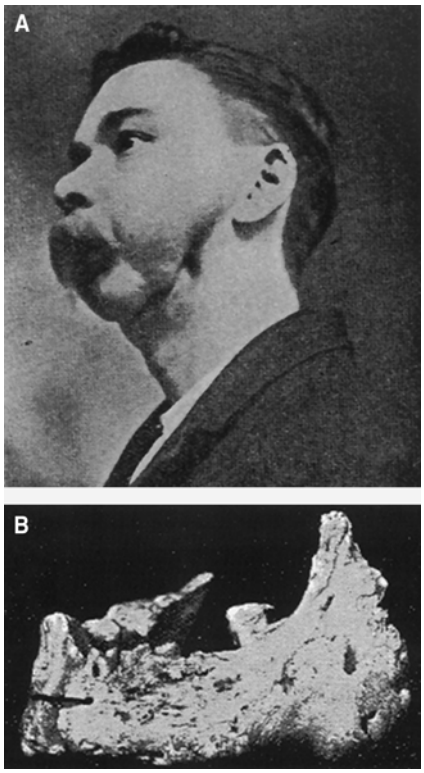
Donald Hunter, British doyen of occupational medicine, commented: "It was the most distressing of all the occupational diseases because it was very painful and was accompanied by a foul fetid discharge that made its victims almost unendurable to others. It was obstinate and chronic, the treatment was agonising and the final result was a distressing disfigurement. It was this disfiguring effect plain to every observer that made phosphorus poisoning so notorious and led to determined efforts for its abolition in every civilised land."[5]

In 1906, several European countries banned the manufacture and importation of white phosphorus matches at the Berne Convention.[4,5] A safe substitute, sesquisulfide, had been discovered by a French chemist and successfully used for manufacture of strike-anywhere matches in 1898.[4,6]

In the United States, John Andrews published a report in 1910 of 150 cases of phossy jaw from 15 of 16 match factories then in operation.[4,6] The Diamond Match Company, which held the American patent rights for sesquisulfide, waived their rights, thereby allowing the entire US match industry to use this alternative.[6] Congress then passed the Esch law, which imposed a prohibitive tax on white phosphorus matches and banned their import and export.[4,6]

Eventually safety matches were developed that used amorphous red phosphorus, which did not have the toxic properties of white phosphorus.[5]

Phosphorus necrosis of the jaw



A *Deformity resulting from excision of entire lower jaw in a case of phosphorus necrosis. (Case of Dr John P. Andrews,* The Occupational Diseases, *W Gilman Thompson, D Appleton & Co, New York, 1914).*

B Phosphorus necrosis *of entire lower jaw excised by Mr McCarthy in 1884 (London Hospital Medical College Museum).*

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6 Hamilton A. Exploring the dangerous trades. Boston: Little Brown and Company, 1943. ❑

Smoothing the transition to adult care

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To the Editor: We congratulate Lam et al[1] for identifying the major problems in transferring adolescents from the Royal Children's Hospital, Melbourne, to adult care.

The article and the accompanying editorial[2] address a difficult problem relating to the transfer of adolescent patients from a stand-alone paediatric hospital to adult services. Lam et al conclude that there needs to be a change of attitude among adult physicians, and recommend the provision of additional resources to enhance the smooth transition to adult care.

As long as paediatric services remain geographically separated from their adult counterparts in stand-alone hospitals, these problems will continue, regardless of any increase in resources. In New South Wales, tertiary paediatric services have now been incorporated onto the same campus as tertiary adult hospitals in shared-site arrangements. This facilitates the transition process, as adult physicians are more closely linked to their paediatric colleagues via shared clinical and research infrastructures. Such close cooperation allows paediatric and adult physicians to share their care during transition and provides the adult physicians with full access to the patients' medical records and radiology, microbiology, laboratory and pulmonary function data.

At Monash Medical Centre, we have taken this further by totally incorporating our adult and paediatric services into one single Department of Respiratory and Sleep Medicine. This arrangement allows an integrated approach to childhood, adolescent and adult care. The combination of services generates trust between all members of staff (an issue raised in the editorial[2]) and gives adult physicians a greater understanding of the needs of adolescents with complex health problems.

One solution to the difficult problem of transition to adult care is to phase out stand-alone paediatric services with their own costly management infrastructure. A shared campus arrangement allows greater integration of the full range of tertiary paediatric and adult services and offers many advantages in providing a seamless transition to adult care.

1 Lam P-Y, Fitzgerald BB, Sawyer SM. Young adults in children's hospitals: why are they there? *Med J Aust* 2005; 182: 381-384.
2 Bennett DL, Towns SJ, Steinbeck KS. Smoothing the transition to adult care [editorial]. *Med J Aust* 2005; 182: 373-374. ❑

Riluzole: a glimmer of hope in the treatment of motor neurone disease

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To the Editor: We read with interest the recent article by Kiernan.[1] Many patients with motor neurone disease (MND) are also taking complementary therapies, with the potential for drug interaction with riluzole. A recent patient highlighted this.

A man in his 50s with progressive MND commenced riluzole at the time of diagnosis. Initial liver function tests performed after starting the drug gave normal results. Eight months later, with disease progression, he began taking low-dose naltrexone 50 mg dissolved in 50 mL of water, of which he took 4 mL a day.

Three months later, he began to feel nauseous, with debilitating lethargy, and developed jaundice. Liver function tests showed: alanine aminotransferase level, 3030 U/L; and asparate aminotransferase level, 2074 U/L. On stopping taking both drugs, his symptoms resolved and the liver function test results gradually became normal. No other contributing cause for the hepatotoxicity was found.

From the temporal profile, the hepatotoxicity in our patient was possibly due to the combination of riluzole and naltrexone, although either drug alone could be implicated, or there may have been another mechanism. Riluzole is predominantly metabolised by cytochrome P450 enzymes (CYP1A2), but there is considerable patient variability, and the hepatotoxicity mechanism is largely unknown[2] (see also MIMS Online: http://www.mims.hcn.net.au).

In recent months, low-dose naltrexone has become popular with patients who have MND, although there are no published data of efficacy. Hepatotoxicity caused by naltrexone is dose-dependent and uncommon.[3] Naltrexone is metabolised by glucuronidation in the liver to an active metabolite, but a direct interaction with riluzole through cytochrome P450 enzymes appears unlikely.[4] There have been no clinical studies to evaluate interactions with other drugs of either riluzole or naltrexone (apart from opiates).[3]

This case highlighted for us that patients may be taking other therapies for MND, and that clinicians should be aware of the possibility of serious drug interactions when riluzole is prescribed.

1 Kiernan MC. Riluzole: a glimmer of hope in the treatment of motor neurone disease. *Med J Aust* 2005; 182: 319-320.
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4 Porter SJ, Somogyi AA, White JM. Kinetics and inhibition of the formation of 6β-naltrexol from naltrexone in human liver cytosol. *Br J Clin Pharmacol* 2000 50; 465-471. ❑

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In reply: Henderson and McCombe describe a patient to highlight an issue raised in a recent editorial:[1] that patients with motor neurone disease (MND) may develop abnormal liver function tests for reasons other than riluzole therapy. In their patient, riluzole was prescribed for a year and liver function test results remained stable. Deterioration in liver function coincided with the introduction of naltrexone. Ultimately, riluzole, an established MND therapy, had to be ceased.

Naltrexone is an authority medication, prescribed in the setting of alcohol or opioid dependence. MEDLINE searches failed to find any study or indication for naltrexone in the treatment of MND. An internet search, however, revealed a number of per-

sonal anecdotes, with a curiously Australian emphasis, suggesting an immuno-modulatory role for naltrexone in MND. A few further clicks of the mouse and the naltrexone ordering site with costings appeared.

Patients with incurable diseases commonly seek "alternative" treatments[2] at great personal financial cost, calculated at thousands of dollars per patient with MND.[3] Often there is insufficient, or, as with naltrexone, no evidence that these treatments are effective.[4] Most patients with MND will consider alternative therapy, irrespective of their educational background[5] or understanding of disease pathophysiology. How each physician approaches the use of complementary and alternative therapies by their patients may develop into an important issue in the therapeutic relationship. Certainly, being aware of the possibility may prove critical. In the patient described by Henderson and McCombe, an unfortunate outcome of irreversible liver failure in a patient with NMD was averted through conventional monitoring of liver function.

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3 Wasner M, Klier H, Borasio GD. The use of alternative medicine by patients with amyotrophic lateral sclerosis. *J Neurol Sci* 2001; 191: 151-154.

4 Dwyer JM. Good medicine and bad medicine: science to promote the convergence of "alternative" and orthodox medicine. *Med J Aust* 2004; 180: 647-648.

5 Dobson R. An exceptional man. *BMJ* 2002; 324: 1478. ❑

Allocation concealment and blinding: when ignorance is bliss

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To the Editor: Forder et al conveyed that trials without allocation concealment have the potential to mislead.[1] However, it is not true in any meaningful sense that "Without exception, allocation concealment is achievable in all randomised clinical trials. In contrast, it is not always possible to blind people to study treatments received." Rather, "Masking may be defined as either the process (researchers not revealing treatment codes until the database is locked) or the result (complete ignorance of all trial participants as to which patients received which treatments). A masking claim indicates only the former . . . If masking is possible only some of the time, then clearly reference is being made to the result, and not the process. To be fair, then, one would have to ask if the *result* of allocation concealment is always possible . . . only the *process* of allocation concealment, but not its result, can be ensured."[2]

Forder et al also state that certain methods (including sealed envelopes) are considered to be adequate concealment methods. Sadly, this is true, but only if the emphasis is on the word "considered", because sealed envelopes are both imperfect at preventing direct observation of future allocations and useless at preventing the prediction of future allocations, even without direct observation. Because the extent of prediction depends on the specific restrictions used on the randomisation,[3] allocation concealment is not even a binary phenomenon, and so to truly assess allocation concealment in a given trial, one must ask how much prediction is possible in that trial.

Allocation concealment is perfect if no observation or prediction is possible, and only partially effective if some prediction is possible. Many trials use randomised blocks, and smaller block sizes tend to allow for substantial prediction.[3-5] So, while methods aimed only at preventing the direct observations of future allocations may be considered to be adequate, it is clear that in reality they are not.

That the authors failed to use this opportunity to set the record straight indicates their implicit agreement with the incorrect statement that methods aimed only at preventing the direct observations of future allocations are not only considered adequate, but actually are adequate. Pretending that allocation concealment is binary, and hence that it suffices to use methods aimed only at preventing the direct observations of future allocations, represents ignorance that may be bliss, but certainly is not harmless.

1 Forder PM, Gebski VJ, Keech AC. Allocation concealment and blinding: when ignorance is bliss. *Med J Aust* 2005; 182: 87-89.

2 Berger VW, Christophi CA. Randomization technique, allocation concealment, masking, and susceptibility of trials to selection bias. *J Mod Appl of Stat Methods* 2003 2: 80-86.

3 Berger VW, Ivanova A, Deloria-Knoll M. Minimizing predictability while retaining balance through the use of less restrictive randomization procedures. *Stat Med* 2003; 22: 3017-3028.

4 Berger VW. Quantifying the magnitude of baseline covariate imbalances resulting from selection bias in randomized clinical trials (with discussion). *Biometr J* 2005; 47: 119-139.

5 Berger VW. Selection bias and covariate imbalances in clinical trials. Chichester: John Wiley & Sons, 2005. ❑

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To the Editor: In their article on controlled trials, Forder et al[1] described the trial by Karlowski et al on vitamin C and the common cold[2] as an example of how patients' or investigators' preconceptions about the value of the treatment may affect a trial's results. However, their presentation of this trial is misleading in two respects.

Firstly, the Karlowski et al trial was reanalysed and the "placebo-effect explanation" of the original authors was shown to be erroneous.[3] For example, their subgroup analysis of "blinded" and "non-blinded" participants excluded 42% of all episodes of colds, even though the subgroups were presented as complementary; numerous further problems are detailed elsewhere.[3] Thus, the trial by Karlowski and colleagues cannot be seen as an example of the placebo effect in action. The concept of large and omnipresent placebo effects can be traced back to an early article by Beecher, who chose "15 illustrative studies" covering such conditions as, "severe postoperative wound pain, cough, headache, seasickness, etc."[4] Beecher calculated that the "average placebo-effect" was 35.2% (SE, ± 2.2%). However, these studies did not use a control group. The comparison was "before–after", which is affected by the regression to the mean phenomenon as most of these conditions are self-limiting. Thus, Beecher's studies did not measure the "effect" of placebo. A recent meta-analysis of 114 trials comparing a placebo group with a no-treatment group found no evidence of placebo effect on binary outcomes, and only a rather small effect on pain, thus disproving Beecher's notion of great and universal placebo-effects.[5] This empirical evidence was disregarded by Forder and colleagues. Although there are reasons to use placebo whenever practicable, the bias caused by the absence of a placebo control should not be exaggerated, and the "placebo effect" should also not be misused to support investigators' own preconceptions.[3]

Secondly, the trial by Karlowski et al was focused on the effect of vitamin C on the common cold,[2] and thus the "placebo effect explanation" in this particularly influential trial is crucial to the biological question. A

recent meta-analysis of 55 placebo-controlled trials found that regular vitamin C supplementation had no effect on the incidence of colds in the general population (relative risk [RR], 0.98; 95% CI, 0.95–1.00), but reduced the incidence of colds in people exposed to substantial physical or cold stress (RR, 0.50; 95% CI, 0.38–0.66).[6] Also, regular vitamin C intake reduced the duration of colds in adults by 8% (95% CI, 3%–13%) and in children by 13.5% (95% CI, 5%–21%). Although further studies are needed to evaluate the practical significance of these findings, it is evident that the interpretation by Karlowski and colleagues that the effect of vitamin C on the common cold may be explained by the break in the double blind[2] is false and should not be reiterated.

1 Forder PM, Gebski VJ, Keech AC. Allocation concealment and blinding: when ignorance is bliss. *Med J Aust* 2005; 182: 87-89.
2 Karlowski TR, Chalmers TC, Frenkel LD, et al. Ascorbic acid for the common cold: a prophylactic and therapeutic trial. *JAMA* 1975; 231: 1038-1042.
3 Hemilä H. Vitamin C, the placebo effect, and the common cold: a case study of how preconceptions influence the analysis of results. *J Clin Epidemiol* 1996; 49: 1079-1084; discussion in 1085-1087.
4 Beecher HK. The powerful placebo. *JAMA* 1955; 159: 1602-1606.
5 Hrobjartsson A, Gøtzsche PC. Is the placebo powerless? An analysis of clinical trials comparing placebo with no treatment. *N Engl J Med* 2001; 344: 1594-1602; discussion in 2001; 345: 1276-1279.
6 Douglas RM, Hemilä H, D'Souza R, et al. Vitamin C for preventing and treating the common cold (Cochrane Review). In: The Cochrane Library, Issue 4, 2004. Abstract available at: http://www.cochrane.org/cochrane/revabstr/AB000980.htm (accessed Apr 2005). ❑

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In reply: Allocation concealment refers to ignorance of future treatment assignment *before* randomisation whereas masking or blinding is most commonly used to refer to the concealment of treatment assignment *after* randomisation.[1]

There are two criteria for successful concealment of allocation: (i) physical concealment of the process of random assignment to treatment, and (ii) concealment of any pattern of consecutive assignments. Successful concealment of the *process* must prevent unauthorised access to randomisation lists, envelopes or algorithms; the best way is to use a centralised or remote service for randomisation, whereby an independent party other than the clinician or investigator accesses a secure sequence list or a secure computer system to generate the next allocation.[2,3] Successful concealment of the *pattern* of random assignments prevents investigators from predicting a future treatment assignment on the basis of pattern recognition of allocations to date. Identifying a pattern of previous allocations can occur in open-label trials, in which all parties are aware of allocated treatments after randomisation, or if the blinding of patients and investigators has been compromised. The likely success of concealing the allocation process can reasonably be judged by its description in most trial reports (usually found in the Methods section). However, it is usually more difficult to assess the likelihood that investigators could have predicted future allocations.

Unsuccessful concealment of treatment assignment after randomisation (masking or blinding) should be detailed in the trial report. In circumstances where the blinding has been substantially compromised, exploring the results of treatment separately among participants who were unblinded and those who remained blinded, should be considered, although these are no longer randomised comparisons. In the study by Karlowski et al,[4] the placebo did not match the active treatment in taste, which alerted the investigators to the likely occurrence of significant unblinding within the study. To their credit, the investigators sought to quantify the extent of unblinding by means of a questionnaire at study close-out, and reported their findings by results of these responses. The particular grouping of responses, however, has been the subject of some discussion,[5,6] and while the interpretation of a possible placebo effect has been challenged, it has not necessarily been disproven. (The absence of a placebo effect could be proven only if information concerning perceived benefits of vitamin C related more to cold frequency than cold symptoms. Biologically, it is far more plausible for a placebo effect to result in fewer cold symptoms reported than fewer colds reported.) This trial highlights the impact of compromised blinding in the reporting of trial results, emphasising the importance of maintaining adequate blinding for reliable and unbiased trial results.

Good quality reporting of trials, in accordance with the CONSORT statement,[7] includes describing the processes in enough detail to assure readers that any pattern of randomisation is not predictable. Authors should report issues relating to allocation concealment, blinding (where appropriate) and randomisation sufficiently to convey the message that these essential trial principles were successfully achieved.[8]

1 Schulz KF, Chalmers I, Altman DG. The landscape and lexicon of blinding in randomized trials. *Ann Intern Med* 2002; 136: 254-259.
2 Beller EM, Gebski V, Keech AC. Randomisation in clinical trials. *Med J Aust* 2002; 177: 565-567.
3 Forder PM, Gebski VJ, Keech AC. Allocation concealment and blinding: when ignorance is bliss. *Med J Aust* 2005; 182: 87-89.
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5 Chalmers TC. Discussion. To the preceding article by H Hemilä. *J Clin Epidemiol* 1996; 49: 1085.
6 Hemilä H. Discussion. To the dissent by Thomas Chalmers. *J Clin Epidemiol* 1996; 49: 1087.
7 Altman DG, Schulz KF, Moher D, et al for the CONSORT Group. The revised CONSORT statement for reporting randomised trials : explanation and elaboration. *Ann Intern Med* 2001; 134: 663-694.
8 Schulz KF, Grimes DA. Allocation concealment in randomised trials: defending against deciphering. *Lancet* 2002; 359: 614–618. ❑

Achieving equal standards in medical student education: is a national exit examination the answer?

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To the Editor: We read with interest the article by Koczwara and colleagues proposing a national exit examination for all Australian medical school graduates.[1] It is refreshing to see interest in educational *outcomes*, a distinctly different trend from earlier reforms that shifted curricular focus from *content* to the learning *process*, exemplified by problem-based learning (PBL). Although the early process-focused programs were based on sound pedagogy current at their time, their educational outcomes have been relatively disappointing, with marginal or no demonstrable improvements in knowledge structures, clinical skills, or generic capabilities such as self-direction.[2] Rather than an indictment of PBL, the results may reflect what was missing in those programs: explicit focus on educational outcomes, alignment of assessments with outcomes, and attention to the learning environment.

There is widespread agreement on the outcomes desired by medical schools. They include teamwork, effective communication, critical evaluation and reflective practice, as well as more traditional outcomes.[3] Unfortunately, assessment methods have been slow to match curricular reforms, as these outcomes require new approaches, such as group and assignment work, peer assessment and portfolio examination, which are only now emerging in Australia.[4] A national exit examination for Australian graduates is unlikely to adequately measure this range of outcomes.

While Koczwara and colleagues recognise that a national examination "might need to include a clinical component" and "would necessarily entail the explicit statement of professional values and expectations", they support a multiple-choice question examination, suggesting such performance "can correlate well with clinical skills and future performance in multiple disciplines".[1] While this might "complement rather than replace" other medical school assessments, the message sent by its failure to address personal and professional attributes would be invidious.

In recognition of the limitations of multiple-choice questions, national examinations in North America now include a clinical component.[5] This has major resource implications and, like all high-stakes assessments, uses relatively reliable, but much less valid measures — standardised or simulated clinical encounters. This is at odds with current initiatives in medical schools, which are moving to clinical assessments with higher face validity, such as the mini-CEX (mini-clinical examination exercise).[6] It would be near impossible to adequately measure generic outcomes, such as teamwork, communication and reflection, in a single national examination. Koczwara et al recognise that insufficient attention has been paid to ensuring that achievement of educational outcomes is embedded in reform of medical curricula. Their solution is overly simple for a highly complex set of issues.

1 Koczwara B, Tattersall MHN, Barton MB, et al. Achieving equal standards in medical student education: is a national exit examination the answer? *Med J Aust* 2005; 182: 228-230.
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TO THE EDITOR: Foreign medical graduates sitting for the Australian Medical Council (AMC) examinations are expected to achieve a standard comparable to that of Australian medical students. If we do not measure the level of knowledge and problem-solving ability nationally, there is no reasonable basis for presuming that the AMC examination is fair. A uniform examination-based assessment should be passed by all potential medical practitioners before registration in Australia. I would be in favour of a national exit examination.[1]

1 Koczwara B, Tattersall MHN, Barton MB, et al. Achieving equal standards in medical student education: is a national exit examination the answer? *Med J Aust* 2005; 182: 228-230. ❑

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TO THE EDITOR: The recent article by Koczwara and colleagues proposing a national exit examination for medical students[1] prompted me to recall a 1970 trial of a national examination in surgery.[2] Seven of the then eight medical schools participated. Interstate differences were wide for some questions; separate analyses of the 15 teaching hospitals showed variation to be even wider within a university than between universities. Do local differences still undermine the validity of a national examination?

It is still uncertain what is actually tested by questions on paper. Context-free, standardised questions and answers assume clinical teaching and practice are standardised. However, clinical teachers writing examination items know well that many colleagues choose the "wrong" answer. Consensus may be imposed on those who differ. Teachers then forget their disparity, but expect candidates to choose only one "true" answer!

Clinical performance is interactive, multifaceted, situation-specific and value-laden. Complex judgement and decision-making cannot be measured by ticking predetermined boxes. Clinical experts develop personal subsets of specific evidence, and seek different data for diagnosis and management. But separate, context-free tasks, as in an objective structured clinical examination (OSCE), naively assume they do not.[3] OSCE even standardises scoring; examiners become recorders rather than assessors.

Reductionist standardisation reflects a pseudoscientific attempt to apply objectivity, consistency and precision to complex human interactions around incomplete evidence and uncertainty, approximations, judgements, trade-offs and locally-determined decisions.[4] Internal consistency of measuring instruments does not confer external validity in real world clinical practice.

The inexorable growth of medical knowledge and technological opportunities continuously expands "what every doctor should know". Medical learning today embraces a mix of science-based, problem-based and work-based learning experiences, with community-based experiences[5] increasingly included. In their recent article, Koczwara and colleagues identified gaps in oncology education,[2] a field ranging from molecular processes to euthanasia. Is oncology managed and taught consistently across different medical schools and hospitals across Australia? Which facets would you test in a national exit examination?[6]

Clinical performance today includes patient/person management, case management, health system management and self-management. Clinicians can judge student performance consistently. However, formal clinical examinations lack the range of cases and open-ended time that allow examiners to observe all the patient-care skills espoused by today's curricula.[7] Assessment of performance in case management and procedural skills within hospital practice can be conducted simpy by paired examiners.[8]

1 Koczwara B, Tattersall MHN, Barton MB, et al. Achieving equal standards in medical student education: is a national exit examination the answer? *Med J Aust* 2005; 182: 228-230.
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In reply: We appreciate the insightful responses to our proposal.[1] Lawson-Smith alludes to one of the most significant justifications for a national examination — fairness. One cannot expect foreign medical graduates to attain a standard comparable with that of Australian graduates if we do not measure this standard. We propose that we owe fairness not only to foreign graduates coming to Australia, but also to Australian medical students who have a right to confidently expect an education that will lead to similar knowledge, skills and attitudes, irrespective of which university they choose. And finally, we owe fairness to society, which would also expect the same standards of graduates irrespective of where they come from. Unless we consider what are acceptable standards, we operate within an environment where standards of outcome differ from place to place and as we do not measure outcomes uniformly, we do not know how they differ nor have a system to address potential deficiencies.

McNeil and Grimm point out that medical education has focused less on outcomes and more on process, and raise concerns that assessment methods lag in the sophistication necessary to assess outcomes. We wonder whether the reason for this lack of sophistication lies in the relative lack of interest in this field, and also in the lack of agreement on what constitutes acceptable outcomes. The process of outcome assessment is indeed complex, and the first step is national consensus on appropriate outcomes to be uniformly achieved.

McNeil and Grimm also point out that some of the desirable outcomes, such as teamwork, effective communication, critical evaluation and reflective practice, may be harder to test than medical knowledge. While this is certainly the case, reliable assessment methods do exist, such as the Moral Judgment Interview[2] and Rest's Defining Issues test.[3] The mini-CEX (mini-clinical examination exercise) that McNeil and Grimm refer to, allows testing of judgement, professionalism, communication, organisation and efficiency.[4]

Cox questions whether a written examination can test the complex judgement and decision-making process that is better tested in the clinical setting by experienced clinicians. We propose that the national examination is not meant to replace clinician-based assessments and ongoing learning and feedback. Furthermore, a national examination does not need to be conducted only in the written form. Specialty exams already conducted nationally incorporate a clinical element and are conducted in multiple locations. The main objective of a national examination is to ensure the comparison of outcomes against agreed acceptable national standards. This objective should not impose specific limitations on the structure of the examination.

Cox warns of the risk of "reductionist standardisation" and asks whether oncology is taught consistently across different medical schools in Australia. We acknowledge that uncertainty is ever present in medical decision-making, but remain hopeful that there exist core knowledge, skills and attitudes that patients can expect and that can provide a foundation for national standards. Oncology is *not* taught consistently across different medical schools in Australia today. While its mode of delivery may differ, we propose that its outcomes should not. And we hope that agreement on national outcome standards and a national process of assessment of these outcomes will be a first step in achieving that objective.

1 Koczwara B, Tattersall MHN, Barton MB, et al. Achieving equal standards in medical student education: is a national exit examination the answer?

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