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LETTERS

Occupational exposure to HIV: response to a system failure

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TO THE EDITOR: Root-cause analysis is an established, retrospective, structured investigative technique1 that was first introduced into wide clinical practice in public hospitals in Victoria in 2001.2 It is usually reserved for investigating infrequent and significant adverse events and explores the nature and causes of organisational systems failures.1 It is important for all healthcare professionals to understand this technique, as it is used increasingly by healthcare organisations.

Cooper and Blamey’s Lesson from Practice described the outcome of an investigation using root-cause analysis of an occupational exposure to HIV from a needlestick injury.3 We commend the analyses that identified multiple failures of the system for reporting and responding to occupational exposures to hazardous material. The practice changes that ensued at Southern Health demonstrate the value of root-cause analysis.

However, more information and analysis is required about the mistaken use of the stored serum samples. As the authors explain, the initial information was that the source patient tested negative for HIV antibodies. Some time later, it was discovered that the specimen tested was not from the source patient but from a patient of the same surname in the same ward. This caused a 3-day delay between the initial test and the Infectious Diseases Unit being notified that the source patient had twice tested positive for HIV antibodies. Some time later, it was discovered that the specimen tested was not from the source patient but from a patient of the same surname in the same ward. This caused a 3-day delay between the initial test and the Infectious Diseases Unit being notified that the source patient had twice tested positive for HIV antibodies. We contend this is an important and common systems failure that usually makes headlines of the form “Wrong site, wrong procedure, wrong person surgery”. The Joint Commission on Accreditation of Healthcare Organisations developed a universal protocol with the intention of highlighting the causative systems failures and minimising the frequency of recurrences of wrong-site surgery.4

The information given by Cooper and Blamey does not clearly explain why (ie, the root cause) the incorrect specimen was tested initially. The pathology department’s review identified the presence of unacceptable “informal norms” in the practice of blood collection and labelling. The suggested remedy that “all serum should be collected with strict adherence to blood collection and labelling protocols” is unlikely to prevent a recurrence. Exhortation to do better rarely solves the underlying problem. It is therefore important to understand why health professionals violate procedures and protocols.

The experiences of Cooper and Blamey demonstrate that some of the limitations and benefits of root-cause analysis depend on the depth of the investigation. The early and unquestioning acceptance that strict adherence to an existing protocol will prevent another “wrong person” error is not convincing. This contrasts with the well-conducted inquiry and subsequent management of occupational exposure to needlestick injuries.


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IN REPLY: Emmett and colleagues request more information and analysis about the mistaken use of stored serum samples.

The pathology staff member correctly labelled the specimen of the patient being bled but did not follow the protocol in identifying that the patient was the same as on the request slip. It had not been highlighted that there were two patients with the same surname (but different first names) in the ward, and blood was collected from one patient with a request slip labelled for another. At specimen reception, the protocols were again not followed, as the staff did not check that the minimum identifiers on the specimen label and request form matched.

It is recognised that violations of procedures are not root causes and are not directly manageable. The cause of the procedural violation must be managed.1 The collection and labelling of blood protocols were reviewed after this incident and found to be appropriate. The root-cause analysis identified that the protocols were not followed and that unacceptable “informal norms” had become practice in the collection and labelling of specimens. Staff training was examined and revised to ensure that staff were aware of the content of the protocols and that they followed them accordingly. All staff members were counselled about the importance of following correct procedures and the consequences of not doing so.

Up to 1000 specimens are received at specimen reception each weekday. New “front end processing” technology is to be introduced at the end of 2003. This electronically scans the specimen and request slip to ensure details match.

In the interim, in recognition that mislabelling will occur, all specimens relating to occupational exposures are collected at the time of the incident. Previously available results and serum stored in the laboratory are not relied on.


Management of healthcare workers after occupational exposure to hepatitis C virus

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TO THE EDITOR: The article by Charles and colleagues5 is an interesting contribution to the development of Australian protocols for healthcare workers infected with hepatitis C virus (HCV). To date, most European countries have

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no national policy for HCV-infected healthcare workers, and existing guidelines are advisory in nature and poorly enforced. A panel of European and American experts recently failed to reach consensus on management of HCV-infected healthcare workers who perform exposure-prone procedures, and concluded that screening for HCV infection and restricting infected healthcare workers is not justified, based on current published data.2

Today, the effectiveness of guidelines relies solely on self-assessment of HCV status from healthcare workers. However, collaboration of healthcare workers might be problematic if management criteria are not defined, and workers’ rights are not guaranteed. Issues such as practice restriction, disclosure of serological status to patients, privacy and discrimination need to be resolved. Given the risk of HCV transmission from healthcare workers to patients is not clear, the burden of uncertainty rests entirely with healthcare workers. Because of the fear of discrimination, needlestick injuries may be under-reported, and infected workers may not seek diagnosis and treatment because they have greater legal protection if they can honestly say that they did not know their serological status.3 Moreover, the largely asymptomatic nature of HCV infection may leave healthcare workers unaware of their infective status.

The results of Charles and colleagues suggest up to tenfold underreporting of occupational injuries with blood exposure in Australian healthcare workers.1 With this number of unreported exposures, there may be two or three new cases of HCV infection in healthcare workers in metropolitan hospitals in Melbourne each year — a figure similar to the prevalence of occupational HCV infection from notified injuries.4

Paradoxically, the prevalence of HCV infection in healthcare workers and the transmission risk for patients cannot be assessed without compulsory testing of healthcare workers, but without risk assessment there is no reason for this compulsory testing. Overcoming this Catch-22 with well-targeted epidemiological studies may help create broad consensus about policies for HCV-infected workers.


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IN REPLY: Magnavita correctly points out the need for a national policy on the management of healthcare workers who are either infected with or occupation-ally exposed to hepatitis C virus (HCV).

Compulsory testing of healthcare workers is neither practical, logical nor fair in the current Australian healthcare environment. Firstly, most healthcare institutions do not currently have adequate staff health systems in place to ensure that staff are appropriately vaccinated against readily preventable diseases, such as hepatitis B, measles and varicella, let alone test all staff for a disease such as hepatitis C, for which there is no vaccine. Secondly, awareness about important issues, such as healthcare worker transmission-risk assessment is embryonic (at best) in most institutions. The risk of HCV transmission from infected healthcare workers to their patients is generally considered to be extremely low, but probably depends on a number of factors, including the nature of the patient’s procedure and the healthcare worker’s injury and level of viraemia at the time. Simplistic legal opinions about such matters rarely help.

Finally, we agree that the rights of healthcare workers are often neglected in this era of litigation-driven medicine. If these rights are not considered, and infected or exposed healthcare workers are simply excluded from all types of work without any appropriate risk assessment or compensation, then compliance with any form of postinjury testing is unlikely. However, rather than ignoring this important workplace issue, as we believe many Australian institutions currently do, a logical assessment of potential transmission risk is possible that is both fair to the patient and the infected worker. Stratification of healthcare workers according to their level of HCV viraemia and whether they are involved in exposure-prone procedures is a logical start — this we have attempted in our proposed guidelines.1

Some healthcare workers may avoid being tested so that they can have the protection of not knowing their serological status.2 However, recent studies suggesting the efficacy of early treatment of acute HCV infection3 mean that it will actually be in healthcare workers’ interest to know if they have recently acquired HCV infection — as long as they are treated in a manner that protects their health and workplace rights, while also protecting the rights of their patients.

significant, number of haemorrhages were prevented by this treatment.

Given that 9.2% of grand multiparous women still had a postpartum haemorrhage, any prospective randomised controlled trial that allowed some of these women not to have active management of the third stage would be unethical. Numerous other studies have shown an association between grand multiparity and postpartum haemorrhage. 2-4 These include the study of Babinszki and colleagues, which limited study subjects to upper-class, private patients and thereby eliminated many confounding variables. 4

Humphrey’s study does not report the severity of the postpartum haemorrhages that did occur; even a small number of life-threatening haemorrhages could justify continuing very active preventive measures in grand multiparous women. The incidences of anaemia and previous postpartum haemorrhage, not recorded in the study, would also be of interest; both are independent reasons to actively manage the third stage in women of any parity, and there are good reasons to believe that both may be more common in grand multiparous women.

Humphrey also states that grand multiparous women did not have higher perinatal mortality rates or poorer maternal outcomes than women of lower parity. However, whenever possible throughout the period of the study, grand multiparous women were identified on booking into antenatal care and treated by senior obstetricians. Many had conditions such as diabetes, hypertension, anaemia and heart disease managed carefully to ensure as good a pregnancy outcome as possible. This focused obstetric care could well have counteracted any natural tendency of grand multiparous women towards poorer outcomes, and thus it is not possible to draw any conclusions about perinatal results from the data provided.

What is clear from these data is that grand multiparous women in far north Queensland are often economically and socially disadvantaged compared with women of lower parity. This is a common finding in almost all studies of grand multiparity. 2,3,5 We should remember that these women are taking home a new baby to conditions that may already be quite compromised. They deserve the best obstetric care we can offer them, and we should be very cautious when reviewing protocols that we do not increase risks to these women or their babies.


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IN REPLY: I thank de Costa for her interest in the debate about excessive medicalisation of normal childbirth in women with significant parity. The obstetric protocol manual of Cairns Base Hospital did not, at any time, discriminate in the details of management of the third stage of labour between grand multiparous and non-grand multiparous women.

The statistical analysis in my report shows that, once confounding factors are accounted for, grand multiparous women who labour spontaneously are twice as likely as their less parous counterparts to have a spontaneous vaginal birth, with no statistically greater risk of a postpartum haemorrhage requiring transfusion. 1 The purpose of multivariate analysis is to remove, as far as possible, the influences of the confounding factors that de Costa’s appraisal relies on.

The recommendation from my study is not that grand multiparous women be ignored, but that, if labour occurs spontaneously at the end of an uncomplicated pregnancy, they be treated no differently to their less parous sisters in terms of venous cannulation and blood cross-matching. I am on record as strongly recommending sensible, routine oxytocin-based management of the third stage of labour in all pregnancies. 2

In the end, the question is whether or not evidence wins out over an individual’s historically influenced clinical beliefs.


To the Editor: On opening the MJA Focus document “Prevention of cardiovascular disease: an evidence-based clinical aid”, 1 I expected to find useful and contemporary guidelines for general practice. However, I was surprised to read two of the recommendations — for the use of antihypertensive and antplatelet drugs in “low risk” patients (those without risk-associated clinical conditions or end-organ damage).

The document recommends drug treatment only if systolic blood pressure is > 180 mmHg or diastolic blood pressure is > 100 mmHg in people under 60 years, or systolic blood pressure is > 160 mmHg in people over 60 years. While Fulcher et al 1 volunteered that this was at odds with clinical practice, they supported the recommendations by stating that they were in accordance with published guidelines. The data for these conservative hypertension parameters were published nearly 10 years ago, 2 or derived from textbooks, 3 and are at odds with the 1999 WHO/ISH guidelines, 4 and even more at odds with the excellent Joint National Committee (JNC 7) report. 5 The latter publication recommends, after lifestyle recommendations, drug treatment for a blood pressure of 140–159/90–99 mmHg for those without end-organ damage.

Furthermore, the focus document recommends primary prevention with aspirin in those with a calculated annual cardiovascular event risk > 3%. Hayden et al 6 suggest antplatelet treatment should be offered to those with a 5-year cardiovascular event risk > 3%, which equates to a > 0.6% annual risk. The benefit to harm ratio needs to be explained to the individual, and therapy should only be initiated once blood pressure is controlled.

As an interested general practitioner and user of evidence-based guidelines, I

Prevention of cardiovascular disease: an evidence-based clinical aid
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usually check the funding of publications, and, with the heavy emphasis on the use of ACE inhibitors (in particular ramipril) and statins (which are produced by Aventis Pharma), it is difficult to rely on the evidence as presented. I would hope that independent bodies such as the National Prescribing Service or Australian Prescriber could take the pharmaceutical lead and produce desktop top references with a more unbiased opinion on the latest collection of evidence that is shaking us up in primary care medicine.

I would again refer readers to the excellent hypertension guidelines mentioned above,6,3 (in particular the JNC 7 reference card available on the Internet at www.nhlbi.nih.gov/guidelines/hypertension/jnc7card.htm), and caution them to remain wary of easy-reference desktop items funded by pharmaceutical companies.


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In REPLY: We thank Cradick for his critical review of our publication.1 His interesting points illustrate some of the reasons we were keen to publish this work.

Firstly, the average practitioner is confused about which guidelines (both national and international) to follow. Cradick refers to the JNC 7 report and WHO/ISH guidelines (the committee is familiar with these), but which to follow? The committee decided a priori that Australian national guidelines, if they existed, would be referenced in preference to overseas ones. We mentioned that current Australian hypertension guidelines should be revised, and that clinical practice was probably at odds with these recommendations.

Secondly, guidelines quickly become dated. Cradick cites the recommendation of Hayden et al that aspirin should be introduced if the annual risk of a cardiovascular event is > 0.6%.2 The latest Australian guidelines3 recommending a > 1% annual risk as the treatment threshold were published just after we received Cradick’s letter. The Hayden article is thus (for some patients) at odds with current local recommendations. Consistent with our approach, we will include the Australian recommendations in the first update of our document (July 2004).

Cradick’s comments imply bias in the presentation of the data; yet he fails to substantiate this. Neither the clinical trials quoted (nearly all of which are funded by the pharmaceutical industry) nor the conclusions or inferences drawn have been challenged. He refers to the “heavy” emphasis on the use of ACE inhibitors and statins, implying that this is inappropriate. We would argue that, given the strength of the evidence, this emphasis is appropriate, and reflects current specialist practice in tertiary centres. Careful reading of the National Prescribing Service publications will show that “heavy” emphasis is made to the 4S,4 LIPID,5 CARE,6 and WOSCOPS7 studies (for example, NPS News 20 February 20028), as well as to the prescribing information for Lipitor (Pfizer), Pravachol (Bristol-Myers Squibb) and Zocor (Merck Sharp & Dohme). We must caution against “throwing the baby out with the bath-water”. The HPS, CARE, LIPID, WOSCOPS, HOPE, PROGRESS, 4S, CURE, and CREDO studies represent substantial and widely acclaimed clinical trials that have had a positive impact on clinical care. They have all been funded by the pharmaceutical industry.

Finally, about 130 GPs in clinical practice had some input in compiling and formatting this document. The document was tested before publication in over 30 000 patients, and was peer reviewed by several clinicians. It is a pity that Cradick was not a participant in any of these processes.


To the EDITOR: Gow and colleagues recently attributed fulminant hepatic failure in an Australian patient to an over-the-counter herbal product containing kava (Piper methysticum) that is extensively used in the community.1 We are not convinced that kava caused this patient’s liver failure. The hepatotoxicity could have been due to other unidentified contaminants of the herbal preparation or to chromium (from the mineral supplements also taken by the patient).

We tested a sample of the product Kava 1800 Plus (Eagle Pharmaceuticals, Batch No. 10711) by thin layer chromatography (TLC) and high pressure liquid chromatography (HPLC) methods, and assessed it against defined standards. Kavalactones were identified and quantified by normal-phase HPLC. Kava was positively identified, with a content of about 47 mg of kavalactones per tablet (the product label specifies 60 mg per tablet). However, flavonoids from Scutellaria lateriflora, analysed by reverse-phase
HPLC, could not be found. *Passiflora incarnata*, analysed by TLC, was also not found, with none of the typical flavonoid bands being detectable.

The absence of two ingredients listed on the product label, *P. incarnata* and *S. lateriflora*, raises a new concern that there may be batch variations, as well as the possibility of unknown adulterants or contaminants being present. The possibilities of contaminants and batch variation were not excluded in the case report by Gow and colleagues. The Therapeutic Goods Administration established that Kava 1800 Plus did not contain common germander (*Teucrium chamaedrys*), a known adulterant for *S. lateriflora*. However, *Teucrium* species are known to have hepatotoxic effects and there are about 100 species, any one of which could have been a contaminant.

Gow et al refer to 68 international case reports of suspected hepatotoxicity with the use of kava. An independent analysis of these 68 cases concluded that only two were probable kava-associated hepatotoxities. An incidence calculation from these case reports indicates that hepatotoxicity from kava occurs in 0.008 cases per million daily doses, which represents an extremely low risk of adverse reactions associated with kava.

Recently, a study on the potential hepatotoxicity of kava found that the aqueous extract of kava does not affect results of liver function tests in rats. Extracts were administered in daily dosages of 200 or 500 mg of active kavacolones per kilogram of bodyweight for 2 or 4 weeks. Sera were assayed for four enzymes that are markers of liver toxicity, and liver homogenates were assayed for malondialdehyde formation, which indicates changes in lipid peroxidation. Kava did not elevate malondialdehyde or the enzymes alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase or lactate dehydrogenase. In fact, in certain instances, these enzymes were significantly reduced, suggesting a lack of toxic effect of kava on the liver. TLC analyses have shown that there is no qualitative difference between aqueous, acetone or ethanol extracts of kava.

We propose, given that patients can consume over-the-counter products such as chromium (shown to have hepatotoxicity at high concentrations), that all products consumed by patients must be evaluated before causality can be attributed to a particular herb. Reports of case records associated with hepatic failure due to kava require proper verification and documentation of all the findings.


**LETTERS**

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**IN REPLy:** Thomsen et al raise several points regarding our recent report of a death following ingestion of a preparation containing kava.

The first is that the results of their analysis of the herbal preparation differ from those of the Therapeutic Goods Administration (TGA). The TGA found *Passiflora incarnata* in the tablets, whereas Thomsen and colleagues did not. The explanation for this lies in the fact that different batches of the product were analysed by the two laboratories. The analysis we reported relates to batch 13861, which was the source of the patient’s tablets. Thomsen and colleagues have analysed material from a different batch that may have contained different components.

Thomsen et al also raise the possibility that *Teucrium*, a known adulterant of *Scutellaria*, may have been present in the preparation. With respect to this issue, the TGA tested for both *T. chamaedrys* and *T. canadensis* and found none present. They also tested for the presence of the chemicals teucreside and verbascoside, both of which are found in many *Teucrium* species. Neither was present. The raw materials used to make the tablets were also analysed, and no evidence of *Teucrium* substitution or contamination could be found.

The next point raised is whether the association between kava use and hepatotoxicity is causal, and they quote an analysis published in the journal *Phytomedicine*, in which the authors claimed that kava was the probable cause in only two of 68 reviewed cases. In marked contrast, the *Journal of Hepatology*, in July 2003, published an analysis of 36 cases of hepatitis associated with the use of kava. The review concluded that kava was the certain or probable cause of the hepatitis in 24 of the 36 cases. Worryingly, eight of these patients required liver transplantation.

Finally, Thomsen et al wonder whether the patient’s illness may have been due to chromium toxicity. However, the illness was not suggestive of chromium poisoning. There is no reason to believe that chromium was present at other than normal background levels.


**LETTERS**

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**To the Editor:** The Australian Drug Evaluation Committee (ADEC) has recommended that thalidomide be approved for the management of erythema nodosum leprosum. This recommendation has now been accepted by the Therapeutic Goods Administration. Was ADEC unaware that the World Health Organization no longer recommends thalidomide in the management of this complication of lepromatous leprosy?


**LETTERS**
Thalidomide should never be used by women who are pregnant or who could become pregnant whilst taking the drug, or could become pregnant within four weeks after stopping the drug. Even a single dose can cause severe birth defects.

I have reviewed the WHO documents referred to by Crawford,1,2 and consulted Medline. I have also had access to a review article in press in the Lancet.1 I believe the evidence indicates that thalidomide is superior to steroids in controlling ENL. Britton and Lockwood state that thalidomide is the drug of choice for men with ENL,3 however, they comment that using thalidomide in women with ENL is a difficult decision for a woman and her doctor. The WHO documents emphasise that any benefit from thalidomide must be balanced against its known toxicity, and conclude that experience has shown that it is virtually impossible to develop and implement a foolproof surveillance mechanism to combat thalidomide toxicity.

ADEC concludes that thalidomide is an effective and useful drug in the management of ENL, and that the risk management program which is to be established in Australia, together with the inclusion of a boxed warning, will ensure that the risk–benefit profile of thalidomide use in ENL is favourable.


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