LETTERS

Self-inflicted superglue injuries

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To the Editor: We are concerned about the recent number of patients presenting to our hospital after accidentally applying superglue to their eyes. Of the four cases in February and March 2004, two arose from patients mistaking cosmetic nail adhesive for their regular ocular lubricant, and applying it to the inferior ocular fornices, creating a tarsorrhaphy.

Superglues are cyanoacrylate derivatives. Those used domestically are lower-alkyl derivatives than those designed for medical use and have higher tissue toxicity.

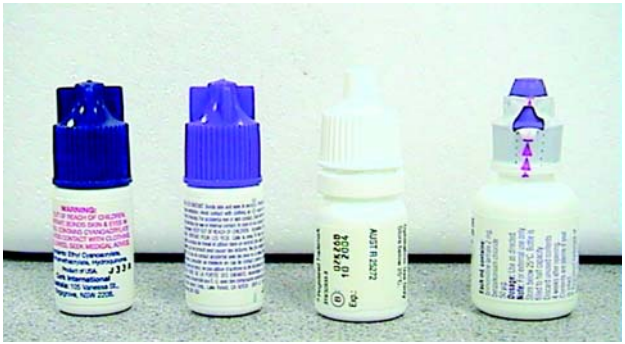
The two patients who mistook nail glue for ocular lubricant both required surgical separation of the upper and lower eyelids, and both had significant corneal abrasions, periocular dermatitis and temporary loss of lashes as a result of the reparative surgery. Both were treated with chloromycetin ointment until the abrasions had healed.

We examined the bottles containing the nail adhesives. They were remarkably similar to many ocular lubricant bottles, with no significant difference in size, colour or feel (Box). As both products are often kept together in a cosmetics area of the bathroom, accidental ocular application can occur. Similar cases have been reported in other countries over the past 20 years.[1-3]

The risk of accidental ocular (or potentially aural) application could be reduced by changes to bottles containing superglue, including:

- childproof cap to prevent conventional opening of the bottle;
- colour coding of the bottles;
- different bottle shape; and
- distinctive odour and/or colouring of the glue.

Superglue and eye lubricant bottles



Examples of bottles of synthetic nail adhesive (two on left) and eye lubricant (two on right), showing similar appearance and feel.

1 Campbell JK. Accidental use of Superglue in the eye. *N C Med J* 1983; 44: 305.

2 McLean CJ. Ocular superglue injury. *J Accid Emerg Med* 1997; 14: 40-41.

3 Mandal A, Imran D, Erdmann MW. Inadvertent application of superglue as eye ointment. *Ir Med J* 2003; 96: 310-311. ❑

Revision of guidelines for the management of gestational diabetes mellitus

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To the Editor: Consensus guidelines for the management of gestational diabetes mellitus (GDM) were prepared by the Australasian Diabetes in Pregnancy Society in 1997–1998 and subsequently published in the Journal.[1]

Since that time, there have been two minor revisions to these guidelines. The first, in relation to the recommended frequency of follow-up testing of women identified as having GDM, was detailed in a letter to the Editor in 2002.[2]

The second concerns the timing of delivery of women with GDM. At the request of the Royal Australian and New Zealand College of Obstetricians and Gynaecologists (RANZCOG), the original recommendation that "continuation of the pregnancy in uncomplicated GDM to 10 days beyond term is acceptable provided that indications from fetal monitoring are reassuring" has been modified by replacing "10 days beyond term" with "full term" to bring this into line with current practice.

The initial guidelines were arrived at by consensus of Australasian practitioners involved in the care of women with GDM. The Australasian Diabetes in Pregnancy Society recognised, both at the time and subsequently, that the level of evidence available to guide clinical decision-making fell well short of that necessary for a definitive statement on the timing of delivery.

It is noteworthy that no international consensus exists concerning the optimal timing of delivery in pregnancies complicated by GDM. The American Diabetes Association, in its Clinical Practice Guidelines, recommends delivery "during the 38th week ... unless obstetric considerations dictate otherwise".[3] The European Association of Perinatal Medicine does not make a recommendation, instead stating that "the optimal time of delivery and need to induce labour are still controversial."[4]

There is currently a paucity of quality evidence on which to confidently base recommendations. We hope that current studies, such as the Australian Carbohydrate Intolerance in Pregnancy Study and the Hyperglycemia and Adverse Pregnancy Outcome Study,[5] will provide this evidence.

1 Hoffman L, Nolan C, Wilson JD, et al. Gestational diabetes mellitus – management guidelines. The Australasian Diabetes in Pregnancy Society. *Med J Aust* 1998; 169: 93-97.
2 Simmons DS, Walters BNJ, Wein P, Cheung NW. Guidelines for the management of gestational diabetes mellitus revisited [letter]. *Med J Aust* 2002; 176: 352.
3 American Diabetes Association. Position statement. Gestational diabetes mellitus. *Diabetes Care* 2003; 26 Suppl 1: S103-S105.
4 European Association of Perinatal Medicine. Diabetes and pregnancy update and guidelines. Perrugia, Italy: EAPM, 2003.
5 HAPO Study Cooperative Research Group. The Hyperglycemia and Adverse Pregnancy Outcome (HAPO) Study. *Int J Gynaecol Obstet* 2002; 78: 69-77. ❑

"Doctor shoppers": at risk by any other name

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To the Editor: The article by Martyres et al on drug-seeking behaviour by young heroin users,[1] leading to the deaths of 202 people over 5 years, leads to an inescapable conclusion. Too often, the medical profession is part of the problem rather than the solution, and as a result young people die. Here is an issue where the admonition *primum non nocere* should be foremost in our practice.

After working with drug users and prescribing methadone for 22 years in a variety of settings, my experience is that drug users use drugs! Whether it is logical, safe or appropriate, or not, this group seeks drugs to modify or modulate their state of being. They often have serious medical and mental health issues, but have sadly decided on their preferred treatment without much knowledge of the diagnosis or of alternative interventions. They are often very skilled in obtaining drugs.

So, we must perform our role equally well. Doctors are not drug dealers. Our duty is not to promote or support intoxication, or even relaxed happiness if that increases the risk of misadventure. It is to promote and support health. It is difficult to see how a prescription for 50 benzodiazepines to a young person (or even an older person) can ever be construed as healthcare. To do it again next day, next week, on and on, is almost unbelievable, yet the data indicate that is exactly what is happening.[1] Even publicans have rules prohibiting serving intoxicated patrons.

That one was "offering the customer what he asked for", a common excuse for this sort of prescribing practice, would not be a suitable defence in the Coroner's Court if insulin, digitalis, or even vitamin A, had been prescribed on request. But appeals to good practice and commonsense, along with current regulatory strategies, are apparently not sufficient to protect this vulnerable group. Kamien points out that data from the HIC could be used to provide immediate information to doctors about whether a patient is a "doctor shopper".[2] The data are already collected and could easily be made available if the will existed. Potential prescribers could at least gain accurate and timely information on which to base their decisions. There would be less excuse for "convenience store" prescribing, and more chance of ethical behaviour. At present these data remain largely useless in preventing avoidable deaths. But, surely, learning nothing from the deaths of 202 young Australians is not an option?

1 Martyres RF, Clode D, Burns JM. Seeking drugs or seeking help? Escalating "doctor shopping" by young heroin users before fatal overdose. *Med J Aust* 2004; 180: 211-214.
2 Kamien M. "Doctor shoppers": at risk by any other name [editorial]. *Med J Aust* 2004; 180: 204-205. ❑

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To the Editor: I write to share my concerns about the "doctor shoppers" in our community.[1] The large medical group in which I practice has long been tormented by the demands of a constant stream of drug addicts, and I feel that we have now lost a very useful tool for dealing with these patients.

I refer to the loss of access to the "Doctor Shopping Hotline". This has resulted in increased aggravation for both staff and doctors. The problem is compounded by our practice being open at weekends and public holidays, when these patients arrive with the familiar story of not being able to get their benzodiazepines and opiates because their own doctors are not available.

The Health Insurance Commission recently notified me about a patient who had attended our surgery, and many others, during a 3-month period last year. During this time, he saw more than 30 doctors and was prescribed more than 300 Pharmaceutical Benefits Scheme (PBS) items (6000 benzodiazepines and more than 2000 opiates [Panadeine Forte]).

I strongly feel that the hotline should be reinstated — for the benefit of the doctors and the patients, and to help reduce a totally unwarranted drain on the PBS.

1 Kamien M. "Doctor shoppers": at risk by any other name. *Med J Aust* 2004; 180: 204-205. ❑

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In reply: I note the concerns expressed by Hart in relation to the discontinuation of the Doctor Shopping Hotline, and his call for the reinstatement of such a service.

The Doctor Shopping Project, which was funded to the end of June 2002, focused on a limited selection of nervous system medications. It has been replaced by the Prescription Shopping Project, which is much broader in scope, as it encompasses all medicines on the Pharmaceutical Benefits Scheme (PBS). The new project aims to reduce the number of patients obtaining PBS medicines in excess of therapeutic need, and provides the opportunity for more informed prescribing across all categories of PBS medicine.

The Health Insurance Commission (HIC) recognises the value of an information service for medical practitioners under the Prescription Shopping Project. An independent researcher has been engaged to explore the reactions and attitudes of medical practitioners and consumers to implementing such an information service. The research also aims to gain insight into medical practitioners' intentions of using such a service, and their expectations of the scope and delivery of the service. Findings were presented to the HIC in early July 2004. The HIC will now convene a forum of relevant peak bodies to consider the scope and delivery of an information service in light of the findings.

The HIC looks forward to working with the profession to establish an information service for medical practitioners under the Prescription Shopping Project. ❑

Epidemic of γ-hydroxybutyrate (GHB) ingestion

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To the Editor: The epidemic of recreational use of γ-hydroxybutyrate (GHB; also known as γ-OH) is a cause for concern, as it is a basal anaesthetic agent (ie, it renders the patient unconscious, with analgesic supplementation required for surgery). It is not surprising that people taking too much of it are becoming unconscious.[1]

GHB was introduced in France as a basal anaesthetic agent by Laborit about 1960. It has a slow onset of action (up to 10 minutes when given intravenously, thought to be due to conversion to an active metabolite, γ-butyrolactone).[2] It causes bradycardia, sometimes requiring atropine administration to maintain cardiac output, and raises blood pressure. Respiration is slow and deep, so that alveolar ventilation is not reduced.

Trials of GHB as an anaesthetic were conducted in Melbourne by Dr William Cole and myself in the late 1960s,[3-5] and it was used for microlaryngeal surgery for several years. Its major problems were prolonged sleep (1–3 hours after 40–100 mg/kg in children) and a high incidence of postoperative vomiting, adding the danger of aspiration in unconscious patients.

GHB was also tried as an anaesthetic in Dunedin, New Zealand, where it was found that the sleep time could be reduced by intravenous administration of physostigmine.[6]

The fact that this drug is a basal anaesthetic needs to be more widely publicised.

1 Five in hospital after party drug overdose. *The Age* 2004; 23 May. Available at: www.theage.com.au/articles/2004/05/23/1085250859718.html?oneclick=true (accessed Jul 2004).
2 Bessman SP, Skolnik JJ. Gamma hydroxybutyrate and gamma butyrolactone: concentration in rat tissues during anaesthesia. *Science* 1964; 143: 1045-1047.
3 Brown TCK. Gammahydroxybutyrate in paediatric anaesthesia. *ANZ J Surg* 1970; 40: 94.
4 Cole WHJ. Observations on the pharmacology of gamma hydroxy sodium butyrate, with special reference to microsurgery of the larynx. *Med J Aust* 1970; 1: 372.
5 Thompson JR, Greer CH, Cole W. Surgical microscopy of the larynx. (Microsurgery of the larynx, microlaryngology, microlaryngoscopy). (Endolaryngeal microsurgery). *Laryngoscope* 1971; 81: 772-783.
6 Henderson RS, Holmes CM. Reversal of the anaesthetic action of sodium gamma-hydroxybutyrate. *Anaesth Intensive Care* 1976; 4: 351-354. ❑

Screening sigmoidoscopy for colorectal cancer

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To the Editor: The editorial by Viiala and Olynyk on screening flexible sigmoidoscopy (FS)[1] is a welcome reminder that there are alternative colorectal neoplasia (CRN) screening strategies to the Australian National Health and Medical Research Council's preferred option of annual faecal occult blood testing. The availability of tests for CRN screening raises the issue of whether screening tests should be dictated by government or professional bodies, or requested by the consumer. FS and colonoscopy remain potential alternatives to faecal occult blood testing in Australia, as reflected by US screening guidelines[2] and recent local data.[1,3]

However, it is unreasonable for Viiala and Olynyk to compare the risks of screening FS (generally diagnostic only) in average-risk subjects (perforation rate, 1/50 000) with the risks of colonoscopy (both diagnostic and therapeutic) in Western Australian tertiary hospital outpatients with symptoms or other risk factors for CRN (perforation rate 1/1000).

Firstly, it is important to recognise that the perforation risk associated with screening FS comes not just from the diagnostic screening test (1/50 000), but also from follow-up colonoscopy and subsequent polypectomy in patients with distal adenomas seen on FS.

Secondly, in the WA tertiary hospital cohort,[4] the estimated perforation rate for diagnostic colonoscopy is about 1/2800 (and about 1/420 for colonoscopy accompanied by polypectomy). Asymptomatic subjects having screening colonoscopy are likely to have a lower risk than patients with symptoms or other significant comorbidities having investigative colonoscopy. Recent data from colonoscopic screening programs (which include subjects having polypectomy) have shown an overall perforation rate of less than 1/3000.[5]

Medical practitioners arranging colonoscopy, and people having this procedure, should be informed about the risks involved and, importantly, be aware that these risks are likely to vary according to the setting in which colonoscopy is performed.

1 Viiala CH, Olynyk JK. Screening sigmoidoscopy for colorectal cancer: further pieces in the jigsaw [editorial]. *Med J Aust* 2004; 180: 493-494.
2 Ransohoff DF, Sandler RS. Screening for colorectal cancer. *N Engl J Med* 2002; 346: 40-44.
3 Scott RG, Edwards JT, Fritschi L, et al. Community-based screening by colonoscopy or computed tomographic colonography in asymptomatic average-risk subjects. *Am J Gastroenterol* 2004; 99: 1145-1151.
4 Viiala CH, Zimmerman M, Cullen DJE, Hoffman NE. Complication rates of colonoscopy in an Australian teaching hospital environment. *Intern Med J* 2003; 33: 355-359.
5 Nelson DB, McQuaid KR, Bond JH, et al. Procedural success and complications of large-scale screening colonoscopy. *Gastrointest Endosc* 2002; 55: 307-314. ❑

Algal toxins or copper poisoning — revisiting the Palm Island "epidemic"

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To the Editor: In their brief review of water and public health, Leder et al[1] uncritically attributed the Palm Island "epidemic" of 1979[2] to algal toxicity, commenting that it was the only recorded manifestation of this phenomenon in Australia. The original report described a hepatitis-like illness (associated in many with dehydration and bloody diarrhoea) in 138 children and 10 adults of Aboriginal and Torres Strait Islander descent living on Great Palm Island, northeast of Townsville, Queensland.[2] No causative agent was actually identified.

My investigation in the early 1980s of *Toxocara pteropodis*, a parasite of flying foxes, excluded it as a likely aetiological agent in the Palm Island outbreak, and compelled a critical reanalysis of other possibilities, which led me to conclude that subacute copper toxicity was the most plausible explanation. My rationale was published as a hypothesis.[3] Sadly, discretion (to protect local technicians) compelled me to withhold critical information that explained how the community had been inadvertently exposed to excessive levels of copper in its water supply. Now that water management is becoming a major societal concern and algal blooms seem to be increasing in frequency, the issue needs to be resolved — and sufficient time may have elapsed for details to be revealed without impugning individuals.

In 1985, having concluded that copper poisoning was the most likely explanation, I contacted the environmental health personnel who had overseen the mixing of algicide into the Palm Island water supply in 1979. They were aware that the actual volume of water to be treated had probably been grossly overestimated, because Solomon Dam's water level was very low at the time. This meant that an excessive dose of copper sulfate was added to the dam, but it was assumed that this would be "erring on the safe side". Further, the copper sulfate was not distributed uniformly through the water in the dam: a local resident with a dinghy had been contracted and instructed to spread the bags of copper salt around the dam, but had instead dumped it all at one place — immediately over the outlet pipe which carried the island's drinking water.

This would readily explain how the community encountered a sustained pulse of high copper levels in its tap water. While chronic copper poisoning can lead to infantile hepatic cirrhosis,[4] acute gastrointestinal symptoms (as manifested during the Palm Island episode) are also well documented.[5,6]

In the absence of laboratory confirmation of copper toxicity, the cause of the "Palm Island mystery disease" must remain speculative. However, in any future similar outbreaks, copper poisoning should be excluded before attributing the cause to algal toxicity.

1 Leder K, Sinclair MI, McNeil JJ. Water and the environment: a natural resource or a limited luxury? *Med J Aust* 2002; 177: 609-613.
2 Byth S. Palm Island mystery disease. *Med J Aust* 1980; 2: 40-42.
3 Prociv P. Palm Island reconsidered: was it copper poisoning? *Aust N Z J Med* 1987; 17: 345-349.
4 Muller-Hocker J. Pathomorphology of the liver in exogenic infantile copper intoxication in Germany. *Eur J Med Res* 1999; 4: 229-232.
5 Stenhammar L. Diarrhoea following contamination of drinking water with copper. *Eur J Med Res* 1999; 4: 217-218.
6 Eife R, Weiss M, Barros V, et al. Chronic poisoning by copper in tap water: I. Copper intoxication with predominantly gastrointestinal symptoms. *Eur J Med Res* 1999; 4: 219-223. ❑

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