conditions are ballerinas and gymnasts (both frequently maligned as having "menstrual problems" — amenorrhoea).

In fact, a female should not expect to have to endure more than a handful of episodes of menstruation in a lifetime.

Just as surgery was the mainstay of treatment of peptic ulcer until it was replaced by modern medication, so the need for surgical removal or destruction of the chronically stimulated endometrium may well dissipate with the advent of more sophisticated hormone therapy (which will probably be more physiological than what we currently accept as "normal"). It is time that excessive menstruation and its sequelae were recognised as yet other lifestyle diseases and managed accordingly by the best means available, be it surgery at present or physiological regulation of the hypothalamus in the future.

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Hypophosphataemia in a comatose alcoholic

To the Editor: Dr Matthew Naughton and Dr John Grant call attention to severe hypophosphataemia as the likely cause of coma in an alcoholic patient.[1] They also comment on the lack of reports linking phosphate depletion and coma. The results of a study in which mineral homoeostasis was investigated in a large series of adult Thai patients with acute falciparum malaria have been published recently[2] and suggest that diagnoses other than severe hypophosphataemia should be considered in unconscious patients with very low serum phosphate concentrations (<0.3 mmol/L). Of 172 patients with slide-positive falciparum malaria, 83 were classified as severe.[3] Of these 83 severely ill patients, 25 were considered to have true cerebral malaria (being unable to exhibit a purposive response to painful stimuli). Three of these 25 patients with strictly defined cerebral malaria (12%) had severe hypophosphataemia, while eight of the remaining 58 severely ill cases (14%) were also markedly hypophosphataemic (serum phosphate concentration range 0.10–0.28 mmol/L); there was no significant difference between the frequency of severe phosphate depletion in patients with cerebral malaria and those without (Fisher's exact test, $P>0.1$).

These results indicate that very low serum phosphate concentrations *per se* do not necessarily cause coma. In the case of the patient reported by Naughton and Grant, metabolic factors other than hypophosphataemia (such as hyperbilirubinaemia, hypomagnesaemia and an elevated serum ammonia concentration) may have been primarily responsible for her altered level of consciousness. To ascribe neurological improvement in a single comatose patient to improvements in one of several metabolic disturbances is open to question. As severely ill patients commonly have vital organ dysfunction which can lead to neurological effects, the clinical signs of severe hypophosphataemia can be difficult to identify with certainty. This would include a tendency to convulsions and diminution of tendon reflexes, features not found in the case under review.[1] Nevertheless, none of our eight patients with non-cerebral but severe falciparum malaria and marked phosphate depletion[2] suffered a witnessed fit and the majority had increased muscle tone at presentation.

The case reported by Dr Naughton and Dr Grant reinforces the need to assess mineral homoeostasis in all severely ill patients, but even if severe hypophosphataemia is found the search for other causes of an altered conscious state should continue.

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An epidemiological study of snake bite envenomation in Papua New Guinea

To the Editor: Dr Currie and colleagues, in their recent study of snake bite in Papua New Guinea, referred to the current rarity of the Papuan black snake *(Pseudechis papuanus)* in Central Province and the absence of a positive identification since 1977.[1] We have recently identified two dead adult male specimens measuring 137 cm and 163 cm in length killed near Vaifa'a, two hours' drive northwest of Port Moresby.

Over 80% of the patients admitted to Port Moresby General Hospital with systemic envenomation following snake bite have taipan *(Oxyuranus scutellatus canni)* venom antigen detectable in their serum by enzyme immunoassay.[2,3] These patients generally present with incoagulable blood and severe neurotoxicity. In our current series, 27% of the 103 patients admitted to Port Moresby General Hospital with signs of envenomation following snake bite required ventilation despite treatment with antivenom.[3] In contrast, the pattern of envenomation in patients admitted to Vaifa'a Health Centre appears to be different. Over a 20-month period, 58 patients were admitted with signs of systemic envenomation: 47 of these had neurotoxicity and 20 incoagulable blood. Only one patient (1.8%) required ventilation during this period.

At present there is no conclusive evidence of a patient being envenomed by a Papuan black snake in the last four years. There seems little doubt that there has been a change in the snake population of Central Province over the last 20 years, and that there has been a decline in the numbers of Papuan black snakes over this period, largely attributable to the cane toad. However, the confirmation of the presence of the Papuan black snake in the Vaifa'a area and a clinical pattern of envenomation which differs in the severity of the neurotoxicity from that observed in Port Moresby, suggest that the Papuan black snake may still be of significance in this part of the province.

We are currently conducting a prospective study of all victims of snake bite throughout Central Province of Papua New Guinea. This should help to further clarify the distribution of venomous species and the clinical syndrome that each species produces.

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Lead: subtle forms and new modes of poisoning

To the Editor: The article by Alperstein et al. serves well to remind the clinical physician and intern that either premeditated or accidental chemical poisoning still needs to be borne in mind in diagnostic decisions.[1] From the viewpoint of a medical practitioner dealing with occupational and environmental lead issues, it is a pity that these areas are not more fully dealt with in the undergraduate curriculum. This may change as public, environmental and occupational medical issues come more to the fore.

The article indicates that in older residential areas where lead-based painting systems persist, the possibility of lead affecting the health of children or even adults is real, especially if paint is flaking or if renovating is being done. Unfortunately, the article seems to intermingle the issues related to adult occupational exposure (either as a worker or home renovator) and general environmental exposures from past applications of lead that are now rapidly declining, as is the average blood lead level of the general population. Leaded petrol additives and lead-based painting systems are amongst the most common of these applications. Both are being phased out. Other applications of lead provide little chance for exposure to the general population.

A vast amount of material has been written on lead toxicology, especially the subclinical effects. It could be argued that Alperstein et al. display a lack of balance in their selection of quoted literature. Although they hint at the debate that is currently raging worldwide over the many aspects of lead toxicology, little real credence is given to the strengths or weaknesses of the research work in each area.

It can be stated, with little doubt, that lead affects many bodily systems, but it is not so much the scientific existence of these effects that is important but the risk to population groups. This risk must ultimately be judged against the controllable health risks from other voluntary and involuntary exposures, be they chemical, biological, physical, or social. For perspective, one blood lead test would cost the same as a complete course of hepatitis B vaccinations, two bicycle helmets or ten mumps-measles-rubella vaccinations for a child. Along a similar vein, most of the longitudinal childhood lead studies have shown that nutrition and home environment are far more significant than low level exposures to lead in affecting a young child's IQ.

Finally, the costs quoted for "lead affected children" are from recent United States Centers for Disease Control and Environmental Protection Agency documents, and they are not easily translatable to Australia, even if the simplistic risk assessments upon which they are based are valid.

Although the article by Alperstein et al. reminds us of the need for vigilance in medical practices drawing patients from older areas, it does give an alarmist and selective overview of lead toxicology. Should an alternative literature selection be required for the topics treated in the article, I would be happy to provide it.

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The war against drugs

To the Editor: How can Alex Wodak[1] and others claim that the "war against drugs" is not working? Surely he knows the statistics. With well over 90% of drug deaths in Australia attributed to the legal drugs, alcohol and tobacco, and only a tiny percentage to the illegal, surely it appears that the illegality of the latter has been and continues to be eminently successful in containing their potential for harmful effects on society. When suffering and ill health induced by the illegal drugs reaches the levels produced by the legal ones, only then will it be clear that our "authoritarian" efforts to contain them have been unsuccessful.

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