to investigate the indication for prophylactic administration of atropine in pediatric anesthesia.

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Regulation of body temperature after acute organophosphate poisoning

To the Editor:

We would like to add some data regarding temperature regulation following acute organophosphate (OP) poisoning.[1] A 34-yr-old woman, admitted one hour after she ingested 50 ml Trimethox® (100 $g \cdot L^{-1}$ dimethoate, 162 $g \cdot L^{-1}$ diazinon, 175 $g \cdot L^{-1}$ methoxychlor), was found stuporous in her room, but was conscious on arrival. The rectal body temperature was 33°C but normalized within one hour after passive rewarming. The patient exhibited typical signs of organophosphate poisoning with miosis and diffuse fasciculations. Plasma cholinesterase concentration was 60 $IU \cdot L^{-1}$ (reference 600–1200) and red blood cell cholinesterase 830 $IU \cdot L^{-1}$ (reference values, 5000–7000). Atropine was given for five days (maximum infusion rate, 1.5 $mg \cdot hr^{-1}$) obidoxime for four days. During atropine therapy, no signs of overdosage could be detected and body temperature was normal. The clinical course was uneventful and the patient was discharged from the ICU on day 7. Muscarinic and nicotinic symptoms had disappeared. Erythrocyte cholinesterase had increased to 2435 $IU \cdot L^{-1}$ on the fourth day. By the 18th day after admission, while the patient did not receive any medication, the body temperature increased to 38.9°C in the morning and 40.4°C a few hours later. The patient only complained from abdominal discomfort and presented one episode of diarrhoea. The general physical examination was unrevealing. Neurological status was normal, without rigidity. Creatinine-phosphokinase remained within the reference values. Several bacteriological samples (blood, urine) were sterile; C-reactive protein concentration did not increase. At the time of hyperpyrexia, plasma cholinesterase concentration was 91 $IU \cdot L^{-1}$ and red blood cholinesterase 1635 $IU \cdot L^{-1}$. Body temperature decreased spontaneously within 48 hr (38.5°C on day 19, and 36.6°C on day 20). The patient was discharged without sequelae few days later.

Hypothermia is seldom reported in cases of acute OP poisoning, and the real incidence of hyperthermia is not established, as these patients generally received atropine which might also interfere with temperature regulation.[2] Fever may last for several days and it is unlikely that such a long-term thermoregulatory response could be only related to acetylcholinesterase inhibition.[3] In the present observation, the delay of 18 days before hyperthermia is unusual. One of the compounds, methoxychlor, an organochlorine derivative, is known to have a prolonged elimination half-life, but no previous case of delayed hyperthermia has been reported.

Extrapolations from animal studies suggest that a lowering of body temperature can be observed following acute exposure to high doses of anticholinesterase agents. In contrast, with low doses or during recovery from high doses of these substances, hyperthermic response was described under certain circumstances in rodents.[4]

We feel that this patient presented a biphasic thermoregulatory response to OP intoxication. The absence of muscle rigidity may be helpful to rule out neuroleptic malignant-like syndrome.

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Ketamine sedation obviates the need for general anaesthesia in children having laser ablation of facial port-wine stains

To the Editor:

Providing satisfactory amnesia and analgesia for children having laser ablation of facial port-wine stains can be an anaesthetic challenge. While general anaesthesia is a treatment option, it exposes children to risks that are avoidable.[1] For

over two years, we have been using an alternative protocol that we think is worth sharing:

1 Parents apply EMLA® cream to their child's hands one hour before the procedure.
2 The child receives 0.75 $mg \cdot kg^{-1}$ midazolam *po* 30 min before the procedure.
3 The child sits on the parent's lap for the procedure while breathing room air. The usual paediatric monitors are applied. Oxygen and resuscitation equipment are always available.
4 The anaesthetist removes the EMLA®, inserts a 24 G intravenous cannula in the hand and gives 0.01 $mg \cdot kg^{-1}$ glycopyrrolate with 2 $mg \cdot kg^{-1}$ ketamine.
5 The dermatologist proceeds with the ablation. Usually only a single bolus of ketamine is required for a treatment session. Repeated 1 $mg \cdot kg^{-1}$ boluses of ketamine are occasionally required for a large lesion. If intravenous access is unsuccessful, the anaesthetist reverts to an *im* injection of 5 $mg \cdot kg^{-1}$ ketamine with 0.01 $mg \cdot kg^{-1}$ glycopyrrolate.
6 At session's end, the patient is transported to the recovery room with mask oxygen, a pulse oximeter and Ambu® bag.

Intravenous ketamine has not caused apnoeas in our patients. Oxygen saturations have been constantly 97–100%. Others using propofol by infusion with fentanyl boluses admit to frequent episodes of apnoea requiring intervention.[2] Our patients receiving *iv* ketamine recover quickly and are usually discharged within one hour of their recovery room admission. Patients requiring *im* ketamine take longer to wake.

Children with seizures still receive general anaesthesia. However, for the majority of our children with port-wine stains, boluses of *iv* ketamine have eliminated the need for general anaesthesia with all its attendant risks and costs.

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Do "Venflon" sideport valves leak?

To the Editor:

Syringes can become contaminated with blood when used to inject into intravenous giving sets,[1] but can injections into the sideport of a "Venlon" intravenous cannula (BOC Ohmeda AB, Helsingborg, Sweden) result in similar syringe contamination?

We studied three groups of "Venlons". These were inserted into silastic tubing containing 5 $mmol^{-1}$ saline, filled by aspiration, and then capped. Then, 2 ml ultrapure water (resistivity, 18 MOhms) were injected through the sideport from a syringe whose tip was then washed in a further 5 ml ultrapure water. This water was then analysed for sodium by atomic emission spectrophotometry. In the Control Group the tubing contained ultrapure water, and in the Blank Group the 5 ml aliquots of ultrapure water were tested for sodium. The sodium concentrations ($\mu mol \cdot L^{-1}$) in each group are plotted as a scattergram (Figure) and recorded as median and (interquartile range) below:

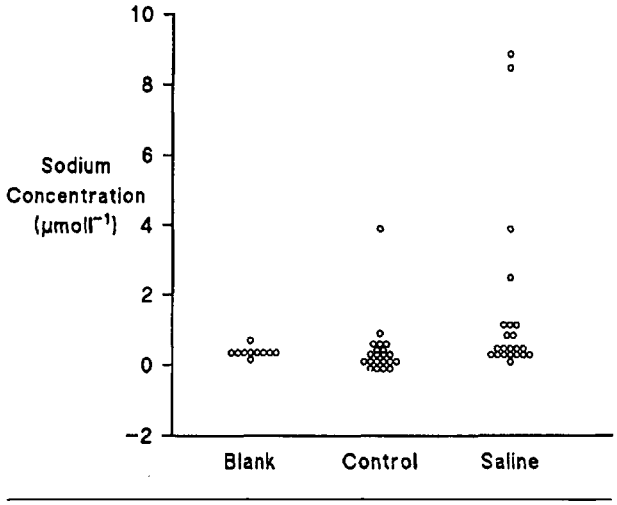

FIGURE Scattergram of sodium concentration (mean of two estimates) in the 5 ml of ultrapure water after washing the syringe tip.

Blank group	0.35	(0.30–0.40)
Control group	0.30	(0.05–0.60)
Study group	0.50*	(0.50–1.25)

Sodium concentrations near 10 $\mu mol \cdot L^{-1}$ were recorded in the Study Group in 9% of trials. If this sodium resulted from reflux through the sideport valve, the volume of saline involved would have been about 0.01 µl. A similar volume of blood is involved in the needlestick injury, which carries a risk of transmitting infection (e.g., hepatitis B and HIV).[2,3]

Although blood behaves differently from saline, this study provides evidence that syringes may become contaminated with blood after injection into such sideports.

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*Significant differences between control and study groups at $P < 0.01$ using Mann-Whitney test.