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