Correspondence

Should atropine be routine in children?

To the Editor:

In a recent article examining the cardiovascular effects of succinylcholine in 41 children, ages 1–12 years, McAuliffe *et al.* concluded that the "incidence of bradycardia after succinylcholine ... appears to be less than previously estimated" and offered no specific recommendations regarding the use of atropine before succinylcholine administration.[1]

Several factors may have influenced the incidence of bradycardia observed in this study. The parasympathetic nervous system has been described as "fully functional" at birth, whereas "the sympathetic nervous system does not mature completely until 4–6 months of age".[2] Therefore, the age group targeted by the study may have been less likely to develop bradycardia than neonates and infants in the first months of life. The incidence of bradycardia following succinylcholine is also influenced by anaesthetic agents, being more common with halothane than isoflurane and less common after administration of thiopentone.[3–5] In addition, nitrous oxide stimulation of the sympathetic nervous system may have further reduced the incidence of bradycardia.

We are curious why McAuliffe *et al.*, limited their study to such a small number of patients since earlier work has demonstrated that thiopentone may reduce the incidence of cardiac arrythmias following the administration of succinylcholine.[3–5]

Shorten *et al.*, compared the cardiovascular effects of single dose succinylcholine administration in 24 children (6–16 yr) after pretreatment with two different doses of atropine (0.01 $mg \cdot kg^{-1}$ vs 0.02 $mg \cdot kg^{-1}$) *iv*.[6] Anaesthesia was induced with thiopentone, and the authors found no differences in haemodynamic variables between the groups. In particular, bradycardia did not occur in any of the 24 patients. In light of the low incidence of bradycardia reported by McAuliffe *et al.*, it is likely that Shorten *et al.*, did not study a large enough population to demonstrate a difference between two different doses of atropine with respect to the incidence of succinylcholine-induced bradycardia. Thus, their conclusion that it is not necessary to administer >0.01 $mg \cdot kg^{-1}$ of atropine before succinylcholine should be interpreted with caution.

Douglas C. Guyton MD
Steven M. Scharf DO
Department of Anesthesiology
University of Mississippi Medical Center
Jackson, MS 39216-4505

REFERENCES

1 *McAuliffe G, Bissonnette B, Boutin C.* Should the routine use of atropine before succinylcholine in children be reconsidered? Can J Anaesth 1995; 42: 724–9.
2 *Schieber RA.* Cardiovascular physiology in infants and children. *In:* Motoyama EK, Davis PJ (Eds.). Smith's Anesthesia for Infants and Children. St. Louis: Mosby, 1990: 77–104.
3 *Schoenstadt DA, Whitcher CE.* Observations on the mechanism of succinylcholine-induced cardiac arrythmias. Anesthesiology 1963; 24: 358–62.
4 *Williams CH, Deutsch S, Linde HW, Bullough JW, Dripps RD.* Effects of intravenously administered succinylcholine on cardiac rate, rhythm, and arterial blood pressure in anesthetized man. Anesthesiology 1961; 22: 947–54.
5 *Badgwell JM, Cunliffe M, Lerman J.* Thiopental attenuates dysrhythmias in children: comparison of induction regimens. Tex Med 1990; 86: 36–8.
6 *Shorten GD, Bissonnette B, Hartley E, Nelson W, Carr AS.* It is not necessary to administer more than 10 $mg \cdot kg^{-1}$ of atropine to older children before succinylcholine. Can J Anaesth 1995; 42: 8–11.

REPLY

It is appropriate to mention that infants <6 mo may have parasympathetic dominance[1] and explains why we chose to study children aged 1 to 12 yr.

Although, thiopentone may attenuate the incidence of dysrythmias in children,[2] atropine is still administered by paediatric anaesthetists to children between one to twelve years of age before induction of anaesthesia. However, many believe that atropine in this age-group might not be necessary and this motivated this study. The most frequently used inhalational agent in our institution for induction of anaesthesia is halothane. However, in view of an increased risk of masseter muscle rigidity when succinylcholine is administered after halothane,[3] thiopentone was chosen. Isoflurane might have contributed to the low incidence of arrhythmia but, this inhalational agent was not used in this study.

Although, the effect of nitrous oxide on the sympathetic nervous system activity remains controversial in adults,[4–5] in infants and children, the sympathetic nervous system cannot be demonstrated.[6] Previous studies reporting an increased incidence of succinylcholine-induced bradycardia in children have used nitrous oxide.

Shorten et al.[7] demonstrated that 0.1 $mg \cdot kg^{-1}$ atropine was as efficient in preventing succinylcoline-induced bradycardia as was 0.2 $mg \cdot kg^{-1}$. In this study, vecuronium was used instead of succinylcoline because until then, the administration of succinylcoline without atropine in children was received with reluctance by the Ethics Committee at the Hospital for Sick Children.

We acknowledge that the number of patients in our study is rather small for the importance of this clinical observations. The relevance of a zero incidence of bradycardia was extensively discussed. It was also reiterated that the "true" incidence of succinylcholine-induced bradycardia in children aged one to twelve years is not clear and that the role of routine atropine administration is also unclear.

As indicated, the routine use of atropine before succinylcholine in children aged from 1 to 12 years deserves to be reconsidered, however, until true risk and benefits are established, the recommendation was that anaesthetists not change their current practice at this time whether or not this includes the routine use of atropine. The only suggestion is to continue

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

Georgina McAuliffe MBBS
Bruno Bissonnette MD
Christine Boutin MD

REFERENCES
1 *Hirsch EF.* Innervation of the Vertebrate Heart. Springfield: Charles C Thomas, 1970: 125–33.
2 *Badgwell JM, Cunliffe M, Lerman J.* Thiopental attenuates dysrhythmias in children: comparison of induction regimens. Tex Med 1990; 86: 36–8.
3 *Hannallah RS, Kaplan RF.* Jaw relaxation after a halothane/succinylcholine sequence in children. Anesthesiology 1994; 81: 99–103.
4 *Smith NT, Eger EI II, Stoelting R, Whayne TF, Cullen C, Kadis LB.* The cardiovascular and sympathomimetic responses to the addition of nitrous oxide to halothane in man. Anesthesiology 1970; 32: 410–5.
5 *Sakabe T, Kuramoto T, Inoue S, Takeshita H.* Cerebral effects of nitrous oxide in the dog. Anesthesiology 1978; 48: 195–200.
6 *Leon JE, Bissonnette B.* Transcranial Doppler sonography: nitrous oxide and cerebral blood flow velocity in children. Can J Anaesth 1991; 38: 974–9.
7 *Shorten GD, Bissonnette B, Hartley E, Nelson WS, Carr AS.* It is not necessary to administer more than 10 $\mu g \cdot kg^{-1}$ to older children before succinylcholine. Can J Anaesth 1995; 42: 8–11.

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.

Philippe Hantson MD
Philippe Hainaut MD
Michel Vander Stappen MD
Paul Mahieu MD
Brussels, Belgium

REFERENCES
1 *Ochi G, Watanabe K, Tokuoka H, Hatakenaka S, Arai T.* Neuroleptic malignant-like syndrome: a complication of acute organophosphate poisoning. Can J Anaesth 1995; 42: 1027–30.
2 *Cupp CM, Kleiber G, Reigart R, Sandifer SH.* Hyperthermia in organophosphate poisoning and response to PAM. J S C Med Assoc 1975; 71: 166–8.
3 *Hirshberg A, Lerman Y.* Clinical problems in organophosphate insecticide poisoning: the use of a computerized information system. Fundam Appl Toxicol 1984; 4: S209–14.
4 *Gordon CJ.* Thermoregulation in laboratory mammals and humans exposed to anticholinesterase agents. Neurotoxicol Teratol 1994; 16: 427–53.

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