



**Figure 2**  
Picture of heart rate (top), 'ST-elevation' (middle), and non-invasive blood pressures on the trend display of the monitor.

either flushed in (2) or the dead space needs to be accounted for when calculating the test-dose volume. We chose a test-dose volume of 1 ml. Assuming a catheter dead space of 0.2 ml, this resulted in an actual test-dose volume of approximately  $0.22 \text{ ml}\cdot\text{kg}^{-1}$  containing  $1.1 \mu\text{g}\cdot\text{kg}^{-1}$  epinephrine. This large dose of epinephrine containing local anesthetic allowed to unmistakably identify intravascular catheter position by immediate and dramatic but brief T-wave elevation in a neonate.

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## Pediatric-compatible train-of-four monitoring

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**SIR**—For both adult and pediatric patients, train-of-four (TOF) monitoring is frequently useful for assessing the depth of neuromuscular blocking agents (1,2). Typically, standard adult ECG electrode pads are used with fixed-current stimulators. However, these adult ECG pads can be excessively large in infants and small children. This may possibly render the monitoring inaccurate. Furthermore, reduced amounts of electrical current may also be necessary in assessing TOF ratios in the pediatric population (3).

The electrical connectors, which are attached to Kendall pediatric ECG pads (part number 30710001; Kendall, Chicopee, MA, USA), are compatible with several variable-current stimulators (models Digi Stim 2 plus, 3 plus, and III; Neuro Technology, Houston, TX, USA). This is shown in Figure 1.

Together, this arrangement provides both smaller pads and an adjustable current source.



**Figure 1**  
The connectors, on the pediatric ECG pads manufactured by Kendall, are both electrically and mechanically compatible with several of Neuro Technology's variable-current train-of-four stimulators. This combination is useful in assessing pediatric neuromuscular blockade.

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## Preoperative apnea in a preterm infant after caudal block with ropivacaine and clonidine

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**SIR**—Caudal clonidine  $1 \mu\text{g}\cdot\text{kg}^{-1}$  provides excellent perioperative analgesia but appears to be the cause for early postanesthetic apnea in preterm infants.

The risk of apnea in preterm infants increases with general anesthesia (1,2). Awake caudal anesthesia for inguinal hernia repair is suggested to avoid this respiratory complication. Addition of clonidine to local anesthetics for caudal block is used to prolong duration of analgesia. However, respiratory depression and postoperative apnea are side effects of clonidine (3).

We present a case of preoperative apnea after caudal clonidine in preterm infant who received light inhalation anesthesia.

A 4-week-old male preterm infant weighing 2.8 kg was scheduled for inguinal herniorrhaphy. He was born prematurely at 35 weeks gestation with a weight of 2.7 kg. No apneic event or congenital heart disease were reported and hemoglobin was  $14.1 \text{ g}\cdot\text{dL}^{-1}$ . No premedication was given and he received air/O<sub>2</sub> (50%/50%) plus sevoflurane, to a maximum of 2% via facemask while breathing spontaneously during the caudal puncture. A peripheral IV line was secured and dextrose 5% in 0.45 NaCl was infused at a rate of  $4 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{h}^{-1}$ .

Heart rate, pulse oximetry and noninvasive blood pressure were monitored. The baby was warmed using warming blankets. Caudal block was quickly performed with the patient in the left lateral position using a 22-gauge short-beveled needle under sterile conditions with 'no turn technique' (4). After negative aspiration for blood or cerebrospinal fluid, a mixture of  $1 \text{ ml}\cdot\text{kg}^{-1}$  ropivacaine 0.2% with  $1 \mu\text{g}\cdot\text{kg}^{-1}$  clonidine was injected. The infant was turned into the supine position and the sevoflurane turned off. We gave supplemental oxygen ( $\text{FiO}_2 = 0.4$ ), heart rate ranged from 135 to  $143 \text{ b}\cdot\text{min}^{-1}$ , mean blood pressure from 40 to 47 mmHg and SpO<sub>2</sub> 97–98%. Fifteen minutes after caudal block and before the beginning of the surgical intervention, respiratory depression with a decrease in tidal volumes, respiratory rate and oxygen saturation (below 90%) occurred with concurrent bradycardia (HR

$88 \text{ b}\cdot\text{min}^{-1}$ ). Oxygen supplementation and manual ventilation via facemask were given with resolution of the episode. Herniorrhaphy lasted 45 min during which time no apneic episode occurred.

The infant was observed for 24 h. Heart rate, SpO<sub>2</sub> and blood pressure were monitored continuously and no other apnea was recorded. No further analgesic medication was required.

Postoperative apnea after general anesthesia in former preterm infants is a well-known phenomenon, related to an immature respiratory musculature and central control mechanism, an unstable elastic rib cage and a lower airway prone to collapse. Awake regional anesthesia for inguinal hernia repair is suggested to avoid life-threatening respiratory complications. To prolong duration of anesthesia and to reduce postoperative need for analgesics in these infants, caudal clonidine has been considered useful. Although clonidine-induced respiratory depression is uncommon in the dose range normally used ( $1\text{--}2 \mu\text{g}\cdot\text{kg}^{-1}$ ), this adjuvant reduces the ventilatory response to carbon dioxide (5,6). The respiratory depression has been associated with differential recruitment of upper airway muscles and continuous activation of laryngeal and pharyngeal muscles in animal studies. Clonidine has been shown to stimulate central alpha-2 adrenoceptor with a differential effect on baroreflex HR and vasomotor regulation. Alpha-2 Adrenoceptor stimulation greatly augments baroreflex-mediated bradycardia and exerts a tonic inhibitory influence on respiratory rhythm in the awake goat. These effects can be reversed by selective Alpha-2 Adrenoceptor blockade (7). In our case, the infant had no prior history of apnea. We found no metabolic abnormalities including electrolyte, glucose and calcium levels, hypothermia or anemia. No premedication, muscle relaxants, sedatives or opioids were given and inspired sevoflurane was given at low concentrations. Therefore, the role of prematurity together with caudal clonidine, apnea and bradycardia seems linked in this case. The respiratory depression occurred 15 min after the caudal injection while in other reports the apnoeic events were described later coinciding with peak plasma levels of clonidine previously reported in a study (age range: 1–9 year; weight range: 9–41 kg; peak plasma levels between 48 and 193 min) (8). We also believe that the younger age of our case allowed a more rapid absorption of the caudal clonidine. Single caudal block of ropivacaine  $2 \text{ mg}\cdot\text{ml}^{-1}$  ( $1 \text{ ml}\cdot\text{kg}^{-1}$ ) administered to children aged 0–12 months is well tolerated and plasma concentrations are well below the threshold for toxicity in adults (9). In addition, no paradoxical rib cage movement related to a high level of caudal anesthesia was observed. Our report suggests that dose range normally used ( $1\text{--}2 \mu\text{g}\cdot\text{kg}^{-1}$ ) may be responsible for early preoperative apnea in preterm infants. We agree with other authors that caudal clonidine should not be used in neonates and small infants undergoing minor surgical procedures (3,10).