Prolonged arterial hypotension due to propofol used for endotracheal intubation in a newborn infant
Wagner B, Intensive Care Unit, University Children’s Hospital of Bern, Switzerland
During early pregnancy, the newborn’s mother had had malaria with hemolytic crisis but had recovered well under antimalarial treatment in Africa. At a gestational age of 37 4/7 weeks, an elective cesarean section (spinal anesthesia) was performed because of breech presentation. Apgar scores were 2 and 4 at 1 and 5 minutes, respectively, mainly due to poor respiratory effort. The newborn male infant was quickly intubated without premedication. On transport to the regional hospital, there was accidental extubation. Upon arrival at the hospital, the cyanotic and apneic newborn received 8 mg/kg of propofol, 10 mcg/kg of fentanyl and muscle relaxation and he was reintubated.

On arrival at our NICU, the patient was deeply sedated with small pupils, hypoventilation and with motor reaction only to painful stimulation. Birth weight, body length and head circumference were within normal range for age. In addition, there was arterial hypotension (mean 25-30 mmHg), which was treated with volume 20 ml/kg and dopamine 5 mcg/kg/min over 24 hours. His cardiovascular status remained otherwise completely normal. The patient was weaned from the ventilator within another 24 hours and showed no respiratory distress. He never showed any signs of a systemic inflammatory response syndrome or of infection. Chest X-rays, head ultrasound and laboratory examinations (hemoglobin, leucocytes, electrolytes, renal function and plasmodium falciparum antigen) remained normal, except for a positive Coombs test due to AO-incompatibility. He went home at the age of 6 days with a normal neurologic and cardiopulmonary status.
The etiology of the infant’s poor respiratory effort immediately after birth remains unclear. The newborn might have suffered from side effects of maternal anesthesia. Neonatal encephalopathy due to maternal infection may present with poor respiratory effort, but we would expect prolonged hypoventilation and additional signs of neonatal encephalopathy.

Prolonged arterial hypotension seen after the second endotracheal intubation, however, is most probably due to the medications used for intubation. Arterial hypotension seen after a single bolus of propofol is probably due to overdosing (maximal recommended dose is 3 mg/kg bodyweight), limited hepatic clearance in the newborn infant, and fentanyl induced increase in propofol serum concentration (1-3).

Considering the increasing popularity of propofol use in neonatal and pediatric anesthesia, it is important to remember the following facts:

1. The “propofol-infusion syndrome” does exist: it is rare but has a high fatality rate; propofol should therefore not be used for long-term sedation in neonatal or pediatric intensive care (4).

2. A single bolus of propofol reduces myocardial contractility and systemic vascular resistance (similar to thiopental). There is an increase in right-to-left shunting through fetal shunts with worsening of
cyanosis (5-8). Propofol should therefore not be used for intubation or sedation in conditions associated with cardiopulmonary compromise, such as newborns with respiratory distress syndrome, pulmonary hypertension, sepsis, cardiomyopathy or congenital heart disease. In these patients other medications for intubation have been shown to be much safer, such as ketamine or low dose benzodiazepines.

3) Recent studies suggest that there is propofol-related neurotoxicity (9-11).

In conclusion, despite the current enthusiasm for propofol, there are well documented and potentially lethal side-effects of propofol. This requires a differentiated approach to the use of propofol. Especially in the new-born infant, there is no place for propofol.


4. Arzneimittel-Kompendium der Schweiz 2000;689-691


