Heart block after intravenous lidocaine in an infant

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A 14-day-old infant received intravenous lidocaine (2 mg·kg⁻¹) at the conclusion of cataract surgery to prevent coughing from tracheal tube stimulation. Within 30 seconds the infant developed high-grade AV heart block and a ventricular rate of 40. Following brief resuscitation efforts, the patient had a normal cardiac rhythm, blood pressure and respiratory pattern. Intravenous lidocaine may be followed by major disturbances in cardiac rhythm and rate. Cautious titration of small doses should decrease the potential for adverse effects.

Key words

Intravenous lidocaine has been advocated to prevent "bucking" or coughing following general anaesthesia with an endotracheal tube.[1-3] We present a case of heart block and bradycardia following a bolus of intravenous lidocaine given to a 14-day-old infant to minimize reactivity to an endotracheal tube at the conclusion of a general anaesthetic.

Case report

A two-week-old, three kg female was admitted for cataract removal from the left eye. She was the product of a full-term uneventful pregnancy. Following delivery via repeat Caesarean section, Apgar scores were eight and nine at one and five minutes, respectively. An otherwise normal physical examination showed bilateral cataracts. Consul-

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tants in genetics and ophthalmology discovered no underlying disorder and felt the cataracts were idiopathic.

At age six days, the child underwent general anaesthesia for cataract removal from the right eye. No premedication was used and the anaesthetic consisted of enflurane/nitrous oxide in oxygen. Although two episodes of heart rate slowing occurred in response to surgical manipulation, the heart rate never fell below 90 beats per minute. Administration of atropine 0.05 mg IV increased the heart rate to 120 beats per minute each time. Blood pressure remained stable at 45–50 mmHg systolic throughout the procedure. The remainder of the anaesthetic and operation was uneventful.

At age 14 days, the child was readmitted for cataract extraction from the left eye. No premedication was given prior to arrival in the operating room. Atropine 0.1 mg IM was administered immediately before induction with halothane/nitrous oxide in oxygen.

After anaesthesia induction and intubation, cataract surgery was begun. While receiving one per cent halothane and 70 per cent nitrous oxide in oxygen the patient had a spontaneous respiratory rate of 30–40 breaths/minute, blood pressure of 50 mmHg systolic, and heart rate of 160–170 beats per minute. After 35 minutes of surgery, halothane was discontinued and when surgery was completed ten minutes later, nitrous oxide was discontinued and 100 per cent oxygen begun.

Lidocaine 6 mg (2 mg·kg⁻¹) was administered IV over five seconds. Approximately 10–15 seconds after lidocaine administration, the heart rate decreased to 90 beats per minute (sinus rhythm). In spite of controlled ventilation, the heart rate continued to decrease to 40 beats per minute. The electrocardiogram showed high-grade AV block.

No change in heart rate or rhythm occurred despite IV administration of atropine 0.1 mg, calcium chloride 100 mg, and bicarbonate 1 mEq.

Sternal compressions at a rate of 100/min were begun and lip colour improved. One minute after resuscitation was begun, atropine 0.1 mg and epinephrine 10 μg IV were administered. One minute following administration of these drugs, heart rate increased to 155 beats per minute (sinus rhythm), and blood pressure was 90 mmHg systolic. After three minutes of controlled ventilation with 100 per cent oxygen, the patient was moving and breathing spontaneously. A chest roentgenogram showed the endotracheal tube tip to be 1 cm above the carina. Arterial blood gas analysis ten minutes later, with the child still intubated and breathing 100 per cent oxygen spontaneously, showed: pH 7.38, PO_2 215 mmHg, PCO_2 30 mmHg, and B.E. -1 mEq·L^{-1}.

The trachea was extubated and the child taken to the recovery room while breathing 40 per cent oxygen via a hood. Respiratory rate was 32 breaths/minute, and HR 140–155 bpm. An uneventful recovery period followed, and the child was discharged home the next day.

Discussion

Intravenous lidocaine (1–2 mg·kg^{-1}) may attenuate cardiovascular and airway responses to intubation at the beginning and end of general anaesthesia.[1-3] For this reason the practice is now considered routine by many anaesthetists.

Toxicity from lidocaine includes a spectrum of reactions focusing on the central nervous system and the heart.[4] Lidocaine produces a slight but significant increase in sinoatrial conduction time in patients with sinoatrial dysfunction.[5] Sinus bradycardia resulting from lidocaine may be due to the development of sinoatrial block.[6] Lidocaine can induce second or third degree heart block in patients with bundle branch block,[7] and thus the drug should be used only with extreme caution in the presence of heart block and bradycardia.[8]

Many reports of bradycardia or heart block following intravenous lidocaine have appeared over the past two decades.[7-17] All have involved patients who were elderly, had significant heart disease, or were concurrently receiving drugs such as procainamide, quinidine, or digoxin.

Congenital cataracts may arise from a number of aetiologies (intrauterine infections, metabolic and chromosomal disorders, dysmorphic syndromes) that are associated with congenital heart disease.[18]

However, the infant we describe had no evidence of cardiac anomalies or conduction disturbances beyond mild bradycardia coincident with eye manipulation (oculocardiac reflex) in an earlier operation.

In this child, heart rate began to decrease 10–15 seconds after lidocaine 2 mg·kg^{-1} IV was given, and continued to fall for the next minute until high-grade AV heart block ensued with a ventricular rate of 40 beats per minute.

It is unlikely the bradycardia in our patient was due to vagal stimulation, since the heart rate remained low in spite of atropine administration. Peak arterial plasma lidocaine levels in adults have been shown to occur 60 seconds following a 20-second IV lidocaine injection of 1–1.5 mg·kg^{-1}. This dose, resulting in therapeutic anti-arrhythmic blood levels (2–6 μg·ml^{-1}), initially produced levels over four times the maximum accepted therapeutic level, well in the toxic range (mean 26.6 μg·ml^{-1} in 32 patients).[19] The time course of the heart dysfunction in the case we report above parallels the expected peak blood level of lidocaine.

Animal and human newborn data suggest infants can tolerate the same lidocaine dose as adults. Newborn sheep need the same blood lidocaine concentrations as adult sheep to demonstrate toxic symptoms.[20] Neonates have an increased total volume of distribution compared with adults,[21] which may counteract their decreased plasma protein binding of lidocaine.[22] In fact, one authority has stated "... reducing [lidocaine] dosage in the neonate solely out of concern for toxicity is not justified."[23] This recommendation may need to be tempered.

Although lidocaine-mediated cardiac dysfunction in the neonatal period has been reported only with massive accidental overdose,[24,25] our patient received a dose (2 mg·kg^{-1}), at the upper limit of recommended dosage when administering lidocaine as an IV bolus to children.[26] No pharmacokinetic data exist in children for intravenous lidocaine. Extrapolating dosages from adult data is often dangerous. Thus, lidocaine 2 mg·kg^{-1} IV in an infant may be an overdose, leading to a toxic effect, rather than an idiosyncratic effect.

Conversely, the newborn conduction system may respond to lidocaine as does the diseased adult conduction system. The cardiac conduction system continues to develop after birth. Both the arterial

supply and the collagen framework of the sinus node undergo important changes; the His bundle undergoes a molding and shaping with resulting cell death.[27] All these changes are vital for the morphology of the normal adult conduction system to appear. In addition, newborn rabbit hearts have low epinephrine stores and decreased sympathetic innervation.[28] Because the newborn has an immature conduction system and sympathetic innervation she may be susceptible to toxicity even from accepted therapeutic doses of lidocaine. Indeed, foetal hearts bathed with therapeutic levels of amide local anaesthetic solutions demonstrate a decreased spontaneous contraction rate and increased A-V conduction times.[29]

Halothane may depress A-V conduction.[30] Drugs such as lidocaine, which decrease conduction through the A-V node, may increase the likelihood of heart block during halothane anaesthesia.[31]

Acidosis may have contributed to the potential for A-V block in our patient. Spontaneously ventilating neonates during halothane/nitrous oxide/oxygen anaesthesia have been shown to exhibit respiratory acidosis.[32] Foetal hearts bathed in solutions of low pH (either from increased PCO_2 or decreased $NaHCO_3$) demonstrate reduced spontaneous contraction rates and prolonged intraventricular conduction velocities.[33] Lowering the pH also exaggerates the delayed conduction seen with amide local anaesthetics.[29,33] Thus, bolus injection of lidocaine in a neonate with possible respiratory acidosis following halothane anaesthesia may account for the unusual arrhythmia we observed.

In summary, lidocaine $2\ \mathrm{mg \cdot kg^{-1}}$ IV was given as a bolus to a 14-day-old infant at the close of general anaesthesia. Sinus bradycardia and high-grade AV block occurred approximately one minute after lidocaine injection and required cardiopulmonary resuscitation. Administration of "therapeutic" lidocaine doses may be followed by major disturbances in cardiac rate and rhythm. Cautious titration of small doses should decrease the potential for toxic effects or idiosyncratic complications.

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Résumé

A la fin d'une chirurgie pour cataracte, un enfant de 14 jours a reçu une injection intraveineuse de lidocaïne (2 mg·kg^{-1}) afin de prévenir la toux provoquée par le tube endotrachéal. En dedans de 30 secondes, l'enfant a développé un bloc AV avec un rythme ventriculaire de 40. Aprés des manœuvres brèves de réanimation, le rythme cardiaque, la pression artérielle ainsi que la respiration retournèrent à la normale. L'injection intraveineuse de lidocaïne peut être suivie par des perturbations majeurs dans le rythme et la fréquence cardiaque. La précaution dans le calcul des petites doses à administrer doit diminuer le risque potentiel des effets secondaires.