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Case report

Complete atrioventricular block during neonatal intubation: a case report

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Abstract

Preterm infants often experience side effects from intubation. Desaturation and sinus bradycardia are frequent. Atropine as premedication mitigates these risks.

We report the occurrence of severe bradycardia related to atrioventricular block during intubation. The infant experienced severe bradycardia not responsive to effective endotracheal ventilation. The electrocardiogram trace displayed an initial 2:1 Mobitz II block with a rapid progression to complete atrioventricular block.

Congenital atrioventricular block is mostly related to atrioventricular node lesions by maternal anti-SSA/anti-SSB antibodies, but, in some cases, atrioventricular block may be paroxysmal and vagally mediated.

Although most rhythm disturbances secondary to intubation are sinus bradycardias, other bradyarhythmia, such as atrioventricular block, should be considered and treated rapidly. Clues for non-sinus bradycardia include initial sharp decrease in heart rate, and a fixed low heart rate despite adequate ventilation.

Keywords: Neonate, Intubation, Atropine, Bradycardia, Atrioventricular block

Introduction

Given their limited respiratory reserve and increased parasympathetic tone, preterm infants often experience side effects during intubation. In an observational cohort study, Neches et al. reported that desaturation and sinus bradycardia occur in approximately 45% and 27% of procedures, respectively.[1] Other sides effects and physiological deleterious responses include oesophageal intubation, mainstem intubation, laryngospasm, oral/airway trauma, vomiting, hypo or hypertension, intracranial hypertension and pain or agitation[2]. Several factors have been associated with a reduced risk of these adverse events, including the use of a video laryngoscope, higher training level of the first airway provider and appropriate premedication.[1–3] Current guidelines recommend premedication with an analgesic, a muscle relaxant and a vagolytic agent for all intubations outside life-threatening emergencies[3–4]. Neonatal studies found that using atropine as premedication helps mitigate risks of desaturation and bradycardia. It significantly increases heart rate without provoking ventricular arrhythmias, reduces bradycardia, effectively prevents conduction disturbances, and contributes to safer intubation.[1,5] Here, we report a less common complication of intubation: the occurrence of severe bradycardia related to atrioventricular block (AVB), despite premedication with atropine.

Case report/case presentation

A growth restricted preterm girl born at 28 weeks of gestation was intubated at 10 days of life for digestive bleeding. Extubation failure related to symptoms of upper airway obstruction led to reintubation at 12 days. Premedication prior to intubation included atropine (0,02 mg/kg), leading to an increase in heart rate (HR) to 175 bpm, fentanyl, and mivacurium. Videolaryngoscopy allowed good airway exposure, but the infant experienced severe bradycardia at 35 bpm within 10 s. HR did not rise with T-piece ventilation, and an experienced operator rapidly intubated the infant. HR remained at 35 bpm despite effective endotracheal ventilation with 100% oxygen. Chest compressions were started and intravenous adrenaline administered, followed by a prompt return to a normal heart rhythm. The

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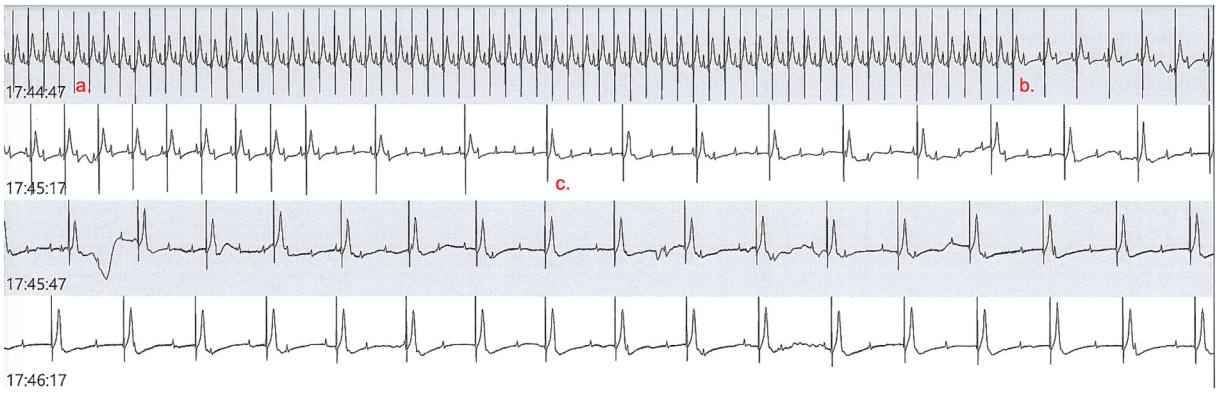

Fig. 1 – Two minutes of continuous ECG recorded during the procedure. (a) Atropine-related sinus tachycardia. (b) 1:2 Atrio-ventricular block followed by 3:1 and 4:1 blocks.(c) Complete AV block with ventricular bradycardia and progressively slowing atrial beats.

ECG trace (Fig. 1) displayed an initial 2:1 Mobitz II block with a rapid progression to 3:1, 4:1, and to complete AVB with short QRS complexes, despite premedication with atropine. Maternal history was negative for autoimmune disease, calcium channel blockers or recent betablockers; anti-SSA and −SSB antibodies were negative. Subsequent echocardiogram, ECG and 24 h Holter were normal. There was no recurrence of arrythmia during the hospitalization. A diagnosis of transient complete AVB secondary to laryngoscopy-associated vagal hypertonia was considered.

Discussion

Neonatal arrhythmias have an estimated prevalence of 1 to 5%, and mostly consist of premature supraventricular beats, typically resolving during the first month of life, and in sinus bradycardia. Bradycardia has a wide range of etiologies, which may be either cardiac or extracardiac and can be secondary to various neonatal conditions, including metabolic disturbances, intracranial hypertension, hypoxia, hypothermia, hypopituitarism, drugs, and others. In preterm infants, immaturity of the central nervous system leads to an exaggerated vagal tone. This can trigger sinus bradycardia in response to stimuli including apnoea's, airway obstruction, endotracheal intubation, airway suctioning, insertion of gastric catheters, or any other event causing vagal stimulation. Although sinus bradycardia is common, other potentially dangerous forms of arrhythmias, such as AVB, should not be underestimated. AVB occur in 1 in 15,000 to 20,000 living births.[6] Congenital AVB is mostly related to AV node lesions by maternal anti-SSA/anti-SSB antibodies, associated with structural heart disease, or can be genetic. Additionally, AVB can also result from infections, ischemia, metabolic disease, inflammatory/infiltrative disease or cardiac procedures.[7] In other cases, most often unrecognized, AVB may be paroxysmal and vagally mediated, as in our patient.[6,7] Vagally-induced AVB resulting from intubation has already been documented in the adult population and highlights the importance of being aware of potential cardiac complications during airway management.[8] Laryngoscopy and endotracheal intubation are well recognized causes of vagal stimulation. In vagally-mediated AVB, there is a simultaneous depression of both sinus and AV node function due to parasympathetic activation. In such instances, the AVB is often preceded by a prolongation of the PR interval.[9] In some situations, cessation of vagal stimulation is followed by recovery of a sinus rhythm. If insufficient, suggested therapy for acute AVB starts with administration of atropine, which helps reduce vagal input in AV node by acting as an antagonist of acetylcholine on postsynaptic muscarinic receptors.[5] Our patient received this as premedication. This may have contributed to the rapid restoration of sinus rhythm once the vagal stimulation induced by laryngoscopy decreased.[5,7] Isoprenaline or Adrenaline, through their agonist action on the beta-1 adrenergic receptor at the AV node, can also be used as a treatment for AVB. It improves AV conduction and helps reverse the sympathetic/parasympathetic imbalance induced by vagal hypertonia.[7] In our case, due to severe bradycardia and compromised hemodynamic, adrenaline bolus was preferred. Given the almost immediate return of a sinus rhythm following the injection of adrenaline, it is difficult to determine whether it actually played a role in resolving the AVB.

Conclusion

Although most rhythm disturbances secondary to intubation are sinus bradycardias, other rhythm disturbances, such as AVB, should be considered and treated rapidly.[5] Promptness of treatment is especially important as severe bradycardias may be associated with changes in cerebral hemodynamic potentially leading to development or worsening of intraventricular haemorrhage, ischemia and poorer long-term neurodevelopmental outcomes.[1] Clues for non-sinus bradycardia include initial sharp decrease in HR, and a fixed low HR despite adequate ventilation.

Statement of ethics

Written informed consent was obtained from the parent of the patient for publication of this case report and any accompanying images.

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CRediT authorship contribution statement

Justine Dauby: Writing – original draft, Investigation, Conceptualization. **Caroline Jacquemart:** Writing – review & editing, Validation, Supervision. **Sophie Tribolet:** Writing – review & editing, Validation, Supervision. **Vincent Rigo:** Writing – review & editing, Validation, Supervision.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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