CASE REPORT

Nasal CPAP and preterm bradycardia: cause or cure

Andrew Torrance MacLaren,¹ Colin Peters,² Peter D MacDonald²¹Neonatal Unit, NHS Greater Glasgow and Clyde, Glasgow, Scotland, UK²Neonatal Unit, Southern General Hospital, Glasgow, Scotland, UK

Correspondence to

Dr Andrew Torrance MacLaren, andrew.maclaren@nhs.net

Accepted 6 May 2014

SUMMARY

Nasal continuous positive airway pressure (nCPAP) is widely used for the treatment of respiratory distress syndrome and apnoea of prematurity. Complications related to fixation devices have been well documented. We report a clinically well preterm baby suffering intermittent, profound episodes of bradycardia without any prior associated apnoea or desaturation. We believe these episodes were due to the oculocardiac reflex related to orbital compression from the continuous positive airway pressure (CPAP) fixation straps. Bradycardia was replicated by gentle ocular compression and the episodes resolved after repositioning the CPAP straps. Vagal overstimulation has previously been reported in preterm babies but we believe this to be the first case in which pressure from CPAP strapping has been reported to trigger bradycardia. However, we suspect that similar cases could easily go unrecognised. Careful positioning of CPAP securing straps may prevent accidental vagal overstimulation contributing to episodic bradycardia.

BACKGROUND

Nasal continuous positive airway pressure (nCPAP) is widely used for the treatment of respiratory distress syndrome and apnoea of prematurity. It has been shown to be a relatively safe treatment reducing the risk of reintubation, length of oxygen requirement and need for mechanical ventilation.¹ However, it does not come without side effects and pressure-related complications particularly related to the fixation of devices have been well documented.²

CASE PRESENTATION

We report a case of a 920 g male infant born at 27 +6 weeks of gestation with respiratory distress syndrome, who was initially intubated and treated with exogenous surfactant. The baby was extubated to biphasic nCPAP (Viasys Infant Flow Driver SiPAP) at 26 h of age (corrected gestational age—28 weeks) receiving a mean airway pressure of 6–7 cm H₂O and with an oxygen requirement of 25–35%. We would expect to see episodic apnoea, desaturation and bradycardia events in an infant of this size and maturity but, following extubation, this infant was noted to have intermittent and unusually profound episodes of sudden bradycardia without any preceding apnoea or desaturation. We define bradycardia events as a periodic slowing of the heart rate to below 100/min. Such events were occurring at least hourly and over a 24 h period the nursing staff recorded at least 5 more severe bradycardia events with the heart rate dropping to the 60 s. Periods of desaturation or shallow breathing occurred after some episodes of bradycardia but a characteristic feature of these events was the abrupt

onset of slowing of heart rate. This preceded any change in respiratory pattern suggesting that the respiratory depression was a secondary phenomenon and the primary event was a cardiac slowing.

INVESTIGATIONS

The infant was screened for infection and treated with 48 h of vancomycin and gentamicin. The blood cultures subsequently proved to be sterile and the C reactive protein (CRP) was repeatedly normal. The baby was also treated with caffeine citrate and the dose was optimised to 10 mg/kg/day. Despite these measures the periods of atypical marked bradycardia persisted. A full 12-lead ECG was performed, showing normal sinus rhythm with no evidence of any conduction defect and with normal PR and QT intervals. The ECG trace during the events showed a normal QRS complex with preceding P waves. An echocardiogram showed a structurally normal heart.

DIFFERENTIAL DIAGNOSIS

We examined the ECG during these bradycardic episodes. We noted that there was no change in the QT interval, no change in the QRS pattern and the baby remained in sinus rhythm. The baby was briefly changed to a high-flow humidified oxygen delivery system (*Vapotherm*) and, interestingly, these episodes ceased. However, due to increasing respiratory effort and poorer capillary blood gases the baby was placed back on biphasic nCPAP. Although there was improvement in respiratory symptoms, the episodes of atypical bradycardia returned. We noted that the straps of the nCPAP mask were positioned in a way as to be applying pressure to the child's orbits. The degree and character of the bradycardia without prior respiratory depression in the context of a clinically well baby led us to speculate that this might be a primary vagal phenomenon.

Figure 1 The position of continuous positive airway pressure mask straps as replicated on a mannequin.

CrossMark

To cite: MacLaren AT, Peters C, MacDonald PD. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2013-202289

Figure 2 Printout of cardiorespiratory monitoring of baby during application of gentle pressure to the orbit. Line 1—ECG trace; Line 2—respiratory pattern; Line 3—arterial line trace.

Gastro-oesophageal reflux, apnoea of prematurity and infection are very common causes of apnoea and bradycardia events in infants. The events did not appear to have any primary respiratory element and there was no evidence of infection. There was no vomiting or behavioural evidence of gastro-oesophageal reflux, either at this time or at any later point in the course of the infant's stay in the neonatal unit. The baby was receiving a maximum of 2 mL of maternal expressed breast milk every 2 h and the episodes persisted in spite of the cessation of feeds.

TREATMENT

Figure 1 shows the position of the straps on the baby duplicated on a mannequin. We hypothesised that these straps were causing vagal overstimulation secondary to orbital pressure. This was tested by a member of consultant staff applying pressure for 2–3 s to the lower eyelid and bradycardia was replicated. An ECG rhythm strip and arterial line trace at this time demonstrated the bradycardia (figure 2) with the same abrupt marked slowing of heart rate as was seen in the spontaneous bradycardia events. Ocular pressure was followed by an immediate slowing of the heart rate and then a short period of sinus arrest lasting almost 5 s. There was some change in the respiratory pattern which followed after the onset of bradycardia. The test was repeated a little later with the same outcome and as a result of these observations the CPAP straps were repositioned away from the orbits. Following this the atypical episodes of primary bradycardia resolved. No further ocular compression tests were carried out as it was considered inappropriate to subject a very tiny immature infant to unnecessary handling.

OUTCOME AND FOLLOW-UP

The baby continued to improve and was discharged well at 38 weeks of gestation. He continues to do well and was recently reviewed in clinic at 7 months of age (corrected gestation 4 months) with normal neurodevelopmental progress to date.

DISCUSSION

Transient episodes of bradycardia are common in preterm infants, however, periods of sinus arrest are relatively rare.³ Reflex anoxic seizures or 'Pallid syncopal attacks' are well recognised in older infants and children and have recently been reviewed by Iyer *et al.*⁴ They are mediated through vagal overstimulation and paediatricians will be very familiar with vagal-mediated bradycardia/sinus arrest in this context. Although the trigger of vagal overstimulation in our case is different, the physiological mechanism leading to bradycardia and transient sinus arrest is similar and can be demonstrated by stimulating the oculocardiac reflex, which produces vagal-mediated

bradycardia following pressure applied to the orbits. Vagal phenomena have been reported in preterm babies⁵ but suction and airway instrumentation is the commonest context in which this is considered. The oculocardiac reflex leading to bradycardia and arrhythmia has previously been described in neonates.⁶

Episodic bradycardia is common in preterm infants and CPAP support usually helps to reduce the frequency and severity of such events. However, this generally occurs in the context of events that are mediated through hypoventilation/apnoea and hypoxaemia. The events we witnessed were quite atypical with the striking feature being the abrupt onset of cardiac slowing in the absence of any initial respiratory element. We believe this to be due to pressure from CPAP strapping triggering the oculocardiac reflex and while we do not suggest that this is a common cause of bradycardia in preterm infants we do suspect that similar cases could easily go unrecognised. We suggest that the positioning of CPAP securing straps merits closer attention as this may contribute to accidental vagal overstimulation and consequent episodic bradycardia.

Learning points

- ▶ Continuous positive airway pressure (CPAP) is commonly used in the neo natal intensive care unit (NICU) as a treatment for apnoea of prematurity.
- ▶ Bradycardia is a common problem in preterm infants. It is usually a consequence of periodic hypoventilation and hypoxia.
- ▶ Vagal overstimulation leading to reflex bradycardia has been documented in preterm neonates.
- ▶ CPAP fixation straps may be positioned in such a way as to cause vagal overstimulation via the oculocardiac reflex, with resultant atypical bradycardia events.
- ▶ CPAP securing straps should be positioned to avoid ocular pressure and potential vagal overstimulation.

Acknowledgements The author would like to thank the Medical Illustration at the Southern General Hospital for producing the pictures.

Contributors ATM was involved in writing the article and abstract as well as producing figure 2. PDM was involved in printing out of the images as well as proof reading and further contribution to the wording of the article. CP was involved in the proof reading and wording of the article. All three authors, led by PDM, were involved in the identification of the clinical problem described in this case, followed by its investigation and resolution.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Davis PG, Henderson-Smart DJ. Nasal continuous positive airways pressure immediately after extubation for preventing morbidity in preterm infants. *Cochrane Database Syst Rev* 2003;(2):CD000143.
- 2 Fischer C, Bertelle V, Hohlfeld J, *et al.* Nasal trauma due to continuous positive airway pressure in neonates. *Arch Dis Child Fetal Neonatal Ed* 2010;95: F447–51.
- 3 Dorotskar PC, Arko MK, Baird TM, *et al.* Asystole and severe bradycardia in preterm infants. *Biol Neonate* 2005;88:299–305.
- 4 Iyer A, Appleton R. Management of reflex anoxic seizures in children. *Arch Dis Child* 2013;98:714–17.
- 5 Phillips SJ, Agate FJ, Silverman WA, *et al.* Autonomic cardiac reactivity in preterm babies. *Biol Neonate* 1964;6:4–5.
- 6 Kirsteen MS, Wishart HY. The oculocardiac reflex. *Proc R Soc Med* 1976;69:373–4.

Copyright 2014 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- Submit as many cases as you like
- Enjoy fast sympathetic peer review and rapid publication of accepted articles
- Access all the published articles
- Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow