Case Report

# Epicardial pacemaker insertion in a preterm very low birth weight neonate – An anaesthetic challenge

Avneet Singh, Ganesh Kumar, Kulbhushan Saini, Gangadharan Prabhakaran[1]

Departments of Anaesthesia and Intensive Care, and [1]Paediatrics, Postgraduate Institute of Medical Education and Research, Chandigarh, India

**ABSTRACT**

Congenital complete heart block (CCHB) has an incidence of one in 20,000 live births and carries a 20% risk of mortality. The hemodynamic instability due to bradycardia and asystole due to the increasing metabolic demands can be avoided by appropriate antenatal planning, timely delivery and initiation of medical treatment and early pacemaker insertion. In this report, we discuss the anaesthetic challenges of permanent epicardial pacemaker insertion with good outcomes in a 32-week gestational age 1380 grams neonate within a few hours of birth.

**Keywords:** Permanent pacemaker, preterm, very low birth weight

**Address for correspondence:** Dr. Ganesh Kumar, Department of Anaesthesia and Intensive Care, Room No. 4014, 4th Floor, Advanced Cardiac Centre, Postgraduate Institute of Medical Education and Research, Chandigarh - 160 012, India.
E-mail: mganeshkumar19@gmail.com
**Submitted:** 26-Apr-2020 **Revised:** 29-May-2020 **Accepted:** 21-Jun-2020 **Published:** 21-Jan-2022

## INTRODUCTION

Congenital complete heart block (CCHB), with an incidence of one in 20,000 live births has 20% mortality risk.[1,2] Amongst these, 53% are diagnosed at 16-24 weeks and 24% at 25-30 weeks of gestation as fetal bradycardia. Early pacemaker insertion reduces the consequences of bradycardia in these babies.[3] We present a case of a premature neonate born at 32 weeks of gestation, planned for permanent epicardial pacemaker insertion within few hours of birth.

## CASE HISTORY

A 32 weeks gestational age neonate, weighing 1380 grams was brought to our operating room (OR) 3 hours after birth. Child was born out of emergency cesarean section for premature rupture of membranes to a primigravida mother, who was a known case of anti-Ro antigen positive Sjögren syndrome. Fetal echocardiography revealed complete heart block with an atrial rate of 160 and ventricular rate of 55 beats per minute (bpm) with a structurally normal heart. The perinatal details have been shown in Figure 1. Echocardiography done after birth revealed dilated right atrium/ventricle with mild pericardial effusion. The umbilical vein and artery were catheterized and an additional 26 G venous access was taken on left hand, exclusively for drugs. 10% dextrose was started at 2.5 ml/hour for maintenance. Isoproterenol infusion was started at 0.2 mcg/kg/min and patient got shifted to OR with the heart rate (HR) of 50 bpm. The ambient OR temperature was increased to 24°C, warming mattress and forced air warmers were used to keep the baby warm. After attaching American Society of Anaesthesiologists standard monitors, the child was induced with graded doses of fentanyl upto 5 mcg/kg and ketamine 0.5 mg/kg. The

| Access this article online | |
|---|---|
| Quick Response Code: | Website: www.annals.in |
| | DOI: 10.4103/aca.ACA_94_20 |



**How to cite this article:** Singh A, Kumar G, Saini K, Gangadharan P. Epicardial pacemaker insertion in a preterm very low birth weight neonate – An anaesthetic challenge. Ann Card Anaesth 2022;25:93-6.

| VITALS | AT 1st hour of life |
|---|---|
| HR | 48bpm |
| RR | 52/min |
| SpO2 | 98% |
| CP/PP | Well palpable |
| CFT | 2 sec |
| Color | pink |
| Temp. | euthermic |

**GENERAL PHYSICAL EXAMINATION**
Well looking
Active, vigorous cry
No cutaneous or periorbital rashes

**SYSTEMIC EXAMINATION**
CVS- S1S2 +
No audible murmur
RS- Bilateral AEE+
No added sounds
P/A- No organomegaly
CNS- Cry/Tone/Activity- normal
Neonatal reflexes- appropriate for gestational age

**Figure 1:** Vitals and physical examination findings of the neonate after birth

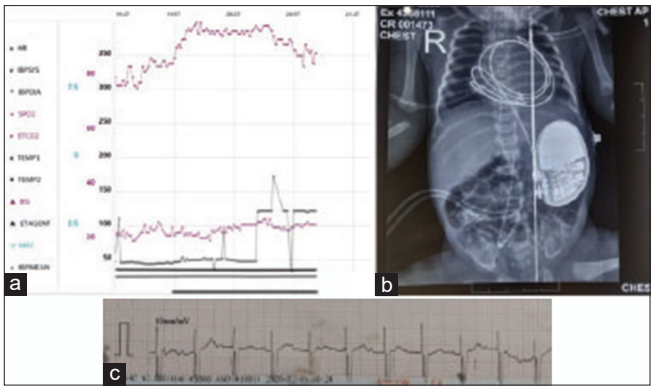


**Figure 2:** Intraoperative hemodynamic parameters (a), postoperative babygram showing the pacemaker generator in the epigastric region (b), postoperative electrocardiogram showing normal electrical capture with heart rate of 120 bpm (c)

trachea was intubated with 3.5 mm uncuffed tube after giving atracurium 0.5 mg/kg. Anesthesia was maintained with Sevoflurane at 0.5 minimum alveolar concentration with air-oxygen mixture to maintain saturation of 94%. Core temperature was monitored using nasopharyngeal probe and epinephrine (0.1 mcg/kg/min) was started to support the cardiac output. Arterial blood gas showed pH 7.41, pO2 64.60, pCO2 33, bicarbonate 20.50, base deficit 3.10 and serum lactate 3.98. An epicardial pacemaker (Mode: VVIR, rate: 120 BPM) was implanted through subxiphoid incision [Figure 2b] and isoproterenol was subsequently stopped. Hemodynamics were stable throughout the procedure [Figure 2a] except drop in core temperature (33°C). Child got shifted to Intensive Care unit (ICU) and the normothermia (37°C) achieved using warmers, following which the epinephrine infusion was tapered and stopped. Even though the child was extubated on day 3, he developed bronchopulmonary dysplasia requiring oxygen support for 31 days. The child got discharged on 38th day and is doing well.

## DISCUSSION

CCHB has an incidence of 2-8% in neonates born to mothers of Sjogren Syndrome.[1,2,4] Thirty percent of these children have congenitally corrected transposition of great arteries, atrioventricular defects, single ventricle disease and left sided isomerism. In the absence of structural heart defect, CCHB is related to maternal systemic lupus erythematosus, Sjogren's syndrome or mixed connective tissue disease.[1,2] The lesser common causes of CCHB are foetal myocarditis, mitochondrial disease, and 18p-syndrome.[2] The risk factors for poor outcomes are prenatal diagnosis, hydrops, prematurity, low birth weight, low ventricular rate (<55 bpm) not responding to medical therapy, structural heart disease, and neonatal lupus.[2]

The antenatal treatment of CCHB includes beta-sympathomimetics, dexamethasone, plasmapheresis, digoxin or furosemide.[3,4] Children with inadequate response (<20% increase in HR) to beta-sympathomimetics undergo temporary or permanent pacemaker insertion. The early pacing prevents low cardiac output and metabolic acidosis, ineffective myocardial stimulation and hemodynamic compromise in the milieu of increasing metabolic demands,[1,2,4] The guidelines for permanent pacing in children are (1) wide QRS escape rhythm, complex ventricular ectopy, or ventricular dysfunction (2) ventricular rate <50-55 bpm or CHB with ventricular rate <70 bpm.[5] The small size of the neonate pose a technical challenge for the surgeon, making an initial temporary pacemaker as a safer management approach.

We managed our patient under a controlled environment from the antenatal period, bridging with isoproterenol. The child had a structurally normal heart, good APGAR scores with normal peripheral perfusion, which made us to decide on a permanent epicardial pacemaker insertion within few hours of delivery. The transcutaneous pacing carried the risk of thermal injury and transvenous pacing is associated umbilical vein thrombosis and infection. This child posed anesthetic challenges due to the physiological changes occurring within the first few hours of birth [Table 1], in addition to prematurity and very low birth weight.[6,7]

The lung protective mechanical ventilation strategy- 4-5 cm H2O of positive end expiratory pressure (PEEP), tidal volumes 6-8 ml/kg, intermittent recruitment maneuvers prevents atelectasis, volutrauma and barotrauma. Mild hypercapnia -PaCO2 45-55 mm Hg, is permissible, but hypocapnia (PaCO2 <39 mm Hg) and hypercapnia (PaCO2 >60 mm Hg) is avoided to prevent intraventricular hemorrhage.[6] The

**Table 1: Physiological changes in preterm neonates**

| System | Physiology |
|---|---|
| Cardiovascular system | Flip-flop transitional circulation |
| | Immature cardiac myocytes (a) disorganized contractile proteins (b) calcium dependent sarcoplasmic reticulum (c) poor compliance |
| | Rate dependent cardiac output |
| | Parasympathetic nervous system is more mature in relation to sympathetic system |
| | Blood pressure varies with gestational age and normalizes 36 hours after delivery. Mean arterial pressures should not be allowed to drop below gestational age in weeks or an absolute value of 30 mm Hg |
| Respiratory system | Reduced surfactant causing respiratory distress syndrome |
| | Oxygen toxicity- bronchopulmonary dysplasia |
| | Low functional residual capacity and positive end expiratory pressures and prone to desaturation |
| | Obligate nasal breathers |
| | Vulnerability to apnoea |
| | Prolonged ventilation- tracheomalacia, BPD, retinopathy of prematurity |
| Cerebrovascular system | Low autoregulatory reserve -risk of intracranial haemorrhage and cerebral ischemia |
| | Fragile capillaries- risk of intraventricular haemorrhage and periventricular leukomalacia |
| Renal system | Glomerular filtration continues to increase with gestational age and improves further from 4 days after birth at any gestational age |
| | Nephron development ceases between 28-36 weeks, neonates born at 32 weeks may not have complete nephron development |
| Hepatic system | Immature hepatic clearance via P450 isoenzymes |
| | Immature glycogen storage- risk of hypoglycaemia |
| Hematopoietic system | Haemoglobin is lower 13-15 g/dl |
| | Foetal haemoglobin is 70-80% |
| | Low levels of pro-coagulants - prolonged coagulation tests |
| | Greater risk of bleeding, needing routine administration of vitamin K |
| | Low levels of anti-coagulants |
| Thermoregulation | More prone to ambient heat loss |
| | (a) Larger body surface-to body weight ratio (b) poorly developed subcutaneous tissue (c) absent shivering thermogenesis mechanism |
| | Non shivering thermogenesis |
| Blood and extracellular volume | Total body water 80% of body weight, with >50% as extracellular fluid, blood volume 90-100 ml/kg |
| | Apparent increase in volume of distribution |
| | Reduced drug binding to albumin and alpha-glycoprotein, increasing free plasma drug concentration, necessitating increased drug loading doses |

saturation should be 88-94%, with minimum possible FiO2 to avoid bronchopulmonary dysplasia and retinopathy of prematurity. Neonates with CCHB show compensatory adaptation to the slow ventricular rate in form of increase in fractional shortening and ventricular size.[4] A sudden increase in preload or systemic vascular resistance can cause cardiac failure due to the fixed HR.[1-4,6] High dose opioid based anesthesia is safe for premature patients, with structural heart disease and poor ventricular functions with disadvantages like delayed extubation and longer ICU stay.[4] Term neonates, without ventricular dysfunction or heart defects, can undergo sevoflurane or ketamine anesthesia.[4] Isoflurane depresses myocardial contractility and thiopentone and propofol decrease both contractility and SVR, and should be avoided.[1,2] The hypotension is prevented by preloading of the patient with 5% albumin, epinephrine and dopamine or use of a temporary pacemaker until the permanent pacemaker is installed.[2] Other complications are non-responsiveness to atropine bolus and ventricular fibrillation.[1] The pre-operative insertion of an umbilical artery catheter was used for continuous blood pressure monitoring and an umbilical vein catheter for catecholamine infusions. These catheters carry the risk of infection, thrombosis, bleeding and portal venous obstruction.[8] Hypoxia, hypercarbia, acidosis, hypothermia, hypo/hyperglycemia, hypocalcemia or sepsis may cause shift to transitional circulation, and hence pre-ductal and post-ductal saturations were monitored. A short duration, single exposure of general anesthesia does not cause cognitive impairment and neuromonitoring in form of bispectral index and near infrared spectroscopy may be done.[9] The intravascular volume status replenishment begins in the pre-operative period with 0.9% normal saline or ringer lactate with 1-2.5% dextrose, continued intraoperatively and an additional 4-7 ml/kg/hr added for third space losses due to thoracotomy.[10] Neonates have a hemoglobin of 14-17 gm/dl with 70-80% in fetal form. The blood losses are to be replaced with fresh packed red cells when hemoglobin falls below 12 g/dl at 15-20 ml/kg.[10] The decreased mean arterial pressures and increased capillary refill time are danger signs. The OR should be pre-warmed to 24°C and warming mattress, forced air warmers and fluid warmers to be used to prevent hypothermia. Both axillary and core temperatures need to be monitored.

## CONCLUSION

The CCHB in preterm very low birth weight infant is both an anesthetic and surgical challenge. The hemodynamic instability and adverse outcomes can be avoided by appropriately planned permanent epicardial pacemaker insertion with good anesthetic outcome in a preterm, very low birth weight neonate.

### Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

### Acknowledgement

We thank the operation theatre technicians and nursing staff who helped us in managing the case efficiently.

### Financial support and sponsorship

Nil.

### Conflicts of interest

There are no conflicts of interest.

## REFERENCES

1. Glatz AC, Gaynor JW, Rhodes LA, Rychik J, Tanel RE, Vetter VL, *et al*. Outcome of high-risk neonates with congenital complete heart block paced in the first 24 hours after birth. J Thorac Cardiovasc Surg 2008;136:767-73.
2. Kussman BD, Madril DR, Thiagarajan RR, Walsh EP, Laussen PC. Anesthetic management of the neonate with congenital complete heart block: A 16-year review. Paediatr Anaesth 2005;15:1059-66.
3. Nakanishi K, Takahashi K, Kawasaki S, Fukunaga H, Amano A. Management of congenital complete heart block in a low-birth-weight infant. J Card Surg 2016;31:645-7.
4. Donofrio MT, Gullquist SD, Mehta ID, Moskowitz WB. Congenital complete heart block: Fetal management protocol, review of the literature, and report of the smallest successful pacemaker implantation. J Perinatol 2004;24:112-7.
5. Gregoratos G, Abrams J, Epstein AE, Freedman RA, Hayes DL, Hlatky MA, *et al*. ACC/AHA/NASPE 2002 guideline update for implantation of cardiac pacemakers and anti-arrhythmia devices–summary article: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (ACC/AHA/NASPE Committee to Update the 1998 Pacemaker Guidelines). J Am Coll Cardiol 2002;40:1703-19.
6. Bang SR. Neonatal anesthesia: How we manage our most vulnerable patients. Korean J Anesthesiol 2015;68:434-41.
7. Miller, Ronald D. Miller's Anesthesia. 7th ed. Philadelphia, PA: Churchill Livingstone/Elsevier; 2010.
8. Butler-O'Hara M, Buzzard CJ, Reubens L, McDermott MP, DiGrazio W, D'Angio CT. A randomized trial comparing long-term and short-term use of umbilical venous catheters in premature infants with birth weights of less than 1251 grams. Pediatrics 2006;118:e25-35.
9. Vutskits L, Culley DJ. GAS, PANDA, and MASK: No evidence of clinical anesthetic neurotoxicity! Anesthesiology 2019;131:762-4.
10. APA consensus guideline on perioperative fluid management in children v 1.1 September 2007 - APAGBI Review Date August 2010.