Anaesthetic Management of a Patient with Sick Sinus Syndrome with Transvenous Pacemaker, Posted for Gastrojejunostomy and Vagotomy

■ Shruti AB<sup>1</sup> ■ Senthil Nath M<sup>2</sup> ■ Vishwanathan PN<sup>3</sup>.

### SUMMARY

A patient with bradycardia is always the one who needs intensive monitoring and care under the influence of anaesthesia. These patients are at a high risk of going in for cardiac arrest. We encountered one such patient with sick sinus syndrome with a **tranvenous pacemaker** in situ. After induction and intubation the patient had a cardiac arrest. He was **resuscitated** and revived. We shall here discuss the intra operative management of this case.

## **KEY WORDS**:

Sick sinus syndrome, Trans venous pacemaker, Resuscitation.

#### **INTRODUCTION:**

Sick sinus syndrome is characterized by a variety of arrythmias and may present with palpitation, dizzy spells or syncope, due to intermittent tachycardia, bradycardia or pauses with no atrial or ventricular activity.<sup>1</sup> A preoperative history of syncope or documented bradycardia introduces the possible need for prompt of a prophylactic transvenous cardiac pacemaker before induction of anaesthesia.<sup>2</sup>

#### **CASE REPORT :**

A fifty one year old male patient presented with epigastric discomfort and vomiting after consumption of food from ten days. He was diagnosed to have gastric outlet obstruction and was posted for gastrojejunostomy and vagotomy. On pre anaesthetic evaluation it was found that the patient had bradycardia with a heart rate of 46-48 beats per minute. On further evaluation the Electrocardiograph showed sinus bradycardia with no other abnormalities. Despite the vomiting the serum electrolytes were impeccable. All other parameters including renal, thyroid and liver parameters were normal.

1. Post graduate student

2. Post graduate student

3. Prof and HOD, Department of anaesthesiology, JSS Medical college and hospital, Mysore, Karnataka.

• Karnataka Anaesthesia Journal

The patient was referred to the cardiologist where a single lumen temporary pacemaker was placed in the internal jugular vein. It was paced at 80 beats per minute. The patient was evaluated and taken up for the proposed surgery. He had a Ryle's tube in situ. He received no feeds 6 hours prior to the surgery and was given Tablet Ranitidine 150mg and Tablet Alprazolam 0.5mg through Ryle's tube the previous night. The next morning he was shifted to the operation theatre. The preoperative vitals showed a pulse rate of 84 beats per minute (pacemaker in pacing mode), blood pressure of 124/84 mmHg and saturation of 100%. The an oxygen Electrocardiograph showed pacemaker spikes. He was premedicated with intravenous Midazolam 0.5mg, Fentanyl 100 µg, 2% Plain Xylocaine 60mg and Ondansetron 4mg. The patient was preoxygenated for 3 minutes with 100% oxygen. He was induced with Thiopentone 5mgkg<sup>-1</sup>. The muscle relaxant used was Vecuronium 0.1mgkg-1. He was intubated with a 9.0mm cuffed portex endotracheal tube. It was connected to Bain's circuit and the air entry was bilaterally equal. The patient at this point witnessed a witnessed cardiac arrest. He developed a ventricular tachycardia at the rate of 170 beats per minute. No pulses were palpable. A DC shock of 200 joules was given along with injection adrenaline intravenously. The Electrocardiograph continued to show the same and the shock was repeated after three minutes with adrenaline. The third shock was followed by sinus rhythm at the rate of 120 beats per minute. The surgeon was asked to proceed with the surgery. The surgery lasted for 2 hours without any intraoperative complications. The patient was reversed using Neostigmine 0.05mgkg<sup>-1</sup> and Atropine 0.02mgkg<sup>-1</sup> and shifted to the Intensive care unit with endotracheal tube in situ. The pacemaker continued to be in pacing mode. Later the pacemaker was changed to sensing mode and finally switched off. The patient was successfully weaned off the ventilator and extubated. The pacemaker was removed and the heart rate was maintained at 80 beats per minute.

# **DISSCUSSION :**

The name Sick sinus syndrome was first used in 1968 by Ferrer. Cardiac output is determined by the product of stroke volume and heart rate. Bradycardia may result in insufficient heart rate to sustain cardiac output and hence oxygen delivery to the tissue beds. In older patients bradycardia may reflect an ischemic myocardium which is unable to maintain a stable electrical rhythm. A prompt response to bradycardia under anaesthesia as some causes are rare and/or obscure, and the hemostatic mechanism may be impaired by anaesthetic agents.<sup>3</sup>

Various anaesthetic drugs cause numerous effects on the cardiovascular system. Hence, it becomes vital to analyze the action of these drugs. For example, in this case, Ondansetron a  $5HT_3$  selective serotonin receptor antagonist used as premedication is known to cause atrial fibrillation, cardiac dysarryhthmias, fatal ventricular tachycardia and severe bradycardia.<sup>4</sup>

On the other hand the induction agent used that is Thiopentone sodium is known to cause an increase in the heart rate(10-36%) resulting from the baroreceptor mediated sympathetic reflex as the response to decrease in cardiac output and blood pressure.<sup>5</sup> This negative ionotropic effect may lead to increase in myocardial oxygen demand. Fentanyl is known to have positive ionotropic effects whereas Midazolam does not have much effects on the cardiovascular system.

It is interesting to know that Vecuronium by itself does not cause bradycardia but its non vagolytic action goes unopposed when used with opiods(e.g Fentanyl) leading to bradycardia and even asystole. Thus, this could have been the cause of asystole in this patient. As the patient was on pacemaker in pacing mode the occurrence of asystole becomes obscure. Pacemaker failure could have been the cause as a result of lead failure during repositioning which leads to under sensing or over sensing of impulses. Physiological changes during intubation include increase in heart rate and blood pressure which can be blunted by deepening the plane of anaesthesia and smooth laryngoscopy.

In this case the cause of ventricular tachycardia seemed to be more due to the drugs and probable lead failure. Despite of these reasons the patient was operated relieved of his symptoms and returned home. To avoid such circumstances from recurring it is advisable to do preparation for a cardiac surgery even though it is a non cardiac surgery which includes the presence and involvement of both the anaesthesiologist and a cardiologist. Hence, it is a challenge to anaesthetize a cardiac patient for non cardiac surgery being managed by a non cardiac anaesthesiologist.

## **REFERENCES :**

- 1. Bloomfield P, Bradbury A, Grub NR, Newby DE., Cardiovascular disease in : Davidson's principles and practice of medicine, 20<sup>th</sup> edition. Pg;569
- 2. Stoelting's Anaesthesia and co- existing disease,5th edition. Pg ; 253
- 3. M Watterson, RW Morris, RN Westhorpe, JA Williamson. QUAL SAF Health Care 2005; 14 eq
- 4. Mark JB, Slaughter TF., CVS Monitoring in : Miller RD, Fleisher LA, John RA, Savarese JJ, Wiener-Kronish JP, Young WL. Miller's Anaesthesia 6<sup>th</sup> edition. Churchill Living stone, Philadelphia. Pg ; 1265-1362
- Reeves JG, Petersa Glass, David A, Lubarsky Mathew D, Mc, Mc Evoy, Richardo Matinez-Ruiz., Miller's Anaesthesia 7<sup>th</sup> edition Churchill Living stone. Philadelphia. Pg ; 733-34, 883