Negative Pressure Pulmonary Oedema after Sedation in a Patient Undergoing Pacemaker Implantation

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ABSTRACT
Negative pressure pulmonary oedema (NPPE) is a manifestation of upper airway obstruction and is seen after extubation postoperatively. The large negative intrathoracic pressure generated by forced inspiration against an obstructed airway is thought to be the principal mechanism involved. This negative pressure leads to an increase in pulmonary vascular volume and pulmonary capillary transmural pressure, creating a risk of disruption of the alveolar–capillary membrane. The early detection of the signs of this syndrome is vital to the treatment and to patient outcome. It is rare and usually occurs in a clinical setting of postextubation, laryngospasm, epiglottitis, croup, endotracheal tube obstruction, post tonsillectomy/adenoectomy and other conditions. The Authors report here a case of negative pressure pulmonary oedema occurring after intravenous sedation in an elderly male undergoing dual chamber permanent pacemaker implantation procedure.

CASE REPORT
An 86 year male with symptomatic Mobitz Type 2 AV block was prepared for creation of a left side pectoral pocket to implant a dual chamber permanent pacemaker under local anaesthesia with strict aseptic precautions in the cath lab. Patient had no past history of cardiac or pulmonary disease in the past and remained asymptomatic except for one episode of syncope coinciding with Mobitz Type 2 AV block one day before being taken up for dual chamber permanent pacemaker implant procedure. Inj. Lignocaine (1 %) 15 ml was infiltrated locally into the skin along the length of the intended incision as well as more deeply and medially with an intent to achieve adequate anaesthesia. As patient did not experience adequate analgesia after additional infiltration of Inj. Lignocaine to a maximum permitted dose of 3mg/kg, patient was administered Inj. Fentanyl 50 mcg intravenously to achieve better analgesia. Incision was placed for creation of pectoral pocket. Soon, it was noticed that the oxygen saturation on pulse oximetry was falling to less than 70 % and lung auscultation revealed bilateral crepitations. The heart rate and blood pressure remained unchanged. The patient was drowsy and sedated and it was noticed that the tongue had fallen back to cause obstruction to the upper airway. Patient underwent immediate endotracheal intubation with artificial ventilation. Pink frothy sputum was noticed in the endotracheal tube. Simultaneously, patient received intravenous Frusemide 40 mg to treat acute pulmonary oedema. The oxygen saturation improved quickly after the above measures and pacemaker implantation procedure was completed successfully. A perioperative chest x-ray confirmed presence of acute pulmonary oedema. (Figure 1). Patient was extubated uneventfully after six hours with no further clinical events and was discharged from the hospital after three days.

DISCUSSION
The first description of the pathophysiological correlation between creation of negative pressure and the development of pulmonary edema was in 1942 by Warren et al.1 The relationship between pulmonary edema and upper airway obstruction in two children, who had croup and epiglottitis was reported by Capitanio et al.2 The first report on the clinical significance of this phenomenon in three adult patients, who experienced onset of pulmonary edema minutes to hours after severe acute upper airway obstruction was by Oswalt et al.3 Negative pressure pulmonary edema (NPPE) is also called post obstruction pulmonary edema (POPE). The presentation of NPPE can be immediate or delayed.4,5 Negative pressure pulmonary oedema is classified as Type I or Type II.6,7 Type I NPPE develops immediately after onset of acute airway obstruction and Type II NPPE develops after the relief of chronic upper airway obstruction. As Type I NPPE develops usually with upper airway acute obstruction or after manipulation of the airway surgically, some authors call it laryngeal spasm-induced pulmonary edema.6 Other factors that increase the risk of Type I NPPE are hanging, strangulation, upper airway tumors, foreign bodies, epiglottitis, croup, choking, migration of Folly’s catheter balloon used to tamponade the nose in epistaxis, near drowning, endotracheal tube (ETT) obstruction, goitre, and mononucleosis. Type II NPPE can result after relief of upper airway obstruction caused by big tonsils, hypertrophic adenoids, or a redundant uvula. According to one study, the incidence of developing Type I NPPE associated with acute postoperative upper airway obstruction is 9.6 – 12%, whereas the incidence of developing Type II NPPE is 44%. In adults about 50% of NPPE occurrences are due to postoperative laryngospasm.8 NPPE is mainly described in a postoperative clinical situation wherein non-cardiogenic edema results from laryngospasm or other forms of upper airway obstruction following extubation.4,2 NPPE begins with a significant
negative pressure pulmonary oedema can occur with sedation due to falling back of the tongue and early recognition is life saving.

CONFLICT OF INTEREST
Nil

ACKNOWLEDGMENT
Nil

ABBREVIATIONS USED
NPPE: Negative pressure pulmonary oedema; PEEP: Positive end expiratory pressure.

REFERENCES
Venkatesh et al.: Negative Pressure Pulmonary Oedema after Sedation