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Periprocedural Management during Therapeutic Cardiac Catheterization in Patients with Sleep Apnea Syndrome: Report of Three Cases and Review of Literature

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Abstract

Most of the patients with sleep apnea syndrome (SAS) also known as sleep disordered breathing are not diagnosed before undergoing any cardiac interventional procedure. Many of them can safely undergo outpatient procedure under sedation or anesthesia. Few of them with moderate to severe grade of SAS, who are not optimized medically, may create problem and need special consideration. We managed three such cases in cardiac catheterization laboratory; two of them were not diagnosed before. The periprocedural problems we faced in these patients are narrated in this article along with review of literature. Some suggestions for management of such patients undergoing therapeutic cardiac catheterization are also highlighted.

Keywords
- AICD
- ASD device closure
- sleep apnea

Introduction

Cardiac catheterization can be diagnostic or therapeutic and mostly done as an outpatient procedure. It is indispensable that some form of anesthesia is required during this procedure, which varies from mild sedation to general anesthesia (GA). Patients with additional medical problem need special attention regarding the effect of anesthetics on other organ system leading to morbidity and mortality. One among these groups is the patient suffering from sleep apnea syndrome (SAS). This syndrome comprises of a spectrum of disorders characterized by excessive, abnormal breathing abnormalities during sleep which varies from respiratory effort-related arousal to apnea and hypopnea leading to intermittent disruption in gas exchange and interruption of sleep. These patients are often not diagnosed during preintervention period and may cause periprocedural issues. No literature is available about the management of such cases in cardiac catheterization laboratory. In the past, we managed three such cases, among whom two had undergone transcatheter device closure for atrial septal defect (ASD) and artificial implantable cardioverter defibrillator (AICD) was implanted in the third patient. The details of these cases are presented below. We too discussed various anesthetic aspects of such patients undergoing interventional procedure and literature review about the periprocedural management of such patients.

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Case Reports

Case 1

This 57-year-old male, posted for interventional closure of ASD. He was a heavy smoker, known case of chronic bronchitis (15 years), and SAS (2 years). His respiratory and sleep symptoms were nearly controlled with salbutamol, frusemide, bromhexine, and intermittent nasal continuous positive airway pressure (nCPAP) therapy. On physical examination, he was 165 cm tall and weighing 80 kg (body mass index [BMI] = 29.4 kg/m^2). His hemogram and blood chemistry values were within normal range. Arterial blood gases (ABGs) were: pH 7.41, PaO_2 92 mm Hg, and $PaCO_2$ 43 mm Hg in room air. The polysomonographic picture (8 months old) is presented in ►**Table 1**.

Verbal reassurance and explanation was given during the preoperative visit. No premedicants were advised. The airway was Mallampati grade II. Except frusemide, the other drug therapies were continued. On arrival in the cardiac catheterization laboratory, continuous electrocardiogram (ECG) and pulse oxymetry monitoring was started. Angiography was performed under local anesthesia after insertion of intravenous and intra-arterial lines, and baseline hemodynamic measurements were obtained. The coronaries and ventricular function was found to be normal. Transesophageal echocardiography (TEE) was performed under topical oral and pharyngeal anesthesia supplemented with ketamine in a dose of 1 mg/kg body weight plus glycopyrrolate 0.1 mg intravenously, to analyze the suitability of the defect for a device closure. The TEE probe was then taken out and oxygen via venti mask was started. The patient was calm throughout, started snoring, and developed sudden apnea and bradycardia (< 30 beats/min) 20 minutes after ketamine administration. During this period no cardiac intervention was going on. His cardiac condition was immediately improved with assisted mask ventilation using FiO_2 1.0. But as he was sleepy, continued to snore and had repeated episodes of apnea with fall in oxygen saturation when assisted ventilation was stopped, his trachea was intubated under the cover of O_2:sevoflurane. On laryngoscopy, the vocal cords were found to be relaxed, the oropharyngeal soft tissue was normal. During the subsequent procedure anesthesia was maintained with O_2:air:sevoflurane (0.5–2%). The septal defect was closed with Amplatzer septal occlude of size 23 mm. Once it was implanted and before releasing it, a repeat TEE evaluation assessed the adequacy of its position, the persistence of residual shunt, and the stability of the device within the septum and then sevoflurane was discontinued. His spontaneous respiratory efforts were regained after 25 minutes, but were not adequate. He was drowsy, unable to lift his head with verbal commands. He was shifted to coronary care unit, ventilated in synchronized intermittent mandatory ventilation mode at a rate of 8 breaths per minute with a positive end-expiratory pressure of 5 cm of H_2O for 5 hours followed by CPAP mode for 4 hours and then extubated while he was fully awake, and his respiratory efforts were adequate. He was discharged after 48 hours with nCPAP on.

CASE 2

He was a 32-year-old male, admitted for ASD device closure on the day of intervention. A short history revealed that he was a restless sleeper at night, as noted by his wife. X-ray chest and laboratory examination revealed no abnormalities. He was 86 kg and 175 cm in height (BMI = 28.1 kg/m^2). The possibility of sleep disordered breathing (SDB) was kept in mind and also a high BMI. All preparation for handling any airway emergency was done. The preanesthetic protocol, monitoring, and angiographic procedure were similar to that of the first patient. He was given injection ketorolac tromethamine 30 mg intravenous stat as the sole analgesic. Before the TEE, injection propofol was given at a dose of 0.2 mg/kg. He became drowsy but his respiratory effort was adequate. Propofol was continued at a dose of 10 to 20 µg/kg/min to keep the sedation score between 2 and 3 as described by Ramsay et al.[1] The ASD was closed with an Amplatzer Umbrella of size 25 mm. Propofol infusion was discontinued after a repeat TEE confirmation of the device position. Oxygen supplementation (to provide a FiO_2 between 0.4 and 0.6) was done via venti mask during the procedure. In the initial 10-minute period, the respiratory rate was decreased from 16 to 8 to 10/min and then returned to normal. The ABG during this interval showed a PO_2 of 94 mm Hg and PCO_2 of 55 mm Hg; the PO_2 was increased to 118 mm Hg and PCO_2 to 35 mm Hg at 30 minutes. The oxygen saturation by pulse oxymetry remained between 94 and 96% throughout the procedure. There was no disturbance in cardiac rhythm and blood pressure during the period of propofol infusion. He responded to verbal commands 32 minutes after the discontinuation of the infusion. His respiratory rate, clinically estimated tidal volume, and respiratory pattern were found to be normal. The plan was to keep him under close supervision but without any oxygen therapy for a further period of 24 hours. At 18th hour he suddenly started snoring vigorously and suddenly awake. There was sudden tachycardia but no pain. He was oxygenated overnight. His heart rate became

Table 1 Polysomnography findings of the patients

Case	Study duration (h)	AI (per hour)	Lowest SaO_2 (%)	Maximum SaO_2 with O_2 (%)	Oxygen desaturation index/h of sleep
1	8.35	35	76	80–93	5
2	7.5	10	85	96	1
3	7.8	8	86	98	1

AI: Apnoea Index.

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normal but he had frequent arousals on that night. His vital function and ABG revealed no abnormalities during the hospital stay for the next 48 hours. A sleep study was done a week later and demonstrated mild degree of sleep apnea (►**Table 1**).

CASE 3

This patient was a 65-year-old male, 165 cm tall weighing 65 kg (BMI 23.9 kg/m^2); suffering from pleomorphic ventricular tachycardia following a myocardial infarction, posted for AICD implantation. He was a known case of diabetic mellitus and on glibenclamide therapy. He had undergone a subtotal thyroidectomy 2 years back for hypothyroid goiter and presently on thyroxine therapy. The thyroid function test as repeated a week before was found to be normal. As he arrived on the morning of the procedure, he was taken straight to the laboratory, but only after being explained regarding the intervention and requirement of anesthesia. Aspiration prophylaxis and injection ketorolac 90 mg intramuscularly was given 30 minutes before the intervention. The monitoring was same as the other two patients.

Left subclavian vein approach was done under local anesthesia for implantation of the device. The electrodes were inserted under fluoroscopic control. The device was anchored with sutures to pectoral muscle and deep fascia and skin was closed. During defibrillation testing, injection propofol 500 µg/kg was given as bolus to produce deep sedation (Ramsay score 5 to 6). Sudden hypotension and bradycardia occurred during the test and recovered immediately with fluid therapy. He received O_2 via face mask while under propofol. Recovery from propofol was satisfactory, he was fully conscious, breathing normally, the ABG and hemodynamics were normal. The procedure was finished within 30 minutes. Immediately after he developed severe snoring and intermittent periods of apnea and hypopnea frequently and a marked fall in oxygen saturation. He was intubated under transtracheal injection of 1% lidocaine (to avoid muscle relaxant). During laryngoscopy his false vocal cords were found to be congested and hypertrophied, but oropharyngeal soft tissue was normal. He was mechanically ventilated under the cover of O_2:air (50–50) for a further period of 45 minutes till the spontaneous respiratory efforts came back and then extubated. The postprocedure monitoring was same as others and he was ready for home after 72 hours. During this time a sleep study demonstrated abnormal polysomnographic picture suggesting SAS.

Discussion

Patients with additional medical problems along the cardiovascular system pose a challenge for the anesthesiologist during cardiac interventional procedure. SAS is a common but often undiagnosed condition,[2] the earliest description of which was made early in the 19th century and subsequently it was recognized as a public health problem.[3]

There is yet no broad consensus regarding standard definition for this disorder. It is characterized by the association of apnea and or hypopnea during sleep with a combination of symptoms and signs related to sleep fragmentation and hypoxic exposure. Though snoring is the cardinal feature of SAS, normal individuals too have pause in ventilation during sleep and one-quarter of adult population is thought to snore.[4] But existence of apnea for greater than 10 seconds during both stages I and II rapid eye movement (REM) sleep with fall in SaO_2 with each apnea, increase in CO_2 during night, and improvement of day time symptoms and general performance with treatment are the features by which a patient can be differed from normal. Apnea in this context is defined as a cessation of airflow of at least 90% that has to exceed 10 seconds' duration and hypopnea is defined as a reduction in respiratory effort or airflow (at least 30%) for more than 10 seconds accompanied by a desaturation of 3% or more and/or electroencephalographic evidence of arousal.[5] The apnea-hypopnea index is the number of apneas and hyperpnea per hour of sleep and is used more or less interchangeably with the term respiratory disturbance index.[6]

The apneas may be obstructive (persistent effort without airflow), central (effort is absent), or mixed (combination of obstructive and central component). The syndrome gets resolved when the sleep-induced respiratory disturbance is eliminated. Although it occurs in all ages it is more common in the late middle age. In spite of the fact that the obese male population is commonly affected, it is also recognized that the patient need not be obese or somnolent to have significant sleep fragmentation or airway obstruction, which happened in our third patient. He had no history of sleep apnea, did not appear to be obese, and had no abnormal features suggestive of difficult intubation. During the subtotal thyroidectomy the intubation was successful and the anesthesia course was uneventful. However, this time there was severe snoring fall in O_2 saturation and repeated apnea-hypopnea happened after the procedure.

Apart from the history different techniques are now available for recognition of SAS. Among which "saw-tooth sign" in spirometry and polysomnography (PSG) are the most reliable one.[6,7] Home respiratory polygraphy can be used as an alternative to PSG in diagnosis of SAS. Some authors advocated the use of PSG only in patients with concomitant disease or where Home Respiratory Polysomnography (HRP) results are inconclusive.[8,9] Nagappa et al in a recent meta-analysis concluded that in ambulatory patients one should use the STOP (snoring, tiredness, observed apnea, and high blood pressure)–Bang (body mass index, age, neck circumference, and gender) questionnaire to identify suspected patients with SAS, so that periprocedural management can be more focused and appropriate.[10] This as a screening tool also has been described by several other authors. This has 4 self-reporting (STOP) and 4 demographic (Bang) items. In the initial validation study a score of at least 3 demonstrated sensitivity of 84, 93, and 100% to detect all grades of SAS (mild, moderate, and severe, respectively).[11]

Problems during anesthetic management have been described by various authors, which include difficult intubation and maintenance of airways because of associated anatomical and physiological problems.[12]

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To our knowledge the anesthetic management of patients with SAS undergoing therapeutic cardiac catheterization procedure has not been described before. As these patients get admitted usually on the same day of the procedure and a limited time is available for a complete review correct diagnosis is most often missed, so also the proper preanesthetic preparation. Hence, special attention should be paid if a snoring patient is of middle age and have associated diseases like chronic obstructive pulmonary disease, diabetes mellitus, hypothyroidism, craniofacial anomalies, and if he is a chronic alcoholic or smoker.[13] An assessment of the airway is a must even if the preanesthetic visit is short and preparation for the management of difficult airway should be kept ready. Associated compromised respiratory and cardiovascular status should be optimized before as per any other cardiac patient undergoing routine surgical procedure. No sedative premedication as a rule. A detailed explanation regarding the procedure and anticipated anesthetic problem should be explained to the patient. Only the first patient had received nCPAP for a period of 2 months, a month before the procedure and a month after the procedure. nCPAP therapy is recommended in SAS patients to reduce the tongue volume and upper airway edema.[14] It also acts as a pneumatic splint to the upper airway and reduce nasal inflammation.[15] The normal appearance of the glottis and oropharyngeal structure in the first patient is a proof to this fact.

We used ketorolac for analgesia because it is not only a potent analgesic but also has negligible effect on the respiration, hemodynamic, and central nervous system.[16–18] Ketamine has been chosen in the first patient because this drug has a very minimal effect on central respiratory drive, with an unaltered ventilator response to carbon dioxide, causes bronchodilation, and also improves pulmonary compliance.[19] This patient did not develop any decrease in respiratory rate, clinically estimated tidal volume, and oxygen saturation during the first 20 minutes of ketamine administration, but had a slow depression of respiration and apnea subsequently. While McArdle has a query regarding the role of ketamine in sleep apnea patients, Thangathurai and Roffey find it as a safe drug in this population.[20,21] We opted for sevoflurane during intubation of this patient not only to avoid the muscle relaxant but also anticipating an early recovery. Olverio et al have described case of delayed recovery till 40 minutes with inhalational anesthesia, and in our case it lasted for 5 hours.[22] The use of propofol along with remifentanil without any problem has been noted by Fassbender et al in a cohort of SAS patients undergoing surgery for moderate duration.[23] The dose of propofol required to produce conscious sedation in normal patients varied from 0.025 to 0.15 mg/kg/min.[24] Our second patient needed a dose of 0.2 mg/kg followed by 10 to 20 µg/kg/min to maintain a Ramsay sedation score between 2 and 3, which was lesser than the normal patients. He was comfortable and had no respiratory jeopardy during the procedure. As propofol can depress the ventilatory response to hypoxia, we administered supplemental oxygen to our patient during the drug infusion. The recovery time was also short (32 minutes) and the quality of recovery was satisfactory.

The development of apnea and hypopnea after an interval of 35 minutes of propofol administration and associated comorbidities, his family members were enquired later regarding any symptom of sleep apnea and history of snoring justified for further investigation which revealed the presence of SAS.

The appearance of SDB and adverse cardiovascular events is not uncommon in the recovery room as it has been reported before.[25,26] So these groups of patients should be closely supervised for a further time period with monitoring of the respiratory function. Postprocedural nocturnal oxymetry is advisable. Oxygen therapy should be supplemented with caution as the patient may be dependent on the hypoxic respiratory derive. Additionally, oxygen does not prevent apnea but reduces, with only limited success, desaturation and the duration of apnea. A safe period for discharge is an important concern to avoid undue rehospitalization.

Though the management of such cases is gradually getting standardized in general surgical settings, there is no literature available for guidance regarding which patient can be safely managed in cardiac cath laboratory during procedural sedation.

Degree of desaturation is more important in these patients rather than the number of respiratory events, because the former group have minimal respiratory reserve and more prone for adverse events. Postintervention, the patients are transferred to cardiac recovery unit and usually kept under 24-hour observation. SAS patients, however, need a longer period of stay because most of the events happen in the first 48 hours, as noted in our patients. The goal of postprocedural monitoring is not only to diagnose the adverse events, but also to document the degree of oxygen desaturation in patients while they are sleeping with or without oxygen, so that complications can be prevented or treated if it happens.

Aspiration/reflux precaution is a must especially in obese patients because they have a larger volume of gastric fluid and lower gastric pH and are at increased risk of aspiration during emergency intubation or extubation.[27] Before any intervention, patients are sleep deprived due to anxiety. This is more pronounced in this group of patients. Moreover, once the effect of anesthetic agents in over, patients are more likely to have a rebound of delta and REM of sleep and have more chance to get into severe sleep apnea. Local anesthesia or monitored anesthesia care is mostly preferred for these patients in outpatient procedures. Careful monitoring during sedation is mandatory because these patients are more sensitive to sedatives. GA with a secure airway is preferred if patients require moderate deep sedation.[28]

It is essential to improve the sleep quality prior to a procedure under anesthesia to reduce the incidence of postprocedural apnea episodes. Patients who are on CPAP or bilevel PAP should be asked to use this machine, weeks prior to and after a procedure under anesthesia. Routine use of narcotics prior to a procedure should be avoided because of chance of sudden death in preoperative holding area. Life-threatening hypoxemia by narcotics and reduction of upper airway dilator muscle tone by benzodiazepines increase the

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duration of respiratory events in such cases, hence if at all these drugs are required, patient should be monitored carefully. Optimal control of comorbidities prior to a major intervention, hopefully decrease the postprocedural complications. All the SAS patients to be admitted a day prior to the procedure for a detailed anesthesia plan. In case GA is required, preoxygenation for 3 to 5 minutes' period is a must to improve oxyhemoglobin saturation before intubation.

In case of severe SAS, a difficult intubation cart is to be kept ready and modern concepts for teleconsultation should be discussed. A planned tracheostomy should be considered in those with severe sleep apnea, failure of CPAP, life-threatening cardiac arrhythmias, and severe oxygen desaturation.[29] Use of local anesthetics on oropharynx should be used carefully. Even though these agents reduce narcotic requirement they may worsen apnea due to their effect on airway mechanoreceptors that contribute to the arousal stimulus and apnea termination.[30]

Postcardiac interventional procedures usually do not require any analgesia. If at all required especially patients who have an incision (AICD insertion or pacemaker insertion) can be managed with non-narcotic analgesics, for example, acetaminophen, tramadol hydrochloride, nonsteroidal anti-inflammatory agents (ibuprofen, naproxen, ketorolac tromethamine), or the cyclooxygenase 2 agents (celecoxib). Topical anesthetics are also useful supplements to control pain. Dexmedetomidine is a useful sedative in these patients because of its opioid-sparing effect and lack of respiratory depression.

Close monitoring for hypoxemia or other complications is important. Continuous monitoring with the help of pulse oximeter, ECG, and noninvasive blood pressure is essential. If possible, it is always better to place the patient in nonsupine position postprocedure to decrease the severity of apnea. Patients with confirmed diagnosis of SAS and who are on prior CPAP should use their CPAP in the postintervention period though there are no randomized trials/reports on this. Most important is that a formal sleep evaluation should be done for these patients after discharge from the hospital.

Extubation is another critical period for which the general acceptance (extubated awake) should be followed and extubation to be done in the presence of appropriate personnel and equipment so as to be able to replace the tube if necessary.[31] Narcotic use should be minimized, because their effect may persist during postextubation period leading to unanticipated complications. Postintervention monitoring is important and the first 48 hours is the most critical time for complications. Incidences of deaths are also reported, which can be due to the accumulated effect of narcotic medications, sleep deprivation, and REM rebound.[32]

Conclusion

SAS is highly prevalent and undiagnosed in medical and surgical population most of the times because snoring is often unnoticed and ignored. Patients with SDB are at high risk of unexpected complications during invasive interventions under sedation because sedation, anesthesia, opioids, and REM sleep rebound have been shown to cause worsening of sleep apnea in periprocedural period that may lead to increased risk of complication. Screening questionnaires such as the Berlin, STOP-Bang, or American Society of Anesthesiologists checklist are easy to administer preoperatively and have shown to identify high-risk patients. A detailed explanation need to be given to the patients/guardian regarding the possible periprocedural complications. For any emergency an algorithm for management should be established. The high-risk patients should also have a formal sleep evaluation for the long-term management of their sleep apnea after discharge from the hospital. It has become increasingly apparent that SDB is associated with heightened perioperative risk. Furthermore, the condition still remains undiagnosed in patients presenting for cardiac catheterization procedure. Anesthesiologist must distill information from clinical report to make key decisions for optimizing periprocedural care in interventional cardiac catheterization laboratory. An anesthesiologist should understand the basic information and salient feature of a typical PSG report relevant to his/her management.

Conflict of Interest
None declared.

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