BCG instillation into the urinary bladder may take one of the two forms: (1) a hypersensitivity reaction producing interstitial pulmonary infiltrates as its radiographic finding and a lymphocytosis on BAL characterized by activated T helper cells, and (2) a more classic BCG mycobacteremia with resultant pulmonary infection and clinical illness, mililiary chest radiographic pattern and granuloma formation which can be documented by lung biopsy, as shown in our patient.

The treatment, like the pathogenesis of the pulmonary complication of intravesical BCG, remains controversial. Based on the postulated two separate forms of reaction in the lung, it seems reasonable to consider observation alone or the use of corticosteroids with isoniazid prophylaxis in the hypersensitivity reaction. However, in patients who demonstrate classic micronodular (miliary) pattern of chest x-ray film abnormality and in whom granulomas are found, with or without BCG organisms on lung biopsy, two-drug antituberculosis therapy is suggested. Until more information is available, treatment with isoniazid and rifampin for nine months seems a reasonable course.

REFERENCES

Cardiac Failure Presenting as Sleep Apnea*

Elimination of Apnea following Medical Management of Cardiac Failure

A patient with symptoms of sleep apnea syndrome had signs of congestive cardiac failure. A sleep study fulfilled the criteria for sleep apnea. Features of Cheyne-Stokes respiration coexisted. Management of the cardiac failure by weight loss principally due to diuretic use eliminated the symptoms of sleep apnea. (Chest 1988; 94:1298-1300)

Cheyne-Stokes respiration is one of the well-recognized respiratory disorders associated with cardiac failure. Findley and colleagues' found that this breathing disorder was associated with 100 percent mortality over the following six months. Another respiratory disorder associated with cardiac failure is paroxymal nocturnal dyspnea, which when severe, can result in insomnia.2 Dark et al3 recently evaluated the prevalence of respiratory disorders during sleep in six patients with cardiac failure. Factors predisposing to the sleep apnea syndrome such as weight greater than 125 percent of normal were excluded; all six patients had breathing pattern abnormalities, central apnea being the most common. To our knowledge, no patient with cardiac failure has presented with the symptoms of sleep apnea. We describe such a case.

CASE REPORT

This 55-year-old man, who had a four-vessel coronary artery bypass for angina in 1983, was referred to the Fresno VA Medical Center for evaluation of possible sleep apnea. He gave a history of heavy snoring for ten years, with an increase in intensity over the past two years. Over the previous two months, these symptoms had progressed. The patient's wife noted that when the patient was sleeping, he would stop breathing for up to a minute at a time. This was followed by loud snoring, choking, and gasping, and the patient would repeatedly wake up at night. Other recent symptoms apart from insomnia included daytime somnolence. His sleepiness was so worrisome that the patient stopped driving for fear of falling asleep at the wheel. He also admitted to increased irritability, recent development of impotence, peripheral edema, and decrease in exercise tolerance. He had gained approximately 13.5 kg over the previous two to three months. He was obese, weighing 108 kg (42 percent of ideal body weight) and had signs of congestive heart failure. This was confirmed radiographically.

A nocturnal sleep study was performed on the night of his admission. A strain gauge was used to evaluate chest wall movement, nasal thermistors were used to evaluate airflow, and a pulse oximeter (Ohmeda Biox II) was used to evaluate oxygenation; an EEG was not included. The sleep study began at 9:15 pm on the night of his admission. The patient fell asleep clinically within minutes of applying the recording materials. Soon afterward, cyclic falls in oxygen saturation from 95 percent to a nadir of 80 percent were noted. These desaturations occurred at a frequency of approximately one per minute and a duration of 30 seconds. The apneic index was 60 for several hours of the sleep apnea test. During these apneic episodes, chest wall movement was absent, indicating that the apneic episodes were principally central in origin, although there may also have been an obstructive element. A representative sleep record is shown in Figure 1. This abnormal pattern of breathing persisted throughout the night.

Results of other investigations included a gated equilibrium blood pool radionuclide ventriculography measurement indicating a left ventricular ejection fraction of 17 percent and a right ventricular ejection fraction of 20 percent. The left and right ventricles were diffusely hypokinetic. Arterial blood gas values with the patient breathing room air on admission were as follows: pH, 7.46; Pco2, 32 mm Hg; Pao2, 70 mm Hg; bicarbonate, 23; base excess, +1; oxygen saturation, 94 percent. Management of his

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Cardiac Failure Presenting as Sleep Apnea (Baylor et al)
cardiac failure was initiated the following day and included use of a diuretic, and an afterload reducing agent. He lost 17.1 kg within 18 days. His symptoms of sleep apnea completely resolved. Results of repeated nocturnal saturation test, nine days after the initial study while breathing room air, revealed that oxygen saturation remained greater than 90 percent throughout the study. Pulmonary function studies following recovery of cardiac failure showed FEV, 3.49 L (107 percent predicted); FVC, 4.30 L (103 percent predicted), and TLC, 6.92 L (109 percent predicted).

**Discussion**

The clinical diagnosis of sleep apnea syndrome requires the combination of appropriate clinical symptoms with an arbitrary number of at least 30 apneic episodes of at least ten seconds' duration, resulting in significant desaturation that occurs over a seven-hour sleeping period.4 Snoring, which is a common early feature of sleep apnea, does not constitute sleep apnea unless associated with other clinical features of sleep apnea and an abnormal sleep study test result as defined above. The event that transformed our patient from being a snorer to the development of symptomatic sleep apnea appears to have been the development of congestive heart failure.

Dark et al5 found an inverse correlation in a group of six patients with congestive heart failure between the left ventricular ejection fraction and the presence of breathing pattern abnormalities. In a group of 15 patients with left ventricular failure studied by Finkley et al,6 40 percent demonstrated five or more apneas per hour. Alex et al6 showed that of six patients with Cheyne-Stokes respiration due to congestive cardiac failure who were evaluated during daytime nap studies, five had evidence of sleep-induced upper airway occlusion during Cheyne-Stokes respiration similar to mixed apnea. None, however, had significant desaturation during these nap studies, and no patient had symptomatic sleep apnea. Upper airway obstruction occurred at the nadir of respiratory drive in Cheyne-Stokes respiration. This decrease in respiratory drive to the muscles of respiration will decrease upper airway dimension, especially in patients who are anatomically predisposed (i.e., history of snoring) to develop airway occlusion. Thus, although apnea is not uncommon in the presence of cardiac failure, it has not, to our knowledge, resulted in the development of symptomatic sleep apnea. This may be related to the frequency, duration, and degree of desaturation in cardiac failure, which, if severe enough, will result in symptomatic sleep apnea as in the patient described above. That significant weight loss and management of cardiac failure resulted in elimination of the symptoms of sleep apnea suggests that cardiac dysfunction played a major role in the etiology of sleep apnea.

Two methods have been used to diagnose definitively obstructive sleep apnea: (1) an invasive method, which required swallowing an esophageal balloon, although the associated discomfort limited its general application; and (2) a noninvasive method that is based on surface inductive plethysmography which measures chest and abdominal wall movement. We only measured chest wall motion; accordingly, a more marked degree of obstructive apnea may have been missed. We were surprised by the clinical and radiographic evidence of cardiac failure and its temporal relationship to the symptomatic sleep apnea syndrome. Although cardiac failure is known to be associated with apnea during sleep, symptomatic sleep apnea related to the development of cardiac failure and its elimination by management of cardiac failure is unusual. It is possible that our patient fulfilled the criteria for asymptomatic sleep apnea prior to the development of congestive heart failure, that congestive heart failure resulted in symptomatic sleep apnea, and that resolution of cardiac failure eliminated the symptoms of sleep apnea. We have not completely excluded the fact that the patient may have continued to have fulfilled some of the criteria for asymptomatic sleep apnea following resolution of his symptoms.

**References**

2 Rees PJ, Clark TJH. Paroxysmal nocturnal dyspnea and periodic respiration. Lancet 1979; 2:1315-17
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Arterial Oxygen Saturation during Nd:YAG Laser Photoresection of Endobronchial Tumors Under Local Anesthesia*

Use of Intermittent Supplemental Oxygen with Pulse Oximetry Guidance

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Although Nd:YAG laser photoresection of endobronchial lung tumor can result in signiﬁcant arterial oxygen desaturation, oxygen supplementation during procedures is often limited due to fear of intrabronchial combustion. We gave intermittent pulse supplemental oxygen to ten patients during 26 laser procedures performed under local anesthesia using SaO2 measured by a pulse oximeter as a guide. In four procedures (15.4 percent), severe oxygen desaturation contraindicated performing or completing laser phototherapy. In the remaining 22 procedures (84.6 percent), laser photoresection was safely and successfully performed without incident. Thus, pulse oximetry is a valuable tool and intermittent oxygen supplementation with pulse oximeter guidance an effective technique for maintaining adequate oxygenation during laser photoresection. (Chest 1988; 94:1300-01)

Safely providing adequate oxygenation during laser photoresection can be a difﬁcult task. The risk of hypoxemia1,2 with its related morbidity must be weighed against the danger of intratracheal combustion3,4 which is enhanced by high oxygen concentrations. This has created a therapeutic dilemma—the bronchoscopist may wish to give supplemental oxygen to avoid dangerous hypoxemia but cannot give too much oxygen for fear of ﬂame. Although the exact risk of intratracheal combustion is unknown, the bronchoscopist often limits supplemental oxygen. Dixon5 and Casey et al2 have stated that if the patient requires an FIO2 greater than 0.50, "the risk of ﬂame may outweigh the beneﬁt of the procedure." We now report our experience of giving limited supplemental oxygen to spontaneously breathing patients while monitoring oxygen saturation with a pulse oximeter.

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PROCEDURES

Twenty six procedures were initiated on ten patients (nine men) averaging 55.1 ± 27.7 years of age (range 41-83). Arterial blood gas values were measured as clinically indicated on room air (11, at least once for each patient) or on supplemental oxygen. The patients received sedation (meperidine, 50 to 100 mg and midazolam, 1 to 5 mg) and atropine (0.4 to 0.6 mg) preoperatively. All were given topical anesthesia with lidocaine and were then orally intubated using the technique of Hodgkin et al.6 Cardiac rhythm and SaO2 were continuously monitored, the latter with a pulse oximeter. The SaO2 as measured by pulse oximetry diﬀered from that measured by arterial blood gas analysis by no more than 1 percent for any measurements (16 measurements). Mechanical ventilation was used for one procedure. For the remaining 25 procedures, patients breathed spontaneously. Supplemental oxygen was administered via a "T-tube" at a rate of 4 to 6 L/min when the laser was inoperative. We have found 4 L/min O2 to correspond to an FIO2 of 0.37 – .41 and to 6 L/min to an FIO2 of 0.47–53. Oxygen was discontinued just prior to initiating laser phototherapy and re instituted each time the procedure was stopped to debride, suction, or allow the lasered area to cool. A precipitous decline in SaO2 or a decrease below 85 percent were considered an indication to temporarily cease laser phototherapy and reintroduce oxygen supplementation.

All laser procedures were performed through a ﬁberoptic bronchoscope using a Nd:YAG laser. Each laser pulse was limited to a maximum energy of 40 watts and a maximum time of 0.5 seconds.

Three sessions were terminated without application of laser therapy because of severe oxygen desaturation (less than 60 percent) upon introduction of the bronchoscope while the patient was still receiving supplemental oxygen (6 L/min). One procedure was halted after only 258 joulies had been delivered because of severe oxygen desaturation (SaO2 52 percent). In that patient, laser phototherapy with continuous supplemental oxygen still produced dangerous oxygen desaturation complicated by multifocal atrial tachycardia.

The remaining 22 procedures were successfully completed utilizing an average of 1,245 ± 599 joulies (mean ± SD) per procedure. For the 22 sessions in which laser phototherapy was completed, initial SaO2 on supplemental oxygen prior to procedure was 96.2 ± 5.7 percent. The SaO2 decreased 8.1 ± 6.8 percent to a nadir of 88.1 ± 11.0 percent.

The maximum decrease in oxygen saturation during procedures was inversely correlated with the room air PaO2 measured before the procedure (p = 0.003, Fig 1). There was no signiﬁcant correlation.