and antineutrophil. Cellular immune hyporesponsiveness is present, as evidenced by cutaneous anergy. T-suppressor lymphocytes have been demonstrated in the peripheral blood that are capable of depressing immunoglobulin synthesis and T-cell proliferation. Perhaps these immunosuppressive effects played a role; however, they are present in patients who do not have this complication.

The microbial etiology of the epiglottitis in adults is often unclear. Khilani et al reviewed 158 cases of adult epiglottitis; an infectious etiology was identifiable in only 29. The necrotizing epiglottitis in this case seemed most likely due to anaerobic infection arising from the normal pharyngeal flora. This was supported by the foul halitosis and by the cultures of pharyngeal, epiglottic tissue, and sputum growing only mixed normal oral flora. That anaerobic organisms can cause epiglottitis has been supported by a recent case report. The Epstein-Barr viral pharyngitis may have caused sufficient local tissue damage to lead to anaerobic superinfection.

The pneumonia appeared to be due to aspiration of oropharyngeal contents. Her poor dental hygiene was very likely an additional contributing factor. Although posttinal sepsis occasionally complicates infectious mononucleosis, negative blood cultures in our patient make this diagnosis less likely. Epstein-Barr virus may cause primary pneumonia, but the prompt resolution of the infiltrates with antibiotic therapy suggested a bacterial, rather than viral etiology. Furthermore, with recovery of her voice, she was able to provide a history of aspirating oropharyngeal secretions.

Finally, the dysphagia was likely due to a combination of factors. The oral motor examination showed neuromuscular dysfunction. This may have been due to local tissue edema and damage or to cranial neuropathy. The pharyngeal and glossal edema, as well as the loss of the epiglottis and valleculae, resulted in the disruption of the normal function and lack of function of the pharynx.

In summary, this case illustrates that infectious mononucleosis may be associated with necrotizing epiglottitis, dysphagia and aspiration pneumonia.

ACKNOWLEDGMENTS: We wish to thank Dr. B. Pressnail, Royal Victoria Hospital, Barrie, Ontario, and Dr. W. Weiser, St. Michael’s Hospital Department of Diagnostic Imaging, for performing repeated videofluoroscopy.

REFERENCES

Recurrent of Sleep Apnea Syndrome following Tracheostomy

A Shift from Obstructive to Central Apnea

Eugene C. Fletcher, M.D., F.C.C.P.

This report describes an unusual case of severe obstructive sleep apnea and alveolar hypoventilation leading to hypoxemia and cor pulmonale, which were corrected by tracheostomy. Four years later, after a 22.5-kg weight gain, nocturnal apneas of similar frequency, duration, and depth of desaturation reappeared but were totally central in origin. The central apneas were eliminated with home nocturnal positive-pressure ventilation via cuffed tracheostomy tube. Each time the patient's apneas were corrected (obstructive: tracheostomy; central: mechanical ventilation), daytime alveolar hypoventilation disappeared rapidly. Yearly right heart catheterizations and radionuclide ejection fractions documented pulmonary hypertension and right heart failure, with resolution following tracheostomy and recurrence after appearance of central apneas. The changes in hemodynamic status corresponded to the patient's weight, presence of apnea, daytime alveolar hypoventilation, and treatment of nocturnal oxyhemoglobin desaturation. This case illustrates the theory of a common etiology of both central and obstructive apnea through abnormal respiratory controller gain and points to several roles obesity may play in apnea.

Monographs and book chapters describing sleep apnea typically define obstructive and central apnea as etiologically unrelated events. In reality, it is common for both central and obstructive events to appear together in the same individual during sleep, suggesting a common etiology. Recent studies have addressed possible central respiratory control factors in the etiology of both "central" and "obstructive" apneas. Hypoxia has been shown to play a pivotal role in periodic breathing, whether induced by hypoxic gas mixtures or ascent to altitude. The hypocapnia induced by compensatory hyperventilation in this setting may reduce the drive to breathe (apneic CO₂ threshold), creating central pauses in respiration. The changes in central drive could alter neuromuscular tone of the upper airway, increasing pharyngeal resistance and predisposing to obstructive apnea.

This report describes a patient with severe obesity in whom obstructive apnea and hypoventilation created severe hemodynamic abnormalities corrected by tracheostomy. Four years later, corresponding to a substantial increase in weight and despite a patent tracheostomy, profound apneic desaturation and right heart failure reappeared. The occurrence of obstructive apnea followed by severe central apnea suggests a relationship between apnea etiologies.

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Recurrence of Sleep Apnea following Tracheostomy (Eugene C. Fletcher)

Recurrence of Sleep Apnea following Tracheostomy (Eugene C. Fletcher)

FIGURE 1. Apneic desaturation during NREM and REM sleep, 1984 to 1988. (A) Breathing room air before tracheostomy. Baseline (nonapneic) SaO₂ falls about 7 percent during REM sleep. (B) Following tracheostomy, nearly all apneic desaturations disappear, but longer nonapneic desaturations (up to 15 mins) persist. (C) Supplemental oxygen at 4 L/min through the tracheal stoma reduces REM desaturations. (D) Four years after tracheostomy, recurrent right heart failure and pulmonary hypertension. Apneas of similar duration and density to 1984 apneas. Polysomnography shows them to be central. (E) Nocturnal positive pressure ventilation (FIO₂ = 0.21 percent) through cuffed tracheostomy tube again eliminates apneic desaturation. Slight tendency to desaturate during REM sleep is noted.

CASE REPORT

The patient first presented in February, 1984 with dyspnea, severe daytime hypoxemia, morbid obesity, shortness of breath, and ankle swelling for three years. Onset of symptoms corresponded to a 45-kg weight gain. He had a lifelong history of heavy snoring and smoked one pack of cigarettes per day for 40 years. He exhibited many signs of right heart failure, including elevated jugular venous pulse, "P" pulmonale on lead 2 of the ECG, cardiomegaly on chest roentgenogram, and leg edema. His admission weight was 191.2 kg, which decreased to 153.4 kg after diuresis. Following stabilization (Table 1, February, 1984), gated radionuclide study documented a reduced right ventricular ejection fraction (RVEF), and right heart catheterization confirmed elevation of pulmonary artery pressure (PA). Spirometric study results showed no obvious expiratory airflow obstruction, but this was suggested by a marked elevation of residual volume (199 percent predicted). Polysomnography revealed 500 to 650 obstructive apneas per night (apnea index = 114/hr), with decrement in oxyhemoglobin saturation (SaO₂) to around 90 percent during rapid eye movement (REM) sleep (Fig 1, panel A). There were no central apneas, and fewer than 5 percent of apneas had any mixed component, which was always less than 10 s in duration. Tracheostomy was performed February, 1984. Polysomnography then showed no apneic desaturation, but during REM sleep, longer periods of desaturation previously described in patients with lung disease were observed (Fig 1, panel B). These were ameliorated with 4 L/min supplemental oxygen over the tracheostomy stoma (Fig 1, panel C), and the patient was discharged home with nocturnal supplemental oxygen and diuretics.

During the next two years, the patient remained symptom free, and two subsequent right heart catheterizations and radionuclide studies (March/85, May/86) showed normalization of the Ppa and RVEF (Table 1). Arterial blood gas values improved with resolution of alveolar hypoventilation, but a widened alveolar-arterial oxygen gradient, P(Al−SaO₂) persisted. At the end of the second year after tracheostomy (May, 1986), the patient's weight was noted to have increased (dietary counseling was of no avail). At the end of the third year following tracheostomy, (May/87) the patient complained of a return of daytime hypoxemia and peripheral edema, and he showed evidence of alveolar hypoventilation by blood gas analysis, and his weight had increased (162 + kg, exceeded clinic scale limit). Patency of the tracheostomy tube was ascertained by fiberoptic visualization. The patient was again urged to lose weight. One year later (May, 1988), at a weight of 164.25 kg, the patient's blood gas values had deteriorated. Dependent edema was treated with diuretics. Ppa and RVEF had again deteriorated (Table 1, June/88). Polysomnography (May, 1988) with the tracheostomy open and unobstructed showed apnea desaturation of duration and nadir SaO₂ (apnea index = 50/hr) similar to his original study of February/84 (Fig 1, panel D). However, all apneas were central in that no chest wall or abdominal activity was present (Fig 2). The ventilatory response to hypoxic hypercarbia was virtually flat. Supplemental oxygen did little to ameliorate the apneic fall in SaO₂. His metal tracheostomy was changed to a cuffed plastic tube and he received ventilation with a pressure cycle ventilator at night (FIO₂ = 0.21 percent) to achieve adequate oxygenation (Fig 1, panel E). Following one and three weeks of home nocturnal ventilator use (June/87, June/88), his daytime arterial PaCO₂ had decreased from 58 to 45 to 34 mm Hg, and his PaO₂ had increased from 49 to 59 to 64 mm Hg (confirmed by multiple blood gas analyses). Repeated polysomnography during spontaneous respiration after three weeks of nocturnal ventilator use showed no central apneas, a non-REM SaO₂ of 88 to 90 percent, and nonapneic REM desaturation to 75 percent (similar to Fig 1, B). Following weight loss to 137.7 kg, nocturnal mechanical ventilation was discontinued (after three months), and subsequent nocturnal oximetry during spontaneous respiration showed continued resolution of apneic desaturation. The RVEF and arterial blood gas values likewise improved (September, 1988).

DISCUSSION

The patient initially presented in severe congestive failure with hypoxemia. Following diuresis and stabilization, obvious daytime alveolar hypoventilation and abnormal gas exchange were present (Table 1, Feb/84). After correction of severe obstructive apneas by tracheostomy, correct of residual REM sleep-related hypoxemia by nocturnal supplemental oxygen, and a 13.5-kg weight (dry) loss, the patient's daytime arterial blood gas values appeared typical of morbid obesity, i.e., abnormal gas exchange with probable V/Q mismatch.

This case is interesting from several standpoints. First, over the study period, the patient evolved from hemodynamically compromising obstructive apneas to hemodynamically compromising central apnea. Second, the deterioration in cardiovascular status correlated well with body weight and the presence of daytime alveolar hypoventilation. Third, the daytime hypoventilation was directly related to the presence.
Table 1—Evolution of Weight, Blood Gases, and Hemodynamic Parameters*

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*Parentheses indicates dry weight following stabilization of right heart failure. With the exception of columns 2/84 (acute cor pulmonale) and 5/87, 5/88 (clinically stable but edematous), blood gases were drawn during clinical stable periods, free of edema.
††Parameters after one and three weeks of home nocturnal mechanical ventilation, respectively.

Figure 2. Portion of polysomnographic sleep tracing from May, 1988 showing central apnea. Top to bottom: EEG (C3A2), left EOG, right EOG, chin EMG, ECG, SaO₂, tracheostomy airflow, ribcage and abdominal wall motion (Respitrace). Such periods of absent central respiratory drive were regularly recurrent throughout sleep (Fig 1, D). Despite paradoxic motion of abdomen and chest wall, reflected in appropriate channels, clearly no obstruction was present, since functioning tracheostomy (confirmed by fiberoptic bronchoscopy while awake and thermistor flow during sleep) precluded airflow obstruction. Such paradoxic chest wall/abdominal wall motion is frequently seen in very obese patients and those with respiratory muscle failure.
of nocturnal episodic desaturation and corrected quickly with abolition of the apneas (Feb/84, June/88) before substantial dry weight loss (↓ body mass) occurred. Finally, shortly after institution of nocturnal mechanical ventilation, the central apneas disappeared during spontaneous nocturnal breathing, and remained resolved following return to a weight of 137.7 kg and discontinuation of the ventilation therapy.

This patient's response to tracheostomy and later to correction of central apnea is similar to that of the "corrector" group described by Rapoport and colleagues. In that study, eight severely apneic patients with pickwickian syndrome, matching weights, apnea indices, arterial blood gas values (daytime hypoventilation), and chemosensitivity drove were separable into two groups when half became eucapnic within two weeks of tracheostomy. Analysis of minute ventilation with EEG monitoring showed both groups to be alveolar hypoventilators during what appeared behaviorally to be an awake state but in actuality was a mixed wake/microsleep state. When the awake state was confirmed by ECG, minute ventilation of "correctors" was normal before and after tracheostomy, whereas that of "noncorrectors" was low. In this patient, the common denominator accompanying daytime hypoventilation both times (pretracheostomy and prenocturnal mechanical ventilation) appeared to be nocturnal episodic hypoxemia, since, after correction, daytime hypercarbia reversed before a substantial loss of body mass occurred. The loss of edema fluid would not appear to account for the resolution of apneas or hypoventilation as all blood gas values and oximetry tracings in Table 1 and Figure 1 were performed after acute diuresis and stabilization to near "dry weight." However, maintenance of the "corrector" state following tracheostomy appeared to correlate with a lower "dry" body weight (139.95 kg). On increasing weight to over 162 kg, symptoms of hypersonomolence, signs of right heart failure, and daytime alveolar hypoventilation returned, presumably in part because of the return of central apneas with profound, episodic nocturnal hypoxemia.

Many investigators propose a common factor leading to both obstructive and central nocturnal apneas. Concepts examining the relationship of both types of apnea were recently summarized by Dempsey and Skatrud. Periodic breathing and central apneas are proposed to result from the interaction of hypercarbia and hypoxemia, which increases controller gain. Sleep resets the CO₂ set point to a few mm Hg above the waking level. Hypoxemia during sleep stimulates central chemoreceptors to mild hyperpnea. If the hyperpnea drives the PaCO₂ below this new set point, an "apneic CO₂ threshold" is exceeded, where respiratory controller output stops and central apnea ensues. Subsequent hypoxemia induces oscillations in chemical stimuli, resulting in a hyperpnea-hypopnea (apnea) cycle very similar to Cheyne-Stokes breathing.

In hypoxic patients who snore, a link is seen between central periodic breathing and the propensity to obstruct the upper airway. Sleep-induced relaxation of the airway increases resistance ten times that of wakefulness. As PaCO₂ falls with cyclic oscillation, ventilatory drive to the diaphragm (and probably the pharyngeal constrictors) may fall further, increasing upper airway resistance. Hudgel et al. showed that a portion of patients may be expected to respond to periodic breathing of hypoxemia with increased upper airway resistance and in some cases, by upper airway obstruction. Thus, subsequent inspiratory efforts meet with partial airway collapse (obstructive hypopnea) and lower SaO₂. As long as the PaCO₂ does not fall below the "apneic CO₂ threshold," the drive to take a breath may be reduced but not eliminated. Upper airway muscle tone remains reduced while inspiratory efforts continue and cyclic obstructive hypopneas (or apneas) occur. If the hypoxemia becomes greater (as in our patient), the resulting hyperpnea causes PaCO₂ to go below the apneic CO₂ threshold, resulting in central apneas.

The following scenario is proposed to account for the appearance of severe obstructive apnea and, later, central apnea in this patient. Morbid obesity caused chronic hypoxemia through abnormal gas exchange and, at weights above 157.5 kg, alveolar hypoventilation. The chronic hypercarbia and hypoxemia predisposed to periodic breathing during sleep through increased respiratory controller oscillation. With the history of lifelong snoring, anatomic or functional narrowing of the upper airway set the stage for obstruction as forces collapsing the airway were greater than the central stimulus to maintain patency. With correction of the nocturnal desaturation by tracheostomy, oxygen, and weight loss to 140.0 kg, improved alveolar ventilation decreased the propensity to develop nocturnal periodic breathing. As the patient regained weight (above 157.5 kg), central alveolar hypoventilation returned. Because of the functioning tracheostomy, central apneas became the manifest expression of widened respiratory controller oscillation. Elimination of episodic nocturnal hypoxemia by mechanical ventilation corrected the alveolar hypoventilation, improved nocturnal SaO₂, and eliminated periodicity when restudied during spontaneous breathing. Rapid correction of alveolar hypoventilation following treatment of nocturnal desaturation has been reported.

Data supporting a central mechanism for the development obstructive apnea is growing. Cases such as this one lend support to a common mechanism for all sleep apneas. The ultimate goal will be to find nonsurgical treatment for both types of apneas that will attack the problem at the controller level rather than at the site of obstruction, as do nasal CPAP and surgery.

References
4. Longobardo GS, Gothe B, Goldman MD, Cherienvaik NS. Sleep apnea considered as a control system instability. Respir Physiol 1982; 50:311-33
7. Cherienvaik NS, Longobardo GS. Cherie-Stokes breathing: an
Combined Aortic and Mitral Valve Replacement in an Adult with Scheie’s Disease*

Samuel M. Butman, M.D., F.C.C.P.; Linda Karl, M.D.; and Jack G. Copeland, M.D., F.C.C.P.

Mitral, aortic, and coronary arterial disease have been described in the various mucopolysaccharidoses. We report the first successful combined aortic and mitral valve replacement in an adult female patient with severe aortic and mitral stenosis due to Scheie’s syndrome, a mucopolysaccharide storage disease. Both annulae were of sufficient integrity for good prosthetic placement, and the patient had an uneventful postoperative recovery.

(Chest 1989; 96:209-10)

Scheie’s disease, first described by Harold G. Scheie and co-workers in 1962 as a forme fruste of Hurler’s disease, is a rare cause of valvular disease in adult patients with heart disease. Mitral, aortic, and coronary diseases have been described in the various forms of this inherited disorder, and successful valvular replacement has been infrequently described. We report the first successful combined aortic and mitral valve replacement in an adult with severe aortic and mitral stenosis due to Scheie’s disease.

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FIGURE 1. Aortic valve specimen obtained at surgery. Fusion of commissures and nodularity are notable.

CASE REPORT

A 42-year-old woman with Scheie’s disease, documented by high levels of chondroitin sulfate B in the urine and typical clinical features, was admitted for diagnostic studies after recent admissions for chest pain and congestive heart failure. Physical examination was notable for auscultatory findings compatible with significant aortic and mitral stenosis. A faint murmur of aortic insufficiency was also present. The remainder of the history and physical examination was significant for blindness with bilateral corneal opacities, status postventriculoperitoneal shunt for hydrocephalus, bilateral carpal tunnel syndrome in the past, and flexion deformities of both wrists. She was receiving ketoconazole for bipolaris spicifer chronic meningitis.

The ECG revealed normal sinus rhythm, left atrial enlargement, and poor R-wave progression. Echocardiography confirmed the clinical impression of aortic and mitral stenosis. A cardiac catheterization was performed, which revealed an aortic valve gradient of 100 mm Hg, with a calculated aortic valve area of 0.5 sq cm, and a mitral valve gradient of 16 mm Hg, with a calculated area of 0.9 sq cm. The mean pulmonary capillary wedge pressure was 20 mm Hg. There was no significant valvular regurgitation, and there was no evidence of coronary artery disease. At surgery, the valves were severely diseased and calcified. The aortic valve was severely stenotic and calcified and had irregularities on the edges of the valve, resembling dysplastic nodules (Fig 1). The mitral valvular chordae were thickened, and the valve was foreshortened and stenotic (Fig 2). The aortic valve was replaced with a 20-mm Hall

FIGURE 2. Mitral valve specimen obtained at surgery. Thickening and nodularity are seen.