just proximal to the fistula, normal pulmonary parenchyma is spared. In our patient, surgical removal of a significant number of fistulas was not possible. Percutaneous transcatheter intravascular occlusion of pulmonary arteriovenous fistulas with coil springs was first reported by Taylor et al. in 1978. Subsequently, occlusions of pulmonary arteriovenous malformations were performed with detachable balloons. The ability to place balloons accurately and to assess the adequacy of placement before detachment makes them attractive. We used spring coils, which are easier to use, appropriate for small-vessel arteriovenous malformations, and less expensive. All major malformations in our patient were occluded with minimal loss of normal parenchyma. She had an improved PaO₂ on room air and FIO₂ of 100 percent. Should oxygenation deteriorate or the residual fistulas enlarge, they can be treated by repeat percutaneous embolization.

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Obstructive Sleep Apnea following Bilateral Carotid Body Resection*

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A patient who had undergone bilateral carotid body resection five years earlier for palliation of chronic airflow obstruction was found to have severe obstructive sleep apnea. He presented with hypercapnic respiratory failure, which improved after tracheostomy. A physiologic mechanism is proposed to explain this association. Previously reported studies of anesthetized animals suggest that loss of peripheral chemoreceptor activity could selectively decrease neural output to the genioglossus, the main protractor muscle of the tongue, predisposing the upper airway to inspiratory occlusion.

The dilator muscles of the upper airway have been shown to play a critical role in maintaining patency of the oropharynx during inspiration. Obstructive sleep apnea (OSA) has been shown to occur in man when inspiratory activity of the genioglossus, the main protractor muscle of the tongue, is not adequate to overcome the negative pressure generated by the chest wall muscles. Several investigators have noticed differences in the magnitude and pattern of responses of the genioglossus and diaphragm to hypoxia and hypercapnia, and there is convincing evidence that neural output to the genioglossus is stimulated to a greater degree than drive to the diaphragm by input from the peripheral chemoreceptors, while the diaphragm is preferentially stimulated via the central chemoreceptor. This latter phenomenon may be of critical importance to the control of upper airway resistance in patients who have undergone surgical removal of the carotid bodies, a procedure which has been advocated by some for palliation of obstructive airways disease. We recently encountered such a patient who had clinically severe OSA after having undergone such "therapeutic" carotid body resection.

CASE REPORT

This 61-year-old man had a long history of chronic obstructive pulmonary disease and bronchiectasis which had been treated medically until five years prior to this admission when he underwent bilateral carotid body resection in a California hospital. He was not obese, and there was no history of snoring or hypersomnolence prior to surgery. His condition did not improve, but remained stable until several months prior to his admission to another hospital with dyspnea and lethargy. On physical examination, he was thin, with decreased breath sounds and coarse crackles, and with mild peripheral edema. Initial arterial blood gas levels showed a pH of 7.30, PaCO₂ of 94 mm Hg, and PaO₂ of 35 mm Hg. He was intubated and mechanically ventilated. After extubation, he remained hypercapnic and dyspneic. He was also quite somnolent, with witnessed snoring.

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OSA after Carotid Body Resection (Parisi et al)
and frequent apneas during sleep. He was transferred to Middlesex General University Hospital for evaluation of his sleep-disordered breathing.

Upon transfer, his arterial blood gases while awake receiving oxygen via nasal cannula at 2 L/min showed a pH of 7.32, Pco2 of 57 mm Hg, and Paco2 of 58 mm Hg. Pulmonary function tests demonstrated airflow obstruction (FEV1/FVC = 31 percent) with a normal total lung capacity (6.04 L, 82 percent of predicted). Ventilatory response to hypoxia was determined while awake and was found to be absent, confirming the lack of peripheral chemosensitivity. A nocturnal sleep study was performed with the patient receiving supplemental oxygen. A total of 2 hours, 46 minutes of sleep were observed, all of which were spent in stages 1 and 2 non-REM sleep, punctuated by frequent arousals. His respiratory pattern during sleep showed episodes of periodic breathing with cyclic hypopneas. A total of 21 apneas were observed, all obstructive in type, which ranged in duration from 10-135 seconds (mean = 40 s). Arterial oxygen saturation, which averaged 86 percent in the awake state, fell to a mean of 60 percent and as low as 26 percent following apneic episodes. A representative sleep record is shown in Figure 1. It is noteworthy that bradycardia did not occur even during prolonged apneas with severe hypoxia, providing further evidence of absent hypoxic chemosensitivity.4

A tracheostomy was performed. His condition improved markedly after the procedure, and his daytime sleepiness resolved. Arterial blood gas levels while awake breathing 40 percent oxygen showed a pH of 7.46, Pco2 of 41 mm Hg, and Paco2 of 61 mm Hg. A sleep study performed 11 days after the tracheostomy showed a significant improvement in sleep profile. A representative portion of the record is shown in Figure 2. Breathing remained regular, and no apnea or hypopnea was noted.

**DISCUSSION**

Unilateral carotid body resection for the treatment of asthma was reported in 1961 separately by Nakayama7 and Overholt.8 Bilateral resection was later advocated by Winter.9

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**Figure 1.** Representative recording from the initial sleep study. A 90-second obstructive apnea is shown, during which arterial oxygen saturation decreased to a nadir of 46 percent. From top to bottom, the tracings represent arterial oxygen saturation, airflow detected by a capnograph, thoracic and abdominal volume signals from the respiratory inductive plethysmograph, electrocardiogram (EKG), chin electromyogram (EMG), electroencephalogram (EEG), and electrooculogram (EOG).
who also advocated this procedure for patients with chronic obstructive pulmonary disease. Despite serious doubts about the efficacy of this procedure and lack of general acceptance, serious consequences have been reported to be rare when baroreceptor afferents were left intact.

To the best of our knowledge, this is the first report of OSA in a patient following carotid body resection. Eichenhorn and co-workers reported a young woman with impaired peripheral chemoreception initially suspected to have sleep apnea, but later noted to become apneic only while awake due to voluntary breathing. It may be that the association in our patient was in part due to the presence of markedly impaired pulmonary function with deterioration of gas exchange during sleep. It has been established that patients with respiratory insufficiency while awake are at risk for further hypoventilation during sleep. When chemoreceptor function is intact, sleep hypoventilation manifested as simultaneous hypoxia and hypercapnia would be expected to result in balanced stimulation of the genioglossus and diaphragm. However, the absence of hypoxic chemosensitivity in this case may have produced disproportionate stimulation of the diaphragm via the central chemoreceptor sufficient to overcome the upper airway dilators and result in obstructive apnea.

The significance of this report is two-fold. First, it appears to support the finding from studies in anesthetized animals which indicates preferential stimulation of hypoglossal motorneurons by activation of the carotid bodies in contrast to the relatively greater increase in phrenic nerve output following central chemoreceptor stimulation. Second, it raises the possibility that carotid body removal may predispose to obstructive sleep apnea, especially in patients who are apt to hypoventilate during sleep.

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Pulmonary Artery Obstruction due to Giant Cell Arteritis*

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Giant cell arteritis is often referred to in the context of polymyalgia rheumatica with temporal artery involvement. There are, however, more malignant forms of presentation of this necrotizing arteritis involving either the great vessels of the aorta or, occasionally, the pulmonary arteries. Our case relates to giant cell arteritis presenting as pulmonary artery obstruction in a patient without polymyalgia rheumatica or extensive aortic or proximal great vessel involvement.

While the association of giant cell arteritis (GCA) with polymyalgia rheumatica is well described, more unusual, life-threatening presentation of this idiopathic arteritis include acute myocardial infarction, aortic arch vessel involvement, stroke syndromes, aortic aneurysm and dissection and even aortic insufficiency. We report a case of giant cell arteritis presenting as pulmonary artery obstruction in an elderly woman without extensive great vessel arteritis.

CASE REPORT

A 77-year-old caucasian woman was admitted for evaluation of progressive dyspnea, fatigue, weakness and right-sided chest pain without fever over approximately six months, with no history of chronic headaches or muscle pain to suggest polymyalgia rheumatica. Physical examination disclosed an elderly woman in no acute distress, blood pressure 90/60 mm Hg, pulse 110 bpm and regular, mild jugular vein distention to 12 ml water, a two-over-six systolic ejection murmur at the lower left sternal border with a normal second sound and no peripheral edema.

Laboratory examination results were normal with the exception of mild anemia, a hemocrit of 36, and a Westergren sedimentation rate of 46 mm/hr. Electrocardiographic examination showed right bundle branch block and a vertical QRS axis. Ventilation lung scan showed a posterior segment of right upper lobe defect and a questionable right lower lobe wedge-shaped defect consistent with pulmonary embolism.

Thallium-201 examination showed right ventricular enlargement with a right ventricular volume overload pattern and three-plus tricuspid regurgitation by Doppler study. Left ventricular ejection fraction was calculated in the 70 to 80 percent range. The pulmonary artery was not well visualized and the aortic, mitral, and tricuspid valves were normal. There was a small pericardial effusion.

Because of worsening symptoms, the patient underwent cardiac catheterization which showed a right atrial pressure a wave of 15 mm Hg, mean pressure of 10, RV equal to 74/17, pulmonary artery pressure of 107 with a pulmonary capillary wedge pressure of 6, cardiac output of 1.8 L/min with a cardiac index of 1.1 L/min. There was a 64 ml gradient between the pulmonary artery and RV just above the pulmonary valve. Pulmonary artery saturation was 43.8 percent with an aortic saturation of 88.5 percent. Left heart pressure was normal, as was coronary arteriography. Pulmonary arteriogram disclosed marked narrowing of both the right and left pulmonary arteries with what appeared to be extrinsic compression of the main pulmonary artery and both of the proximal pulmonary artery segments. CT scan of the chest was essentially unremarkable with

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