is confident that an invasive procedure such as tracheostomy is indicated. Documenting just the obstructions may not help decide on therapy, and probably offers little advantage over simply watching the patient and listening to the patient while he or she is sleeping.

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To the Editor:

In the absence of complete nasal obstruction, a single thermistor threaded through nasal prongs and properly positioned in the anterior nares has proved adequate to document airflow during sleep in over 30 patients, as well as in many previous studies of airflow in our laboratory.

We are in complete agreement with Kryger that one does not perform tracheostomy without a clear understanding of the clinical significance of that apnea. On the other hand, surgery for sleep-related apnea is inappropriate without objective monitoring of the type and duration of apneic episodes. Sleep monitoring along with history, physical examination, and laboratory assessment, are all essential in arriving at a valid therapeutic decision.

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Tidal Volumes and Volume Ventilators

To the Editor:

In response to Graybar's communication entitled "The Most Advanced Respiratory Life Support System Available," the differences in set and delivered values for tidal volume (TV) need further clarification. We do not disagree that the values of TV delivered by the particular ventilator (Puritan-Bennett MA-1) were exactly as reported by Graybar; however, all volume ventilators are designed to produce a set TV. The difference between the set and delivered values for TV is then influenced by the compliance factor of the tubing used. The operating instructions for the particular ventilator (Puritan-Bennett MA-1) state: "To read patient tidal volume, correction must be made to the spirometer and 4 to 5 ml/cm H2O with disposable tubing. Our ventilators' flow sheets indicate the following: set TV = observed TV — (4 ml x observed pressure — PEEP), where PEEP is positive end-expiratory pressure and where pressures are in centimeters of water. The volumes recorded by the Dixie Test Lung (Michigan Instruments TTL) reflect the true TV.

Lamont A. Reddington, R.R.T., Director, and Robert E. Hillberg, M.D., F.C.C.P., Medical Director, Respiratory Services, California Hospital Medical Center, Los Angeles

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The Diagnosis of Aortic Stenosis in a Patient with an Artificial Demand Pacemaker

To the Editor:

We report the effect of an artificial transvenous demand pacemaker and atrial contraction on the intensity of the murmur in valvular aortic stenosis.

Case Report

An 84-year-old white man was admitted to the hospital with a history of electrocardiographic and physical findings that were consistent with significant valvular aortic stenosis. The patient also had previously received a permanent artificial transvenous demand pacemaker.

The patient had a grade-4/6 harsh systolic ejection murmur heard best over the second right intercostal space and the apex. On further examination, we noted that the intensity of the murmur abruptly decreased from grade 4/6 to 1/6 for 10 to 15 seconds and then abruptly increased to its previous intensity. This periodic waxing and waning continued in a regular cyclic manner and was unrelated to respiration or the position of the patient.

By physical examination, the heart rate of approximately 72 beats per minute was regular and unchanging; however, the rhythm strip of the electrocardiogram revealed intermittent artificial pacemaker-induced rhythm alternating with sinus rhythm. Phonocardiograms revealed that the intensity of the systolic ejection murmur correlated with the changes in the cardiac rhythm. The murmur's intensity increased during sinus rhythm and diminished markedly during artificial demand pacemaker-induced rhythm; however, the contour of the murmur remained diamond-shaped.

Hemodynamic studies showed that during sinus rhythm the left ventricular systolic and left ventricular end-diastolic pressures were 220 mm Hg and 22 mm Hg, respectively. The aortic pressure was 165 mm Hg, resulting in a systolic pressure gradient of 55 mm Hg. During artificial demand pacemaker-induced rhythm, corresponding values for left ventricular systolic, left ventricular end-diastolic, and aortic pressures were 204 mm Hg, 5 mm Hg, and 145 mm Hg, respectively, with a systolic pressure gradient of 59 mm Hg. No significant change occurred in the systolic pressure gradient between sinus rhythm and artificial demand pacemaker-induced rhythm (Fig 1).

Discussion

Normally the atria contribute between 15 and 30 percent to ventricular preload and aortic blood flow; however, in patients with valvular aortic stenosis, the atrial contribution to ventricular preload is of much greater significance than in patients with normal hearts. Abruptly eliminating the atrial function as a "booster pump" in patients with valvular aortic stenosis can result in abrupt clinical deterioration.

In our patient, since the peak systolic pressure gradient remained unchanged during normal sinus rhythm and pacemaker-induced rhythm, the markedly increased intensity of the systolic ejection murmur during normal sinus rhythm seems to be due to increased flow across the aortic valve.

In a similar patient, Harris et al showed that during continuous right ventricular pacing in a patient with valvular aortic stenosis, the systolic ejection murmur increased in...
intensity when atrial contraction contributed to left ventricular filling. Harris et al attributed this finding to augmentation of left ventricular pressure due to the atrial contraction but did not implicate flow across the valve or the systolic pressure gradient in causing the increased intensity of the murmur.

In a patient with valvular aortic stenosis and atrioventricular dissociation (but no artificial demand pacemaker), Braunwald and Frahm showed that as the P waves lost their normal temporal relationship with the QRS complexes, the murmur of valvular aortic stenosis decreased; however, in contrast to our patient, the peak pressure gradient decreased from 91 to 74 mm Hg.

CONCLUSION

In a patient with an artificial demand pacemaker and unexplained symptoms of syncope or angina pectoris who has a seemingly soft "innocent" or "functional" systolic ejection murmur, the important physical signs of significant valvular aortic stenosis, such as the time of onset and shape of the systolic murmur and any delay in the carotid upstroke, must be searched for diligently. The artificial demand pacemaker conceivably could be responsible for life-threatening delays in the diagnosis and treatment of significant valvular aortic stenosis by disguising the loud harsh murmur of valvular aortic stenosis as an innocent "flow murmur."

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