suspicion of a traumatic pulmonary AV fistula. A contrast-enhanced TTE using agitated saline solution is a quick, inexpensive, and simple test to perform. Systemic air embolism is a potential risk with this technique. This may be minimized by the avoidance of multiple injections.

Conclusions

To the best of our knowledge, this is the first report of the bedside use of contrast-enhanced TTE in confirming the diagnosis of a pulmonary AV fistula secondary to a gunshot wound to the chest. Despite its rarity, traumatic pulmonary AV fistula should be considered while evaluating unexplained hypoxemia in patients with penetrating chest injuries. The evaluation of these patients should include bedside contrast-enhanced echocardiography using agitated saline solution.

References


Identification and Treatment of Bronchoconstriction Induced by a Vagus Nerve Stimulator Employed for Management of Seizure Disorder*

Jagdeep S. Bijwadia, MD, FCCP; Robert C. Hoch, MD, FCCP; and Donn D. Dexter, MD

We evaluated a 63-year-old woman who developed dyspnea with a sensation of chest tightness that was temporally associated with discharges from a vagus nerve stimulator that had been implanted for the control of intractable seizures. Spirometry demonstrated the development of significant airflow obstruction associated with the firing of the stimulator. Adjustment of the stimulator settings resolved the discharge-associated bronchoconstrictive phenomenon. These findings highlight an important association between vagus nerve stimulators and dyspnea that should be considered in the differential diagnosis of patients with these devices who present with dyspnea and/or chest tightness. The relative importance of vagal stimulation to bronchoconstriction is suggested by the findings.

(CHEST 2005; 127:401–402)

Key words: bronchoconstriction; dyspnea; seizure; vagus nerve stimulator

Vagus nerve stimulators are relatively new devices that are finding increased use in the nonpharmacologic management of seizure disorders. They are implanted in the subcutaneous tissues of the chest, with a wire lead inserted into the vagus nerve to deliver regularly timed cycles of electrical pulses that suppress epileptogenic foci.¹ We report a case in which a vagus nerve stimulator had the unintended effect of inducing dyspnea that was associated with the activation of the device, which was found to temporally correlate with bronchoconstriction as demonstrated by serial pulmonary function assessments. Nerve stimulator setting adjustments were found to alleviate the bronchoconstriction and associated dyspnea.

Case Presentation

A 63-year-old, right-handed white woman with a 20-year history of psychomotor seizures with intractable features, despite management with levetiracetam and carbamazepine, underwent implantation of a vagus nerve stimulator (NeuroCybernetic Pros-
Discussion

We believe this to be the first report of dyspnea attributable to airflow obstruction that is temporally associated with a discharge of a vagus nerve stimulator. Reports of the unintended consequences of vagus nerve stimulators have included descriptions of voice change secondary to stimulation of the laryngeal nerve, and dyspnea has been reported in up to 25.3% of patients with higher (ie, 30 Hz) vagus nerve stimulation compared with 10.7% of patients receiving lower (ie, 1 Hz) stimulation, although previously this has not been linked specifically to bronchoconstriction and airflow obstruction. Our findings suggest an important role for the vagus nerve in determining airway constriction independent of other factors. Prior studies have suggested the selective importance of vagus stimulation in the parasympathetic regulation of airway tone in humans. It is noteworthy that pretreatment with inhaled ipratropium bromide did not attenuate the fall in airflow with nerve stimulator discharge in our patient, suggesting that factors other than acetylcholine may be mediating the bronchoconstriction seen with vagus nerve stimulation. The identification of such factors may provide important insights into new pathways for the treatment of vagus nerve-mediated bronchoconstriction. Although further studies are needed to define these new pathways, an intriguing report has indicated that vagally mediated nonadrenergic, noncholinergic excitatory innervation plays a role in allergen-induced bronchoconstriction. Neurokinin-2 receptors were involved in this bronchoconstrictive process, which was blocked by specific inhibitors.

Conclusions

Our findings confirm that vagus nerve stimulator-associated dyspnea may be corrected by reprogramming the settings of the stimulator, which in the case of our patient resolved the bronchoconstriction. As vagus nerve stimulators become more commonplace in the management of seizure disorders, an awareness of the association between their use and the presence of dyspnea with bronchoconstriction, as well as the potentially simple correction of this problem with stimulator reprogramming is needed. The unintended, but relatively pure, demonstration of significant airflow obstruction induced by vagal stimulation provides evidence of an important contribution of this nerve to airway tone.

References

3 Schacter S. Vagus nerve stimulation therapy summary: five years after FDA approval. Neurology 2002; 59(suppl):S15–S20

Table 1—Spirometry Values Before and During Vagus Nerve Stimulator Discharge*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Baseline</th>
<th>During Stimulator</th>
<th>Change, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>FEV&lt;sub&gt;1&lt;/sub&gt;, L</td>
<td>1.87</td>
<td>1.52</td>
<td>-19</td>
</tr>
<tr>
<td>FVC, L</td>
<td>2.40</td>
<td>2.09</td>
<td>-13</td>
</tr>
<tr>
<td>FEF&lt;sub&gt;25–75&lt;/sub&gt;/FVC, %</td>
<td>78</td>
<td>73</td>
<td></td>
</tr>
<tr>
<td>FEF&lt;sub&gt;25–75&lt;/sub&gt;, L/s</td>
<td>1.67</td>
<td>1.24</td>
<td>-26</td>
</tr>
</tbody>
</table>

*FEF<sub>25–75</sub> = forced expiratory flow measured during expiration of 25 to 75% of vital capacity.