sis, distal more than proximal, with sensory loss which suggested the clinical syndromic diagnosis of motor sensory neuropathy. Dispersion of motor unit potentials along with the increase of amplitude in both the proximal and distal muscles of the upper limbs suggested neurogenic muscle involvement possibly caused by a peripheral nerve demyelinating process. The majority of the upper and lower limb nerves could not generate motor action potentials while the right ulnar motor nerve had decreased nerve conduction velocity of more than 40 percent of the normal control value. There was also delay in the terminal motor latency. These findings of nerve conduction studies favor a demyelinating insult rather than axonal degeneration. Acute massive exposure to arsenic poisoning mostly results in axonopathy; however, subacute arsenic poisoning can cause segmental demyelination or complete blocking of the conduction as seen in this patient. Clinical and electrophysiologic presentation largely depends upon the duration of the insult. In subacute or prolonged cases, the nerve conduction studies may reveal a picture like LGB syndrome. Generalized loss of compound muscle action potential or of sensory nerve action potential in the distal muscles of the upper and lower limbs could have suggested an axonal degeneration. However, as this loss is associated with significant dispersion of motor unit potentials, increased amplitude of motor unit potentials, delay in terminal motor latency, and decreased nerve conduction velocities, it suggests a demyelinating process in our patient. Arsenic was found in abnormally high levels in both nails and urine. In many patients, the source of arsenic remains a mystery; however, a new batch of opium detected to have high arsenic content was the source of poisoning in this case. It is the most commonly recognized heavy metal poisoning neuropathy in the Indian subcontinent. It is usually subacute prolonged poisoning which affects the peripheral nervous system along with characteristic skin changes such as hyperkeratosis of palms and soles, hyperpigmentation or raindrop- or teardrop-shaped depigmentation over the skin of the trunk, and Meer's lines over the nails. Arsenic poisoning in the West is mostly accidental or homicidal; however, in the East, arsenic is still used in pesticides, therapeutic powders, and pills by indigenous medical practitioners. In the Indian subcontinent, it is also used in tincture of ginger for potentiating the action of alcohol or opium, which resulted in arsenic poisoning in our case. Arsenic neuropathy usually appears within one to two weeks following ingestion.

In this patient, respiratory discomfort, paradoxical movements of the abdominal wall during inspiration and of the diaphragm in fluoroscopic examination demonstrated diaphragmatic palsy, while prolongation of phrenic nerve conduction time suggested demyelinating insult of the nerve. There were no antecedent events or predisposing factors other than arsenic poisoning which could be identified as the underlying cause of phrenic nerve involvement. The phrenic nerve is predominantly a motor nerve and originates mainly from the fourth cervical segment of the spinal cord. It is augmented by the fibers from the third and fifth cervical motor nerve roots. Bilateral phrenic nerve involvement in association with the motor sensory neuropathy probably reflects a more diffuse process caused by arsenic poisoning. This is further suggested by clinical and roentgenographic improvement which followed d-penicillamine therapy. Diaphragm palsy accompanying brachial neuritis, root avulsion, injury, and leprosy is, most of the time, unilateral. In severe arsenic neuropathy, one must be aware of such a complication for timely respiratory support. Phrenic nerve conduction helps in confirmation or detection of subclinical involvement.

REFERENCES

The Thoracic Vent*
Clinical Experience with a New Device for Treating Simple Pneumothorax

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We report recent experience with a new device, the thoracic vent, in the management of simple pneumothorax. There were 16 patients aged 19 to 73 years who suffered pneumothorax due to spontaneous (4), traumatic (3), or iatrogenic (9) causes. Ease of insertion, patient tolerance, and the presence of a unique signal diaphragm all contributed to patient and physician acceptance of the device. Average

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time to pneumothorax resolution was 2.5 days, and time to thoracic vent removal averaged 3.2 days. There were no immediate recurrences or significant complications. We conclude that the thoracic vent is an effective device for initial and definitive therapy of simple pneumothorax.

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Pneumothorax is a commonly encountered clinical problem which normally necessitates therapeutic intervention. Etiologies include spontaneous rupture of blebs or bullae,iatrogenic injury, and blunt or penetrating trauma. Historically, the incidence of simple pneumothorax has been approximately 15 to 20 per 100,000 population yearly. This has likely increased in recent years due to the rising frequency of Pneumocystis pneumonia, adult survival with cystic fibrosis, increasing use of positive pressure ventilation for the treatment of critically ill patients, and an expanding number of central venous access procedures potentially resulting in iatrogenic pneumothoraces.

Standard therapy for management of pneumothorax consists of insertion of an intercostal drainage tube connected to an underwater seal device with or without negative pressure applied to the tube. While usually successful, this treatment necessitates hospitalization, inhibits patient mobility by virtue of the cumbersome water seal/collection device, and entails moderate patient discomfort. Alternative techniques for the management of simple pneumothorax include aspiration and placement of a small-bore catheter connected to a flutter-valve to prevent air entry into the pleural space. While these latter techniques have not achieved widespread acceptance, their objectives are laudable: (1) effective treatment of simple pneumothorax; (2) ease of use; (3) high patient acceptance due to smaller indwelling tubes and no need for connection to peripheral devices; and (4) potential for outpatient therapy of selected patients.

A new device for treatment of simple pneumothorax, the thoracic vent, has been designed to satisfy these objectives. We herein report the initial clinical experience with this device and make recommendations regarding the potential scope of its use.

METHODS

Patients who experienced simple pneumothorax between October 1989, and December 1990, were considered candidates for treatment with the thoracic vent, irrespective of etiology of the pneumothorax. Patients were excluded if any of the following existed: hydropneumothorax or hemothorax; tension pneumothorax; need for positive pressure ventilation; or complete pneumothorax. Hospital records were reviewed, and the following data were recorded: patient sex and age; size and etiology of pneumothorax; complications from use of the thoracic vent; duration of pneumothorax; and duration of thoracic vent use.

The thoracic vent consists of a flexible 13 French urethane catheter, with removable in-line trocar, connected to a one-way valve (Fig 1). A unique signal diaphragm reflects pressure changes in the pleural space and indicates initial entry of the trocar into the pleural space during insertion. A valved aspiration cannula is provided to allow immediate evacuation of air with a 60 ml syringe, and tubing is also included for possible connection of the device to a suction apparatus or water seal system.

All thoracic vents were inserted under local anesthetic in the second intercostal space in the midclavicular line. Insertions were performed at the bedside by the resident staff after instruction in the use of the thoracic vent. Daily chest roentgenograms were obtained, and the thoracic vents were removed 24 h following complete reexpansion of the affected lung and stabilization of the signal diaphragm in a depressed state, indicating maintenance of negative intrapleural pressure.

RESULTS

During the period of review, 16 patients underwent placement of a thoracic vent for management of simple pneumothorax (Table 1). There were eight men and eight women with a mean age of 43.6 years (range 19 to 73 years). Etiologies included spontaneous pneumothorax in four patients,iatrogenic pneumothorax in nine patients, and traumatic pneumothorax in three patients. Associated conditions consisted of terminal lung cancer in one patient, cystic fibrosis complicated by pneumonia in one patient, and rib fractures in three patients.

Table 1—Characteristics of Patients Treated for Pneumothorax

<table>
<thead>
<tr>
<th>Patient/ Sex/Age</th>
<th>Size of Pneumothorax, cm</th>
<th>Etiology of Pneumothorax*</th>
<th>Days to Vent Removal</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/F /19</td>
<td>4</td>
<td>S</td>
<td>3</td>
</tr>
<tr>
<td>2/M/63</td>
<td>4</td>
<td>I</td>
<td>2</td>
</tr>
<tr>
<td>3/F/53</td>
<td>2.5</td>
<td>T</td>
<td>2</td>
</tr>
<tr>
<td>4/F/29</td>
<td>2</td>
<td>T</td>
<td>2</td>
</tr>
<tr>
<td>5/F/50</td>
<td>2</td>
<td>I</td>
<td>2</td>
</tr>
<tr>
<td>6/M/52</td>
<td>1.5</td>
<td>I</td>
<td>1</td>
</tr>
<tr>
<td>7/M/22</td>
<td>4</td>
<td>T</td>
<td>2</td>
</tr>
<tr>
<td>8/M/73</td>
<td>2</td>
<td>S</td>
<td>5</td>
</tr>
<tr>
<td>9/M/21</td>
<td>5</td>
<td>S</td>
<td>4</td>
</tr>
<tr>
<td>10/M/43</td>
<td>4</td>
<td>I</td>
<td>3</td>
</tr>
<tr>
<td>11/F/50</td>
<td>3</td>
<td>I</td>
<td>3</td>
</tr>
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<tr>
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<tr>
<td>16/M/47</td>
<td>2</td>
<td>S</td>
<td>1</td>
</tr>
</tbody>
</table>

*S, spontaneous; I, iatrogenic; T, traumatic.

Figure 1. The thoracic vent illustrating the urethane catheter, plastic chamber containing the one-way valve, and adhesive panels.
Size of pneumothorax at initiation of therapy averaged 3.0 ± 0.3 (SEM) cm from the visceral pleura to the apex of the pleural space (range 1.5 to 5 cm). Time to complete lung reexpansion was 2.5 ± 0.5 days (range one to six days), and time from insertion to removal of the thoracic vent was 3.2 ± 0.4 days (range one to seven days). The thoracic vents were very well tolerated, permitting patients to ambulate in the hospital corridors, and were associated with minimal discomfort both during insertion and thereafter. One patient who had terminal cancer experienced therapeutic failure of pneumothorax management following initial chest tube insertion. A thoracic vent was placed, permitting the patient to be discharged without fear of lung collapse, and he died at home five days later.

There were no recurrences of pneumothorax during one month of follow-up. Three complications occurred in this small series of patients. Two patients were disoriented and removed their thoracic vents within 24 h of insertion. Their pneumothoraces had resolved and did not recur. Another patient was thrombocytopenic and developed a small hematoma at the thoracic vent insertion site which required no further therapy.

**Discussion**

Standard therapy for simple pneumothorax, insertion of a chest tube connected to an underwater seal device, is highly effective in achieving initial resolution of the thoracic space. The incidence of recurrence is low except for spontaneous pneumothoraces, which have published recurrence rates of 40 to 50 percent.9,10 Despite this largely successful initial therapy, new techniques are being sought for management of simple pneumothorax due to current therapeutic requirements for hospitalization and the need for cumbersome devices.

New methods for treating simple pneumothorax consist primarily of insertion of small-bore catheters into the pleural space to permit air evacuation, connecting the catheters to small one-way valves to prevent reentry of air into the pleural space.1 Although normally successful, these devices have not achieved widespread use for several reasons, including concerns regarding the effectiveness of the one-way valve and reports of catheter kinking.8

In this article, we report experience with a new device, the thoracic vent, for managing simple pneumothorax. The thoracic vent was designed to overcome shortcomings reported in other small-bore catheter systems. The thoracic vent catheter is made of urethane and is very resistant to kinking, particularly at body temperature. The self-contained one-way valve was designed to maximize air exit and eliminate the possibility of its reentry. The signal diaphragm is a unique feature which reflects alterations in intrapleural pressure. It allows safe and certain catheter placement in the pleural space and indicates when stable negative intrapleural pressure has been achieved, permitting removal of the vent. Should application of negative pressure be necessary, tubing is supplied for connection to such a device.

We found the thoracic vent to be very effective for treatment of simple pneumothorax. There was no instance of failure in our patients, and the device was tolerated very well. While long-term follow-up is not available, it is unlikely that recurrence rates for spontaneous pneumothorax in our patients will differ significantly from rates reported for patients treated by chest tube alone. Given these results, we feel that the thoracic vent is suitable for initial definitive therapy for most simple pneumothoraces.

Our favorable initial experience with the thoracic vent must be tempered by a note of caution. This device may not be appropriate for use in patients who are expected to have large volume or protracted air leaks, especially those who are on positive pressure ventilation. Further information is necessary before the relative safety of the use of the thoracic vent in such circumstances can be determined.

One of the goals of pneumothorax therapy is to select patients who are candidates for safe and effective outpatient therapy. The thoracic vent, being self contained, is ideally suited for such treatment, and its signal diaphragm provides a potential means for patients to monitor the progress of therapy themselves. Our experience with this technique is limited, but successful. Further information is necessary to better define which patients are candidates for such outpatient therapy.

**References**