(18 mm Hg) produced the bulging septum and the abnormal echocardiographic findings.

Because of the improved ability to visualize the inter-atrial septum, the 2-dimensional echocardiographic study was the significant factor in excluding every other diagnosis. An aneurysmal bulge of the interatrial septum into the left atrium in a structurally normal heart in a newborn infant should suggest a high-flow lesion such as an arteriovenous malformation.

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Pacemaker System Failure Secondary to Air Entrapment within the Pulse Generator Pocket*

A Complication of Subclavian Venipuncture for Lead Placement

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Air entrapment within the pulse generator pocket may produce pacemaker system malfunction if the anodal contact plate becomes insulated by the accumulation of air. Unipolar pulse generators are predisposed to this complication. We describe a pacemaker-dependent patient who, early after implantation, experienced pacemaker system failure as a complication of subclavian venipuncture. This patient had an unsuspected pneumothorax that progressed to subcutaneous air entrapment within the pulse generator pocket. Management of this previously unreported complication of subclavian venipuncture is rapid, noninvasive and effective. With the growing use of subclavian venipuncture technique for lead placement one should avoid the predisposing factors that can lead to subcutaneous air entrapment.

The insertion of permanent endocardial pacing lead electrode systems utilizing a modification of the Seldinger technique for subclavian venipuncture with a peel-away introducer has been described by littleford et al1-2 and others.3-5 Since March 1979, this has been our technique of choice for transvenous lead implantation, because of the low incidence of complications while providing less surgical trauma, decreased operative time, and a reliable vein site for more than 95 percent of all patients receiving single or dual-chamber pacing systems. However, various potential complications of the subclavian venipuncture technique must be recognized, treated, and avoided.6,7 For this reason, "air entrapment," a previously unreported complication of the subclavian venipuncture technique, is described. Air entrapment secondary to other factors (failure to evacuate air from the generator pocket at the time of closure, and pocket infection with gas-producing bacteria) has been previously reported as an unusual cause of pacemaker system malfunction.1,8

Pacemaker system failure due to air entrapment is limited to unipolar pacing modes and occurs when subcutaneous air is entrapped within the pulse generator pocket, resulting in insulation of the unipolar anodal plate (indifferent electrode) from the subcutaneous tissue.

CASE REPORT

On November 7, 1979, a 77-year-old pacemaker-dependent man with symptomatic complete heart block had his second pacemaker operation within a two-year period. His current pacing system consisted of a nine-year-old bipolar

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lead that had been converted to a unipolar system two years earlier because of a single conductor wire fracture and a two-year-old normally functioning lithium-powered unipolar pulse generator (Medtronic 5973) that had been fitted with a Silastic boot to prevent intractable pectoral muscle stimulation. Reoperation and pacemaker system revision were undertaken because of asymptomatic intermittent failure to capture and discomfort due to inferolateral migration of the pulse generator.

At operation, the proximal lead connector pins were isolated, loss of unipolar and bipolar conductor wire integrity was confirmed, a new unipolar lead was positioned in the apex of the right ventricle using the subclavian venipuncture entry technique, and the same unipolar pulse generator was reimplanted. To accomplish these goals, an inferior transverse incision was necessary to gain access to the migrating pulse generator while a second transverse incision was made 3 cm below the right clavicle for subclavian vein entry and fashioning of a new subcutaneous pocket. The proximal portion of the fractured electrode system was removed; however, fixation within the central venous channel prevented complete removal of the distal portion of the lead. A new pulse generator pocket was fashioned medially between the pectoralis muscle and the thin subcutaneous tissue. The Silastic boot was retained. The old pocket was obliterated and both wounds were irrigated and closed. The pulse generator rested between the two compression dressings covering the two wounds.

Five hours after implantation, while being monitored in an intensive care setting, the patient had an episode of paroxysmal coughing, followed by loss of ventricular capture and a short asystolic period. Hemodynamics stabilized with the emergence of an idioventricular escape rhythm of 37 beats/min. Physical examination revealed subcutaneous emphysema over the right thorax and neck. A discrete air bubble was palpable over the pulse generator. Compression over the generator restored pacemaker function. Normal pacemaker function was permanently restored by removal of the inferior and superior compression dressings and placement of a pressure bandage directly over the generator. A chest roentgenogram confirmed a stable electrode position, a small right pneumothorax, and resolving subcutaneous emphysema (Fig 1). During the next two days, a compression dressing was maintained over the generator and the pneumothorax resolved without the need for a chest tube. The patient was dismissed six days after the procedure and has maintained uncomplicated normal pacemaker function during a 12-month follow-up period.

**DISCUSSION**

Pacemaker malfunction shortly after implantation has, until recently, occurred in 5 to 21 percent of patients in most previously reported series and has been as high as 36 percent in some series. The recent improvements in lead fixation characteristics combined with the multi-parameter programmable pulse generators has reduced lead-related failures to less than 5 percent. Accompanying this reduction in lead-related complications has been an increase in unusual pacemaker system failures. Most of these failures relate to technical errors such as use of faulty set screws, incompatible lead pulse-generator connections, inadvertent surgical disruption of the lead insulation, sensing failures due to “cross talk” of retained endocardial leads, and noninvasive programming errors at implantation. Added to this list of unusual causes of early pacemaker system failure is air en-

![Figure 1. Portable anteroposterior chest film taken shortly after pacemaker malfunction. Note the presence of a unipolar pacing electrode with tip in the right ventricular apex and connected to pulse generator. The old bipolar electrode has been amputated proximally, and the tip remains in the right ventricle. There is a fracture of one lead electrode near the apex of the right lung. White arrows point to right hydro pneumothorax. Dark arrows outline extensive subcutaneous emphysema of right chest wall.](http://journal.publications.chestnet.org/pdffileaccess.ashx?url=/data/journals/chest/21321/ on 06/21/2017)
the indifferent electrode within a Dacron pouch has been effective in reducing troublesome pectoral muscle stimulation in some patients.

The frequency of pneumothorax complicating the subclavian venipuncture technique varies from 0 to 6 percent.\textsuperscript{5,6} Its frequency is significantly influenced by the technical experience of the physician, the urgency required for obtaining venous entry, and the study methods for identifying its occurrence. A comprehensive review of complications of subclavian venipuncture has been reported recently by McGoon et al\textsuperscript{8} (Table 1); however, pacemaker malfunction related to this procedure has not been described. In our experience\textsuperscript{11} and that of Littleford and Spector,\textsuperscript{4} pneumothorax occurred in two of 70 patients and two of 74 patients, respectively. All four of these patients had spontaneous resolution of the pneumothorax without associated malfunction of the pacemaker system. As illustrated in the present case report, malfunction of the pacemaker system due to air entrapment requires multiple predisposing factors. These factors include a unipolar pulse generator with the anodal plate facing the subcutaneous surface, creation of a pleural air leak, and absence of a compression dressing over the pulse generator. Additional contributing factors are redundacy of the pocket and a nonconductive covering. The implantation technique should include avoidance of the initial pneumothorax, careful fashioned of the pacemaker pocket, thorough irrigation before wound closure, and avoidance of infection and hematomas, which may contribute to bacterial gas formation.

The diagnosis and management of air entrapment due to pneumothorax can be noninvasive, rapid, effective, andpermanent. A high index of suspicion should be maintained when using unipolar generators that have nonconductive material limiting the indifferent electrode to an "anterior window." When associated with pneumothorax, subcutaneous emphysema should be evident and an air bubble palpable above the generator. Although confirmation can be obtained with a chest roentgenogram taken at appropriate tangential angles,\textsuperscript{10,12} the diagnosis can be confirmed more rapidly by observing the immediate resumption of normal pacemaker function in response to compression and displacement of the air bubble. The electrocardiographic manifestations of air entrapment are variable and include failure to sense, failure to capture, and variations in pulse amplitudes. Total absence of pacemaker activity also has been reported.\textsuperscript{7,8} Amplitude variation in combination with one or more of the other abnormalities is highly suggestive of this disorder.

The rapidly growing use of the subclavian venipuncture for permanent transvenous lead placement, combined with the expanding use of smaller pulse generators that have nonconductive coatings to decrease the likelihood of muscle stimulation and myopotential inhibition, increases the likelihood of acute failure of the pacemaker system due to air entrapment. Awareness of this potential complication should enhance its effective diagnosis, treatment, and prevention.

| Table 1—Complications of Subclavian Venipuncture |
|--------------------------|--------------------------|
| Pneumothorax             | Air embolus              |
| Air embolus              | Hemotherox               |
| Hemomediastinum          | Hydrotherox              |
| Hydrothorax              | Subclavian artery perforation |
| Thrombosis               | Catheter sepsis          |
| Brachial nerve palsy     | Cellulitis               |
| Cellulitis               | Pneumothorax             |
| Pneumothorax             | Air embolus              |
| Pneumothorax             | Hemotherox               |
| Pneumothorax             | Hydrotherox              |
| Pneumothorax             | Subclavian artery perforation |
| Pneumothorax             | Thrombosis               |
| Pneumothorax             | Catheter sepsis          |
| Pneumothorax             | Brachial nerve palsy     |
| Pneumothorax             | Cellulitis               |
| Pneumothorax             | Pneumothorax             |
| Pneumothorax             | Air entrapment in pacemaker pocket with pacemaker malfunction |

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Fatal Pulmonary Henoch-Schönlein Syndrome

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A patient with Henoch-Schönlein syndrome involving joints, gastrointestinal tract, kidneys, and skin presented with pulmonary involvement. Immunohistochemical studies revealed extensive granular deposition of IgA along the alveolar septa, similar to that found in the skin and kidneys. This histologically unreported observation lends support to the notion that IgA is the main pathogenic antibody in Henoch-Schönlein syndrome.

Henoch-Schönlein syndrome is a clinicopathologic entity characterized by diffuse, necrotizing vasculitis involving joints, skin, gastrointestinal tract, and kidneys. Pulmonary involvement in Henoch-Schönlein syndrome is extremely rare. There are only two cases described in the literature with examination of the lungs but without immunohistochemical studies. We report our post mortem observations in a patient with Henoch-Schönlein syndrome who died in acute respiratory insufficiency.

Case Report

A 57-year-old white man was seen first in 1975 because of joint pains and swelling, which affected knees, ankles, and wrists. The symptoms were controlled with aspirin during four years.

In January of 1979, he developed an acute respiratory infection with recurrence of joint pain, a painful ulceration in the buccal mucosa and a diffuse purpuric rash over the lower extremities. The patient had a temperature of 99.5°F (37.5°C) and blood pressure of 140/80 mm Hg. Marked swelling and tenderness of the shoulders, elbows, wrists and interphalangeal joints was observed, along with an erythematous, nonblanching rash on both lower extremities, involving the anterior surface of the legs, ankles and feet.

Laboratory Studies

Urinalysis yielded moderate proteinuria and hematuria with finely granular and hyaline casts. No red blood casts were seen. Twenty-four hour urine protein excretion was 400 mg, serum creatinine 1.5 mg percent, BUN 25 mg, and sedimentation rate of 110 mm/hour. Rheumatoid factor was 1:40. Coombs test, cryoglobulin, antinuclear antibody and hepatitis B surface antigen were all negative. Serum complement levels were normal.

After admission, the BUN rose to 67 mg percent and creatinine to 3.9 mg percent. A diagnostic needle biopsy of the kidney was performed and revealed changes consistent with Henoch-Schönlein purpura. Biopsy of the skin rash revealed acute leukoclastic vasculitis. The patient was placed on 60 mg prednisone daily with improvement in renal function; he was discharged only to be readmitted a few days later with complaints of blurred vision. Examination at this time revealed bilateral papilledema. Blood pressure at this time was 134/80 mm Hg. CT scan of the head was negative. A diagnosis of pseudotumor cerebri was made; the patient was started on diuretic therapy (Diamox), with improvement in symptoms. He continued treatment as an outpatient with gradual tapering of prednisone and the diuretic.

He was readmitted 11 months later with nausea, vomiting and recurrence of joint pains. The BUN was 106 mg percent and serum creatinine was 8 mg percent. He improved on IV fluids and was discharged three days later.

The patient was readmitted within 48 hours with chills, low grade fever, nausea, vomiting, productive cough, and hemoptysis. Chest x-ray examination revealed diffuse bilateral infiltrates interpreted as bronchopneumonia (Fig 1). The BUN was 106 mg, serum creatinine 9.5 mg, and hemoglobin 6.9 percent g. He was treated with IV fluids, oxygen and antibiotics. The morning of the third day he collapsed and all efforts to resuscitate him were unsuccessful.

Gross Pathology

At postmortem examination, the main findings were in

![Figure 1. Extensive bilateral infiltrates present on chest film, more pronounced in the right lung.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21321/ on 06/21/2017)