ance of tumors with epicardium involvement and pericardial effusion\(^1\) is quite similar to the intrapericardial thrombus. However, a tumor can be seen on the pre-pericardiocentesis 2D-echo. The 2D-echo appearance of intrapericardial fibrin band has been described previously.\(^2\) This structure is a dense linear echo adherent to the visceral and/or parietal pericardium with an undulating motion synchronous with the cardiac cycle, as seen in our first patient (Fig 2). This characteristic motion of the fibrin band and its presence before pericardial tapping will differentiate it from a thrombus.

Although detection of an intrapericardial thrombus formation is a reliable sign of heart puncture, the 2D-echo is not yet a proven sensitive method for detection of such complications. However, these are reasonable areas for investigation, because a reliable method for detecting or ruling out heart puncture would find a clinical use in the decision of surgical intervention. In addition, repeated echocardiographic examinations might detect continuous pericardial bleeding with the gradual increase of the thrombus size.

REFERENCES
4 Mann W, Miller JE, Glauser FL. Bloody pericardial fluid, the value of blood gas measurements. JAMA 1978; 239:2151-52
7 Mano BV, Panidis IP, Kother MN, Mintz GS, Ross J. Two-dimensional echocardiographic detection of right atrial thrombus. Am J Cardiol 1983; 51:615-16

Cadmium Chemical Pneumonitis*

Scott Barnhart, M.D., and Linda Rosenstock, M.D., M.P.H.

Metal fume exposures are common to a number of trades and may result in the self-limited acute toxic syndrome of metal fume fever. Acute inhalational cadmium toxicity may mimic metal fume fever on initial presentation, but may have a markedly different clinical course. We report a case of cadmium-induced chemical pneumonitis in a welder who, over four years, has shown persistent pulmonary function abnormalities. The potential for acute cadmium toxicity needs to be considered in any patient suspected of having metal fume fever.

Exposure to metal fumes is common among many workers involved in smelting, welding, or cutting metals. The inhalation of metal fumes is known to result in several clinical syndromes including metal fume fever, chemical pneumonitis, and asthma. The diagnosis of these syndromes is often difficult since there is usually no immediate symptom and the delayed onset of the symptom may mask the relation between exposure and the syndrome.\(^*\) Common metal fume exposures include iron, steel, zinc, manganese, chromium, nickel, vanadium and cadmium. Cadmium, a lustrous white metal, has broad industrial uses ranging from electroplating steel to antifriction bearings to solder used in welding and brazing. The cadmium oxide fumes which result from burning cadmium are readily generated in many processes including welding, smelting and refining.\(^4\) The acute inhalation of cadmium fumes has been reported to cause both metal fume fever and chemical pneumonitis.\(^5\) Chronic cadmium exposure by inhalation has been reported to cause emphysema, pulmonary fibrosis, renal insufficiency and to be carcinogenic.\(^6\) Fortunately, acute inhalation exposures are rarely sufficient to cause metal fume fever or chemical pneumonitis unless the work is being done in poorly ventilated areas such as welding in an enclosed space.

Metal fume fever is a syndrome of fever, general malaise, and chest tightness which is benign and self-limited. Chemical pneumonitis is potentially more serious and may result in death, persistent restrictive ventilatory defects or may resolve entirely. We report a case of cadmium chemical pneumonitis with long-term follow-up.

CASE REPORT

A 34-year-old man was referred to the Occupational Medicine Clinic for evaluation of asbestos exposure. His history revealed that he had had minimal asbestos exposure, but that four years earlier he had been silver soldering for about one hour in an enclosed, unventilated small tank with an opening only large enough to admit his upper body. At the time of exposure, he noted only diplopia. Later that evening, he developed dyspnea, cough, myalgias and felt febrile. He did not seek medical attention at the time. Because of persistent cough and dyspnea he was seen about two weeks later by a local pulmonary physician who ordered a chest x-ray film and serial pulmonary function tests, and told the patient he had metal fume fever. His cough and dyspnea resolved over a period of about four weeks. When referred to our clinic, he was asymptomatic and had normal findings on physical examination. Review of his chest x-ray films obtained two weeks after his acute exposure revealed bilateral infiltrates (Fig 1). Chest x-ray evaluation four years later was normal. Results of pulmonary function tests (Table I) revealed a moderate restrictive impairment and moderately decreased single breath diffusing capacity (Dco). His Dco returned to normal within two months and his TLC has shown continued improvement, but remains below normal nearly four years after exposure.

DISCUSSION

While this patient was originally diagnosed as having metal fume fever, his presentation, laboratory findings and course

\*From the Division of Respiratory Disease and Critical Care, and the Occupational Medicine Program, Department of Medicine, University of Washington, Seattle. 
Reprint requests: Dr. Barnhart, Harborview Medical Center, 325 Ninth Avenue, Seattle 98104
are much more characteristic of chemical pneumonitis from cadmium exposure that resulted from silver soldering in an enclosed space. Metal fume fever is a common occupational illness among welders. It usually results from exposure to zinc oxide fumes, but is also reported following exposure to fumes from copper, aluminum, antimony, iron, manganese, nickel and cadmium. Significant exposures usually occur in poorly ventilated or enclosed spaces. Symptoms usually follow exposure by 4 to 12 hours and are characterized by a metallic taste, fever, malaise, joint pains, cough, sore throat, chest tightness and fatigue. The fever may reach 102°-104°F (38.8°-40.0°C) and the syndrome lasts one to two days. Polymorphonuclear leukocytosis is commonly present and the chest radiograph is usually normal. Re-exposure within one to two days usually does not cause recurrent symptoms. However, later exposures may once again elicit the syndrome; for this reason, metal fume fever is known among workers as Monday fever, as it is more likely to occur after several days away from work. The mechanism of this tolerance remains unexplained. In contrast to chemical pneumonitis, the syndrome of metal fume fever has no known long-term adverse effects.

Chemical pneumonitis can result from multiple toxic exposures ranging from irritant gases to toxic liquids to metal fumes. Multiple cases of chemical pneumonitis following exposure to cadmium fumes have been reported. Exposure may result in symptoms ranging from mild throat irritation to severe dyspnea associated with hemorrhagic pulmonary edema that may be fatal. The exposure usually results in minimal or no immediate symptoms. Initial presenting symptoms that occur several hours after exposure are consistent with metal fume fever including cough, throat irritation, fever, malaise. Unlike metal fume fever, however, the chest radiograph is frequently abnormal with bilateral infiltrates. Later, severe dyspnea, cough, hemoptysis, wheezing, abdominal pain, headache, renal failure and accompanying signs of pulmonary edema and respiratory failure may develop.

Autopsy studies of patients dying following acute exposure to cadmium fumes have revealed, on gross examination, severe tracheobronchitis and consolidated lungs. On light microscopy the bronchial epithelium is denuded and there is evidence of intra-alveolar hemorrhage and large numbers of macrophages filling the alveoli. Similar findings of pulmonary edema have been reported in animal studies where rats were exposed to cadmium fumes or to a cadmium chloride aerosol. These animals developed pulmonary edema and on microscopic examination were found to have a proliferation of the type 2 alveolar cells.

If the patient survives the acute exposure, there may be a persistent restrictive ventilatory defect. Five cases of cadmium chemical pneumonitis with serial pulmonary function tests for a period of up to four years have been reported. Each of these cases shows a restrictive impairment which improves over time. The greatest improvement appears to be in the first three months (Fig 2).

The diagnosis of chemical pneumonitis secondary to cadmium fume exposure should be suspected in any patient complaining of dyspnea following exposure to metal fumes.

![Figure 1](image) Posterioanterior chest x-ray film two weeks after exposure reveals diffuse bilateral interstitial infiltrates.

### Table 1—Pulmonary Function Over Four Years

<table>
<thead>
<tr>
<th></th>
<th>Predicted</th>
<th>14 days</th>
<th>18 days</th>
<th>21 days</th>
<th>26 days</th>
<th>32 days</th>
<th>44 months</th>
<th>46 months</th>
</tr>
</thead>
<tbody>
<tr>
<td>FEV₁ (L)</td>
<td>3.8</td>
<td>2.2</td>
<td>2.4</td>
<td>2.5</td>
<td>2.7</td>
<td>2.8</td>
<td>2.7</td>
<td>2.9</td>
</tr>
<tr>
<td>(% predicted)</td>
<td>(58)</td>
<td>(62)</td>
<td>(62)</td>
<td>(71)</td>
<td>(74)</td>
<td>(71)</td>
<td>(76)</td>
<td></td>
</tr>
<tr>
<td>FVC (L)</td>
<td>4.4</td>
<td>2.6</td>
<td>2.6</td>
<td>3.0</td>
<td>3.1</td>
<td>3.2</td>
<td>3.2</td>
<td>3.5</td>
</tr>
<tr>
<td>(% predicted)</td>
<td>(59)</td>
<td>(59)</td>
<td>(67)</td>
<td>(70)</td>
<td>(73)</td>
<td>(73)</td>
<td>(76)</td>
<td></td>
</tr>
<tr>
<td>FEV/FVC (%)</td>
<td>86.0</td>
<td>85</td>
<td>92</td>
<td>83</td>
<td>87</td>
<td>88</td>
<td>84</td>
<td>83</td>
</tr>
<tr>
<td>TLC (L)</td>
<td>6.3</td>
<td>3.3</td>
<td>3.8</td>
<td>4.9</td>
<td>4.2</td>
<td>4.2</td>
<td>4.2</td>
<td>5.0</td>
</tr>
<tr>
<td>(% predicted)</td>
<td>(52)</td>
<td>(60)</td>
<td>(76)</td>
<td>(68)</td>
<td>(68)</td>
<td>(68)</td>
<td>(79)</td>
<td></td>
</tr>
<tr>
<td>Dco (ml/min/mm Hg)</td>
<td>29.4</td>
<td>20.8</td>
<td>34.6</td>
<td>27.9</td>
<td>26.7</td>
<td>29.3</td>
<td>29.3</td>
<td>29.3</td>
</tr>
<tr>
<td>(% predicted)</td>
<td>(71)</td>
<td>(118)</td>
<td>(95)</td>
<td>(95)</td>
<td>(95)</td>
<td>(100)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

FEV₁ = forced expiratory volume in one second; FVC = forced vital capacity; FEV/FVC = ratio of forced expiratory volume in one second to forced vital capacity; TLC = total lung capacity.

Cadmium Chemical Pneumonitis (Bamhart, Rosenstock)
Frequently, the worker is unaware that the metal being cut, burned, or brazed contains cadmium. Physical, laboratory and radiographic abnormalities consistent with non-cardiogenic pulmonary edema all support the diagnosis of chemical pneumonitis. The treatment is supportive and although its clinical efficacy is not established some authors have advocated the use of corticosteroids. Determining the cause of the pneumonitis may be more difficult. In this case, the setting strongly suggests cadmium, but phosgene (from chlorinated hydrocarbons and heat) or other gases also need to be considered. Analysis of the fumes or the material being used is best. Biological determinations are difficult to interpret and are much less satisfactory. Blood levels correlate best with acute exposures and urine levels are probably a better reflection of the total body burden. Blood or urine cadmium levels which are above 0.005 mg/L suggest excessive exposure.

Chemical pneumonitis secondary to the inhalation of cadmium fumes is a potentially lethal syndrome and is associated with long-term adverse respiratory sequelae. Given the markedly different clinical course but nearly identical presentation, cadmium pneumonitis needs to be considered in any patient presenting with the signs and symptoms of metal fume fever.

As with most diseases of the workplace, the best treatment is prevention and should include, in addition to technological improvements in the workplace, worker education about potential hazards of welding, appropriate ventilation, and if necessary use of personal protective equipment.

REFERENCES


Systolic Anterior Motion of the Mitral Valve*

An Unusual Echocardiographic Feature of Mitral Valve Endocarditis

Francesco Enia, M.D.; Renato Lo Mauro, M.D.; and Enrico Geraci, M.D.

An unusual M-mode echocardiographic feature of mitral valve endocarditis is described: systolic anterior motion of the mitral valve, likely due to mitral valve vegetations, protruding during systole into the left ventricular outflow tract. The presence of mitral valve vegetation was confirmed at operation.

Systolic anterior motion of the mitral valve (SAM) was the first echocardiographic abnormality observed in hypertrophic cardiomyopathy. This finding was considered to represent echocardiographic evidence of the left ventricular outflow tract obstruction seen in hypertrophic cardiomyopathy and was promoted for the measurement of this obstruction. Subsequently, there were increasing reports of patients who showed SAM without evidence of hypertrophic cardiomyopathy. We describe a patient with SAM as an echocardiographic feature of mitral valve vegetations. We did not find similar reports in the literature, except an unpublished observation by Rakowski and Gilbert, quoted by Gilbert et al.

CASE REPORT

The patient was a 48-year-old man with mitral regurgitation.

*From the Division of Cardiology, Ospedale V. Cervello, Palermo, Italy.
Reprint requests: Dr. Enia, Via F. Lazzaz 47, 90145 Palermo, Italy

CHEST / 86 / 5 / NOVEMBER, 1984 791