bleomycin and oxygen were used together and demonstrated a synergistic effect. Even though Rinaldo et al. found a greater degree of experimental lung injury with oxygen eight vs 21 days following intratracheal bleomycin, Goldiner et al. reported a mean of 9.6 months between bleomycin administration and oxygen therapy during surgery. Thus, the period of time when oxygen administration appears to be safe following bleomycin has not been established.

The mechanism responsible for reactivation, or production of bleomycin pulmonary toxicity by oxygen, is not well understood but presumably is related to the availability of free oxygen radicals to produce lung injury. Why normally well-tolerated concentrations of oxygen produce lung damage in this setting may be related to the inability of bleomycin-injured lung tissue to scavenger released oxygen radicals, similar to the situation in parquat lung toxicity.

The interstitial changes of bleomycin lung have been described as irreversibly progressive and often fatal. Although no controlled studies exist, corticosteroids have been associated with clinical, radiographic and pathophysiologic recovery, particularly when instituted soon after appearance of symptoms.

Our patient, who had an initial favorable response to corticosteroids for bleomycin pulmonary toxicity, developed ARDS following surgery using a relatively low oxygen concentration (FiO2 = 0.33), similar to that reported by Goldiner et al. When corticosteroids were re instituted, our patient experienced a dramatic clinical response.

Individuals with a history of bleomycin toxicity, and even those with previous drug exposure without clinical toxicity, who are undergoing general anesthesia, should be ventilated with ambient air or as low an FiO2 as possible. This principle should apply as well to the recovery phase and any time when hospitalization occurs. When an FiO2 of 0.30 has to be used, short-term prophylactic corticosteroid administration should be strongly considered. If respiratory distress develops postsurgery in this clinical setting, bleomycin toxicity should be suspected and immediate administration of intravenous corticosteroids given, if other causes of ARDS are excluded. The patient should be ventilated with PEEP and the lowest FiO2 to keep a hemoglobin oxygen saturation of 90 percent.

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Acute Mercury Poisoning with Severe Chronic Pulmonary Manifestations*

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This report describes a patient who developed acute chemical pneumonitis following overexposure to metal mercury vapor. The exposure occurred in a gold extraction facility where a gold-mercury amalgam was heated in a confined area. Prompt treatment with penicillamine and corticosteroids was instituted; radiologic pulmonary infiltrates disappeared within a week, but there was little change in the pulmonary function abnormalities (restriction and diffusion impairment) over the period of 11 months of follow-up. This raises the possibility of persistent pulmonary function impairment after metal mercury vapor-induced chemical pneumonitis.

Occupational mercury poisoning due to inhalation of metallic mercury vapor is usually characterized by gingivitis and, in severe cases, stomatitis with sialorhea, fine tremor, irritability and insomnia. The clinical picture in which irritability predominates has often been referred to as mercurial erethism; the central nervous system is the critical organ in chronic industrial mercury poisoning. Rare cases with very high acute exposure to metallic mercury vapor have been reported in which severe respiratory symptoms have dominated the clinical picture. The critical organ in

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these cases is the lung. A review of the English literature in 1969 revealed only 48 such cases and few reports have been published since then.

We report here a case of acute mercury poisoning after exposure to metallic mercury vapor. Severe life threatening respiratory symptoms developed due to acute chemical pneumonitis; impairment of pulmonary function has persisted for one year after resolution of the acute chemical pneumonitis.

CASE REPORT

The patient, a 31-year-old white male resident of New York City, was admitted to the Mount Sinai Hospital (New York) with marked shortness of breath, dizziness, weakness and vomiting. This was his first hospital admission. He worked as the manager of a gold mine in Guyana and as a diamond trader. He came to the hospital directly from the airport on his return from South America. He had been exposed to mercury vapor while attempting to extract gold ore in a process that included mixing the ore with mercury and then heating the gold-mercury amalgam to extract the gold. The extraction took place close to the natural deposits of gold ore in a work area 20 feet by 20 feet by 10 feet, without windows or exhaust ventilation system. This was the first time the patient participated in this operation. Approximately two hours after he started, he experienced vomiting, shortness of breath and dizziness. After four hours he developed chest pain, dry cough, chills and extreme weakness. One other worker, a Guamanese, also experienced nausea and dizziness. The patient left the site for the local airport and flew back to New York, where he immediately sought medical care at the hospital. He was aware that his condition could be critical.

On admission, approximately 12 hours after exposure, the patient was very dyspnecic and complained of chest pain with deep inspiration. His blood pressure was 110/60 mm Hg; pulse, 90/min, with rapid shallow breathing (respiratory rate, 44/min at rest); temperature was normal. Breath sounds were barely perceptible; a few rales were present and the diaphragm was in a high position. There was no cyanosis or clubbing. The rest of the physical examination, including the oral mucosoae and the neurological examination was normal.

Past medical history was noncontributory; the patient had smoked one package of cigarettes a day for the last ten years.

Only a questionable increase in bronchovascular markings could be seen on the initial chest radiograph (Fig 1). Irregular infiltrates with confluence in the upper lobes were seen 48 hours later (Fig 2).

When he was admitted, arterial blood gases (measured on room air) revealed a PaO₂ of 69 mm Hg, a PaCO₂ of 36.2 mm Hg and pH of 7.44. Several attempts to measure his vital capacity were made during the first four days of hospitalization. Values were considerably below 1 L. On the fifth day, the forced vital capacity (FVC) was 1.95 L (38 percent of predicted) with no evidence of airway obstruction (Table I).

His maximum voluntary ventilation was reduced more than expected for a forced expiratory volume in one second (FEV₁) of 1.72 L. The single breath carbon monoxide diffusing capacity (DLCO) was decreased, 14.1 ml/min (49 percent of predicted); D(VO₂) was normal.

The patient's white blood cell count was 24,500 on admission with 79 percent neutrophils and 15 percent band forms; it returned to normal (8,100) with normal differential on the fifth hospital day. Urinalysis on admission was normal and remained so during the hospitalization; no proteinuria was detected. Blood urea nitrogen (BUN) 11 mg/dl and serum creatinin (1.2 mg/dl) levels were normal.

Treatment with penicillin 250 mg orally every six hours for three days started in the emergency room. A 24 hour urine collection contained 1,900 μg/L of mercury on the first day (in nonexposed persons, urinary mercury is less than 20 μg/24 hrs); 930 and 900 μg/L were excreted on the second and third days, respectively. Corticosteroids were given for the first three days as Solu-Medrol 60 mg intravenously, and for the rest of his hospitalization as prednisone 30 mg orally per day. Oxygen was administered via a nasal canula (2 L/min initially and 5 L/min after the severity of the chemical pneumonitis became obvious.

The patient improved slowly. For the first four days, he continued to have severe shortness of breath, chest pain and marked weakness, and was practically confined to his bed. After three days, the only abnormality found upon chest examination was the limitation in respiratory excursions; no areas of consolidation were found on percussion and no rales, rhonchi or wheezing were detected. On the fifth day, his respiratory rate decreased and he was able to perform deeper inspiratory efforts, although he was still short of breath with minimal exercise. A chest radiograph on the sixth day showed a decrease in the infiltrates in both lungs. Pulmonary function tests showed slight improvement in FVC (2.31 L or 45 percent of predicted) and in DLCO (16.5 ml/min per mm Hg or 57 percent of predicted.) Other possible effects of mercury toxicity, such as neurologic abnormalities, stomatitis or proteinuria were consistently absent; BUN and serum creatinin levels remained normal.

The patient was discharged after nine days. His urinary mercury level on the day of discharge was 130 μg/L.

There has been little change in his clinical status in the ensuing

Figure 1. Chest radiograph, posteroanterior projection, showing questionable increase in bronchovascular markings.

Figure 2. Chest radiograph, two days after admission, showing moderate to severe diffuse irregular infiltrates with confluence in the upper lobes.
Table 1—Pulmonary Function* in a Patient Following Acute Heavy Exposure to Mercury Vapor

<table>
<thead>
<tr>
<th></th>
<th>3/14/83</th>
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<th>7/7/83</th>
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<td>Lung volumes(L)</td>
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<tr>
<td>VC</td>
<td>—</td>
<td>2.31 (45%)</td>
<td>2.95 (56%)</td>
<td>2.55 (50%)</td>
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<tr>
<td>ERV</td>
<td>—</td>
<td>0.80</td>
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<td>FRC</td>
<td>—</td>
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<td>2.42</td>
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<tr>
<td>TLC</td>
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<td>5.01 (69%)</td>
<td>4.05 (57%)</td>
</tr>
<tr>
<td>RV</td>
<td>—</td>
<td>1.57 (80%)</td>
<td>2.06 (101%)</td>
<td>1.50 (76%)</td>
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<tr>
<td>Dynamic lung volumes and flows</td>
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<td></td>
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<tr>
<td>FVC(L)</td>
<td>1.95 (38%)</td>
<td>2.31 (45%)</td>
<td>2.95 (56%)</td>
<td>2.55 (50%)</td>
</tr>
<tr>
<td>FEV1(L)</td>
<td>1.72 (43%)</td>
<td>2.04 (50%)</td>
<td>2.44 (60%)</td>
<td>1.73 (43%)</td>
</tr>
<tr>
<td>FEF25-75% (L/sec)</td>
<td>0.86</td>
<td>0.89</td>
<td>0.83</td>
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<td>FEF50% (L/sec)</td>
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<td>0.42</td>
<td>0.38</td>
<td>1.00</td>
</tr>
<tr>
<td>FEF75% (L/sec)</td>
<td>1.93 (44%)</td>
<td>2.75 (63%)</td>
<td>2.97 (58%)</td>
<td>1.28 (29%)</td>
</tr>
<tr>
<td>MVV(L/min)</td>
<td>2.16</td>
<td>3.40</td>
<td>2.96</td>
<td>1.61</td>
</tr>
<tr>
<td>DCO3 ml/min per mm Hg</td>
<td>39.2 (39%)</td>
<td>45.0 (33%)</td>
<td>47.1 (34%)</td>
<td>30.0 (21%)</td>
</tr>
<tr>
<td></td>
<td>14.1 (49%)</td>
<td>16.5 (57%)</td>
<td>16.2 (50%)</td>
<td>18.1 (57%)</td>
</tr>
</tbody>
</table>

*Dynamic lung volumes, flow and MVV were performed using a computerized rolling seal spirometer following the guidelines of the Epidemiology Standardization project. The FRC was performed using a pressure plethysmograph and the DCO3 using a demand value system. Predicted values are Miller's modification of Morris' regressions, a modification of Goldman and Beaklee for TLC and RV, Miller for DCO3 and D/VA and Baldwin for MVV.

year. He cannot walk one flight of stairs without shortness of breath. Chest radiograph reveals only reduced lung volume without infiltrates (Fig 3). Pulmonary function tests showed slight improvement at four months but a decrease in FVC and lung volumes at 11 months, with evidence of airway obstruction (Table 1; mid-expiratory time or FEF25-75% was 1.0). His D/VA remained normal.

DISCUSSION

In this case, the heating of the gold-mercury amalgam to extract gold most probably generated very high concentrations of mercury vapor and caused the acute mercury toxicity observed. The lack of ventilation in the confined space where the extraction took place contributed to the severity of the mercury poisoning. The precise mercury vapor concentration to which the patient was exposed is not known, although an exposure to more than 1-2 mg/m³ for a few hours caused acute mercurial pneumonitis in four patients. 5

![Figure 3. Chest radiograph taken seven months after release, showing only questionable increase in markings.](image)

Respiratory absorption of mercury vapor is rapid due to excellent diffusion through the alveolar membrane. Whereas the central nervous system is the target organ after chronic exposure to mercury vapor, the lung is the critical organ in acute exposure at very high levels. Thus, the symptoms and signs observed in this case, severe shortness of breath, chest pain, dry cough and shallow rapid breathing, were due to acute chemical pneumonitis and, most likely, bronchiolitis. Chronic bronchiolitis is suggested by the persistence of severe dyspnea despite negative radiographic findings and by the advent of airway obstruction in pulmonary function testing. Bronchiolitis is well known to follow acute exposures to toxic vapors. 6,7 Diffuse interstitial pulmonary fibrosis has also been reported in cases of acute mercury poisoning surviving several weeks. 8 Slight interstitial fibrosis was found by lung biopsy five months after the acute toxic exposure in a case similar to the case we report. 8

Prompt treatment with the chelating agent resulted in very high urinary mercury excretion; symptoms and signs of central nervous system effects, stomatitis or renal toxicity were not observed. Penicillamine is generally accepted as an effective chelating agent for mercury. 7 Dimercaptosuccinylamine (DMSA) and BAL is also effective; however, penicillamine has the advantage of oral administration and, possibly, more potent chelation. In our patient, penicillamine was administered starting in the emergency room; the high urinary mercury excretion during the first three days (total approximately 4,000 µg) reflected the high level of exposure and the effectiveness of chelation therapy.

Although mercury is one of the well and long known toxic metals and the hazards of the amalgamation process of gold with mercury have been recognized and reported, 9,10 severe life-threatening acute mercury poisoning still occurs, as illustrated by this patient and several other recent cases. 4 One of the six patients reported by Snodgrass et al 10 developed chemical pneumonitis. As with our patient, the response to chelation with penicillamine and to corticosteroids (prednisone) was good but follow-up was limited to a month, during which pulmonary function test results had not yet re-
turned to normal.
While bronchiolitis obliterans has been observed in patients following penicillamine therapy, those patients have had an underlying collagen vascular disease (usually rheumatoid arthritis) and have been treated with penicillamine for longer periods of time.

Prevention of excessive mercury exposure, especially in small industrial or primitive "cottage industry" facilities, is important and should be given the necessary attention in order to prevent both the acute life-threatening mercury poisoning, with chemical pneumonitis and bronchiolitis as its major manifestations, and the residual pulmonary impairment.

REFERENCES

Polymyositic Heart Disease*
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There is an apparent correlation between the severity and duration of skeletal muscle involvement, cardiac manifestations and the extent of conduction system disease in polymyositis. Cardiac involvement during the course of polymyositis has been recognized as one of the typical features of skeletal muscle myositis. We report a patient with polymyositis in whom bifascicular block, prolonged P-R interval and congestive heart failure appeared three years before any clinical or laboratory evidence of active skeletal muscle myositis. To the best of our knowledge, this is the first report of polymyositis where cardiac manifestations preceded those of skeletal muscle myositis.

Polymyositis is a progressive inflammatory myopathy which can affect the heart. Cardiac involvement during the course of polymyositis has been increasingly recognized in the last decade. Gottdiener et al., in a noninvasive study, found that 76 percent of their patients with polymyositis had evidence of cardiac involvement. Patients with conduction abnormalities are prone to develop complete heart block because of the widespread progressive destruction of specialized conducting tissue and replacement by fibrotic tissue.

CASE REPORT
A 59-year-old man was hospitalized in March 1980 because of chest pain and dyspnea. We noted cardiomegaly, normal heart sounds and no murmurs. Neurologic examination results were normal. Chest x-ray films showed moderate cardiomegaly with mild pulmonary congestion. The ECG revealed bifascicular block (right bundle branch block and left anterior hemiblock) and first degree A-V block. Results of laboratory studies, including erythrocyte sedimentation rate, serum glutamic-oxalacetic transaminase (SGOT), creatinine phosphokinase (CPK) and lactate dehydrogenase (LDH) were all normal. An echocardiogram showed a dilated, poorly contracting left ventricle. Cardiac catheterization revealed an enlarged, poorly contractile, left ventricle, mild mitral regurgitation and 40 percent obstruction of the left circumflex artery. The patient was discharged with a presumptive diagnosis of idiopathic dilated cardiomyopathy.

In February, 1982 he was readmitted because of palpitations and vertigo. The ECG showed the same conduction defects and, in addition, periods of second degree A-V block (Wenckebach periodicity). An electrophysiologic study demonstrated an A-V nodal level of block. No treatment was given. Twenty months later (October, 1983), he was readmitted because of syncope and pulmonary edema. The ECG showed intermittent second and third degree A-V block. Physical examination revealed hepatosplenomegaly and symmetric proximal muscle wasting and weakness in both upper and lower limbs, as well as wasting of the paravertebral muscles. Laboratory studies showed marked elevation of serum muscle enzymes: CPK (of muscle origin, MM band) 2300 mu/ml (normal under 145 mu/ml), aldolase—16 mu/L (normal under 7.6 mu/L), SGOT—127 mu/L (normal under 40 mu/L), LDH—290 mu/L (normal under 225 mu/L). The erythrocyte sedimentation rate, hemogram,

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