given therapy with digoxin. Intravenous dopamine and dobutamine were infused, which increased his systolic blood pressure to 100 mm Hg. The patient was intubated. Echocardiographic study disclosed four chamber enlargement with markedly hypokinetic ventricles and a small pericardial effusion. CT scan of the thorax showed cardiomegaly, enlarged pulmonary artery with prominence of the pulmonary vasculature, distended inferior vena cava and hepatic veins and ascites, without aortic aneurysm or dissection. Portable perfusion lung scan was interpreted as low probability for pulmonary embolism. Right heart flotation catheter revealed a right atrial pressure of 24 mm Hg, pulmonary artery pressure of 37/24 mm Hg, and pulmonary capillary wedge pressure of 24 mm Hg; cardiac output was 3.87 L/min. Despite treatment with diuretics, increasing doses of inotropic agents and ultrafiltration, the patient's cardiovascular status gradually deteriorated. Forty-one hours after presentation he went into complete electromechanical dissociation and attempts at resuscitation were unsuccessful.

On postmortem examination, the patient was noted to have idiopathic hemochromatosis involving the heart, liver, pancreas, stomach, duodenum, adrenal glands, kidneys and skin. Titters for Coxsackie virus, Epstein-Barr virus, echovirus, Mycoplasma and Rocky Mountain spotted fever were negative. Postmortem cultures from myocardium, pericardial and ascitic fluids were also negative. Heavy metal screen was negative. Urine screen was positive for cocaine and/or metabolite.

**DISCUSSION**

Hemochromatosis is a hereditary multisystem disorder characterized by iron deposition in parenchymal cells of the liver, heart, pancreas, and skin. Approximately one-third of patients with hemochromatosis die from slow progression of cardiac myopathy. The pathologic hallmark of this disease is an accumulation of iron in the cardiac muscle, liver, pancreas, and other organs. The iron deposition leads to a variety of clinical manifestations, including liver disease, diabetes, and endocrinopathies. The disease is caused by mutations in the HFE gene, which are associated with increased iron absorption from the gut. The diagnosis is typically made through genetic testing or liver biopsy. It is important to note that hemochromatosis is not a rare disease, with an estimated prevalence of 1 in 200 individuals in the general population. The disease can be managed through phlebotomy, which can result in a significant reduction in serum iron and iron stores. However, the long-term impact of phlebotomy on quality of life and survival is unclear.

Cardiac involvement in hemochromatosis typically results in dilated cardiomyopathy. Restrictive cardiomyopathy is rare. Our patient demonstrated restrictive physiology with equalization of mean right atrial pressure, right ventricular end-diastolic pressure and mean pulmonary capillary wedge pressure. Because of rapid deterioration in this patient's cardiac function, the diagnosis of acute myocarditis or a cardiotoxin was entertained. However, subsequent titers and postmortem cultures were negative. No heavy metals were detected, but a urine drug screen was positive for cocaine and/or metabolite, indicating cocaine use within 48 hrs of presentation.

Cardiovascular effects of cocaine are due to its sympathomimetic and vasoconstrictive properties, resulting in hypertension, coronary artery spasm and tachycardia which, in turn, may produce cerebrovascular accidents, pulmonary edema, myocardial infarction, and sudden death. Our patient had normal coronary arteries and no myocardial infarction at autopsy.

While the relationship between this patient's acute congestive heart failure and cocaine use is unclear, the fulminant course in this patient with idiopathic hemochromatosis is unusual and raises the question of another factor resulting in acute myocardial dysfunction. Since no other cause was identified, it is speculated that cocaine played a synergistic role in this patient by depressing myocardium already impaired by diffuse parenchymal iron deposition associated with idiopathic hemochromatosis. Further observations are needed, however, to determine whether cocaine can result in acute heart failure in patients with underlying myocardial disease.

**REFERENCES**


**Occupational Asthma Due to Fumes of Galvanized Metal***

Jean-Luc Malo, M.D.; and André Cartier, M.D.

Two solderers exposed to fumes of galvanized metal reported a history of shortness of breath and fever which occurred during the evening and night of days spent at work. Specific inhalation challenges performed by asking subjects to do soldering on galvanized iron revealed a late bronchospastic reaction. One subject also demonstrated a significant increase in oral temperature and peripheral neutrophils. Environmental measurements revealed the presence of zinc after soldering on galvanized metal. This contaminant was not present after a control exposure while soldering on iron. Although metal fume fever has been described in workers exposed to fumes of galvanized metal, this is the first account of occupational asthma due to this agent.

Galvanizing operations have been reported to cause metal fume fever but not occupational asthma. Two solderers processing galvanized metal demonstrated late bronchospastic reactions. One of the two also showed fever and leukocytosis which suggests the presence of metal fume fever or hypersensitivity pneumonitis.

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Chest 92/2/August, 1987

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CASE 1

The first worker was a 55-year-old solderer exposed to galvanized fumes for 4½ years. In the past three years, he reported shortness of breath and fever which occurred during the evening and night of days spent at work. He did not mention any thirst or metallic taste in his mouth, or muscular aches. He left work due to his complaints and was seen nine months after. At that time, he still reported dyspnea and wheezing on exercise. He used an inhaled beta-2 adrenergic agent four times per day and PRN. Skin tests done by the prick method with a battery of 15 common inhaled allergens revealed no immediate reaction. Lung function tests showed a FEV1 of 2.4 L (92 percent predicted) and a FEV1/FVC of 0.72 percent (81 percent predicted). Transfer factor for carbon monoxide was 120 percent predicted by the steady state method.2 Mild bronchial hyperresponsiveness to histamine3 was present as the provocative concentration causing a 20 percent fall in FEV1 (PC 20) was 5.0 mg/ml. No significant change in FEV1 was demonstrated by asking the subject to do electric soldering on iron for 15 minutes using a rod heated at approximately 100°C. However, the subject showed a bronchospastic reaction which was maximal 270 minutes after the end of the soldering for 30 minutes on galvanized metal (Fig 1). Oral temperature went from 36.6°C to a maximum of 38.9°C, ten hours after the active challenge. White blood cell counts increased from 10,600/cu mm (78 percent neutrophils, 2 percent eosinophils) to 15,800/cu mm (76 percent neutrophils, 3 percent eosinophils) eight hours after the end of challenge. There was no significant change in transfer factor. Chest roentgenogram was normal ten hours after the end of exposure. The PC 20 histamine dropped significantly4 to 1.5 mg/ml on the following day at a time FEV1 was back to baseline.

CASE 2

This 52-year-old man had been a solderer for 23 years. Four months before his visit, he had done soldering on galvanized metal on two occasions. Two hours after exposure, he reported shortness of breath, dry cough, and wheezing. These symptoms lasted for approximately 12 hours, and he reported chills on the following evening. He did not have any thirst or metallic taste in his mouth or muscular aches after exposure. Skin tests showed immediate reactivity to ragweed pollen, house dust, and D farinae. Lung function tests revealed an obstructive breathing defect, the FEV1 being of 2.33 L (80 percent predicted) and the FEV1/FVC of 61 percent (75 percent predicted). Transfer factor for carbon monoxide was 93 percent predicted.2 The PC 20 histamine by the same method as above was 0.5 mg/ml (moderate bronchial hyperresponsiveness). Asking the subject to do soldering on iron in a similar way as for worker 1 for 15 minutes produced no significant change in FEV1, whereas a late bronchospastic reaction was shown after asking the subject to do similar soldering on galvanized metal (Fig 1). No fever or significant change in white blood counts and transfer factor developed. The PC 20 dropped to 0.18 mg/ml, 48 hours after the exposure, which is a borderline significant change,5 at a time FEV1 was back to baseline.

Environmental Measurements

Assessment of particles with an apparatus placed on a control solderer's shirt, at a distance of 10 to 15 cm of the mouth, revealed the following concentrations of metals in mg/m³:

<table>
<thead>
<tr>
<th>Iron</th>
<th>Chromium</th>
<th>Manganese</th>
<th>Copper</th>
<th>Zinc</th>
</tr>
</thead>
<tbody>
<tr>
<td>Soldering on iron: 1.80 ND 0.32 ND ND</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Soldering on galvanized iron: 2.80 ND 0.47 ND 22.0</td>
<td></td>
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</tr>
</tbody>
</table>

ND indicates not detectable.

DISCUSSION

Metal fume fever is an influenza-like illness that is encountered in solderers. Even if monitoring of spirometry has not been carried out in such workers to our knowledge, there is no mention that such an exposure could cause asthmatic symptoms or significant changes in FEV1 like those found in our workers. These two men reported fever after exposure to galvanized fumes, but this could be demonstrated in only one of the two who also showed leukocytosis. This worker might have had a combination of occupational asthma and metal fume fever or hypersensitivity pneumonitis.

Although the exact mechanism of the reaction seen in our two subjects is uncertain, we think that the bronchoconstriction which we described is specific (nonirritating) and due to exposure to a sensitizing agent. Indeed, so-called late reactions, like those found after exposure to an antigen, were described in both workers. Also, we reported significant changes in bronchial hyperresponsiveness to histamine in one of the two workers and borderline changes in the other. Such changes are encountered after late asthmatic reactions following exposure to an antigen6 or a sensitizing agent at work.7 Environmental measurements revealed the presence of zinc while soldering on galvanized metal but not on iron. It is thus possible that this agent was responsible for the reaction.

ACKNOWLEDGMENTS: The authors want to express their gratitude to Mr. Roger Tremblay of the Department of Community Health of Hôpital du Sacré-Coeur and to the Institut de Recherche en Santé et Sécurité du Travail du Québec for measuring the concentrations of metals.
Two Cases of Dilated Cardiomyopathy Complicated by Left Ventricular Aneurysm*

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This report describes two patients with dilated cardiomyopathy associated with angiographically proven left ventricular aneurysm. There was no apparent history of myocardial infarction and coronary arteries were angiographically normal in both cases.

Localized wall motion abnormality has been reported in dilated cardiomyopathy. However, left ventricular aneurysm is rare. This report describes two adult patients with dilated cardiomyopathy complicated by angiographically proven left ventricular aneurysm.

CASE REPORTS

Case 1

A 62-year-old woman was hospitalized because of dyspnea. She had a history of dyspnea and cardiomegaly which began 16 years prior to admission. There was no history of chest pain suggestive of angina pectoris or myocardial infarction. On admission, she was cyanotic with a cold sweat. The pulse rate was 120 beats per minute and regular, and blood pressure was 144/100 mm Hg. Respiration was labored with orthopnea, and the lungs were filled with bubbling rales. Gallop rhythm and a 2/6 holosystolic murmur were present at the apex. Results of laboratory studies, including cardiac enzymes, were normal. A chest x-ray film revealed marked cardiomegaly with the cardiothoracic ratio of 0.74 and markedly increased pulmonary markings. An electrocardiogram (ECG) showed left ventricular hypertrophy, Q waves in lateral leads, and diffuse ST-T abnormalities (Fig 1, A). These findings did not change on serial ECGs. Short runs of ventricular tachycardia were found on a Holter ECG. At rest, thallium 201 (M-99m Tc) myocardial perfusion studies revealed a persistent perfusion defect at the anterolateral region (Fig 1, B). Studies with technetium 99m (M-99m Tc) pertechnate, first-pass method, revealed a decreased ejection fraction of both ventricles (left ventricle, 17 percent; right ventricle, 30 percent). Cardiac catheterization showed elevated left ventricular end-diastolic pressure (18 mm Hg) and a...