1982, in pulmonary distress. At autopsy, no other localization of epithelial lesions than in the lung was recognized.

**DISCUSSION**

In the reported cases of clinical busulfan pulmonary toxicity to date, duration of therapy has ranged from nine to 120 months with a mean value of 30 months. The total dose of busulfan administered ranged from 500 mg to 5,700 mg with a mean of 900 mg.1 However, one case was published after a six-week low dose busulfan treatment.2 Most of the cases of pulmonary toxicity have occurred while busulfan was being administered, but in at least one case, the onset of symptoms began approximately one month after discontinuation of therapy.3 In our patient, the acute pulmonary symptoms developed following only 140 mg busulfan and with a delay of two months after discontinuation of therapy, which is still unique. It must be stressed that the patient had not previously been exposed to thoracic irradiation or to high dose oxygen therapy, and that he did not receive any drug known to be associated with pulmonary toxicity until April 1981 when he was treated for the first time with melphalan.

The acute pulmonary and general symptomatology which developed six months after the last melphalan course makes it highly unlikely that melphalan therapy is to be considered directly responsible for the acute pulmonary phenomena. Indeed, in the five patients reported to develop the clinical lung toxicity after treatment with melphalan, the delay between drug administration and the clinical events was never so long.4,5 Melphalan produced, nevertheless, infraclinical pulmonary lesions, as demonstrated by Taetle et al,19 who found four patients with atypical alveolar proliferative lesions (AEP) and two patients with the typical picture of lung fibrosis in a retrospective analysis of autopsies of 11 selected multiple myeloma patients treated with melphalan alone for one to 48 months. In the autopsies of 11 untreated myeloma patients and 11 controls, no AEP lesions were discovered. In this respect, the most valid explanation for the events occurring in our patient is that melphalan produced the asymptomatic infraclinical lung damage and that subsequent, although short and intermittently administered, busulfan treatment aggravated these lesions by additive toxicity on the pulmonary tissue.

The administration of melphalan therapy in May 1982 following the evolution of the CML rapidly induced (approximately three weeks) the initial acute symptomatology with fatal outcome, although the dose was again low and the patient received maintenance corticotherapy. Here, a direct effect and probable cross-reaction with busulfan can be hypothesized.

Physicians should be aware of these possible synergistic effects on lung tissue, particularly in patients who have had previous melphalan treatment.

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**Acute Dissection of the Ascending Aorta**

**Initial Presentation as Acute Lower GI Bleeding**

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We present the unusual case of a 56-year-old man with acute dissection of the ascending aorta (De Bakey type I) whose presenting symptoms were those of lower gastrointestinal (GI) bleeding. Surgical repair was successfully accomplished with resection of the aorta with a Dacron tubular graft combined with aortic valvular replacement after obtaining bowel viability.

In 1826, Laennec coined the term "dissecting aneurysm," and 31 years later, Swain diagnosed the condition shortly before the death of his patient. Yet despite more than two decades of advancements in methods of diagnosis, aortic dissection remains extremely difficult to diagnose. Classic symptoms of chest pain or back pain with occasional abdominal pain can be complicated by symptoms of aortic insufficiency, if the dissection has involved the valve, as well as peripheral vascular insufficiency, including diminished pulses in the extremities or symptoms of cerebral or renal ischemia. This patient represents a "forme fruste" with initial presentation being lower GI bleeding.

**CASE REPORT**

A 56-year-old man with a three and one-half year history of hypertension was admitted with initial complaints of abdominal pain and gross hemoptoeza. Primary GI symptoms were compounded by complaints of a shock-like feeling through his body, numbness in the left lower extremity, and vertigo, followed several hours after admission with substernal pain and tightness and blood pressure differential in the upper extremities. Aortograms revealed a circumferential dissection, arising 2 to 3 cm above the aortic anulus, extending to the celiac axis, with involvement of the innominate, right renal, inferior mesenteric, and right iliac vessels, as well as the aortic bifurcation. Portions of the lower abdominal aorta were completely occluded and it was not clear where true and false lumens were. There was severe aortic regurgitation without visualization of the coronary arteries. Hypertension was controlled with intravenous nitroprusside. Colonoscopy revealed no evidence of bowel infarct.
tion, but focal areas of ischemia. About 36 hours after onset of symptoms the patient had aortic valve replacement with a no. 27 Edwards-Carpentier porcine valve and replacement of the ascending aorta with a preoccluded no. 24 Dacron tube graft placed in the supracoronary position with the distal end at the level of the right innominate artery. The patient was discharged one week after surgery and remains stable six weeks later.

**Discussion**

Dissection of the ascending aorta remains among the most catastrophic events affecting the great vessels. Risk of early rupture is influenced by the anatomic site, and there is a persistent risk of late rupture. Thus, surgical intervention has been advocated for both acute and chronic dissections to alter this natural history. Untreated, a few patients die immediately (3 percent). Within 24 hours, this figure rises to 24 percent, within two weeks to 60 percent, and by three months to 90 percent. Medical treatment is associated with serious untoward events and often does not establish satisfactory long-term results. Refinements in surgical procedures and postoperative care have led to improved operative results, initiating a trend in more aggressive surgical management.

Contraindications to surgical management include bowel infarction (100 percent mortality), bilateral renal infarction or severe neurologic deficits. We elected to evaluate the bowel before planning surgical repair of the dissection.

This report describes an unusual presentation of acute dissection and can alert the physician to the potential problem in a patient with a history of hypertension and GI bleeding. The correct diagnosis in this case was reached after secondary symptoms of variable pulses in the left lower extremity, an increasing grade 3/6 diastolic murmur of aortic insufficiency, and chest pain with multifocal ventricular ectopies without evidence of myocardial infarction led to an echogram and then an aortogram to evaluate the patient for possible acute aortic dissection.

**Acknowledgments:** Karen Prewitt, CCRN, made important contributions to the work. Jeannette Freeman was instrumental in the preparation of the manuscript.

**References**


**Right-Sided Native-Valve Endocarditis Caused by Actinobacillus actinomycetemcomitans***

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The clinical and echocardiographic features of a case of endocarditis due to *Actinobacillus actinomycetemcomitans* uniquely involving the native tricuspid valve are presented. The importance of two-dimensional echocardiographic studies in the evaluation of patients with suspected endocarditis is emphasized.

*Actinobacillus actinomycetemcomitans* is an uncommon cause of endocarditis. This organism is a slow-growing, fastidious Gram-negative bacillus normally found in the oral cavity. Approximately 50 cases of endocarditis due to *Actinomycetemcomitans* have been reported since the first case report in 1964, all involving the native left-sided cardiac valves or prosthetic cardiac valves. The purpose of this report is to document a case of right-sided endocarditis occurring on a native valve due to *Actinomyce temcomitans* and to stress the value of two-dimensional echocardiographic studies over standard M-mode techniques in the routine assessment of patients with suspected endocarditis.

**Case Report**

In August 1981, a 48-year-old male dress manufacturer came to the Dallas Veterans Administration Medical Center with three weeks of recurrent fevers, chills, and nausea. He had had a nonproductive cough for two days. His temperature was 38.3°C (100.5°F), with otherwise normal findings on physical examination. The hematocrit reading was 40.9 percent, the hemoglobin level was 14.1 g/100 ml, and the white blood cell (WBC) count was 11,400/cu mm (segmented cells, 59 percent; and band cells, 14 percent). The bilirubin level was 0.9 mg/100 ml, and the serum glutamic-oxaloacetic transaminase (SGOT) level was 28 mU/ml. The chest x-ray film was within normal limits. Three weeks later, the patient was admitted to the hospital because of continued similar complaints, with worsening anorexia and malaise. He admitted to excessive intake of alcohol during the past 15 years and to several gingival surgical procedures from January to May 1981, and denied previous rheumatic fever, cardiac murmur, intravenous drug abuse, or recent antibiotic therapy. The patient's temperature was 40.4°C (104.7°F), the blood pressure was 135/70 mm Hg, the pulse rate was 120 beats per minute and regular, and the respiratory rate was 16/min. Physical examination revealed poor dentition, no retinopathy, normal cardiac findings without murmurs, clear pulmonary fields, no abdominal organomegaly, a mildly enlarged and tender prostate gland, and no rash or cutaneous lesions. Laboratory values were as follows: hematomicrot reading, 27.5 percent; hemoglobin level, 9.1 g/100 ml; reticulocyte count, 0.8 percent; WBC count, 9,900/cu mm (segmented cells, 67 percent; and band cells, 17 percent); bilirubin level, 2.4 mg/100 ml; SGOT level, 19 mU/ml; total protein level, 7.1 g/100 ml; albumin level, 3.1 g/100 ml; and hepatitis B surface antigen, negative. Electrophoresis of the hemoglobin revealed an AA pattern; G6PD was normal. Findings from urinalysis were unremarkable. The chest x-

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Right-sided Native-valve Endocarditis (Hirsh, Nixon)