valve ring abscess and aortic valve replacement. Our animal data suggest that irreversible damage to cells of the conduction system may be brought about by balloon dilatation and that this may be more frequent with larger balloons. While our patient had reasonably maintained blood pressure with ventricular pacemaking, the loss of atrial function in a patient with a hypertrophic noncompliant ventricle may result in a marked decrease in blood pressure and cardiac output (Fig 3). The utilization of dual-chamber pacing in the DDD mode, which provides for an appropriate AV contraction sequence, as well as rate augmentation, seems appropriate under these circumstances.

In summary, we present a case of CHB requiring permanent pacing subsequent to ABV. The irreversible damage consisting of contraction band necrosis in the cells of the conduction system in the canine model may underlie the mechanism of heart block in this patient. The physician performing ABV should be aware of the need for urgent pacemaker therapy and may wish to prophylactically insert a temporary pacemaker in these cases.

References

Cocaine-induced Acute Aortic Dissection*
Dominick Gadaleta, M.D.; Michael H. Hall, M.D.; and Roy L. Nelson, M.D.

While cocaine-induced myocardial infarction has been frequently documented, the differential diagnosis of chest pain should include aortic pathology. The successful management of acute aortic dissection secondary to cocaine abuse has not been previously reported to our knowledge. In a 45-year-old man who presented with typical chest pain and wide mediastinum, the successful management of this disease included early and accurate diagnosis and replacement of the aortic valve as well as the torn portion of the ascending aorta.

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Clinicians are aware of the more commonly occurring cardiac and cerebrovascular consequences of cocaine abuse: ventricular arrhythmia, myocardial ischemia and infarction, cerebrovascular accident, and subarachnoid hemorrhage. However, the differential diagnosis of chest pain associated with cocaine abuse should include aortic pathology. Acute rupture of the ascending aorta has been reported as a fatal consequence of smoking cocaine (freebasing). This article is the first description, to our knowledge, of a successful antemortem diagnosis and repair of a cocaine-induced acute aortic dissection.

Case Report
A healthy 45-year-old male executive, at the end of a 36-h drinking and cocaine-snorting session, presented with severe, sudden, unrelenting, crushing substernal chest pain without radiation. There was associated upper back pain, along with diaphoresis and palpitations, but without lightheadedness or leg, abdominal, or groin pain. The patient was initially evaluated at a nearby hospital and referred to North Shore University Hospital for definitive care.

Past medical history was significant for mild, untreated hypertension. The patient smoked two packs of cigarettes and drank one quart of vodka per day. He had had a recent emergency room visit for drug overdose (IV heroin and cocaine—a speedball). There was no history of Marfan's syndrome or connective tissue disorder, nor were other drugs or other forms of cocaine used during this time.

Physical examination revealed moderate distress. His vital signs were as follows: heart rate, 100 bpm and regularly irregular; blood pressure 145/90 mm Hg; and respiratory rate, 26 breaths/min. Cardiac examination showed a normal S1 and S3, with a decrescendo diastolic murmur at the left sternal border. There was no rub or gallop. The left radial and left femoral pulses were diminished, with no palpable pulses distal to the left common femoral artery. The right radial and femoral pulses were full and bounding. His urine output and laboratory values were within normal limits. The ECG showed sinus rhythm with frequent atrial premature contractions, normal axis, and no evidence of acute infarction nor ischemia. A chest x-ray film showed clear lung fields with a widened mediastinum.

Echocardiogram and CT scan suggested a Stanford type A acute aortic dissection. Angiography revealed a tear starting in the

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ascending aorta. The dissecting hematoma extended to the left common iliac artery (Fig 1). He was stabilized with IV propranolol and nitroprusside and transferred for surgical therapy.

At surgery, the patient was placed on cardiopulmonary bypass by femoral artery and right atrial cannulation. He was cooled systemically to 25°C, and the heart was arrested with cold blood cardioplegia. A transverse tear was noted in the ascending aorta, which extended around one-third of the aorta's circumference anteriorly. The dissection extended deeply into the noncoronary sinus of Valsalva, disrupting the aortic valve. The valve leaflets appeared thin and elongated. There was no annuloaortic ectasia, and the left ventricle was not enlarged. The aortic valve was replaced with a 27-mm Carpentier-Edwards porcine aortic prosthesis. The ascending aorta was completely excised from the level just above the coronary ostia to just beyond the innominate artery. Teflon felt strips were placed inside and outside the aortic cuffs for reinforcement. The outside strips were soaked in salt-poor albumin and baked in an autoclave for 3 min to make them impermeable. A 30-mm, low-porosity woven graft, which had also been soaked with salt-poor albumin and baked in an autoclave was then anastomosed to the two aortic cuffs, proximal first and distal second. After rewarming, the patient was removed from cardiopulmonary bypass, with excellent hemodynamic values and without need for pressor or inotropic support.

The patient's postoperative course was complicated by an opportunistic pulmonary infection causing ventilatory dependence requiring tracheostomy. His workup revealed him to be HIV positive. Discharge occurred on the 47th postoperative day. At nine months postoperatively he has made a complete recovery with no disability.

**DISCUSSION**

Cocaine is a popular drug used in many forms through all social strata. Acute aortic dissection must be considered in any patient with chest pain who admits to recent cocaine use. Previously reported causes of chest pain occurring during and immediately after cocaine use include myocardial ischemia or infarction, myocarditis, and pneumomediastinum. Aortic disruption causing chest pain had not been previously reported, to our knowledge, in this clinical setting. It is impossible to determine the incidence of this potentially lethal complication, as many patients die before they get to a hospital, and a history of cocaine use may not be obtainable. At presentation, the diagnosis may be missed or delayed if the focus is directed toward only the cardiac rather than the vascular systems, since cardiac complications have been the more commonly reported sequelae. Further delay in diagnosis can occur as the patients may have no cardiovascular risk factors. Although toxic levels of cocaine are frequently reported, there is a wide range of blood concentrations measured among cocaine-related fatalities. Therefore, a drug level is rarely useful.

Aortic dissection may masquerade as an acute myocardial infarction if appropriate ECG changes are present. Now that IV thrombolytic therapy is becoming common in emergency rooms prior to cardiac catheterization, it becomes especially important to maintain a high degree of suspicion and a low threshold for echocardiography or CT scanning in such patients when there is a suggestion of a widened mediastinum on routine chest x-ray film.

Cocaine blocks the presynaptic uptake and subsequent breakdown of norepinephrine and dopamine, producing an excess of neurotransmitters at postsynaptic sites. Cocaine also acts to sensitize the heart and peripheral vasculature to the effects of catecholamines, thereby causing more accentuated chronotrophy, inotrophy, and increased systemic vascular resistance. The resultant dose-dependent increase in heart rate and systemic blood pressure, with marked increase in myocardial oxygen demand and increased contractility, lead to an increased shearing force on the aortic wall. It is thought that the extreme and sustained elevation in blood pressure associated with prolonged cocaine use was a significant factor causing aortic disruption and dissection in this otherwise normal patient. Early diagnosis, stabilization of blood pressure, and referral to a tertiary care center for surgical therapy were essential to the successful outcome in this case.
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Ventilator Dependence in Acute Severe Asthma Due to a Variant Presentation of Guillain-Barré Syndrome*

Richard J Hamilton, M.D.; Ralph Puckett, M.D.; and W Carl Bazemore, M.D., F.C.C.P.

A 23-year-old white female asthmatic patient arrived in the Emergency Department in acute respiratory distress, afebrile, with poor air movement and diffuse inspiratory and expiratory wheezes. Neurologic exam was completely normal. Her respiratory condition rapidly deteriorated and she was intubated, paralyzed, sedated and transferred to the Intensive Care Unit. There, she exhibited severe bronchospasm that was resistant to all modes of therapy and kept her ventilator dependent. On hospital day 10, after discontinuing sedation and neuromuscular blockade, she was found to have a flacid quadraparesis. She was able to blink her eyes and move her head. Her sensory and cranial nerve examinations were completely intact. Babinski and deep tendon reflexes were absent.

The results of CSF studies were normal with no cells found; glucose value was 99 mg/dl, and protein level was 31 mg/dl, as well as negative stains, smears, and cultures. The CSF protein electrophoresis as well as myelin basic protein levels were normal. Urine heavy metal screen was negative. Anticholinergic receptor binding antibody levels were zero. Computed tomography and electroencephalography studies were within normal limits. The patient remained afebrile throughout her illness and experienced no increase in acute or convalescent viral titers. Results of serum chemistries, complete blood count, blood cultures, and urinalysis remained within normal limits throughout her illness.

Portable NCS done at that time in the ICU revealed a peripheral demyelination of lower extremities as evidenced by prolonged distal latencies, reduced amplitude, and mild nerve conduction velocity reduction in peroneal nerve testing, as well as focal slowing across the fibular head. (Latencies were measured at 20 ms above the fibular head, 17 ms below, and 9.1 ms at ankle; amplitudes were 1, 1.2, and 2 mV respectively; NCV were 33 and 38 m/s above and below the fibular head.) There was also reduced amplitude of median motor distal responses with a normal median latency and NCV. (Latencies were measured at 8.2 and 4.1 ms at the elbow and wrist; amplitudes were both 1 mV; and NCV was measured at 56 meters/s at the elbow.) Needle EMG of vastus lateralis, anterior tibialis, gastrocnemius, and abductor pollicis brevis showed reduced interference with no evidence for denervation. From these data, and despite normal CSF studies, she was diagnosed as having a variant presentation of GBS and a course of plasmapheresis was begun, consisting of five treatments, averaging 2.5 to 4.0 liters at a time.

Over the ten-day course of this treatment, her neurologic and respiratory status slowly improved. By hospital day 37, she was off the ventilator and walking short distances. A repeat comparison NCS showed generalized decreases in latencies and increases in NCV to normal, and a slight increase in amplitude in peroneal testing. (Latencies were 12.8, 10.6, and 3.8 ms above and below the fibular head and at ankle, respectively; amplitudes were 1.8, 2.0, and 2 mV, respectively.) Median motor and sensory testing showed increase in evoked response to normal (borderline) levels. (Motor latencies were measured as 6.9 and 2.8 ms at elbow and wrist; amplitudes were measured as 5 mV at both sites; NCV was 52 m/s at elbow. Sensory exam showed latency as 3.1 ms and amplitude as 50 μV.) Needle EMG repeated at the previous sites, as well as deltoid, biceps, extensor digitorum, first dorsal interosseus, and peroneus longus demonstrated mild denervation residuals in the peroneal distribution. Generalized axonopathic changes were noted.

Case Report

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GBS = Guillain-Barré syndrome; CIP = critical illness polyneuropathy
NCS = nerve conduction studies

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