Liotta et al successfully bypassed from the ascending aorta to the infrarenal abdominal aorta and resected the infected pseudoaneurysm following surgery for coarctation of the aorta. In 1975, Cooley and Norman described a technique of bypass from the ascending aorta to the supraceliac abdominal aorta as a method of aortic reconstruction for recurrent coarctation. In 1977, Wukasch and Cooley reported the practical use of this technique in nine patients; however, these cases mostly consisted of recurrent coarctation and coarctation with associated anomalies.

We have chosen ascending aorta-supraceliac abdominal aorta bypass as a method for revascularization. The advantages of this procedure are as follows: (1) the procedure for bypass through the heterotopic root is free from infection; (2) proximal anastomosis can be done easily; (3) the supraceliac abdominal aorta offers an adequate length for distal anastomosis; and (4) this procedure requires a shorter graft.

REFERENCES

Sudden withdrawal of oral therapy with hydralazine for reduction of afterload in a patient precipitated severe congestive heart failure. Signs of metabolic encephalopathy evolved due to low cardiac output. Reinstitution of therapy with hydralazine resulted in prompt improvement in cardiac and neurologic status. This case underscores the need for careful follow-up of such patients and argues against sudden withdrawal of vasodilator therapy.

In recent years, vasodilator drugs have been shown to be beneficial in treating patients with heart failure.1-3 The orally administered vasodilator agent, hydralazine, which relaxes peripheral arterioles, relieves the signs and symptoms of circulatory congestion and low cardiac output in certain patients.4-5 The results of long-term therapy with hydralazine in patients with heart failure are not yet available. The status of patients from whom therapy with hydralazine is withdrawn after initial evidence of improvement is not known. We report the findings in a patient in whom sudden withdrawal of therapy with hydralazine resulted in acute, severe heart failure with attendant neurologic dysfunction.

CASE REPORT

A 71-year-old man was diagnosed as having severe aortic insufficiency and congestive heart failure. The patient was placed on therapy with digoxin and furosemide. Over the next 36 years, he had several hospitalizations for shortness of breath, orthopnea, paroxysmal nocturnal dyspnea, and peripheral edema, which responded to increasingly higher doses of digoxin and diuretic drugs.

The patient was referred for further cardiac evaluation. Examination revealed a dyspneic elderly man with heart rate of 76 beats per minute and blood pressure of 110/70 mm Hg. A 10 cm jugular venous distension was present above the sternal angle. Bisbasilar pulmonary rales were audible. Cardiac examination revealed cardiomegaly, a loud pulmonic closure sound, S3 and S4 gallop rhythms, a grade 2/6 systolic ejection murmur, and a diastolic murmur along the left sternal border. The span of the liver was 12 cm. Peripheral edema was also present. A chest x-ray film displayed massive cardiomegaly and interstitial pulmonary congestion. An electrocardiogram demonstrated left ventricular hypertrophy.

Cardiac catheterization revealed high right ventricular (55/12 mm Hg), pulmonary arterial (55/30 mm Hg) and pulmonary capillary wedge (27 mm Hg) pressures. The aortic pressure was 112/70 mm Hg. Left ventriculographic studies showed a markedly enlarged and diffusely hypokinetic left ventricle. The ejection fraction was 10 percent. Moderately severe aortic insufficiency was observed on aortographic studies of the ascending aorta.

The patient was considered a high-risk candidate for surgical correction because of poor left ventricular function. Vasodilator therapy with intravenously administered sodium nitroprusside produced an improvement in left ventricular function. The patient was then given a trial with oral therapy with hydralazine. He had a marked increase in cardiac output, a decrease in pulmonary capillary wedge pressure, and a fall in resistances in the systemic and pulmonary vascular beds without major changes in heart rate and blood pressure (Table 1). After 72 hours of oral therapy with hydralazine, the patient reported a marked diminution in complaints of fatigue and orthopnea. The intensity of the S3 and S4 gallop rhythm and the murmur of aortic insufficiency decreased.

Precipitation of Heart Failure following Sudden Withdrawal of Hydralazine*

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Table 1—Effect of Therapy with Nitroprusside and Hydralazine on Cardiac Hemodynamics

<table>
<thead>
<tr>
<th>Data</th>
<th>Control</th>
<th>Nitroprusside</th>
<th>Hydralazine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate, beats per min</td>
<td>78</td>
<td>68</td>
<td>75</td>
</tr>
<tr>
<td>Mean arterial pressure, mm Hg</td>
<td>85</td>
<td>78</td>
<td>77</td>
</tr>
<tr>
<td>Mean pulmonary arterial pressure, mm Hg</td>
<td>39</td>
<td>26</td>
<td>31</td>
</tr>
<tr>
<td>Mean pulmonary capillary wedge pressure, mm Hg</td>
<td>27</td>
<td>18</td>
<td>21</td>
</tr>
<tr>
<td>Cardiac output, L/min</td>
<td>3.1</td>
<td>4.4</td>
<td>4.1</td>
</tr>
<tr>
<td>Systemic vascular resistance, dyne-sec-cm⁻²</td>
<td>2,194</td>
<td>1,418</td>
<td>1,502</td>
</tr>
<tr>
<td>Pulmonary vascular resistance, dyne-sec-cm⁻²</td>
<td>310</td>
<td>145</td>
<td>195</td>
</tr>
</tbody>
</table>

The patient was discharged on therapy with hydralazine (50 mg every six hours), furosemide (80 mg daily), and digoxin (0.125 mg daily). For several months, he did well, reporting a dramatic increase in exercise tolerance, along with continued resolution of symptoms. Two weeks prior to the present admission, the patient had experienced two brief episodes of dizziness, and his local physician had advised him to take only digoxin and furosemide, but to discontinue hydralazine, despite the absence of hypotension or orthostatic changes. Within two days, the patient became psychotic, with delusions and hallucinations. He became extremely short of breath on walking a few steps, and orthopnea and paroxysmal nocturnal dyspnea returned.

Upon readmission to the hospital, the patient was found to be disoriented. Physical examination revealed a heart rate of 80 beats per minute, blood pressure of 110/70 mm Hg, jugular venous distention to 13 cm, recurrence of bibasilar rales, S₃ and S₄ gallop rhythm, and marked peripheral edema. No focal neurologic signs were present. Serum levels of electrolytes, calcium, magnesium, and glucose were normal. The blood urea nitrogen level (BUN) was 40 mg/100 ml, and the level of creatinine was 2.9 mg/100 ml. The level of digoxin in the serum was 1.0 ng/ml. An electroencephalogram (EEG) demonstrated diffuse slowing and low voltage compatible with metabolic encephalopathy. The EEG was unchanged. The chest x-ray film revealed severe pulmonary congestion but no segmental defect suggestive of infection or pulmonary emboli. Serum levels of enzymes (creatinine phosphokinase, lactic dehydrogenase, and glutamic-oxaloacetic transaminase) were within normal limits for three consecutive days.

The patient was maintained on therapy with digoxin and furosemide, without clinical improvement. Because of adequate levels of digitalis in the serum and clinical evidence of low cardiac output, a nasogastric tube was inserted, and therapy with hydralazine was resumed. By the fifth day of hospitalization, the patient was again well oriented. The heart rate was 75 beats per minute, and the blood pressure was 108/66 mm Hg, with no orthostatic changes. A repeat EEG showed resolution of the diffuse slowing and low voltage present on the earlier record. The BUN decreased to 22 mg/100 ml and the creatinine level to 2.1 mg/100 ml. At the time of discharge, the patient was able to walk several hundred yards without dyspnea, fatigue, or dizziness.

**DISCUSSION**

Reduction of systemic vascular resistance has been shown to be beneficial in patients with a low cardiac output associated with cardiomyopathy and regurgitant valvar lesions. Patients with chronic heart failure, who initially show hemodynamic improvement with vasodilator therapy, require long-term therapy with these agents for maintenance of improved cardiac function.

This case demonstrates the potential dangers of sudden withdrawal of therapy with the vasodilator agent, hydralazine, in patients with severe depression of cardiac output. Oral therapy with hydralazine had been shown earlier to be effective in improving left ventricular function in this patient without a marked change in arterial pressure. Sudden withdrawal of the drug promptly resulted in symptoms and signs of congestive heart failure and metabolic encephalopathy. No clinical or laboratory evidence of pulmonary embolism, infection, or myocardial infarction was present to account for the depression of myocardial function. All other medications, including digoxin and furosemide, had been continued in therapeutic doses. Although precise measurements of cardiac output and other variables of left ventricular function were not made in this patient, it seems reasonable to postulate that withdrawal of therapy with hydralazine resulted in an increase in systemic vascular resistance and secondary deterioration in left ventricular function. The rapid resolution of the patient's problems with reinstitution of therapy with hydralazine further suggests that withdrawal of such therapy was indeed responsible for the precipitation of heart failure.

Usually a vasodilator agent would be expected to result in a decline in arterial pressure and reflex tachycardia; however, a significant change in arterial pressure is seldom observed in patients with heart failure treated with vasodilator agents. Probably, an increase in cardiac output maintains blood pressure and heart rate at the levels before therapy.

This patient has now been observed for nine months while receiving therapy consisting of hydralazine, digoxin, and furosemide and continues to do well. This report stresses the need for close follow-up of patients receiving vasodilator agents in order to evaluate the patient’s compliance and the continued success of therapy.

**REFERENCES**