response differed from the patient group reported by Martinet and coworkers who found in vitro suppression of T lymphocytes but no apparent patient improvement or in vivo suppression of T lymphocyte obtained by bronchoalveolar lavage. This lack of pulmonary physiologic response to therapy may not reflect the actual clinical state of the patient as the radiologic and physiologic assessment of sarcoid alveolitis may not accurately reflect the overall situation. The suppression of animal hemopoietic cells in vitro but not in vivo has been reported and may be due to cytotoxicity as well as to the dosage of cyclosporine used in the in vitro experiments. The response of sarcoidosis to medical therapy is currently not measured by any specific monitoring technique but by clinical responses, and the improvement of the skin sarcoid is certainly an indication of a therapeutic response.

We are of the opinion that selected patients with sarcoidosis may benefit from therapy with cyclosporine. Clearly patients with insulin-dependent diabetes mellitus or with corticosteroid-induced diabetes mellitus may be unsuitable for cyclosporine therapy because of unstable glucose metabolism stemming from abdominal side effects with anorexia or from the direct effects of cyclosporine. The drug may be of benefit in skin sarcoidosis or in progressive sarcoid resistant to conventional therapy. The appropriate indications, the duration, and the dosage of the drug for optimal treatment have yet to be determined and further trials are necessary to determine the place for this drug in the therapy of chronic sarcoidosis.

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Orthostatic Hypotension following Right Ventricular Myocardial Infarction Corrected with Mineralocorticoid Therapy*

Philip E. Hill, M.D., F.C.C.P.; Teresa J. Mason, M.D.; Richard Provo, M.D.; and William A. Neill, M.D.

Severe hypotension while standing became a problem in a patient after discharge from the hospital following right ventricular myocardial infarction. Hemodynamic studies showed that right ventricular systolic function did not maintain adequate left ventricular preload and that the patient did not compensate for cardiac dysfunction by increasing blood volume. Volume expansion by mineralocorticoid therapy corrected the orthostatic hypotension and ameliorated symptoms. Hypotension eventually resolved and therapy was stopped four months after the myocardial infarction.

(Chest 1990; 98:1029-32)

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ight ventricular myocardial infarction (RVMI) occurs as a complication in some patients who have infarction of the inferior or posterior region of the left ventricle due to occlusion of the right coronary artery. The acute hemodynamic abnormalities found in RVMI mimic restrictive and constrictive pericardial disorders, and the need for volume administration is well documented.

We report a case of RVMI in a patient in whom volume administration, pacing, intra-aortic balloon counterpulsation, and pressor agents succeeded initially in supporting the patient. After hospital discharge, however, the patient had severe symptomatic orthostatic hypotension. This clinical problem, which could not be explained as an adverse reaction to medication, responded to mineralocorticoid therapy. This case illustrates

*From the Cardiology Department, MacNeal Hospital, Berwyn, Ill, and the Section of Cardiology, Department of Medicine, Rush-Presbyterian-St. Luke's Medical Center, Chicago.
†Assistant Professor of Medicine.
‡Professor of Medicine.
Reprint requests: Dr. Hill, University of Virginia Health Sciences Center, Box 158, Charlottesville 22908

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the ability of mineralocorticoid therapy to correct low cardiac output and orthostatic hypotension as a late sequela of right ventricular infarction.

**CASE REPORT**

A 52-year-old man was admitted to the hospital with shock and bradycardia two hours following the onset of chest pain. The initial arterial systolic pressure was 40 mm Hg with a heart rate of 30 bpm. The patient was semicomatose, jugular veins were markedly distended, and heart tones were difficult to hear. An initial ECG showed junctional rhythm at 42 bpm, absence of P waves, and acute inferior injury pattern. ST elevation in lead 3 was greater than in lead 2 suggesting RVMI. Within the first two hours of hospital admission dopamine treatment was started, and a temporary transvenous pacemaker, Swan-Ganz pulmonary artery catheter, and intra-aortic balloon counterpulsation catheter (IABP) were inserted. Endotracheal intubation and mechanical ventilation were initiated. With dopamine and the IABP, the initial mean right atrial pressure (RAP) was 20 mm Hg, right ventricular pressure (RVP) was 30/15 mm Hg, pulmonary artery pressure (PAP) was 30/15 mm Hg, mean pulmonary capillary wedge pressure (PCWP) was 15 to 18 mm Hg, and cardiac index (CI) was 1.2 L/min/m² (thermodilution). A prominent V wave suggesting tricuspid regurgitation was not noted on the RAP. The total creatine kinase value peaked at 5,588 IU/L with 16 percent MB fraction. The patient received 11 L of 0.9 percent sodium chloride over the next 48 hours.

A gated nuclear ventriculogram was obtained on the third hospital day and demonstrated a left ventricular ejection fraction of 0.48 with inferoapical and posterolateral hypokinesis and a first pass right ventricular ejection fraction of 0.31 with diffuse hypokinesis (Fig 1). An echocardiogram revealed no pericardial effusion. At the end of seven days the CI increased to 2.5 L/min/m² (thermodilution), normal sinus rhythm returned, and the RAP fell to 8 mm Hg while the PCWP was 12 mm Hg. Cardiac catheterization showed complete occlusion of a large dominant right coronary artery and mild occlusive disease of the left coronary system. The patient was discharged home on the 21st day.

Two weeks after hospital discharge the patient complained of lightheadedness on standing. Supine systolic brachial artery pressure was 90 mm Hg and standing it was 40 mm Hg. He was receiving no medication. Plasma cortisol level was 13.3 μg/dl at 9:45 AM (normal, 4 to 25 μg/dl) and increased to 31.7 μg/dl at 10:45 AM one hour after receiving 250 μg of cosyntropin (Cortrosyn) 1M (ACTH) (normal 2x baseline). One week later the patient remained asymptomatic and was hospitalized for further study.

**SPECIAL STUDY AND RESULTS**

After an overnight fast the right brachial artery and pulmonary artery were cannulated. Measurements were made with the patient in the supine and standing position (1) in the baseline state, (2) after receiving 1 L of 0.9 percent sodium chloride in one hour, and (3) after receiving a second liter of 0.9 percent sodium chloride over the next hour. Care was taken to position the transducer at the same midaxillary position during all pressure measurements. The results are shown in Table 1. The baseline data with the patient supine showed normal arterial blood pressure, low left heart filling pressure (PCWP), and low CI. When the patient stood upright, filling pressures (PCWP and RAP), arterial pressure, and CI fell. Intravenous administration of 1 L of saline solution restored left ventricular cardiac filling pressure (PCWP) to normal, raised CI, and essentially abolished the orthostatic hemodynamic effects. Cardiac index was not restored completely to normal, however, even when the filling pressure in the left ventricle was further increased by administration of another liter of saline solution. When supine (Fig 2), the relationship of left and right heart filling pressures (PCWP and RAP) at baseline and after volume expansion was normal, ie, both filling pressures increased

**Table 1 — Hemodynamic Parameters Supine and Standing before and after Intravenous Saline Solution Administration**

<table>
<thead>
<tr>
<th></th>
<th>t0 Baseline</th>
<th>t1h +1 L 0.9 percent NaCl</th>
<th>t2h +2 L 0.9 percent NaCl</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Supine</td>
<td>Standing</td>
<td>Supine</td>
</tr>
<tr>
<td>Heart rate, bpm</td>
<td>74</td>
<td>96</td>
<td>75</td>
</tr>
<tr>
<td>Rad art BP, mm Hg</td>
<td>118/68</td>
<td>83/44</td>
<td>144/67</td>
</tr>
<tr>
<td>Mean rad art BP, mm Hg</td>
<td>85</td>
<td>56</td>
<td>93</td>
</tr>
<tr>
<td>Mean RAP, mm Hg</td>
<td>0</td>
<td>−4</td>
<td>3</td>
</tr>
<tr>
<td>Mean PCWP, mm Hg</td>
<td>2</td>
<td>−3</td>
<td>9</td>
</tr>
<tr>
<td>CI, L/min/m²</td>
<td>2.1</td>
<td>1.7</td>
<td>2.4</td>
</tr>
<tr>
<td>Plasma volume, mL/kg</td>
<td>43</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total blood volume, mL/kg</td>
<td>61</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*bpm = beats per minute; rad art BP = radial arterial blood pressure; RAP = right atrial pressure; PCWP = pulmonary capillary wedge pressure; and CI = cardiac index. Normal values: mean radial artery blood pressure, 70 to 105 mm Hg; mean RAP, −1 to 5 mm Hg; mean PCWP, 6 to 15 mm Hg; CI, 2.8 to 4.2 L/min/m²; plasma volume, 35 to 45 mL/kg; and total blood volume, 60 to 80 mL/kg.*
Supine PCWP & RAP
Before and after saline administration

and left heart filling pressure remained greater than right heart filling pressure (PCWP>RAP). With standing (Fig 3), the relationship between PCWP and PAP became abnormal. The left heart filling pressure (PCWP) remained low despite a normal right heart filling pressure (RAP) suggesting that the right ventricular systolic function could not maintain left ventricular preload. After administration of 2 L of saline solution the right heart filling pressure was greater than the left heart filling pressure (RAP>PCWP).

THERAPY
These results showed that the patient's orthostatic hypotension could be ameliorated acutely by intravascular volume expansion above the volume level that he achieved as an ambulatory patient with unrestricted fluid and salt intake and without diuretic or vasoactive drug treatment. To simulate the favorable effects of acute volume expansion the patient was started on a regimen of fludrocortisone acetate (Florinef) 0.1 mg daily increasing the dose to 0.3 mg daily at the end of two weeks. The patient became asymptomatic and the orthostatic hypotension disappeared. After another two weeks the mineralocorticoid therapy was stopped and the orthostatic hypotension and symptoms returned. The mineralocorticoid therapy was again started and continued for another two months with beneficial results within one week. Treatment with the medication was stopped four months after his RVMI, orthostatic hypotension was not observed, and the patient remained asymptomatic. Three years after his myocardial infarction the patient is asymptomatic.

DISCUSSION
A common cause of orthostatic hypotension is hypovolemia usually secondary to diuretic administration. This patient, however, was not receiving diuretics and total blood and plasma volume were normal. Autonomic insufficiency (idiopathic orthostatic hypotension, Shy-Drager syndrome) also needs to be considered, but this is progressive, and our patient eventually recovered. Hypoadrenal function was ruled out with normal plasma cortisol levels and a normal response to ACTH. Orthostatic hypotension is seen in severe left ventricular systolic dysfunction, but our patient had only mild left ventricular dysfunction. On the other hand, the right ventricle had a reduced ejection fraction and could not maintain normal left ventricular filling pressures. Right ventricular incompetence was best demonstrated in the upright position after the patient had received 2 L of saline solution and the RAP was greater than the PCWP (Fig 3).

Cohn et al first described the acute hemodynamic abnormality (a high RAP equal to or greater than PCWP) in RVMI and demonstrated that the patients improve with volume expansion. Dell'Italia et al showed that the hemodynamic abnormality (RAP>PCWP) could be elicited with volume expansion in patients without cardiogenic shock and that right ventricular systolic function improved with sufficient time, at least two months after RVMI. Goldstein et al produced RVMI in dogs and demonstrated that pericardiectomy improved cardiac output. Their study suggested that systolic and diastolic dysfunction were important acutely. Our patient had a normal right ventricular filling pressure even after volume loading; therefore, his low cardiac output could not be attributed to noncompliance of the right side of the heart.

We believe that low cardiac output was the cause of orthostatic hypotension in our patient. Poor systolic function of the right ventricle was one aspect contributing to the low cardiac output but it was not the only factor. The other important aspect was the patient's failure to make the normal compensatory adjustment to cardiac dysfunction, ie, expanded blood volume. When blood volume was expanded by saline solution infusion, cardiac output increased and postural hypotension resolved. When blood volume was expanded in the long term by mineralocorticoid administration, his symptoms subsided. A low left ventricular preload associated with hypotension in a patient with recent myocardial infarction may be a sign of a slowly recovering right ventricular infarction, and the use of mineralocorticoid therapy is a logical approach to its correction.

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Resolution of the Adult Respiratory Distress Syndrome following Colectomy and Liver Transplantation

Mohamed Ali, M.D.; and William J. Wall, M.D.

A 32-year-old woman with liver failure from end-stage cirrhosis and ulcerative colitis developed septicemia and severe ARDS. Subtotal colectomy and a successful liver transplantation resulted in complete resolution of the ARDS.

(Chest 1990; 98:1032-34)

ALT = alanine aminotransferase; ESLF = end-stage liver failure

When ARDS occurs as a complicating feature of liver failure from chronic liver disease, the associated mortality rate is reported as 100 percent. We report a case of ARDS in a patient with bacterial sepsis in whom removal of the septic source along with liver transplantation completely reversed the respiratory failure.

CASE REPORT

A 32-year-old woman was admitted to University Hospital for possible liver transplantation. She had a 17-year history of biopsy-proven chronic active hepatitis. The hepatitis progressed to cirrhosis with portal hypertension manifested by esophageal varices, uncontrolled ascites and splenomegaly. Four years prior to this admission she developed bloody diarrhea and was diagnosed as having ulcerative colitis. This was relatively quiescent until a month before admission. At that time bloody diarrhea recurred and became progressively worse. Liver disease was advanced and began to deteriorate. The serum bilirubin value was 663 μmol/L, the serum albumin was 13 g/L and the prothrombin time was 16.5 s (control = 13.5) with no response to vitamin K. The liver function tests were ALT, 93 IU/L; AST, 86 IU/L and alkaline phosphatase, 360 IU/L, respectively. Encephalopathy had worsened. Past history included pulmonary tuberculosis that had been adequately treated.

A physical examination at the time of admission revealed an encephalopathic, malnourished icteric female weighing only 47 kg. Skin appeared dry and jugular venous pressure was zero. There was a right pleural effusion. Ascites and splenomegally were pronounced and the liver was small. There was fresh blood in the rectum. The patient was anemic (Hb, 67 g/L) and appeared hypovolemic. Urine output was less than 10 mL/h. The serum urea value was 21 mmol/L and the creatinine was 122 μmol/L. She was given blood and albumin with an improvement in the urine output.

Endoscopy revealed esophageal varices and mild gastritis. Sigmoidoscopy revealed severe colitis with large ulcers and a colonic biopsy showed inflammatory bowel disease. She was treated with methylprednisolone (10 mg/day), salicylate and hydrocortisone (200 mg/day) enemas.

Over the next three days radiographic and clinical evidence of pulmonary edema developed. Diuresis, fluid restriction and repeated thoracentesis to achieve a daily negative fluid balance were unsuccessful in resolving the pulmonary edema and she required admission to the ICU with intubation and positive pressure ventilation (Fig 1). Arterial blood gas values prior to intubation on a Fio2 of 0.50 were PaO2, 35 mm Hg; PaCO2, 35 mm Hg; pH, 7.45; and HCO3, 24 mEq/L. The ECG was normal. Wedge pressure was 12 mm Hg, cardiac index was 4.3 L/min/m2 and calculated systemic vascular resistance index was low at 983 dynessec cm−1 m−2 (normal, 1,680 to 2,580). At the same time, blood cultures taken three days before were reported to be growing Bacteroides. Sputum and pleural fluid cultures were negative. Abdominal and pelvic ultrasound showed ascites but no abscesses. A diagnosis of ARDS secondary to sepsis in a patient with end-stage liver disease was made. The lung injury score of 2.3 indicated moderate to severe ARDS (Fig 2). Because of the sepsis, liver transplantation was postponed.

Her immediate ICU course was complicated by a pneumothorax from high PEEP (18 cm H2O), recurrent colonic bleeding, coaugulopathy and herpes labialis. She was aggressively supported with ventilation; imipenem-cilastatin, 1 g every 6 h; acyclovir, 200 mg every 12 h; total parenteral nutrition and meticulous nursing care.

Over the next 19 days her pulmonary status improved somewhat (Fio2, 0.55, and PEEP, 13 cm H2O) and blood cultures became negative (Fig 2). At this point, orthotopic liver transplantation and subtotal colectomy with ileostomy and mucous fistula was performed. Intraoperative findings included a small cirrhotic liver, a large spleen and massive ascites. No abscesses was discovered and the external appearance of the bowel was normal. Postoperatively,