Beriberi Heart in Iowa Veterans

By Charles H. Gutenkauf, M.D.

Beriberi heart disease does not constitute a rigid syndrome because the severity of cardiac failure, associated peripheral neuritis, other concomitant signs and symptoms, and response to treatment are variable. Five patients with peripheral neuritis, edema and evidence of cardiac failure are presented. All improved after treatment, and, in two instances, there was marked reduction in cardiac size. Beriberi was thought to be the most likely diagnosis in each case, since there was no evidence of other etiology.

This communication deals with 5 cases of beriberi heart disease recognized at the Des Moines Veterans Hospital among 7,912 admissions between October, 1948 and January, 1950. None of these Iowa residents was a prisoner of the Japanese. Beriberi was considered a disease of the Orient until about 25 years ago. It has been regularly reported in the southern and eastern portion of the United States, but only once (to my knowledge) in the “grain belt” regions.

All 5 cases conform for the most part to Blankenhorn’s diagnostic criteria: (1) enlarged heart with sinus rhythm; (2) dependent edema; (3) elevated venous pressure; (4) peripheral neuritis or pellagra; (5) nonspecific changes in the electrocardiogram; (6) no other cause evident; (7) gross deficiency of diet of three months or more; and (8) improvement and reduction of heart size after specific treatment or autopsy findings consistent with beriberi (see tables 1 and 2).

Case Reports

Case 1. W. B., age 34, was admitted Oct. 23, 1948 with a three year history of taking 10 to 13 ounces of whiskey daily, eating very little and gradually losing 25 pounds of weight. Two months before entry he fell while walking and began to have weakness, exertional dyspnea, cough and call pain. Ankle edema appeared three weeks before admission.

Examination revealed a hyperkinetic, perspiring, mildly dyspneic man. Temperature was 99 F., pulse 104, respirations 24, and blood pressure 120/70. The lungs were clear. The heart was enlarged 2 cm. beyond the left midclavicular line. The apex beat was forceful, and there was a faint pulmonic diastolic murmur. The liver was palpable 3 cm. below the right costal margin, and there was marked pitting edema of the lower extremities. There was definite weakness of the extremities with inability to rise from a squat. The knee and ankle jerks were absent and the calves were tender on pressure.

Laboratory findings (see table 3): The spinal fluid contained 52.5 mg. of protein per 100 cc. Liver function tests were indicative of hepatocellular damage. Basal metabolic rate was minus 4 per cent on October 28.

Chest roentgenograms revealed the cardiothoracic ratio to be 15.25:30 cm., with prominent hilar markings and blunting of the right costophrenic sinus. Electrocardiogram was normal. The arm to tongue circulation time on November 2 was 21 seconds (Macasol), and the venous pressure was 24 cm.

Hospital Course. He was given 2 cc. Salicylan intramuscularly on admission. After six days on a low sodium diet (200 mg. per day), only a trace of ankle edema remained and his weight was constant. At this time administration of thiamine was begun in daily doses of 20 mg. intramuscularly and 20 mg. orally, in addition to administration of choline and other vitamins by mouth and a high protein diet. A neurologist confirmed the diagnosis of B deficiency neuritis, for which physiotherapy was given. The murmur was absent on the second day. Electrocardiogram on Dec. 17, 1948 showed that the previously inverted T wave in lead III was now upright. Clinical improvement continued until discharge on Dec. 17, 1948.

He was readmitted for nervousness on June 15, 1949. There was no evidence of cardiac decompensation. The blood pressure was 104/86, and the reflexes had returned. The cardiothoracic ratio was 12.7:30 cm. The electrocardiogram was normal. The liver had decreased in size, and there was no bromsulfalein retention.

Comment. This patient had a mild degree of cardiac enlargement and neuritis (mixed type), and probably a mild cirrhosis.
### TABLE 1.—Pertinent Data on Five Cases of Beriberi with Cardiovascular Manifestations

<table>
<thead>
<tr>
<th>Case</th>
<th>Initials</th>
<th>Age</th>
<th>Sex</th>
<th>Duration of Dietary Deficiency</th>
<th>Enlarged Heart</th>
<th>Venous Pressure</th>
<th>Edema</th>
<th>Electrocardiogram</th>
<th>Other Signs of Deficiency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Case 1</td>
<td>W. B.</td>
<td>3 yrs.</td>
<td>M</td>
<td>3 yrs.</td>
<td>+</td>
<td>24 cm.*</td>
<td>+++</td>
<td>Inverted $T_1$</td>
<td>Polynearitis, moderate</td>
</tr>
<tr>
<td>Case 2</td>
<td>R. S.</td>
<td>Mild—4 yrs.</td>
<td>Severe—8 months</td>
<td>4 yrs.</td>
<td>?</td>
<td>+++</td>
<td>Low voltage</td>
<td>Polynearitis, severe. Scurvy</td>
<td></td>
</tr>
<tr>
<td>Case 3</td>
<td>J. S.</td>
<td>4 yrs.</td>
<td>M</td>
<td>Cervical veins distended</td>
<td>+</td>
<td>Normal</td>
<td>Polynearitis, mild</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Case 4</td>
<td>D. W.</td>
<td>6 yrs.</td>
<td>M</td>
<td>Cervical veins distended</td>
<td>+++</td>
<td>+</td>
<td>Inverted $T_1$ and $CF_4$ Low Voltage</td>
<td>Polynearitis, severe</td>
<td></td>
</tr>
</tbody>
</table>

* Third hospital day.
† Sixth hospital day.
+ Slight, ++ Moderate, +++ Marked.

### TABLE 2.—Response to Therapy in This Series

<table>
<thead>
<tr>
<th>Case, Initials, Age, Sex</th>
<th>Treatment</th>
<th>Loss of Edema</th>
<th>Decrease in Heart Size†</th>
<th>ECG Changes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Case 1</td>
<td>Thiamine. NaCl 250 mg./day for 6 days. Mercuhydrin</td>
<td>6 days* (Not weighed on admission)</td>
<td>Clinical in 10 days, 2.5 cm. 8 months later.</td>
<td>$T_4$ upright in two months. Normal</td>
</tr>
<tr>
<td>Case 2</td>
<td>Thiamine. High protein diet</td>
<td>8 days* (Not weighed for 2 months)</td>
<td>? (Bedside films)</td>
<td>Increased voltage, $T$ remained diphasic after 6 months treatment</td>
</tr>
<tr>
<td>Case 3</td>
<td>Thiamine. NaCl 2 Gm./day</td>
<td>6 days.* 17 lbs. from 4th to 10th day</td>
<td>Clinically in 20 days.</td>
<td>Not repeated (left against advice)</td>
</tr>
<tr>
<td>Case 4</td>
<td>Thiamine. Mercuhydrin digitalis NaCl 2 Gm./day</td>
<td>4 days.* 20.5 lbs. in 8 days</td>
<td>5.5 cm. in 2 months</td>
<td>Inc. voltage. Normal in 3 months$§$</td>
</tr>
<tr>
<td>Case 5</td>
<td>Thiamine. NaCl 2 Gm./day</td>
<td>2 days.* 36 lbs. in 7 days</td>
<td>4 cm. in 4 weeks</td>
<td>Normal in 3 weeks</td>
</tr>
</tbody>
</table>

* Disappearance of pitting edema.
† As determined by repeated teleroentgenograms, except in cases 2 and 3.
† Pneumoperitoneum given.
§ See comments on fig. 3.
## Table 3.—Pertinent Laboratory Data

<table>
<thead>
<tr>
<th>Case</th>
<th>Erythrocyte Sedimentation Rate</th>
<th>Bromsulfalein retention at 45 minutes</th>
<th>E.S.R. Cutler 45 hrs.</th>
<th>C.C.F. 24 hrs. 46 hrs.</th>
<th>Serum Proteins</th>
<th>Total Albumin Globulin</th>
<th>Chol. Esters</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>S.W. cm.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Case 1, W. B.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10/23/49</td>
<td>4.85</td>
<td>14.0</td>
<td>9 mm./hr</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>6.06</td>
</tr>
<tr>
<td>10/28/49</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>22%</td>
<td>—</td>
<td>—</td>
<td>4.60</td>
</tr>
<tr>
<td>11/18/49</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>2+</td>
<td>—</td>
<td>—</td>
<td>1.46</td>
</tr>
<tr>
<td>12/1/49</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>2%</td>
<td>3.9</td>
<td>4.8</td>
<td>2+</td>
</tr>
<tr>
<td>Case 2, R. S.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6/24/49</td>
<td>3.69</td>
<td>11.0</td>
<td>38 mm./hr</td>
<td>—</td>
<td>neg.</td>
<td>neg.</td>
<td>1.5</td>
</tr>
<tr>
<td>8/5/49</td>
<td>4.01</td>
<td>12.5</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>5.05</td>
</tr>
<tr>
<td>Case 3, J. S.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6/24/49</td>
<td>3.40</td>
<td>11.5</td>
<td>24 mm./hr</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>2+</td>
</tr>
<tr>
<td>6/27/49</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>8.8%</td>
<td>neg.</td>
<td>2.0</td>
<td>8.71</td>
</tr>
<tr>
<td>6/30/49</td>
<td>5.74</td>
<td>16.0</td>
<td>12 mm./hr</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>5.80</td>
</tr>
<tr>
<td>7/6/49</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>5.2%</td>
<td>—</td>
<td>—</td>
<td>2.91</td>
</tr>
<tr>
<td>Case 4, D. W.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7/5/49</td>
<td>3.10</td>
<td>9.5</td>
<td>17 mm./hr</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>8.8%</td>
</tr>
<tr>
<td>7/8/49</td>
<td>4.65</td>
<td>14 Gm.</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Case 5, L. L.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1/9/50</td>
<td>4.33</td>
<td>12.0</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>neg.</td>
</tr>
<tr>
<td>1/12/50</td>
<td>—</td>
<td>—</td>
<td>19 mm./hr</td>
<td>neg.</td>
<td>neg.</td>
<td>1.8</td>
<td>—</td>
</tr>
<tr>
<td>1/13/50</td>
<td>6.09</td>
<td>16.0</td>
<td>18 mm./hr</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>7.0</td>
</tr>
<tr>
<td>1/23/50</td>
<td>—</td>
<td>—</td>
<td>19 mm./hr</td>
<td>12.4%</td>
<td>—</td>
<td>—</td>
<td>3.9</td>
</tr>
<tr>
<td>2/3/50</td>
<td>—</td>
<td>—</td>
<td>14 mm./hr</td>
<td>3.0%</td>
<td>—</td>
<td>—</td>
<td>3.1</td>
</tr>
<tr>
<td>2/13/50</td>
<td>—</td>
<td>—</td>
<td>8 mm./hr</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>2.6</td>
</tr>
</tbody>
</table>

E. S. R. = Erythrocyte Sedimentation Rate.
Bsf. = Bromsulfalein retention at 45 minutes.
C. C. F. = Cephalin-Cholesterol Flocculation.
T. T. = Thymol Turbidity.
Chol. = Blood Cholesterol in mg./100 cc.

Case 2, R. S., age 39, a bartender, was admitted May 27, 1949. He had been drinking from 4 to 20 glasses of beer daily and eating poorly for four years. For eight months he had consumed one-half pint of whiskey, one-half bottle of wine, and thirty cans of beer daily, and had eaten little. Eighteen months before entry he noted weakness of the legs. Sudden syncope occurred six months before admission and

---

The text continues with additional details about the patients' medical history and laboratory findings.
a productive cough appeared. For several weeks he had experienced nausea, diarrhea, back and leg pains, dyspnea, ankle edema and weakness. Abdominal soreness and "numbness" and petechiae on the legs and arms had been present for 10 days.

Examination revealed an apparently well nourished, irritable, sluggish, orthopneic man. The pulse was 120, temperature 101 F., respirations 28, and blood pressure 132/80. The pulse was weak. The heart sounds were muffled, but there was no enlargement or murmur. Moist rales were heard throughout both lungs. A tender liver extended 3 cm. below the right costal margin. There was a moderate amount of ascites and marked edema of the back, scrotum, thighs and legs. An abscess was present on the anterior chest, and there was an ecchymotic area 5 by 8 cm. on the left shoulder. Numerous hyperkeratotic hemorrhagic hair follicles were present on the arms and legs. The gait was dragging, and rising from a chair required great effort. The deep reflexes were absent.

There was a moderate anemia (see table 3), and the sedimentation rate was 38 mm. in one hour (Cutler). The platelet count was 270,000 per cu. mm. Hemolytic Staphylococcus aureus was cultured from the cutaneous abscess. Tubercle bacilli were found in sputum. The spinal fluid protein was 61.5 mg. per 100 cc. Roentgenograms of the chest revealed bilateral infiltrations of far-advanced pulmonary tuberculosis involving less than one-third of each lung field. The electrocardiogram was abnormal with low voltage (fig. 1).

Hospital Course. For 10 days he was dyspneic, weak, and febrile, and was given penicillin and a high calorie, high vitamin diet. After eight days the edema had disappeared and generalized muscular wasting and malnutrition were apparent. The neurologist found muscular tenderness, diminution of all types of sensation in the lower extremities, bilateral foot drop, and absent reflexes, and made the diagnosis of B deficiency neuritis. Gastric analysis showed 100 degrees of free hydrochloric acid.

After June 22, 1949 the patient was given 20 mg. of thiamine intramuscularly daily, large oral doses of vitamins B and C, and one blood transfusion. Two months after admission he had lost 27 pounds. Subsequent roentgenograms showed a normal heart, and no progression of the tuberculosis. Pneumoperitoneum was induced, and physical therapy was prescribed for the neuritis. There was a progressive gain in weight and strength after August, and by October he could shuffle to the bathroom. An electrocardiogram on November 22 was abnormal with diphasic T waves in leads I and CF₄, but there was increased voltage as compared with the previous tracing (see figure 1). He was transferred to a tuberculosis sanatorium on Nov. 22, 1949. Eleven months after admission residual polyneuritis was still present.

Comment. There is no evidence that this patient had cardiac enlargement and the venous pressure was unfortunately not measured. The history, however, together with an advanced degree of typical B deficiency neuritis, associated evidence of scurvy, electrocardiographic changes, edema, orthopnea, hepatic enlargement and response to therapy strongly supports a diagnosis of beriberi heart disease.

Case 3. J. S., age 32, a bartender, was admitted June 21, 1949. Alcoholic intake had been excessive for 15 years. For four years he had been inebriated nearly every day. He ate erratically. An episode of sudden syncope and cyanosis had occurred seven months before entry, followed by exertional dyspnea, shortness of breath, cough, orthopnea, palpitation and ankle edema. For some time he had noted pains in the arms, legs and back, relieved by whiskey. Twelve days prior to admission edema began to increase rapidly.

Examination revealed a flushed, obese, tremulous, perspiring, and dyspneic man. Pulse rate was 110, respirations 22, and blood pressure 178/90. There was slight prominence of the eyes and the tongue showed marginal erythema. The neck veins were distended and rales were heard in the right lung base. The heart was enlarged 3 cm. beyond the left midclavicular line. Faint systolic apical and aortic

![Fig. 1. Case 2. Electrocardiogram on fourth day showing low voltage. After six months there is increased voltage of QRS in leads I and II which may be due to pneumoperitoneum. The diphasic T waves in leads I and CF₄ are thought to represent residuals of beriberi.](http://circ.ahajournals.org/content/32/6/355.full.pdf)
murmurs were noted. Moderate ascites was present, the liver was slightly enlarged, and there was marked edema of the lower extremities, thighs, and buttocks.

Laboratory findings (see table 3): Roentgenogram of the chest revealed a prominent left ventricle, with a cardiothoracic ratio of 16.5:32 cm. and clear lung fields. Electrocardiogram was normal.

Hospital Course. On a low sodium diet he lost twelve pounds from the fourth to the sixth day. At this time he complained of pain and numbness in the lower extremities. Deep muscular tenderness on pressure, particularly in the calves, and hypoactive to absent reflexes were then noted and administration of thiamine (20 mg. intramuscularly and 20 mg. orally) was begun. The murmurs disappeared on the second day.

Ten days after admission he was asymptomatic, free of edema, and had lost seventeen pounds; the heart was normal in size, and carotid sinus irritability could not be demonstrated. On the last three days of his hospital stay blood pressure readings averaged 128/82, and the pulse ranged from 80 to 90. On the twenty-first day he left against advice. Letter follow-up six months later disclosed that he was still drinking, and had fatigue, anorexia and palpitation on exertion.

Case 4. D. W., age 38, laborer, was admitted July 1, 1949. He admitted alcoholism for six years. He ate three candy bars daily. His diet was meager, but only mildly deficient in thiamine. He had had episodes of syncope and faintness for five years. In 1943 and 1945 his blood pressure was slightly elevated. Eight months prior to admission exertional dizziness, dyspnea, and ankle edema began, and a heart murmur was noted. For six months he had numbness and pain in the feet and fingers and muscular soreness. Two months later palpitation and paroxysms of nocturnal dyspnea began. Dyspnea and ankle edema progressed in severity before admission.

Examination revealed a dyspneic, orthopneic and mildly cyanotic man. Pulse rate was 120, respirations 28 and blood pressure 150/80. There was slight cheilosis and the tongue had marginal reddening and papillary atrophy. The neck veins were engorged. There were basilar rales and moderate cardiac enlargement with a systolic gallop and no murmurs. There was mild liver enlargement, ascites, and moderate edema of the lower extremities. The deep reflexes were absent. Muscular tenderness and diminished sensation were present in the extremities.

Laboratory findings (see table 3): Gastric analysis after histamine revealed hypochlorhydria. Electrocardiogram showed borderline QRS voltage, inverted T1, flat T2. Roentgenogram of the chest revealed a cardiothoracic ratio of 16.5:29 cm. (fig. 2, left) and pulmonary congestion. Angiocardiography on the tenth day revealed a normal circulation rate, and a generalized cardiac enlargement, the wall of the left ventricle measuring 2.5 cm. in thickness.

Hospital Course. The patient was digitalized and given morphine and 4 cc. of Mercuhydrin in the first three days. Also, 10 mg. of thiamine was given intramuscularly daily for six days, after which the dose was doubled and large doses of all vitamins were given orally. Edema was absent after five days and by the eighth day he had lost 20 pounds. As the edema subsided the patient complained bitterly of hypersensitivity of the feet and toes and numbness of the extremities. Generalized muscular wasting became evident. The attending neurologist's diagnosis was B deficiency neuritis. Two cc. of crude liver extract were given daily from July 14 to July 26.

Fig. 2. Case 4. Left. Telerontgenogram on 7/5/49. Cardiothoracic ratio 16.5:29 cm. Right, 9/7/49. Cardiothoracic ratio 11.1:29 cm.
There was mental confusion on awakening during the first month, and a "heaviness of the chest" at bedtime, which was unrelieved by nitroglycerine. Electrocardiograms showed progressive inversion of the T wave in leads I and CF, in the first three weeks, suggesting myocardial infarction. The sedimentation rate was 22 mm. in one hour (Cutler) on Aug. 1 and 16 mm. on Aug. 15, 1949. Dicumarol was given from July 26 until Aug. 16. The cardiothoracic ratio decreased from 16.5:29 cm. on July 5 to 15.2:29 cm. on July 15 and 11.1:29 cm. on Sept. 7, 1949 (fig. 2, right). Adhesions were noted above the right hemidiaphragm on Sept. 7, 1949. Digitalis was discontinued on Sept. 26. Electrocardiograms after Oct. 10 were normal (fig. 3). Weakness, numbness and pain on walking persisted until the end of August. The blood pressure on August 22 was 150/86, and the pulse ranged from 80 to 90 at rest. Embryocardia persisted. Angiocardiography on September 2 revealed an unchanged circulation rate, and no change in the thickness of the wall of the left ventricle in spite of the spectacular decrease in heart size.

When discharged on Oct. 25, 1949 the patient still had weakness and minimal dyspnea on climbing stairs. When recalled on June 2, 1950 his appetite had increased and he had no dyspnea. He complained of numbness, tingling, and hypersensitivity of the feet. He appeared hyperkinetic and the tongue and fingers were tremorous. His pulse was 90 and his blood pressure, 128/90. The ankle jerks were absent and there was hypesthesia of the toes. Basal metabolic rates were minus 4 and minus 6 per cent. The cardiothoracic ratio was 11.29 cm. The electrocardiogram was normal (see figure 3).

Case 5. L. L., age 32, a cook, was admitted Jan. 8, 1950. He was disoriented. Relatives stated he had had palpitation and dyspnea for one year, and transient mental confusion for six months. Abdominal distention, nausea, cough and pleuritic chest pain appeared in the last two weeks. He had been drinking heavily for 15 years. For two years he had eaten poorly, and had consumed at least twelve bottles of beer daily, a fifth of whiskey on weekends, and often went three days without food.

Records of Army hospitalization from March 8, 1945 to Feb. 28, 1947 for a malunited fracture of the right femur revealed normal neurologic, cardiovascular, and renal findings.

In September 1948 a routine examination revealed hypesthesia and edema of both feet, absent right knee jerk, pulse 90, blood pressure 170/100 and a normal chest x-ray.

Examination revealed an obese, graying and orthopneic man who fabricated freely. Temperature was 99, pulse 120, respirations 30 and blood pressure 170/100. There were basilar rales and moderate cardiac enlargement with regular rhythm and no murmurs. The abdomen was distended. Bilateral pitting edema extended to the knees, and pigmentation was present on the right pretilial area. Areflexia and calf tenderness were noted. There was diminished sensation and weakness in all extremities.

Laboratory findings (see table 3): Spinal fluid was normal. Histamine gastric analysis two weeks after admission revealed hyperchlorhydria. Oral glucose tolerance curve was suggestive of diabetes.

Electrocardiogram showed flat or absent T waves in all leads. Teleroentgenogram of the chest revealed generalized cardiac enlargement and pulmonary congestion (fig. 4).

Hospital Course. On bed rest, and a diet containing 3 Gm. of sodium chloride, he lost 19 pounds in 48 hours, at which time there was no ankle edema. The liver was then palpable 4 cm. below the right costal margin, pulse was 108, blood pressure 150/100, and he appeared much improved. He had lost 32 pounds by the fourth day and 36 pounds by the seventh day. A daily intramuscular injection containing 210 mg. thiamine, other soluble vitamins, and 2 cc. crude liver extract was given from the fourth to the eleventh day. After the third week he received a high protein diet, large oral doses of all vitamins, 100 mg. thiamine intramuscularly daily, and vitamin B12 15 µg. twice weekly.

Venous pressure on the fifth day was 12 cm. H2O, and circulation time, arm-lung (ether) 5 seconds, and arm-tongue (Decholin), 14 seconds. On the ninth day arm-tongue time was 12 seconds.

Electrocardiograms on the fifth day revealed inversion of T waves in leads I, II, CF4, and CF5. On the eighth day these were upright, and the tracings were normal after three weeks (fig. 5). Teleroentgenograms revealed progressive decrease in cardio-

![Fig. 3. Case 4. Transient T-wave changes for three weeks. Digitalis given for 13 weeks. Normal after 16 weeks.](http://circ.ahajournals.org/)
thoracic ratio from 17.5:31 cm. on admission to 15:31 cm. on the eighth day, and 13.5:31 cm. on the twenty-eighth day (fig. 4). The sedimentation rate five weeks after admission was 8 mm. in one hour.

From the second to fifth week the blood pressure ranged from 140/100 to 130/96, the pulse 100 to 128, with persistent embryocardia. After five weeks the mental status was normal except for mild memory defects. At this time the biceps jerks had returned, but the lower extremities remained areflexic. The patient left against advice on February 18, 1950.

**Fig. 4. Case 5. Left. Teleroentgenogram on 1/8/50. Cardiothoracic ratio 17.5:31 cm. Right. 2/6/50. Cardiothoracic ratio 13.5:31 cm.**

**Fig. 5. Case 5. Transient inversion of T waves with return to normal in three weeks.**

**CLINICAL FEATURES OF CARDIOVASCULAR MANIFESTATIONS OF BERIBERI**

**Etiology.** Clinical and experimental evidence has established that thiamine deficiency is responsible for beriberi. Thiamine is a part of an essential enzyme of carbohydrate metabolism. The diets of the majority of patients who develop beriberi have been low in thiamine but of high caloric value. Diets consisting mostly or entirely of carbohydrates are especially harmful. It is currently estimated that an average young adult requires 0.25 to 0.35 mg. of thiamine per 1000 calories of balanced diet. The importance of the intestinal synthesis of thiamine in the prevention of deficiency states has not been established.

In the Occident, alcoholism and a thiamine-poor diet are jointly responsible for the majority of cases of beriberi.

The dietary history is usually unsatisfactory unless obtained and interpreted by a nutritionist. Digestive disturbances may impair absorption or utilization of food and vitamins. Psychic or other illnesses which limit the choice of foods occasionally may create thiamine deficiency. Overcooking destroys thiamine. Increased metabolism due to thyrotoxicosis, exercise, pregnancy, or infections increases the demand for thiamine in proportion to the added caloric demand. Increased metabolism also increases the heart load. In
48 cases studied by Blankenhorn, alcoholism was the main precipitating factor in 50 per cent, other diseases in 37.5 per cent and no evident cause was found in 12.5 per cent.8

All 5 patients here presented were white men between ages 32 and 39 who consumed excessive amounts of alcohol. The thiamine intake of each (diet and alcohol) had been approximately 0.15 to 0.26 mg. per 1000 calories.

Symptoms and Signs. The symptoms and signs vary with the duration and severity of hypovitaminosis and are modified by associated diseases. One patient in this series had concomitant active pulmonary tuberculosis, which others have reported.5,10 Beriberi has been classified as (1) "dry," or neuritic; (2) "wet," or edematous; and (3) "cardiac," which varies, but may be characterized by severe and fulminating cardiac symptoms.11 Mixed forms of the disease are most frequently seen, and patients may display different forms of the disease at various times.6 Severe polyneuritis is not usually associated with cardiac failure, probably because the neuritis prevents muscular exertion. Mild polyneuritis may permit severe congestive failure to develop rapidly, particularly in patients doing heavy work.5,10 The relationships of cardiac enlargement, edema and polyneuritis in these patients is shown in table 1.

One-third of patients with beriberi show evidence of cardiovascular dysfunction warranting a diagnosis of "beriberi heart."7,12 The 5 patients in this series had dyspnea, orthopnea, edema, hepatomegaly; 4 had elevated venous pressure, and 2 had paroxysmal nocturnal dyspnea. Three patients had palpitation and 3 had syncope prior to admission. Syncope is due to carotid sinus irritability and disappears with thiamine therapy.5 Peripheral edema may be slight or extensive, and may be so evenly distributed, hard, and nondependent that the patient appears robust until he has lost 30 to 40 pounds of fluid.11 Loss of edema is often attended by aggravation of the symptoms of polyneuritis. Tachycardia is nearly always present, and cardiac enlargement is the rule, though occasionally failure occurs in a heart of normal size.5,9 One patient in this series had no evidence of cardiac enlargement and only moderate dyspnea in the presence of marked edema, electrocardiographic changes, and severe polyneuritis. Embryocardiawas frequently noted, and was present in the 2 patients in this series who had marked cardiac enlargement. One of these had gallop rhythm, another common finding. Transient systolic murmurs were heard in 2 of the 5 patients. An overactive heart with a bounding, Corrigan-like pulse, and elevated pulse pressure is found in one-third of patients, but cardiac activity and pulse contour may be normal.10,12 Blankenhorn2 found pistol shot arterial sounds and elevated pulse pressure in only 2 of 9 patients, whereas Gelfand and Bellet3 reported a Corrigan-like pulse in their 10 patients. Of the 5 patients reported here, 1 had a forceful apical impulse but none had a Corrigan pulse. Three had elevation of the systolic blood pressure, which disappeared during convalescence.

Studies of hemodynamics indicate a relatively rapid circulation in a majority of cases.2,9 This has been attributed to generalized arteriolar dilatation which creates a condition similar to multiple arteriovenous fistulas. This view is supported by the findings of rapid circulation time, decreased arteriovenous oxygen difference, and high pulse pressure.5,9,14 Burwell and Dexter14 demonstrated a rapid circulation and a high cardiac output in 1 case by means of cardiac catheterization, and showed a dramatic disappearance of these abnormalities after three weeks of therapy. There are, however, genuine cases in which a slow circulation has been demonstrated. Two of 10 patients in a series reported by Blankenhorn and co-workers12 had markedly prolonged circulation times. This may be due to relatively severe myocardial degeneration with less arteriolar dilatation. In this series, the circulation time was relatively rapid in 1 case, only slightly prolonged in another, and a third had a normal circulation rate on the tenth day.

Symptoms of neuritis consist of aching and numbness of the calves, peripheral muscular weakness, or burning hypersensitive feet and fingers. Typical findings include symmetrical absence of reflexes, calf tenderness, inability to rise from a squat, and patchy or "stocking-and-glove" peripheral sensory loss, particularly
in the lower extremities. Severe cases show extreme muscular wasting. Psychosis is occasionally present. Polyneuritis was found in each patient. One had Korsakoff's psychosis, and 1 with severe neuritis was mildly confused. Symptoms and signs of neuritis became more apparent with loss of edema in 2 patients, and in another case were absent until diuresis occurred.

The appearance of 2 patients suggested hyperthyroidism, but basal metabolic rates were normal. All had anorexia, 3 had abdominal soreness, and 1 had diarrhea in the later stages of his illness. One patient had chilosis, 1 had scurvy, and 2 had mild glossitis.

X-Ray Findings. The heart shadow is usually diffusely enlarged. If irreversible damage has not occurred, the reduction in size during therapy may be striking. In this series 4 patients had cardiac enlargement, which disappeared with therapy. Angiocardiograms in case 4 revealed dilatation and thickening of the wall of the left ventricle, which decreased markedly during two months of therapy.

Electrocardiogram. Electrocardiographic changes are not specific, are often minor, and occasionally are absent.9, 10, 13 Usually there is a sinus rhythm with low voltage of T or QRS or inversion of T waves in one or more limb or precordial leads. Prolongation of the Q-T interval occurs less frequently, and occasionally premature contractions, auricular fibrillation, or bundle branch block is present. The temporary accentuation of T wave changes during therapy, such as occurred in 2 patients, may suggest coronary occlusion.9, 10, 13 Reversion to normal usually occurs within three to four weeks, lagging behind clinical recovery. In this series, 4 patients had flat or inverted T waves in one or more leads. Two had low voltage of the QRS complex. Two had normal tracings, but in one of these the inverted T became upright during therapy. One patient's electrocardiogram became normal in three weeks, and the tracings of 2 patients reverted to normal in less than three months, but the exact time of reversion is unknown. A fourth patient (case 2) had residual abnormalities after six months of treatment.

Laboratory Findings. Hypoproteinemia and anemia are usually found, and evidence of hepatocellular damage is occasionally observed.2, 9, 13 Four patients had moderate anemia, and 2 of 4 patients tested had hypoproteinemia. The milder degrees of anemia disappeared with diuresis. Liver function tests showed only mild bromsulfalein retention in 3 patients. Another had abnormalities suggesting early cirrhosis. One patient had hyperchlorhydria, and 2 had hyperchlorhydria. One had impaired glucose tolerance. Three patients had unexplained mild elevation of the sedimentation rate during the first month. Two of these had x-ray signs suggestive of pneumonitis. The susceptibility of these patients to respiratory infections and the greater likelihood of sudden death in such cases has been noted.8, 9

Diagnosis. Blankenhorn states that patients with slow circulation may closely resemble other forms of degenerative heart disease, but the diagnosis can be made with relative certainty utilizing the combined skills of the nutritionist, cardiologist, and neurologist. The patients in this series conform for the most part to Blankenhorn's criteria (tables 1 and 2). All had evidence of cardiac failure associated with polyneuritis, and no evidence of coronary, rheumatic, syphilitic or thyrotoxic heart disease. Although none of these patients had a control period of thiamine free diet, their response to therapy was typical of beriberi heart disease.

In the differential diagnosis hyperthyroidism, arteriovenous fistula, Fiedler's myocarditis, idiopathic postpartum failure, glomerulonephritis, cirrhosis, lupus erythematosus, scleroderma, periarteritis nodosa and amyloidosis must be considered.

Morbid Anatomy. In chronic thiamine deficiency the heart may be hypertrophied as well as dilated. Wenckebach and others in the Orient observed more right than left heart dilatation.9, 10 This is not the rule.9

Microscopically, vacuolization (hydropic degeneration) of the myocardial fibers, collagenous infiltration, edema, fragmentation, loss of striation, and longitudinal separation of muscle fibers are observed. In severe chronic
cases, considerable interstitial fibrosis is present. These changes may be absent. Degenerative lesions also occur in the nervous system, especially in peripheral nerves.

Treatment. In many cases rest and the usual hospital diet may result in striking improvement within a few hours. Four patients in this series had moderate improvement of signs of congestive failure before parenteral thiamine was given. Specific therapy consists of the parenteral administration of thiamine in doses of 10 to 100 mg. daily. Oral therapy alone may result in relapse. A high-protein diet should be given, with oral and parenteral supplements of all vitamins. Associated pellagra or scurvy require large doses of niacin or ascorbic acid. Excessive and unbalanced administration of vitamins may precipitate other avitaminoses. Digitalis is indicated if the heart is markedly dilated, and mercurial diuretics and moderate sodium restriction are indicated in markedly edematous patients. Thiamine has produced dramatic response when digitalis and mercurials failed. In this series, the time of disappearance of edema and symptoms of congestive failure varied from 7 to 10 days, but 2 patients (excluding the patient with tuberculosis) had persistent tachycardia for several weeks. The time required for maximal decrease in heart size varied from one to two months. The loss of edema may have been accelerated in 4 instances by sodium restriction. Other authors have commented on the variable response to treatment. In general, the neuritis responds much more slowly than the cardiovascular manifestations. In severe chronic cases, even prolonged intensive therapy may result in only slow and incomplete recovery.

Summary and Conclusions

1. Beriberi heart disease occurs in Iowa.
2. Five alcoholic patients with variable amounts of cardiac failure, polyneuritis and cardiac enlargement which conform to the criteria for diagnosis of beriberi heart disease have been presented.
3. Because beriberi is a reversible form of heart disease, as shown by these 5 patients, its early diagnosis is important.

Addendum

Case 2, R. S., was admitted again on Dec. 7, 1950 with minimal cardiac failure, mild neuritis and acute alcoholism. The cardiothoracic ratio was 17:32 cm. as compared to 13.5:32 cm. in Nov. 1949; the tuberculous lesions were unchanged and pneumoperitoneum was absent. Electrocardiogram was now normal. After one week on a thiamine-poor diet there was no change in these findings; the venous pressure was 15 cm. of H2O and the arm-to-tongue time was 8 seconds. He was then given 120 mg. of thiamine daily. Edema gradually cleared, and on the eleventh day of therapy flattening of the T waves was noted in all leads. On the eighteenth day the \\nT waves in leads I, AVL, V6, V5, V4, were inverted or diphasic, and the cardiothoracic ratio was 15.5:32 cm. Five days later he was again transferred to a tuberculosis sanatorium.

Comment: Cardiac enlargement and elevated venous pressure was, therefore, found in all 5 patients; 4 had a normal or only slightly prolonged circulation time, and had electrocardiographic changes during therapy.

Acknowledgments

The author expresses his appreciation to Dr. Daniel J. Glomset for his assistance in preparation of this manuscript, to Dr. William B. Bean for his editorial comment, and to Dr. K. Daum for her analysis of the diet histories of these patients.

References

9. Weiss, S., and Wilkins, R. W.: Nature of the


11 Stroud, W. D.: The Diagnosis and Treatment of Cardiovascular Disease. V. I, II. Philadelphia, F. H. Davis Company, 1940.


Beriberi Heart in Iowa Veterans
CHARLES H. GUTENKAUF

Circulation. 1951;3:352-362
doi: 10.1161/01.CIR.3.3.352

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1951 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/3/3/352

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/