MANAGEMENT OF "INTRACTABLE" ASCITES IN DECOMPENSATED CIRRHOSIS

Favorable Response of Five Patients to Medical Treatment

CHARLES H. BROWN, M.D., MAURO MERLO, M.D.,*
Department of Gastroenterology
and
RICHARD C. BRITTON, M.D.
Department of Vascular Surgery

TRADITIONAL management of a patient with cirrhosis of the liver and associated ascites comprises a high-protein diet with supplemental vitamins and repeated paracenteses when the extent of ascites interferes with respiration, appetite, or comfort. Paracentesis has been given undue credit as a therapeutic measure, inasmuch as improvement in the patient usually has resulted from improved nutrition and hepatocellular function. Repeated paracenteses leach out the patient's plasma protein by plasmapheresis and actually are harmful. A most pitiful patient is the one with so-called intractable ascites, whose course involves progressive muscular wasting and cachexia as the ascites reaccumulates more rapidly after each paracentesis until the onset of jaundice, hepatic coma, hemorrhage from esophageal varices, and finally death.

We believe that too much reliance has been placed on repeated paracenteses as the only treatment for the cirrhotic patient with ascites, and that too little emphasis has been placed on the strict medical treatment. It is not sufficient simply to hand the patient a diet sheet outlining a high-protein and high-caloric intake. The responsible physician must see to it that the patient follows the prescribed diet. It is too easy to think: "This patient has intractable ascites and all we can do is to tap him repeatedly." Strict attention to minute details of medical management of the patient, although tedious, will usually cause a favorable response to treatment.

We shall discuss in this report the treatment of intractable ascites caused by decompensated cirrhosis, and the favorable response to it of five patients referred to us because of the apparently irreversible ascites.

Medical Treatment

The medical treatment of the patient with decompensated cirrhosis and ascites can be outlined as follows. All patients were admitted to the hospital and were given bed rest, and a diet consisting of 120 gm. of protein, 370 gm. of carbohydrate, and from 120 to 170 gm. of fat with no more than 500 mg. of salt daily. Diet diaries were kept of the exact caloric intake. The inclusion of fat in the diet

*Fellow in the Department of General Medicine.

Downloaded from www.ccjm.org on May 11, 2025. For personal use only.
All other uses require permission.

was considered essential for palatability and for supplying fat-soluble vitamins. Supplemental feedings of a salt-poor protein powder* two hours after meals were given. Although probably not essential for those patients receiving a high-protein diet, lipotrophic agents† were given orally. Multiple vitamins with an emphasis on the vitamin-B complex, vitamin B-12, and crude liver extract were administered parenterally; vitamin K was administered to those patients in whom there was a prolonged prothrombin time.

Testosterone propionate (100 mg. intramuscularly once or twice a week) was given for its anabolic effect. Twenty-four-hour urine collections were obtained to determine the gross concentrations of sodium excretion; mercurial diuretics were usually withheld if less than 1 mEq. per liter of sodium was excreted. Later, with increased sodium excretion, from 1 to 4 ml. of a mercurial diuretic was given daily.

When signs of pre-coma, hepatic coma developed, or the amount of blood ammonia increased, treatment was given for ammonia or protein intoxication as previously outlined by Owens, Brown, and Britton.[1]

Rigid avoidance of alcohol was advised. Our five patients had been alcoholics, and strictly avoided further alcoholic intake. Although small amounts of alcohol may be harmless, it is difficult for these patients to limit alcoholic intake to a small amount; consequently, strict abstinence is strongly urged. Carbonic-anhydrase diuretics and ammonium salts were avoided, as they may precipitate hepatic coma.

There is considerable current interest in the role of the portacaval shunt operation for controlling ascites that is truly refractory to medical treatment. Ligation of the hepatic artery[2,3] and bilateral adrenalectomy[4] have also been recommended, but because of the high mortality rate and the difficulty of postoperative management, these procedures are not popular and we consider them contraindicated.

Case Reports

Case 1. A 46-year-old man, an engineer, was admitted to the Cleveland Clinic Hospital on April 4, 1959, because of loss of appetite and 40 pounds in weight during the six months prior to admission. Jaundice and abdominal swelling had been present for one month. A heavy drinker most of his adult life, he consumed about one fifth of whisky daily. Physical examination revealed the patient to be undernourished. Slight icterus of the sclerae and numerous spider nevi on the chest were present. There was pitting edema of the pretibial areas. The abdomen was distended and a fluid wave was palpable. The liver was enlarged, extending 12 cm. below the right costal margin. The spleen was palpable. Roentgenograms gave evidence of normal esophagus, stomach, duodenum, and colon. Abdominal paracentesis was performed for diagnostic purposes and 100 ml. of fluid was obtained. The result of the microscopic examination of the sediment was negative for neoplastic cells.

* *Protinal powder, The National Drug Company; or, Gevral protein, Lederle Laboratories.*

† *Choline dihydrogen citrate, 10 gr. t.i.d.*

Downloaded from www.ccjm.org on May 11, 2025. For personal use only.
All other uses require permission.

During the six weeks in the hospital, the patient was treated with a strict hepatic program. His general condition improved greatly, and the icterus and ascites gradually subsided. At the time of discharge on June 2, 1959, he was advised to continue the program. When last examined on September 16, 1959, he was feeling well, his appetite had increased, and he had gained 17 pounds. No evidence of peripheral edema or ascites remained. The liver and spleen were still palpable under the costal margin but were greatly reduced in size. His clinical improvement was substantiated by laboratory tests showing an increase in the serum albumin from 1.9 to 3.8 gm. per 100 ml. The laboratory data are summarized in *Table 1.*

Table 1.—*Summary of data in case 1—ascites and decompensated hepatic cirrhosis before and after treatment*

Test	1959			
	April 16 (Before treatment)	June 2	June 30	Sept. 16
Hemoglobin, gm./100 ml.	11.4	11.6	11.6	13.3
Bilirubin, mg./100 ml.:				
Direct	2.9	1.7	—	0.4
Indirect	3.6	1.8	—	0.5
Serum albumin, gm./100 ml.	1.9	3.1	3.5	3.8
Serum globulin, gm./100 ml.	6.4	4.1	2.8	3.4
Prothrombin time, percentage of normal control	62%	80%	80%	80%
Bromsulphalein retention in 45 min., per cent	26%	22%	14%	12%
Cephalin-cholesterol flocculation, grade	3+	—	0	—
Thymol turbidity, units	9.3	—	2.6	1.3
Alkaline phosphatase, units	5.7	3.2	2.4	—
Serum transaminase, S. G. O.	60	30	20	30
Ascites, grade	4+	0	0	0
Edema, grade	2+	0	0	0
Weight, pounds	130	—	—	147

Comment. This patient with decompensated cirrhosis, ascites, and peripheral edema responded to medical treatment without paracenteses except for the removal of 100 ml. of ascitic fluid for diagnostic purposes. With improvement in nutrition and increase in albumin, spontaneous diuresis and disappearance of ascites occurred. In spite of the loss of edema and ascites, the patient gained 17 pounds.

Downloaded from www.ccjm.org on May 11, 2025. For personal use only.
All other uses require permission.

Case 2. A 48-year-old man, a truck driver, was admitted to the Cleveland Clinic Hospital on September 7, 1958, because of progressive edema of the ankle for one year, and general weakness, loss of appetite, and increasing abdominal distention for two months. Ten days prior to admission, an abdominal paracentesis was performed at another hospital. An indwelling catheter had been inserted into the right lower quadrant of the abdomen, and approximately 20 liters of ascitic fluid was removed. For 15 years prior to admission the patient's alcoholic intake had been excessive.

On physical examination, numerous spider angiomas were observed on the chest. The abdomen was distended, and a fluid wave was demonstrated. Dilated veins were present in the abdominal wall. The liver was enlarged, extending 6 cm. below the right costal margin. Cytologic examination of the ascitic fluid showed absence of neoplastic cells. The indwelling abdominal catheter was removed. Needle biopsy of the liver was performed and the pathologic report stated that the hepatic architecture was greatly distorted by fibrous tissue with a large number of proliferated bile ducts. The pathologic diagnosis was portal cirrhosis.

Roentgenograms showed evidence of normal chest, esophagus, and stomach, and no evidence of esophageal varices. The patient was given medical treatment only, and was discharged from the hospital on September 25, 1958. He was free of ascites and edema of the ankle when examined one month later (October 24). He was advised to continue the medical treatment and to return at bimonthly intervals for progress studies.

The patient was readmitted to the hospital on May 14, 1959. An acute upper respiratory infection in the patient four weeks previously had been followed by dyspnea, edema, and increasing size of the abdomen. Physical examination showed evidence of a right pleural effusion as well as ascites. Thoracentesis was performed twice, and 2,500 ml. and 1,700 ml., respectively, of clear fluid were removed. Cytologic study of the fluids was negative for neoplastic cells. The patient improved on medical treatment and was discharged from the hospital on May 22 with the advice to follow the same hepatic program. He was examined in July, August, and December, 1959, for progress studies. The ascites and edema gradually disappeared. *(Fig. 1, A and B.)* The laboratory data are summarized in *Table 2.*

Comment. This patient was considered as having intractable ascites: 20 liters of ascitic fluid were removed in the course of 10 days. He responded to medical treatment, but again, eight months later, ascites, peripheral edema, and pleural effusion on the right side developed. He again responded to medical treatment. An indwelling catheter in the abdomen had been ineffective; whereas, strict medical treatment was effective in controlling the ascites.

Case 3. A 47-year-old man, an executive, was admitted to the Cleveland Clinic Hospital on October 29, 1955, because of progressive abdominal swelling, peripheral edema, weight-loss of 30 pounds, and severe generalized malaise of three months' duration. A heavy drinker most of his life, he consumed a pint of whisky daily. In the previous month he had undergone five paracenteses in another hospital, with the removal of 42 liters of ascitic fluid.

On physical examination the patient appeared emaciated, with a loss of muscular tissue of the chest and shoulders, and pitting edema of the legs, scrotum, and presacral

Downloaded from www.ccjm.org on May 11, 2025. For personal use only.
All other uses require permission.

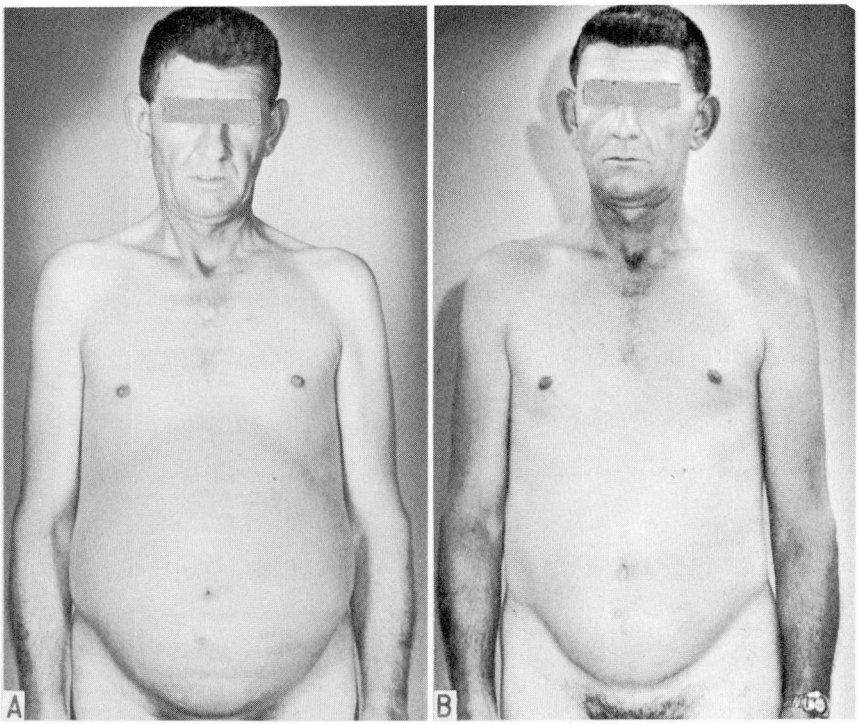

Fig. 1. Case 2. A, September, 1958, before treatment. Note protuberant abdomen and ascites; the umbilicus shifted position and is nearer the symphysis pubis than the xyphoid. This shift in position is characteristic in the cirrhotic patient with ascites, and is evident also in *Figures 2, 3,* and *4.* B, December, 1959, (20 months after treatment). Nutrition has improved and there is increased musculature. Ascites disappeared and did not recur and the patient gained 34 pounds.

area. Palmar erythema, icterus of the sclerae, and spider angiomas on the shoulder were present. The abdomen was distended and a fluid wave was demonstrated. The liver was enlarged, hard, nodular, and extended 5 cm. below the right costal margin. The spleen was not palpable. Dilated veins in the abdominal wall were prominently visible. A roentgenogram of the esophagus showed evidence of esophageal varices. During his stay in the hospital, two paracenteses were performed; 7.4 liters and 13.9 liters of fluid, respectively, were removed to relieve the respiratory distress. The result of the cytologic examination of the sediment was negative for neoplastic cells. The patient was treated with a strict hepatic program and was discharged on November 18, 1955.

He was readmitted to the hospital for progress hepatic studies in August, 1956, and in January and July, 1957. During those two years he followed the dietary program

Downloaded from www.ccjm.org on May 11, 2025. For personal use only.
All other uses require permission.

Table 2.—*Summary of data in case 2—ascites and decompensated hepatic cirrhosis before and after treatment*

Test	1958		1959			
	Sept. (Before treatment)	Dec.	May	July	Aug.	Dec.
Hemoglobin, gm./100 ml.	14.6	—	12.9	—	15.5	15.8
Serum albumin, gm./100 ml.	1.0	2.5	1.82	3.01	2.32	4.16
Serum globulin, gm./100 ml.	5.2	4.2	4.69	3.64	3.71	2.66
Prothrombin time, percentage of normal control	80%	86%	50%	68%	68%	68%
Cephalin-cholesterol flocculation, grade	4+	4+	4+	3+	—	—
Thymol turbidity, units	3.2	—	7.5	—	—	—
Ascites, grade	4+	0	4+	2+	0	0
Edema, grade	2+	0	4+	2+	0	0
Weight, pounds	163-171	182	202-177	192	188	—

muscular strength. He did not require paracentesis, and when he was last examined there was no evidence of ascitic fluid. The liver was still palpable three fingerbreadths under the right costal margin, and the spleen also was palpable. *(Fig. 2, A, B, and C.)* The laboratory data are summarized in *Table 3.*

Comment. This patient had massive ascites: 63 liters of ascitic fluid were removed in the course of six weeks. He responded to medical treatment, had no recurrence of the ascites, gained in muscular strength and tone, and in nutrition. His clinical status improved to the extent that we were considering advising a portacaval shunt, when he suddenly suffered a fatal massive gastrointestinal hemorrhage at home.

Case 4. A 51-year-old unemployed man was first admitted to the Cleveland Clinic Hospital on June 2, 1958, because of ascites and peripheral edema of one and one-half years' duration. He had been treated at another hospital with a low-salt and high-protein diet, multivitamins, and oral diuretics. Response to this treatment had been poor, and he had required monthly paracentesis. Two months prior to admission, two Cooney buttons had been inserted in the lower abdominal wall in an attempt to drain the ascitic fluid. These did not function well. The patient was accustomed to a heavy alcoholic intake most of his adult life, and consumed about one pint of whisky daily.

Physical examination revealed an emaciated man who appeared to be chronically ill. Multiple spider angiomas were on the shoulders and arms, and there was palmar erythema. The abdomen was distended and a fluid wave was palpable; two plastic valves were present in both hypogastric areas. The liver and the spleen were not

Downloaded from www.ccjm.org on May 11, 2025. For personal use only.
All other uses require permission.

palpable. Esophagoscopy revealed a small varix at the cardia. A roentgenogram of the chest showed evidence suggestive of a small amount of fluid in the right costophrenic angle.

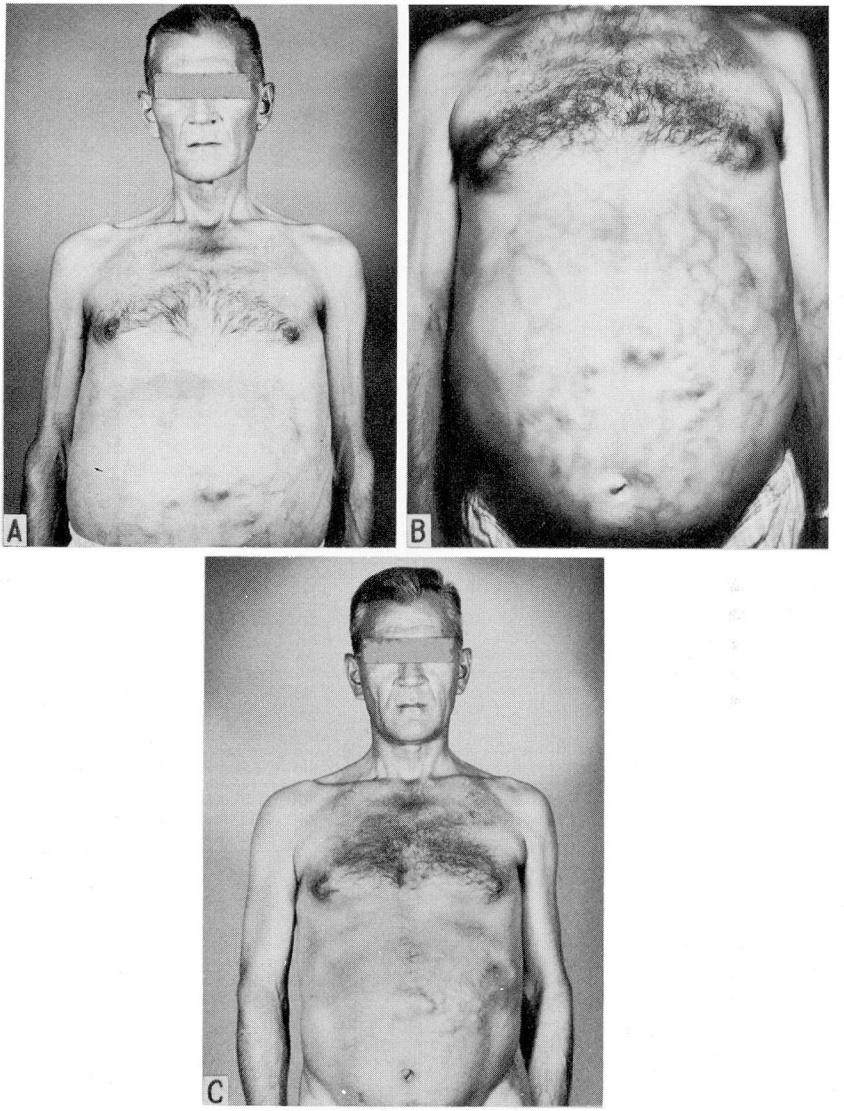

Fig. 2. Case 3. A, October, 1955, showing severe emaciation, loss of musculature, and ascites. B, Infrared photograph showing notable collateral circulation in the abdominal wall. C, January, 1957, showing a gain of 14 pounds in weight (despite loss of ascitic fluid), increased musculature, and improved nutrition.

Downloaded from www.ccjm.org on May 11, 2025. For personal use only.
All other uses require permission.

Table 3.—*Summary of data in case 3—ascites and decompensated hepatic cirrhosis before and after treatment*

Test	1955	1956	1957	
	Nov. (Before treatment)	Aug.	Jan.	July
Hemoglobin, gm./100 ml.	13.4	12.4	13.5	14.5
Bilirubin, mg./100 ml.:				
Direct	0.7	0.6	0.9	1.1
Indirect	2.22	1.46	1.5	2.0
Serum albumin, gm./100 ml.	1.2	1.0	2.45	2.19
Serum globulin, gm./100 ml.	6.2	5.6	5.75	5.80
Prothrombin time, percentage of normal control	54%	72%	62%	58%
Bromsulphalein retention in 45 min., per cent	—	32%	—	38%
Cephalin-cholesterol flocculation, grade	4+	4+	4+	—
Thymol turbidity, units	23	17.5	19	17.5
Alkaline phosphatase, units	6.6	—	8.7	7.0
Ascites, grade	4+	?	0	0
Edema, grade	2+	0	0	0
Weight, pounds	191	197	205	205

On June 6, a specimen from a needle biopsy of the liver showed severe portal fibrosis, and proliferation of the bile ducts. There was chronic inflammatory infiltration of the portal areas. On June 11, under local anesthesia, the Cooney buttons were removed and 7 liters of ascitic fluid was withdrawn from the abdominal cavity. The patient was treated with a strict hepatic program that included mercurial diuretics. He showed a prompt and distinct clinical improvement and was discharged on June 28, 1958, with the recommendation that he continue the same program.

Subsequent examinations have shown his progress to be satisfactory, and except for occasional pain in the lower abdomen, he has felt well. In August, 1958, one paracentesis was performed and 6.3 liters of ascitic fluid was removed. He was last examined in December, 1959, and had had no recurrence of ascites or retention of fluid. The patient had gained 25 pounds in weight, had a good appetite, was working regularly, and felt well. *(Fig. 3, A, B, and C.)* The laboratory data are summarized in *Table 4.*

Comment. This patient had intractable ascites for one and one-half years and had been unable to work. Repeated paracenteses were necessary, and as a last resort, (in another hospital) two Cooney buttons had been placed in the abdominal wall in an

Downloaded from www.ccjm.org on May 11, 2025. For personal use only.
All other uses require permission.

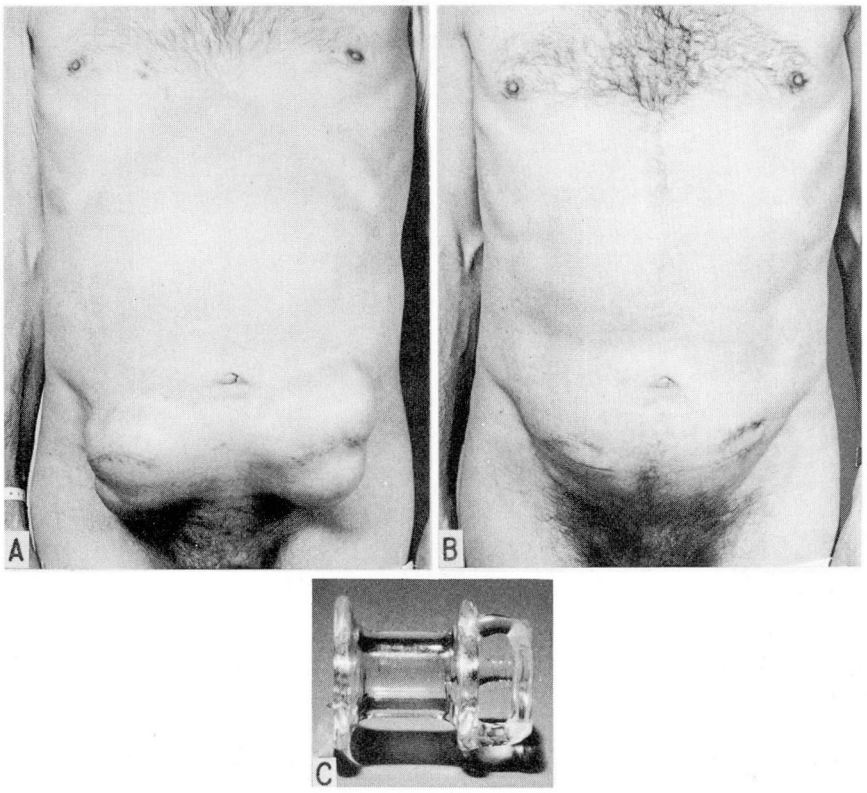

Fig. 3. Case 4. A, June, 1958, showing ascites and bulging in both lower quadrants caused by two Cooney buttons inserted two months previously (in another institution) in an attempt to control the ascites. The Cooney buttons did not function, and caused pain. B, January, 1959, six months after A. No ascites was present, although the patient had gained 13 pounds. C, One of the Cooney buttons that were removed.

attempt to drain the ascitic fluid. The patient responded satisfactorily to strict medical therapy advised by us, and the Cooney buttons were removed. He has had no ascites, has required no paracenteses in the past 16 months, and was able to return to work.

Case 5. A 51-year-old man, a laborer, first entered the Cleveland Clinic Hospital on April 4, 1958, because of jaundice and ascites of two months' duration. He had been treated in another hospital and had had five paracenteses; 8 liters of fluid was removed each time. He had been a heavy drinker for 15 years, consuming about one-half pint of whisky and two or three pints of beer daily.

On physical examination he appeared undernourished. Several spider nevi were on the anterior chest. The abdomen protruded and a fluid wave was palpable. The spleen and liver were not palpable. Pitting edema of the legs was present. Roentgenograms gave evidence of normal chest, esophagus, stomach, and duodenum. On April 19, an

Downloaded from www.ccjm.org on May 11, 2025. For personal use only.
All other uses require permission.

Table 4.—*Summary of data in case 4—ascites and decompensated hepatic cirrhosis before and after treatment*

Test	1958			1959	
	June (Before treatment)	Sept.	Nov.	Jan.	Dec.
Hemoglobin, gm./100 ml.	13.7	14.4	14.4	14.2	—
Bilirubin, mg./100 ml.:					
Direct	0.2	0.4	0.1	—	—
Indirect	0.6	0.7	1.0	—	—
Serum albumin, gm./100 ml.	2.4	2.4	2.7	3.0	—
Serum globulin, gm./100 ml.	3.5	5.1	3.8	3.0	—
Prothrombin time, percentage of normal control	100%	—	—	100%	—
Bromsulphalein retention in 45 min., per cent	29%	24%	23%	21%	—
Cephalin-cholesterol flocculation, grade	3+	—	—	—	—
Thymol turbidity, units	4	—	—	—	—
Alkaline phosphatase, units	8.5	—	—	—	—
Ascites, grade	4+	0	0	0	0
Edema, grade	4+	0	0	0	0
Weight, pounds	160	155	168	171	180

abdominal paracentesis was performed and 6 liters of cloudy fluid was removed. The result of the microscopic examination of the sediment was negative for neoplastic cells. Five days later a specimen of a needle biopsy of the liver showed an irregular nodulation and broad zones of dense fibrous tissue containing numerous small bile ducts. The pathologic diagnosis was inactive postnecrotic cirrhosis.

The patient was treated with a strict hepatic program for 10 days and was discharged from the hospital on May 3. He was advised to continue with the same program, and to report periodically. His progress has been excellent up to December, 1959, when he was last examined. His appetite has been good and he has gained 34 pounds in weight. There has never been evidence of a recurrence of ascites or of peripheral edema. *(Fig. 4, A and B.)* The laboratory data are summarized in *Table 5.*

Comment. This patient had decompensated cirrhosis with intractable ascites, and had had 46 liters of ascitic fluid removed by paracenteses. After institution of strict medical therapy, no further paracenteses have been necessary in the 20 months of treatment. There have been improvement in the amount of serum albumin, in general nutrition, in muscle tone, and an increase in weight.

Downloaded from www.ccjm.org on May 11, 2025. For personal use only.
All other uses require permission.

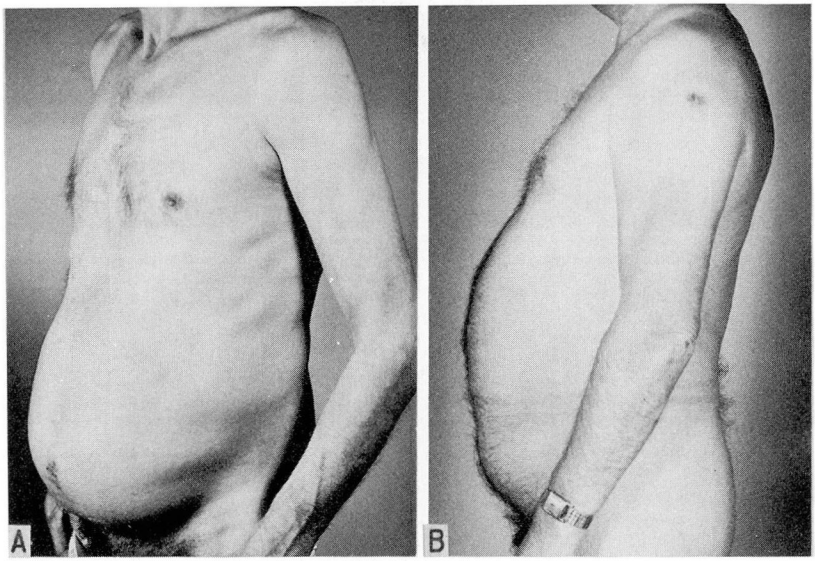

Fig. 4. Case 5. A, April, 1958, before treatment; note muscular wasting of neck, shoulders and arms, and great distention of the abdomen with ascites. B, September, 1959, improvement is evidenced by increased musculature, by the absence of ascites, and a gain of 25 pounds in weight despite the loss of ascitic fluid.

Discussion

Ascites is one of the most prominent and frequent physical signs in the decompensated stage of cirrhosis. The pathogenesis of ascites and edema in the cirrhotic patient is dependent upon factors that include: severity of portal hypertension, state of increased capillary permeability, presence of hypoproteinemia and hypoalbuminemia, retention of sodium, secondary aldosteronism, the stage of hepatic disease, and the type, extent, and cause of obstruction of outflow of hepatic blood. The factors that contribute to the formation and recurrence of ascites recently have come under close scrutiny. As a result, great emphasis is being placed upon the roles of the total body sodium, the type and extent of obstruction of hepatic blood flow, the stage of the hepatic disease, and secondary aldosteronism.

The increasing emphasis upon the importance of the role of sodium in the production and maintenance of ascites is based upon the demonstration that patients with decompensated cirrhosis retain sodium and excrete meager amounts, particularly during active formation of ascites. Farnsworth,[5] and Ricketts, Eichelberger, and Kirsner[6] noted that sodium was almost absent from the urine, and Eisenmenger, Blondheim, Bongiovanni, and Kunkel[7] noted that the sodium content was low in the sweat and saliva of patients who are forming ascites.

Downloaded from www.ccjm.org on May 11, 2025. For personal use only.
All other uses require permission.

Table 5.—*Summary of data in case 5—ascites and decompensated hepatic cirrhosis before and after treatment*

Test	1958		1959				
	April (Before treatment)	July	Jan.	April	July	Sept.	Dec.
Hemoglobin, gm./100 ml.	13.7	16.4	17.8	—	18.4	17.4	16.6
Bilirubin, gm./100 ml.:							
Direct	0.1	0.1	0.1	—	0.6	—	—
Indirect	0.9	0.7	0.5	—	0.4	—	—
Serum albumin, gm./100 ml.	1.7	3.0	3.5	4.3	4.4	4.16	4.94
Serum globulin, gm./100 ml.	3.5	4.1	3.4	3.1	2.9	2.32	2.31
Prothrombin time, percentage of normal control	80%	100%	—	—	—	—	—
Bromsulphalein retention in 45 min., per cent	40%	24%	20%	10%	—	23%	14%
Cephalin-cholesterol flocculation, grade	2+	Neg.	Neg.	—	1+	—	—
Thymol turbidity, units	6	4.1	2.1	—	1.9	—	—
Ascites, grade	4+	0	0	0	0	0	0
Edema, grade	4+	0	0	0	0	0	0
Weight, pounds	157-124	151	165	158	157	158	155½

The electrolyte pattern in patients with cirrhosis and ascites is similar to that seen in hyperaldosteronism, and it has been demonstrated that many cirrhotic patients with ascites excrete abnormally large amounts of aldosterone in the urine.[4,8,9] Consequently, it has been postulated that secondary hyperaldosteronism may be a factor in the development of ascites in cirrhotic patients. Kerr, Read, Haslam, and Sherlock[10] administered an aldosterone antagonist (spirolactone*) to four patients with cirrhosis and intractable ascites, and diuresis of both sodium and water ensued in three of them. On an experimental basis, similar good results from the use of aldosterone antagonists in patients with intractable ascites have been reported by others, although undesirable side-effects have been the loss of appetite, nausea, and vomiting. The use of aldosterone antagonists and spirolactone in the treatment of the decompensated cirrhotic patient with ascites is still experimental, and not yet a part of standard treatment for these patients.

Salt restriction in the past has been inadequately controlled in the patient with decompensated cirrhosis. The so-called low-sodium diets have actually represented a sodium overload for patients with ascites. Restriction of sodium to 500 mg. or less daily is necessary.

* *Aldactone, G. D. Searle & Co.*

Downloaded from www.ccjm.org on May 11, 2025. For personal use only.
All other uses require permission.

During the period in which the patient is actively accumulating ascites he may be refractory to the action of diuretics. Forced diuresis may occur through use of the newer carbonic-anhydrase inhibitors, but this is usually a forced excretion of water and potassium without a corresponding excretion of sodium. Carbonic-anhydrase-inhibiting diuretics given to a patient with decompensated cirrhosis have precipitated hepatic coma. The mechanism by which such diuretics induce hepatic coma may be through lowering the concentration of plasma potassium, as Sherlock[11] reported. The carbonic-anhydrase-inhibiting diuretics, including chlorothiazide*, hydrochlorothiazide†, and acetazolamide‡, are contraindicated in these patients. If a diuretic is used in the patient with decompensated cirrhosis, mercurial diuretics with supplemental potassium chloride intake are preferable. Ammonium chloride, to enhance the diuretic action of the mercurial agent, is absolutely contraindicated, because of the possibility of causing ammonia intoxication and hepatic coma.

The patient with decompensated cirrhosis and ascites, limited to a 500-mg. salt intake daily, in addition to a high-protein diet and other measures, will frequently have no further increase in the ascites. After a period of stabilization lasting from several days to several months, spontaneous diuresis and disappearance of the ascites may occur. It is during stabilization of the ascites that a mercurial diuretic, by causing a diuresis of water and subsequently sodium, may be helpful. When diuresis occurs and ascites disappears, whether spontaneous or aided by mercurial diuretics, hepatic function tests usually indicate that a general improvement in hepatocellular function has also occurred.

The greatest criticism of the traditional management of ascites is the frequent paracenteses that result in a great loss of serum albumin with each tap, a loss of albumin ranging from 20 to 266 gm.[12,13] The high cost of salt-poor serum albumin to replace that lost during paracentesis is formidable, and the supply of free salt-poor albumin available through the Red Cross blood program is greatly limited. Ideally, the amount of serum albumin lost at each paracentesis should be determined, and this amount should be given intravenously, since the periodic loss of this potent osmotic factor can only serve to perpetuate the ascites.§ Paracentesis has been used by us only for the purpose of relieving severe discomfort, to improve the appetite, to rule out carcinomatosis by cytologic study, and in preparation for needle biopsy of the liver.[14] Seldom has more than one paracentesis,

**Diuril (chorothiazide), Merck Sharp & Dohme.*

†HydroDIURIL (hydrochlorothiazide), Merck Sharp & Dohme.

‡Diamox (acetazolamide), Lederle Laboratories.

§Recently in other patients, when paracentesis was necessary, because of the high cost of serum albumin, ascitic fluid was saved. This ascitic fluid subsequently was dialyzed in the artificial kidney to remove salt and water. The concentrated and salt-poor ascitic fluid was then returned to the patient intravenously to replace the albumin. This procedure has been done in six patients with apparently beneficial results. (R. C. Britton, and S. Nakamoto; see page 82 of this issue.)

Downloaded from www.ccjm.org on May 11, 2025. For personal use only.
All other uses require permission.

usually for diagnostic purposes, been necessary. By such restriction of paracenteses and in association with rigid salt restriction, a high-protein, high-carbohydrate and high-caloric diet, suitable vitamins, and other measures, it has been possible to stabilize ascites without surgical intervention.

Summary

Five alcoholic patients had intractable ascites and decompensated cirrhosis. When first seen, four patients had had repeated paracenteses. One patient had had an indwelling catheter placed in the abdomen. In another patient two Cooney buttons had been placed in the abdominal wall. All of the patients responded to medical treatment, obtained relief of the ascites, and improved in general nutrition and in hepatic function. It may be questionable whether ascites caused by decompensated cirrhosis is ever intractable if medical treatment is strict, intensive, and prolonged.

Medical treatment of the cirrhotic patient with ascites is outlined. Repeated paracenteses, carbonic-anhydrase diuretics, and ammonium salts are avoided. Avoidance of alcohol is imperative. A 500-mg. sodium, high-protein, high-caloric diet with supplemental protein feedings between meals is used, and a careful diet diary is kept to determine the actual intake. Supplemental vitamins are administered orally and parenterally, and vitamin-B complex, liver extract, and depotestosterone are injected parenterally. Mercurial diuretics may be given, and serum albumin used when available. With perseverance and persistence on the part of both the patient and the physician, ascites in most cases of decompensated cirrhosis should respond to medical treatment.

References

1. Owens, F. J.; Brown, C. H.; Britton, R. C., and Faulkner, W. R.: Episodic hepatic encephalopathy: problem of all specialties. Cleveland Clin. Quart. **26**: 1-11, 1959.

2. Rienhoff, W. F., Jr., and Woods, A. C., Jr.: Ligation of hepatic and splenic arteries in treatment of cirrhosis with ascites. J.A.M.A. **152**: 687-690, 1953.

3. Berman, J. K.; Habegger, E. D., and Fields, D. C.: Arterial ligations in cirrhosis of liver; selection of patients and results. Connecticut M. J. **18**: 197-203, 1954.

4. Giuseffi, J.; Werk, E. E., Jr.; Larson, P. U.; Schiff, L., and Elliott, D. W.: Effect of bilateral adrenalectomy in patient with ascites and postnecrotic cirrhosis. New England J. Med. **257**: 796-803, 1957.

5. Farnsworth, E. B.: Electrolyte partition in patients with edema of various origins; sodium and chloride. Am. J. Med. **4**: 338-342, 1948.

6. Ricketts, W. E.; Eichelberger, L., and Kirsner, J. B.: Observations on alterations in electrolytes and fluid balance in patients with cirrhosis of liver with and without ascites. J. Clin. Invest. **30**: 1157-1170, 1951.

Downloaded from www.ccjm.org on May 11, 2025. For personal use only.
All other uses require permission.

7. Eisenmenger, W. J.; Blondheim, S. H.; Bongiovanni, A. M., and Kunkel, H. G.: Electrolyte studies on patients with cirrhosis of liver. J. Clin. Invest. **29:** 1491-1499, 1950.

8. Chart, J. J., and Shipley, E. S.: (Abs.) Mechanism of sodium retention in cirrhosis of liver. J. Clin. Invest. **32:** 560, 1953.

9. Leutscher, J. A., Jr., and Johnson, B. B.: Observations on sodium-retaining corticoid (aldosterone) in urine of children and adults in relation to sodium balance and edema. J. Clin. Invest. **33:** 1441-1446, 1954.

10. Kerr, D. N.; Read, A. E.; Haslam, R. M., and Sherlock, S.: Use of steroidal spirolactone in treatment of ascites in hepatic cirrhosis. Lancet **2:** 1084-1087, 1958.

11. Sherlock, S.: Hepatic coma; presented at American Association for Study of Liver Disease, Chicago, Nov. 1959.

12. Berman, J. K., and Hull, J. E.: Experimental ascites—its production and control. Surgery **32:** 67-75, 1952.

13. Lichtman, S. S.: Diseases of the Liver, Gallbladder and Bile Ducts. Vol. 2. Philadelphia: Lea & Febiger, 1953, pp. 609-808; p. 702.

14. Brown, C. H.: Biopsy of liver with Vim-Silverman needles. Mississippi Valley M. J. **81:** 198-200, 1959.

Downloaded from www.ccjm.org on May 11, 2025. For personal use only.
All other uses require permission.