The Relief of Resistant Edema by Utilization of a Sump Phenomenon

By Ferdinand R. Scheffm, M.D. and Augusto A. Camara, M.D.*

Fourteen massively edematous patients with either cardiac disease or nephritis, who had hydrothorax and/or ascites, have proved resistant to the usual diuretic measures, but have been relieved of edema by repeated aspirations of fluid from either the pleural or peritoneal spaces. In the intervals between aspirations the fluid of the interstitial space appeared to seep readily into the sump from which fluid had just been removed. The repeated aspirations did not result in hypoproteinemia or hypoproteineinemia in these cases. As a means of by-passing "reluctant" kidneys the utilization of the sump phenomenon has proved simple, safe and effective in some very obstinate cases.

Despite the more efficacious control of edema brought about by a better understanding of electrolyte metabolism, the application of acid-base and water balance principles and the development of effective diuretics, there remain certain groups of cardiac and nephrotic patients whose edema remains absolutely or relatively intractable. In our experience, one such group of patients manifest what we shall call the sump phenomenon which will be described in this report.

The authors have collected data on 10 cardiac, 3 nephritic and 1 diabetic patients. Each had an accumulation of free fluid in either pleural or peritoneal spaces. Relief of edema in these patients was achieved by repeated aspirations of fluid from this space, which acted like a "low-pressure" area into which edema fluid readily seeped after each aspiration. In the intervals between aspirations there was little or no change in body weight, but signs of fluid in the sump increased, while the degree of peripheral edema decreased. In some of these cases, despite meticulous attention to the regime and repeated doses of mercurial diuretics, no relief of edema could be attained except through repeated aspirations of fluid from the sump. In the other cases, utilization of the sump for the mechanical removal of fluid proved of value in hastening the relief of edema and shortening the period of hospitalization.

It has been observed that in such patients, recurrence of peripheral edema is preceded by reaccumulation of fluid in the sump, and the appearance of edema can be forestalled by repeated tapping.

Most important of all, the authors have seen no deleterious effect of repeated aspirations in these patients.

Method and Material

All but two of the patients reported in this paper were studied in the Metabolic Unit of the Deaconess Hospital. Daily fasting weights in the morning were recorded on a scale sensitive to 100 Gm. Twenty-four hourly urine specimens were collected daily, collections being started and ended at 6 A.M. daily, and their volumes recorded. The daily urinary excretions of chloride, sodium and potassium were determined. Analyses for the same electrolytes were carried out on fluid aspirated from the sump each time. The morning following admission, blood specimens were obtained in the fasting state for initial blood chemistry. The chemical determinations included sodium, potassium, chloride, carbon dioxide combining power, blood urea nitrogen and serum proteins. The same analyses were obtained at appropriate intervals and at the end of treatment.

*This work was done during a tenure of Fellowship of the American Heart Association.
### Table 1.—Clinical Data on Patients Manifesting the Sump Phenomenon

<table>
<thead>
<tr>
<th>Patient</th>
<th>Diagnosis</th>
<th>Sump</th>
<th>Aspirations</th>
<th>Edema Weight Loss</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. F. J., M, 52</td>
<td>Heart disease, rheumatic; mitral stenosis, mitral insufficiency</td>
<td>Pleural cavity, right</td>
<td>7 in 20 days</td>
<td>13.30</td>
</tr>
<tr>
<td>2. K. H., M, 62</td>
<td>Heart disease, rheumatic; aortic stenosis, auricular fibrillation</td>
<td>Pleural cavity, right</td>
<td>5 in 13 days</td>
<td>5.30</td>
</tr>
<tr>
<td>3. G. L., M, 18</td>
<td>Heart disease, congenital; marked hypertrophy &amp; dilatation, right ventricle &amp; right auricle; tricuspid insufficiency</td>
<td>Peritoneal cavity</td>
<td>3 in 15 days</td>
<td>9.00</td>
</tr>
<tr>
<td>4. B. I., M, 50</td>
<td>ASCVD Diabetes mellitus</td>
<td>Pleural cavity, right</td>
<td>6 in 13 days</td>
<td>4.68</td>
</tr>
<tr>
<td>5. B. W., F, 40</td>
<td>Heart disease, rheumatic; mitral stenosis &amp; insufficiency; aortic insufficiency; auricular fibrillation</td>
<td>Pleural cavity, right</td>
<td>4 in 10 days</td>
<td>2.52</td>
</tr>
<tr>
<td>6. B. O., F, 60</td>
<td>Hypertensive ASCVD; auricular fibrillation</td>
<td>Pleural cavity, right</td>
<td>4 in 11 days</td>
<td>2.85</td>
</tr>
<tr>
<td>7. A. L., F, 61</td>
<td>Hypertensive ASCVD</td>
<td>Both pleural cavities</td>
<td>5 in 30 days</td>
<td>4.74</td>
</tr>
<tr>
<td>8. A. W., F, 44</td>
<td>Heart disease, rheumatic; mitral stenosis &amp; insufficiency; auricular fibrillation</td>
<td>Pleural cavity, right</td>
<td>4 in 12 days</td>
<td>2.20</td>
</tr>
<tr>
<td>9. C. B., M, 56</td>
<td>ASCVD</td>
<td>Pleural cavity, right</td>
<td>4 in 25 days</td>
<td>3.23</td>
</tr>
<tr>
<td>10. C. D., F, 45</td>
<td>Heart disease, rheumatic; mitral stenosis &amp; insufficiency; auricular fibrillation</td>
<td>Pleural cavity, right</td>
<td>4 in 8 days</td>
<td>2.93</td>
</tr>
</tbody>
</table>

**In two of the patients, immediately following thoracenteses, 50 ml. of deuterium oxide \((D_2O)\) mixed with an equal amount of 5 per cent dextrose in distilled water was infused subcutaneously into the inner aspect of the thigh. Eighteen to 19 hours later, chest fluid and blood were obtained simultaneously for determination of deuterium oxide \((D_2O)\) concentration. The procedures used in these analyses have been described in a previous paper.**

Four of the patients, in whom one of the pleu-
F. R. SCHEMM AND A. A. CAMARA

**Table 2. Electrolyte and Protein Data on Patients Manifesting The Sump Phenomenon During The Period of Study**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sodium Out</th>
<th>Plasma Sodium</th>
<th>Protein Out Through Sump</th>
<th>Serum Proteins</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Via sump</td>
<td>Via urine</td>
<td>Sump sodium</td>
<td>Before aspiration</td>
</tr>
<tr>
<td></td>
<td>mEq</td>
<td>mEq</td>
<td>mEq/L</td>
<td>mEq/L</td>
</tr>
<tr>
<td>I. Cardiacs</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. F. J.</td>
<td>1,638</td>
<td>147</td>
<td>136</td>
<td>138</td>
</tr>
<tr>
<td>2. K. H.</td>
<td>698</td>
<td>148</td>
<td>129</td>
<td>133</td>
</tr>
<tr>
<td>3. G. L.</td>
<td>1,239 plus</td>
<td>1,474</td>
<td>146</td>
<td>137</td>
</tr>
<tr>
<td>4. B. I.</td>
<td>630</td>
<td>934</td>
<td>141</td>
<td>143</td>
</tr>
<tr>
<td>5. B. W.</td>
<td>338 (chest)</td>
<td>1,262</td>
<td>138</td>
<td>136</td>
</tr>
<tr>
<td></td>
<td>439 (abd)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

|                 |            |               |                          |                  |                  |                  |                  |
| II. Nephritics  |            |               |                          |                  |                  |                  |                  |
| 1. J. P.        | 1,149      | 9             | 140                      | 126              | 8                | 2.7 T            | 3.3 T            |
|                 |            |               |                          |                  | 4 days later     | 1.1 A            | 1.2 A            |
|                 |            |               |                          |                  | 144              |                  |                  |

<table>
<thead>
<tr>
<th></th>
<th>Chloride Out</th>
<th>Plasma Chloride</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Via Sump</td>
<td>Via Urine</td>
<td>Sump chloride</td>
<td>Before Aspirations</td>
<td>After Aspirations</td>
</tr>
<tr>
<td></td>
<td>mEq</td>
<td>mEq</td>
<td>mEq/L</td>
<td>mEq/L</td>
<td>mEq/L</td>
</tr>
<tr>
<td>2. R. W.</td>
<td>2,485</td>
<td>948</td>
<td>101</td>
<td>107</td>
<td>86</td>
</tr>
<tr>
<td>3. R. D.</td>
<td>1,738</td>
<td>3,306</td>
<td>110</td>
<td>95</td>
<td>258</td>
</tr>
</tbody>
</table>

Notes:
- The entry spaces acted as a *sump*, had serial x-ray films of the chest taken during the course of treatment, to show clearing of the fluid after aspiration and its subsequent reaccumulation.
- Of the 10 cardiac patients, five were female, and five were male; all ranging in age from 18 to 62 years. Five of them had rheumatic heart disease with chronic valvulitis, four had arteriosclerotic heart disease with or without hypertension, and one had congenital heart disease.
- The patients in the nephrotic stage of glomerulonephritis, all males, numbered three, with an age range of 3 to 7 years. The one remaining patient in this series was a young man of 32 who had diabetes mellitus and who also presented the syndrome of Kimmelstiel-Wilson.

**RESULTS**

In table 1 the over-all clinical data are presented. It gives the number of aspirations performed during the time it took these patients to attain dry weight, the number and dose of mercurial diuretics administered, and the per
cent of actual weight loss attributable to the amount of fluid removed from the *sump*. Note that among the truly resistant cardiac and nephritic patients, the total amount of fluid aspirated accounted for 75 to 99 per cent of the actual weight loss. In the other patients whose clearing of edema was materially hastened by utilization of the *sump*, this figure ranged from 27 to 52 per cent.

Table 2 gives the electrolyte data on 12 of the patients who were studied in the Metabolic Unit. In two of the earlier patients (R. W. and R. D.), treated before a Beckmann flame photometer was acquired, urinary, aspirated fluid and plasma chlorides were determined instead of sodium. The amount of electrolytes, either sodium or chloride, removed via the *sump* varied from 72 to 99 per cent of the total output (sodium or chloride of urine plus that of *sump* fluid) in the patients with intractable edema, and from 31 to 82 per cent in the other patients whose edema cleared more rapidly as a result of repeated aspirations. Plasma proteins (total and albumin) as well as plasma sodium or chloride, before and after repeated aspirations of *sump* fluid was instituted, are also presented in table 2. This table shows, in those cases where the concentration of protein in the aspirated fluid was determined, the total amount of protein removed with the *sump* fluid; the range was from 8 to 258 Gm. Note that despite repeated taps, no harmful dilution of either blood electrolytes or of blood protein resulted.

The data in table 3 were obtained to show that a tagged material such as D₂O, when introduced subcutaneously into a dependent portion of the body, found its way into the *sump* after a relatively short period of time. D₂O thus administered was found in perfect equilibrium between the chest fluid and blood plasma after 18 to 19 hours.

**Case Summaries**

Case summaries of the 4 patients who presented *truly resistant* edema that could be relieved only by repeated fluid drainage from a *sump* will be reviewed. In the 10 other patients in this series, clearing of moderately resistant edema was significantly accelerated by the same procedure. The essential data on these patients are shown in the preceding tables.

**Case 1.** J. P. was a 3½ year old boy whose trouble started with a series of severe upper respiratory tract infections one year prior to hospital admission on Feb. 20, 1949. Onset of edema came two months later, immediately following tonsillectomy. Up to the time of admission, there were several episodes of diarrhea, abdominal pain, fever, upper respiratory tract infection and urticarial rashes. Edema had appeared intermittently, but became persistent and progressive about three months before admission. On admission he was truly anasarctic, with pitting edema from the toes to the scalp, and had a grotesquely protuberant abdomen tight with ascitic fluid. He presented a full-blown picture of the nephrotic stage of glomerulonephritis with albuminuria, cylindruria, microscopic hematuria, hypercholesterolemia, low serum proteins with inverted albumin-globulin ratio and no uremia (blood urea nitrogen 7 mg. per 100 cc.). Marked relief was obtained after the first paracentesis in which 3200 ml. of fluid were removed. As shown in table 1, during a nine-day period, when a total of 8.2 liters were drained from the abdomen, the total edema weight loss was 9.2 kg., or 20 pounds! The aspirated fluid accounted for 89 per cent of this weight loss. The electrolyte data presented in figure 1 demonstrate strikingly the much greater loss of excess body sodium through repeated drainage of the abdomen as compared with the exceedingly small amounts of this electrolyte excreted through the kidneys, despite repeated injections of Thiomerin. Actual calculations show that of the total loss of sodium, 99 per cent came out through the *sump*. (See table 2.)

This boy was edema-free for a few weeks following

### Table 3.—Data Showing Appearance of D₂O in Sump Fluid Following Its Subcutaneous Administration in Area of Dependent Edema

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sump</th>
<th>D₂O Administration</th>
<th>D₂O Concentration 18 Hours Later</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Site</td>
<td>Chest fluid</td>
</tr>
<tr>
<td>1. F. J.</td>
<td>Pleural cavity, right</td>
<td>ml.</td>
<td>Per cent</td>
</tr>
<tr>
<td></td>
<td>50</td>
<td>Thigh, left</td>
<td>0.124</td>
</tr>
<tr>
<td>2. A. W.</td>
<td>Pleural cavity, right</td>
<td>50</td>
<td>Thigh, left</td>
</tr>
</tbody>
</table>

* No weight change in these two patients during the 18 hours of observation.
discharge. Subsequently, fluid reaccumulated in the abdomen and required tapping every 10 days over a period of many months. At the time of writing, he has been free of edema during the last 12 months.

Case 2. F. J., a 52 year old man, had a high grade mitral stenosis resulting from recurrent episodes of rheumatic fever in childhood. Symptoms of cardiac decompensation started two years before he was seen by us in July, 1952. During the six months immediately preceding our study, he had been repeatedly hospitalized, with no relief following the institution of digitalis therapy, the customary low sodium diet with acid diuretics and frequent doses of mercurial diuretics. Massive right hydrothorax had been aspirated only a few times for mechanical relief of compression of the lung. On admission, he was deeply cyanotic, mentally confused, anasarca; he had peripheral edema reaching half way up the torso, bilateral hydrothorax, ascites and marked hepatic congestion (lower border of the liver 5 inches below the right costal margin).

He was promptly redigitalized, placed in an oxygen tent and on a high-fluid, neutral diet and ammonium chloride regimen. Even after proper hydration, it was found that injections of Thiomerin resulted in only small diureses of sodium; hence a program of repeated aspiration of the right pleural space was begun. Thoracentesis was performed on the right pleural cavity seven times over the period of 21 days shown in figure 2. The amount of fluid removed ranged from 2800 ml. with the second thoracentesis to 1200 ml. removed during the sixth aspiration. In this patient, the total amount of fluid removed via the sump, 13.3 liters, accounted for 87 per cent of the edema weight loss of 15.2 kg. This figure tallies closely with that of 85 per cent of the total excess body sodium removed mechanically through the sump, by calculation from electrolyte data. (See table 2.)

As seen in the accompanying series of chest x-ray films (fig. 3), the right pleural space kept refilling with fluid after repeated aspirations. The first plate taken on Aug. 8, 1952, prior to thoracentesis, shows free fluid; following this film, 1700 ml. of fluid was drained. An x-ray film made the same day, shortly after tapping, shows clearing of virtually all fluid. The film taken on Aug. 20, 1952, shows some reaccumulation of free fluid (he had been drained of 1,800 ml. on Aug. 12, 1,200 ml. on Aug. 14., and 1,300 ml. on Aug. 18, 1952). The picture taken on Oct. 6, 1952 shows massive reaccumulation of fluid and a thoracentesis immediately following yielded 2,000 ml. of fluid.

At the time of discharge, this patient was edema-free, but subsequently the right pleural cavity had to be tapped 25 more times at intervals of three to seven days to prevent recurrence of peripheral edema and hepatic passive congestion. Six more thoracenteses were practically dry taps, resulting in withdrawal of only 4 to 30 ml. of fluid. After having been free from recurrence of hydrothorax and from peripheral edema for several months he submitted to cardiac surgery for mitral valvuloplasty in Boston. He died unexpectedly one hour following surgery.

Case 3. R. W. was 7 years old when he came to us in the summer of 1948 with massive anasarca resulting from glomerulonephritis. He had had several bouts of upper respiratory tract infection during the preceding two winters. Edema set in 15 months before admission; it had been preceded for one month by a noticeable change in disposition. There were intermittent spontaneous remissions and exacerbations of edema, but following an episode of vomiting, diarrhea and fever two months prior to admission, edema became persistent and progressively worse. There had been no gross hematuria.

Upon admission the child was almost unbelievably anasarca (weight 49.2 kg.). The face was full; the palpebral fissures narrowed to mere slits. There was

![Figure 1: Graphic representation of data on patient J. P., male, age 3½.](image)

![Figure 2: Graphic representation of data on patient F. J., male, age 52.](image)
deep edema up to the shoulders and axillae. The abdomen (circumference 39.5 inches) was grotesquely distended with free fluid, with some brawny pitting edema of the abdominal wall itself. The scrotum and penis were practically one contiguous globular mass. The legs were so tremendously and tightly swollen with edema that the child had to spread them apart to be comfortable. The complexion was deathly pale.

Admission urine showed 3 plus albumin with a few granular casts, 2 to 5 red blood cells and 5 to 7 white blood cells per high power field. The red blood cell count was 3.4 million and hemoglobin 11.7 Gm. Blood urea nitrogen was 10.5 mg. per 100 cc. The total protein was only 4.03 Gm. and the albumin 1.44 Gm. per 100 cc., despite the preceding high protein feeding. Cholesterol was 492 mg. per 100 cc.

Despite meticulous care in instituting the diuretic regime and in spite of the frequent and repeated doses of mercurial diuretics, 72 per cent of the excess chloride in the body by-passed reluctant kidneys and was removed by mechanical drainage of the peritoneal space (table 2). Figure 4 shows graphically the weight change in relation to paracentesis. Between taps, the weight remained stationary, but was brought down step-ladder-wise with each tap. Clinically, while the weight remained stationary between paracenteses, signs of peripheral edema diminished while fluid reaccumulated in the abdomen.

This boy went home entirely free of peripheral edema on the thirty third day, the weight then being 28.7 kg. (edema weight loss of 22.5 kg.). He was seen eight months later at which time he was free of edema and the urine showed no albumin. He had mild recurrence of edema once or twice subsequently for about a year, but he is, at the time of writing, a normally developed 13 year old boy, apparently recovered from glomerulonephritis.

Case 4. K. H. was a 62 year old man who had aortic valvulitis with stenosis and insufficiency attributed to preceding rheumatic infection. He had complained of exertional dyspnea for many years, and had known of a heart murmur for about eight years prior to hospital admission in August, 1951. On two previous admissions in 1950 and July 1951, under a different service, right hydrothorax had been found but thoracentesis was not done. He had complained of orthopnea on each admission; there was hardly any peripheral edema.

During this admission he incidentally developed an acute cholecystitis for which cholecystectomy was performed and from which he recovered uneventfully. Postoperatively the right pleural space was drained repeatedly at intervals of two to four days. The data are shown graphically in fig. 5. As in the preceding three cases, relatively small amounts of sodium were coaxed out through the kidneys. Most of it was removed mechanically via the sump.

Following discharge from the hospital, K. H. underwent further repeated thoracenteses at intervals of one to three weeks for a period of five months. He was entirely relieved of dyspnea during that period and felt well. Sudden death
F. R. SCHEMM AND A. A. CAMARA

HISTORICAL COMMENT

Drainage of abnormal accumulations of fluids in body spaces dates back to antiquity. Hippocrates performed it in his day, with often fatal results and the procedure was practiced through the ages and recorded by Galen, Aurelianus, Celsus, Aetius, Avicenna and other notable medical men of history. Among the first to perform abdominal paracentesis during the modern era were Alexander Monro (secundus) and William Hewson, around 1770. Adolf Kussmaul pioneered in the procedure of thoracentesis in modern times, his first operation having been performed in 1888.

The high mortality rate caused by sepsis following either procedure in the early days is understandable. Following the era of Pasteur and Lister, however, abdominal and thoracic paracenteses were not entirely absolved from immediate or remote danger. Capps concluded from his experimental investigations that sudden death following thoracentesis was caused by a pleural reflex resulting in direct cardiac inhibition. Other causes of immediate fatalty following thoracentesis were attributed to air embolism, pulmonary thrombosis following lung puncture with or without embolism to the heart or brain, and acute pulmonary edema.

Literature on the deleterious effects of abdominal paracentesis is confined mainly to observations in ascites associated with cirrhosis of the liver. Sustained and progressive lowering of blood pressure terminating in shock following drainage of large amounts of ascitic fluid is abundantly described in the literature. Lichtman mentions the not too frequent rapidly downhill course following a paracentesis. He attributes this to "... the loss of large amounts of protein, electrolytes, water and other vital substances." Cantrow warned against a steady fall in serum protein concentration due to dilution of blood plasma. On the other hand, Miccoli and Ferroni in 1940, reporting studies on cirrhotics conducted over a period of two to eight months, showed that withdrawal of ascitic fluid produced only a moderate reduction in the protein content of the blood in the majority of instances. Restoration of previous levels usually occurred within a week.

Eisenmenger and co-workers, who carried out extensive electrolyte studies on patients with cirrhosis of the liver, reported that in some patients aspiration of ascitic fluid causes an immediate and precipitous fall of serum sodium followed by gradual return to preaspiration levels. Gabuzda and co-workers as well as Nelson and his co-workers have reported the same observations. The latter emphasized the fact that such a drop in serum sodium following paracentesis is more apt to occur in patients with advanced cirrhosis of the liver.

A careful search of the literature has failed to reveal studies of the effect of repeated aspiration on blood proteins and electrolytes in congestive heart failure and glomerulonephritis. Nevertheless, the argument has been advanced that repeated paracentesis or thoracentesis might have the same deleterious effect in those two conditions as in cirrhosis of the liver.

DISCUSSION

The mechanical drainage of free fluid from serous cavities in congestive heart failure and in the nephrotic stage of glomerulonephritis has heretofore been advocated only for relief of compression of the lungs or of oppressive distension of the abdomen. Usually abdominal paracentesis is performed in nephritic patients with ascites in the hope, often vain, that "decompression" of the kidneys will initiate a diuresis.

Observations on the present series of patients have been described in detail in order to introduce the concept of the sump and to show that utilization of this phenomenon can be of tremendous help in the management of patients with absolutely or relatively intractable edema. In these cases, the stimuli directing the renal tubules to conserve sodium and water (deshyocorticosterone-like and antidiuretic hormones) are presumably so intense as to make elimination of excess body sodium and water

Fig. 5: Graphic representation of data on patient K. H., male, age 62.

came six months later under circumstances not known to the writers.
via the kidneys impossible. Accordingly, to afford relief of edema in such cases, the kidneys have to be by-passed.

The clinical picture of a diminishing peripheral edema while fluid reaccumulated (with no weight increase) in the sump just aspirated; the series of chest x-ray films showing refilling of the sump following aspirations; and finally, the demonstration that a tagged material (D₂O) administered subcutaneously into an area with peripheral edema found its way into the body space involved, all prove unequivocally the existence of the sump phenomenon in the patients described.

In describing the effects of paracentesis in cirrhosis of the liver, Nelson made the same observation that following paracentesis, peripheral edema often decreased during the post-aspiration period while ascites reaccumulated, with no change in body weight. This phenomenon has been observed by many clinicians in patients subjected to periodic aspirations because of ascites due to advanced heart disease or cirrhosis of the liver. There was relief of the tense, shiny distension of the lower extremities after aspiration as the abdominal fullness was recurring. However, the significance of the observation was not grasped, due to the failure to weigh the patients and to appreciate that a shift of edema fluid had occurred. The practice of removing several gallons of fluid only when absolutely necessary was the common practice then, and still is, even now.

It was observed in some of our cases that in order to forestall a recurrence of peripheral edema after dry weight had been attained, it was necessary to aspirate the sump at intervals for some months. Failure to do so after 500 ml. to 1500 ml. had collected in the sump was followed by the reappearance of peripheral edema, indicating that seepage from the sump was occurring in the opposite direction, i.e. out from the sump into the interstitial spaces.

Thoracenteses and paracenteses were performed 77 times in this series of 14 patients, and in no instance was the procedure followed by immediate or remote untoward reactions or complications. In those cases with massive hydrothorax (2800 ml. drained in one instance), the precaution of partial air replacement during the procedure, after aliquots of 400 to 600 ml. had been drained, was observed. No striking dyspnoea nor unrelievable pain developed, and no evidence of "pleural reflex" with or without cardiac inhibition was encountered. The procedure of air replacement prevented, of course, any undue negative pressure within the thorax, and produced a hydropneumothorax in which there was rapid absorption of the air and no excessively rapid refilling of the thorax with fluid.

Tight abdominal binders applied over large packs after abdominal paracentesis prevented the occurrence of shock or untoward disturbances even after drainage at one sitting of huge amounts of ascitic fluid amounting in one instance to 33.5 liters in one of our patients (not in this series), a man 62 years of age with arteriosclerotic heart disease, a record amount not exceeded since, in our experience.

In this series was a 7-year-old boy, R. W. with a body weight of 26.4 Kg (when edema-free) from whom more than 8 liters of abdominal fluid were drawn at one sitting without inducing any untoward reaction.

In almost all instances of abdominal paracentesis a prophylactic antibiotic (penicillin unless contra-indicated) was administered the day of and the day following the procedure. If drainage persisted beyond 48 hours the antibiotic was continued without interruption until the paracentesis site was closed. Similar protection was offered when aspiration of a pleural cavity was performed frequently in a frail or poorly nourished patient.

The fact that neither hypoproteinemia nor hyponatremia developed after repeated drainage of sump fluid in this series of cardiac and nephritic patients needs to be emphasized. Even though significant amounts of protein were removed by repeated aspiration, protein synthesis by the liver evidently was sufficiently rapid for replacement so that hypoproteinemia did not develop in those with normal serum protein concentrations and there was no further lowering of serum protein values in those who had hypoproteinemia at the beginning of treatment. It should be recalled
that the concentration of protein in the edema fluid, including that of chest and abdominal fluid of such patients, is usually not as great as that usually encountered in cirrhotics. This, along with the fact that protein synthesis by the liver in cirrhotics is necessarily impaired, may account for the difference in our own experience from that of others\textsuperscript{11, 12, 13} who have noted a sharp decline in serum protein concentration following abdominal paracentesis in cirrhotics.

With the exception of one (J. P.) who developed a transient lowering of plasma sodium concentration, none of the patients in our series developed hyponatremia as a result of repeated aspirations. This is easily understood if one remembers the fact that the fluid removed mechanically is but an aliquot of the total extracellular fluid, and, therefore, contains water and electrolytes (principally sodium) in the proper proportions.

Hyponatremia developing as an immediate, transient complication following paracentesis in cirrhosis of the liver cannot be denied. It is reported in well-documented and detailed electrolyte studies.\textsuperscript{11, 12, 14} It will be noted from these reports, however, that this complication is more apt to occur only in the far advanced cirrhotics.

A brief review of the current concepts of electrolyte and water metabolism as well as of edema formation might be of help in understanding the occurrence of this reported complication. As Newburgh\textsuperscript{14} has pointed out in an excellent review of the subject, the extracellular fluid in health is characterized by constancy of concentration of each of its inorganic constituents, and also by relative fixity of its fluid volume. These two features are under separate control, working through the kidneys which are the final guardians of the “milieu interne.” The reabsorption of sodium is controlled by an adrenal cortical hormone, but the body content of water is governed by the antidiuretic hormone through regulated reabsorption of water. Constancy of concentration of sodium is maintained in health, so that there must be coordinated activity of these functions, causing the rate of absorption of sodium to keep pace with the reabsorption of water. At the present stage of our knowledge, we do not know the ultimate nature nor the seat of this coordinating mechanism.

In edema, whether it be the anasarca of heart failure, the nephrotic edema of glomerulonephritis, or the ascites of cirrhosis of the liver, the organism has become abnormally geared to maintain an excessively large volume of extracellular fluid. However, except in the far-advanced or deteriorated cases, coordination between the two factors which govern renal tubular reabsorption of sodium and of water is still maintained, as evidenced by persistence of normal concentration of sodium in the extracellular fluid, including edema fluid. In the far-advanced cases, be they cardiacs, cirrhitics, or nephritics or patients with a combination of severe heart, liver and kidney damage, whatever the primary difficulty might be, the stimulus for maintenance of an abnormally large volume of extracellular fluid is so intense that when measures such as dietary sodium restriction and mercurial diuretics are instituted for the prevention of further accumulation and for the elimination of excess body sodium, normal sodium concentration in the extracellular fluid is sacrificed for volume; i.e., such patients do lose body sodium, but retain disproportionately excessive amounts of water, resulting in the picture of edema with hyponatremia. In these patients, loss of sodium with resulting hyponatremia may and does occur from administration of mercurial diuretics, due to abnormal losses through the gastrointestinal tract with diarrhea and/or vomiting, or due to aspiration of thoracic or abdominal fluid accumulations.

What needs to be emphasized here is the fact that in such far-advanced cases, it is not the aspiration of fluid, but any loss of sodium, which can initiate the development of hyponatremia. This fact is often overlooked in attributing hyponatremia solely to aspiration.

Evidently, none of the patients in the series of cardiac and nephritic patients described in this paper falls in the above category. Our patients, with the exception of one who had a transient hyponatremia, all maintained a
normal concentration of plasma sodium after repeated aspirations and while being maintained on a diuretic regimen with moderate dietary sodium restriction.

**Summary**

Fourteen cases of massive peripheral edema with hydrothorax and/or ascites have proven resistant to the usual methods, but have been relieved by successive aspirations of fluid from either the pleural or peritoneal spaces. The repeated aspirations did not lower the plasma sodium nor protein level; nor were they followed by immediate postaspiration diuresis.

From 27 to 99 per cent of the sodium and water eliminated, to achieve a dry weight, was removed mechanically, rather than coaxed out via reluctant kidneys. In some instances dry weight was achieved with a weight loss which corresponded very closely to the weight of the aspirated fluid. In the intervals between aspirations, the signs of fluid in the chest or abdomen increased as the signs of peripheral edema decreased without any change in total body weight. The fluid of the interstitial space and of the untapped space appeared to seep readily into the sump from which the fluid had just been removed.

In most instances after dry weight was reached, the usual regimen, which had been in force from the beginning but had proven ineffective, was effective in preventing reaccumulation of fluid. However, in some instances, it was observed that in order to anticipate or prevent a recurrence of peripheral edema and either hydrothorax or ascites, it was necessary to aspirate the sump at intervals for some months. Failure to do so after 500 ml. to 1500 ml. had collected in the sump was followed by the reappearance of generalized edema, suggesting that seepage from the sump was occurring in the opposite direction.

The advantages of the recognition of the sump phenomenon are obvious in those patients in whom, because of intense hormonal stimulation of the renal tubules to conserve sodium and water, loss of edema is impossible in spite of proper attention to the usual regimen. As a means of by-passing reluctant kidneys the utilization of the sump phenomenon has proven simple, safe and effective clinically in some very obstinate cases.

**Summario in Interlingua**

14 casos de massive edema peripheric con hydrothorax e/o ascites se provava resistente al methodos usual sed eseva alleviata per successive aspirationes de fluido ab le cavitates pleural o peritoneal. Le repetite aspirationes non abassava le nivello plasmatic de natrium o proteina. Illos etiam non eseva sequite per immediate diuresis postaspirational.

In alicun casos le peso del fluido aspirate amontava quasi completamente al excesso que le patiente debeva perder pro restituer su peso non-edematose. In le varie casos, inter 99 e 27 pro cento del natrium e aqua edematose eseva assi eliminate per medios mechanic (e non fortiate a quitar le corpore via le renes re-luctante). Durante le intervallas ab un aspiration al altere, le signos de fluido in le thorace o abdomen accresceva in proportion al reduction de signos de edema peripheric durante que le peso total del corpore non se abassava. Le fluido interstitial como etiam le fluido in spacios non-aspirate pareva filtrar sin dificultate a in le spatio ab que le fluido habeva justo essite removite. Le spatio, de facto, ageva como un specie de "cisterna collector." In le majoritate del casos le peso non-edematose—quando illo eseva establite per le aspirationes de fluido—poteva esser mantenite per medio del regime traditional que—ben que in uso ab le initio—habeva previemente remanite inefficace. Nunc, nonobstante, illo serviva efficacemente a prevenir le re-accumulacion del fluido. Sed in alicun casos il eseva observe que pro prevenir le recurrentia de edema peripheric plus hydrothorace o ascites, il eseva necessari aspirare le "cisterna" a intervallas regular durante un periodo de plure menses. Si isto non eseva facite quando un quantitate de 500 a 1500 ml eseva accumulate in le "cisterna", le consequentia eseva un re-apparition del edema general. Isto indicava que il habeva un filtrage de fluido ab le "cisterna" in le direction inverse.

Le avantages del recognition de iste pheno-meno del "cisterna" es obvie in le caso de
patients in le quales le plus stricte observation del regime usual non pote resultar in le suppression del edema proque un intense stimulation hormonal fortia le tubulos renal a conservar natrium e aqua. Como medio de circumvenir le obstaculo de renes reluctant, le utilization del phenomeno del "cisterna" se ha provate simple, sin risco, e clinicamente efficace mesmo in casos que esseva muito obstinate.

REFERENCES


4 —, Ibid., p. 560.


The Relief of Resistant Edema by Utilization of a Sump Phenomenon
FERDINAND R. SCHEMM and AUGUSTO A. CAMARA

Circulation. 1955;11:411-421
doi: 10.1161/01.CIR.11.3.411

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1955 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/11/3/411

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/