CLINICAL OBSERVATIONS ON THE PORTAL SYSTEM IN LARGE CONSUMERS OF SUGARS

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According to U. S. Department of Agriculture statistics, sugar consumed in United States was 7 pounds per capita in 1821. In 1925 it reached 121 pounds per capita. The consumption of so much sugar is a radical departure from dietetic habits of the past. Has human economy accommodated itself to the new form of carbohydrates?

During the latter part of the 19th century, sugar had come to form a considerable part of American dietary, dietetic observers then advised against its use in hepatic cirrhosis and allied diseases. As per capita consumption of sugar grew apace, evidences of its toxicity multiplied. The portal system consists of veins draining the gastro intestinal canal, spleen, gall bladder, and pancreas, and of accessory portal veins. All the component veins unite to form the portal trunk which divides into two branches, one entering the right and the other the left lobe of the liver. As they course through the substance of the liver they break up into smaller vessels and capillaries forming interlobular and intralobular veins. At this stage branches of the hepatic artery, which have been associated with intrahepatic ramifications of the portal vein, become confluent with them forming sublobular veins and these in turn combine to form hepatic veins which enter vena cava inferior just below the diaphragm.

Portal circulation in its course toward the liver is not a closed system. There are several outlets connecting it with the caval system. Under normal conditions, very little, if any portal blood enters either vena cava or any of its tributaries, but when lumen of the portal vessels are narrowed from any cause, blood seeks an outlet through one or all three main collateral vessels, with the usual result that they become enlarged to accommodate the additional influx. Varices of esophagial and diaphragmatic veins may then form at and above the cardia and often give rise to sudden and fatal hemorrhages. Large varicosities in hemorrhoidal plexus giving rise to severe hemorrhoids, may be due to the same cause, or collateral circulation may develop through para-

umbilical into superficial abdominal, epigastric and thoracoepigastric veins distending them and converting them at times into varices. In the case of portal cirrhosis, this is the most desirable consummation.

In some cases of portal cirrhosis and in other cases where diagnosis of cirrhosis could not be made with certainty, venules are found in the thoracoabdominal region, with some regularity as to position, arrangement, and form. They usually appear at the fifth interspace or below, rarely they may be found as high as the fourth interspace. In a general way they parallel and overlie insertion of diaphragm in anterior thorax and extend as far as the axillary lines. Their distribution at times is symmetrical, suggesting an arch formed of filigree work of delicate rose color, fine stroke and outline. In well marked cases the arch may become a circle, commencing at the fifth or sixth interspace at the sternal line on both sides of the chest, paralleling and including costal insertion of diaphragm as far as mid axillary line and thence curving forward and downward, reaching within 2 centimeters of the umbilicus on both sides. I have observed two such cases out of more than 75. Usually the number, extent, and arrangement of venules are influenced by the size and degree of involvement of the liver. These venules may be less marked on one side than the other, or may be entirely absent on one side. It is possible to estimate the size and degree of involvement of the left and right lobes of the liver from the number and size of venules present on the respective sides of the thorax. They are present more often and are better marked on the left side than on the right, which conforms with the clinical findings that the left lobe of the liver is more often involved and farther advanced in sclerotic processes than the right lobe.

What is the significance of the venules? In some well marked cases of cirrhosis they are absent, in others, equally well marked, they are present. They are also present in cases where portal cirrhosis could not be diagnosed.

I paid careful attention to patients with venules, and soon had a theory. Almost all cases having well marked venules gave a history of consumption of large amounts of sugar over a long period. They were sugar addicts. Under a diet restricted only in matter of sugars, syrups, molasses, etc., almost without exception their subjective symptoms improved and venules became less marked and in some cases completely disappeared. Depletion by salines and fasting for a few days had similar effects. In a few advanced cases of cirrhosis they were uninfluenced, although the patients' subjective symptoms were relieved and improvement in general condition was the rule. A few illustrative cases follow.

L. H., 1926. Since the time he was able to climb chairs and reach the sugar bowl on the dining table, he has consumed sugar by teaspoonsfull at every chance he had. At 5 he had well formed symmetrically placed venules on his lower anterior chest. He was suffering from symptoms of hepatic insufficiency, had visible veins on the abdomen, and an enlarged liver (four inches). Sugar was prohibited but no other restrictions were made. He returned for examination two years

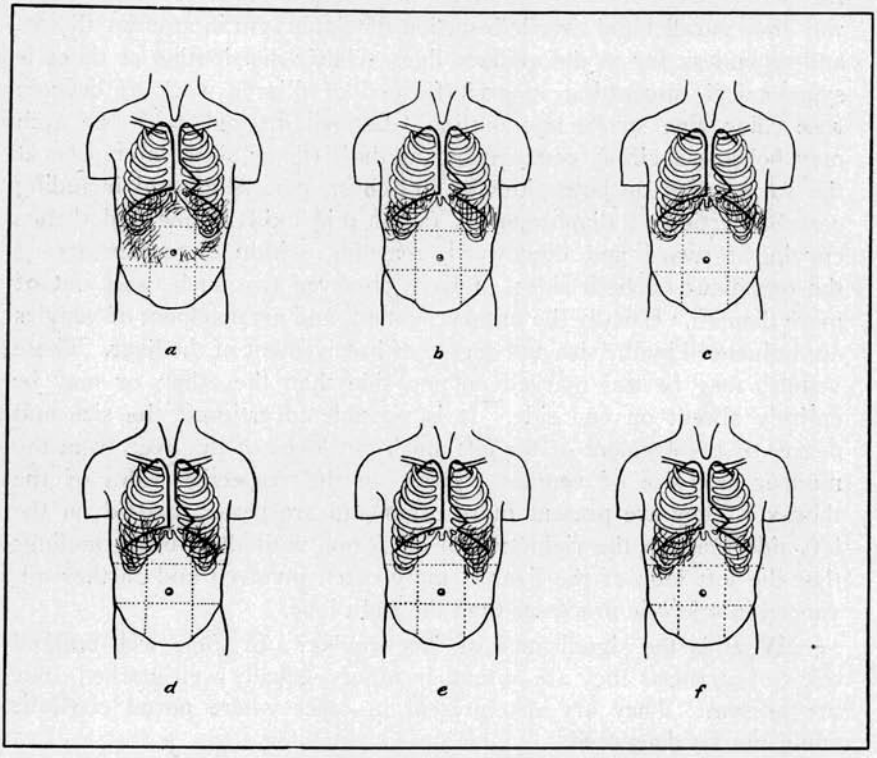

FIG. 1. *(a)* **E. A. as he appeared October 16, 1929; sugar was omitted from his diet.** *(b)* **E. A. as he appeared May 13, 1930.** *(c)* **An ordinary case of venules.** *(d)* **A shrub or dwarf tree type of venules.** *(e)* **Left-sided venules.** *(f)* **Right-sided venules.**

later. Venules had disappeared, liver was one inch smaller, superficial abdominal veins were still large. This child has cirrhosis but progress is evidently checked. He had gained four pounds during last eleven months, liver was reduced one-half inch and he is in better health.

E. A., 50, a farmer, consulted me on October 16, 1929. He had been in poor health for many years, had lost 45 pounds in weight in the last few years. He was pale, coughing and expectorating pro-

fusely and looked spent. Examination of lungs revealed tuberculous fibrosis of both apices, the Von Pirquet test was positive; one sputum examination, negative for tuberculosis; low tension pulse, heart lacked tone. Lower margin of right lobe of liver reached the level of the umbilicus and that of left was slightly higher. Inspection revealed the most marked case of venules I had ever observed; they formed a complete circle, broken only at the umbilicus. A drawn circle would have bisected each venule at about a right angle. Superficial abdominal veins were all dilated, indicating that the portal system was seeking relief, and obtaining it by diverting part of its contents into the caval system, through para-umbilical and superficial abdominal veins. The patient was a sugar addict of the most aggravated type—he ate syrups and molasses, vast amounts of them, every day. Sugars, syrups, and other sweets were prohibited but there were no other dietetic restrictions. Carlsbad salts and iron were prescribed. Change for the better was surprising. He gained seven pounds in thirteen days, and visibly improved in appearance and spirits. In seven weeks the venules had grown faint and the liver was slightly reduced in size. Cough and expectoration were much diminished. He spent the following winter in Arizona, returned May 13, 1930, when examination revealed that the liver was much reduced in size and the lower half of the circle of venules had vanished. Superficial abdominal veins had become less prominent. He had increased 20 pounds more in weight. There were no evidences of clinical tuberculosis in this case.

I. J. M., 70, came under my care August 15, 1930, because of an acute gastro-intestinal upset. Venules were well marked—almost forming a semicircle; superficial abdominal veins, specially thoraco-epigastric veins, were much dilated. He was a chronic sugar addict. Depletion by catharsis and a brief period of abstinence from food reduced the size of veins and venules.

W. D., a retired locomotive engineer 63 years old, consulted me on April 12, 1930. He was a chronic diabetic having a well marked fibrosis in the left pulmonary apex. A sugar addict, he used three teaspoons full of sugar to each cup of coffee and four teaspoons full to each dish of cereal; blood sugar, 310 milligrams per cent. He had well marked venules on both sides and superficial abdominal veins were much enlarged. Liver, three inches palpable and hard, a diabetic cirrhosis. Sugar was ordered dropped from his diet, and the amounts of bread, potatoes, rice and milk were restricted. Six months later he returned, he had gained six pounds, looked well, was able to do several hours of work every day, his veins and venules were unchanged, his liver was hard, palpable and of same size as before.

Areas in which these venules are found are drained by perforating branches of the mammary, thoraco-epigastric, superior and inferior epigastric, abdominal cutaneous and para-umbilical veins. They neither individually nor collectively account for the venules which are enlarged capillaries that have developed in response to the reflected irritative or pathological influences emanating from the liver and overflowing from spinal roots, relayed over intercostal nerves to the area approximately overlying the organ where the irritative influences originated.

Among inveterate candy and fountain drink consumers, a gorge is not uncommonly followed by acute pain in the hepatic region. A girl of seven was subject to acute pain in the hepatic region. It was severe enough to keep her awake at night. She was an inordinate sugar eater. Recovery followed abstention from sugar.

J. B. S., 12 years old (April 9, 1931), has one bilious attack every month. He has a good appetite and is fond of sweets, eats in abundance; he soon becomes weak, nauseated, has sharp pains in the hepatic region, he vomits, has headaches, starves, there is mild acidosis with a large trace of indican and urobilin and a small trace of sugar in the urine. He gradually recovers, appetite returns, all discomforts from the hepatic region disappear to return again with the usual group of symptoms at the regular time. Removal of sugar from his diet caused a marked improvement and the next expected attack did not materialize. Medication consisted of calomel, bismuth, sodium succinate, and bile salts.

W. C., 17 (September 16, 1930), was greatly addicted to fountain drinks and at home was overly fond of sweets. He suffered from a feeling of fullness and discomfort in the hepatic region; soon the liver was tender and enlarged and acute distress supervened. For about one year he had dizzy and blind attacks of about one minute's duration simulating petit-mal, at times he came close to falling. The attacks usually came after meals and sometimes as often as twice a day. All these symptoms cleared soon after sugars were restricted.

In chronic cases the usual course is gradual decline accompanied by a feeling of tenderness on percussion, lack of endurance and ambition, distress and constant fullness in abdomen, unrelated to the time and kind of meals. These symptoms are usually due to hepatic insufficiency and the liver is atrophic in the majority of cases.

Sugars, syrups, molasses, and other sweets were toxic to them in the amounts they consumed. Besides they crowded important articles out of their diet and created a desire for stimulating foods and consequently their diet became deficient in some important factors.

These cases were treated by restriction or prohibition of sugars and in some balancing of the diets. Outside of salines and bile salts the treatment has been symptomatic. All those who remained under observation and treatment were greatly improved.

Venules are observed on the chest at various levels above diaphragmatic insertion. They approximately overlie sclerotic areas in the lungs. They differ in size, number, location, and arrangement from venules over the hepatic area.

In some cases of emphysema venules may be seen along the insertion of diaphragm. Cases of emphysema are rare in the author's practice who has never seen one with venules.

Chronic cholecystitis may at times be confused with hepatic insufficiency. The latter is of very common occurrence whereas the former is comparatively rare. Chronic cholecystitis with its vague digestive disorders, belching, sour eructations, sensation of weight and fullness in the upper abdomen, at times severe pains, chilliness, slight rise of temperature and local circumscribed tenderness should not be difficult to differentiate from hepatic incompetency. Hepatic incompetency may co-exist with cholecystitis and modify the symptoms.

Are the signs and symptoms described in this paper due to ingestion of excessive amounts of carbohydrates or due to irritative qualities of certain forms of carbohydrates, namely sugars? In a few cases I have seen venules with no history of sugar addiction and they confessed to the consumption of large amounts of potatoes, bread, and gravy. They were not well marked cases.

I feel justified in stating that in many cases venules are signs pointing to portal embarrassments and irritations, and in others to actual portal cirrhosis in the various stages of development. Well marked cases have generally been co-existent with sclerotic changes in the liver and the patients have invariably given history of sugar addiction of long duration.