THORACICO-ABDOMINAL VENULES, THEIR ETIOLOGY AND TREATMENT

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For many years I had observed dilated capillaries at various areas on the surface of the human body and speculated about their significance. My attention was particularly centered on the thoracico-abdominal region where they seemed to be found oftener and in greater numbers than elsewhere on the surface of the human body. In some cases there were only a few, scattered haphazard, while in others they were numerous and appeared in bizarre forms and symmetrical groupings, often suggesting an arch, formed of filagree work of delicate rose color, fine stroke and outline. In others they resembled metabolism record charts of a patient with rapid respiration. Some other cases simulated the maps of groups of river basins wherein all the tributaries have been delineated with all their meanderings and zigzaggings. In still others they looked like the ornamental Hebrew script or merely a flush occupying a narrow strip across

the lower anterior thoracic region corresponding approximately to the insertion of diaphragm and thoracic wall.

Individual enlarged capillaries are approximately 2 to 40 millimeters long and 1/5 to ½ millimeter wide. Their area of distribution in the thoracico-abdominal region lies between the fifth rib and the costal margin. They rarely extend as high as the fourth rib and as low as the umbilicus and pubis. In general they parallel and overlie the line of insertion of the diaphragm to the anterior thorax and extend laterally as far as the axillary lines.

The manner of their arrangement suggests the form of a Tudor arch in many cases, in exceptional cases the arch may extend downward and forward from the axillary lines to the umbilicus and make a complete circle. Rarely they may extend laterally down the abdomen reaching the pubis on both sides to form an ellipse. More often the circle and ellipse are fragmentary. In early and mild cases a blotchy erythema is discernible over the liver area which may develop to actual venules or may disappear under proper treatment.

The Portal trunks break up into smaller branches in the substance of the liver and the branches into capillaries at the periphery of the lobules in the space of Kiernan where they become confluent with the capillaries of the hepatic artery. This porto-arterial blood enters into the sinusoids in the substance of the lobule and reaches every hepatic cell. Then these vessels converge to the center to form the central lobular vein.

Inflammation in the portal vessels, even mild, may easily compress the portal capillaries in perilobular area in the space of Kiernan and may, if continuous, partially or completely obstruct them and cause varied degrees of portal hypertension or damming back of the portal blood. Less often the obstruction may be elsewhere in the portal system.

The mild degree of obstruction is compensated by diverting enough of the portal blood, through existing channels, to caval circulation. These anastomoses are through the coronary veins of stomach to aesophagal plexus at cardias; through the superior hemorrhoidal veins to middle and inferior hemorrhoidal veins at rectum and through the para umbilical veins to the veins of thoracico-abdominal region. The lower anterior chest and upper abdominal wall are in the portal domain.

In cases of greater obstruction the glisson's capsule of the liver is hypertrophied and highly vascularized and becomes adherent to diaphragmatic and parietal peritoneum finding outlet through diaphragmatic intercostal, peritoneal, mammary, axillary and other veins. In still higher degree of obstruction abdominal visceral and omental branches of the portal system are highly congested and become adherent to the parietal peritoneum communicating with peritoneal and abdominal plexuses of veins.

The circles and ellipses of venules I have considered to be due to communication between mesenteric veins of transverse, ascending and descending colon, and parietal peritoneal and superficial abdominal plexuses. Lower thoracic arch of venules always form part of the circles and ellipses, but the circle and ellipse were never found together. In the formation of circle and ellipse of venules, pressure on the vena cava may be a contributary factor, but these cases have atrophic liver, no ascitic fluid or any other cause of intra-abdominal pressure discoverable.

On careful examination and inquiry into the dietetic habits of the patients with the thoracico-abdominal venules, I found that, with rare exceptions, they habitually consumed excessive amounts of sugar.

Average annual per capita consumption of sugar in United States during the last ten years was 97 pounds. Sugar is a habit-forming food, the temptation to increased consumption being always present. For the use of this paper, those who consume habitually large amounts of sugar are to be called "sugar addicts." In many such cases blood sugar was not as high nor did urine contain sugar as often as one would expect. It was a surprise to find some of the sugar addicts with comparatively low blood sugar. Excessive sugar absorption may lead to hyperinsulinism. This may account for low blood sugar in some early cases. However, hypoactivity of the pancreas follows sooner or later, and in many of the older cases glycosurea is found to be due to portal hypertension and exhaustion of the pancreas. Reduction of the amount of the sugar consumed did not

bring on symptoms of hyperinsulinism, so far as my observation goes.

Sugar addicts with thoracico-abdominal venules are middle aged or older, suggesting addiction of long duration. Yet intensive degree of addiction and hypersensitiveness to sugar may bring it on earlier. I have seen one at the age of 5, one at 8, one at 14, and another at 18, and several in early twenties. There may be venules in thoracico-abdominal region when there is no demonstrable dilitation of the subcutaneous abdominal veins. The relationship between the venules and portal circulation is suggested because the reduction in sugar ingestion and other measures to relieve the portal circulation, reduces the venules both in size and number and may even cause their disappearance in some cases, reappearing when the addiction is resumed.

1. Each gram of sugar in the process of its conversion into glycogen binds three grams of water which leads to overfilling of the portal system and general plethora.

The following quotations from J. H. P. Paton's article in *The British Medical Journal* of April 29, 1933, on "Relation of Excessive Carbohydrate Ingestion to Catarrhs and Other Diseases" is pertinent to our subject.

"McClendon has demonstrated water retention after high glucose feeding in man. Ramsay has described occurrence of visible congestion and exudate in the retina of the patients whose carbohydrate intake is in excess of what they can tolerate and has pointed out that this is an index of the state of capillaries elsewhere in the body. . . . It may be concluded that the tendency to suffer from a variety of chronic catarrh and exudative processes is associated with intolerance of carbohydrates, especially sugar. As a corollary of this it may be assumed that even in persons of high tolerance a similar liability to chronic catarrhal inflammation will result if the absorption of carbohydrates is sufficiently excessive."

Paton gives statistics of a boarding school for young women on catarrhal diseases of more than seven days' duration.

Average incidence
1904–1913 2.6 Per cent.
1914–1917 0.8 Per cent.

He sees a connection between the reduced consumption of sugar during the Great War and reduction in incidence of catarrhal diseases and finds the degree of prevalence of the same class of diseases proportionate to the amount of carbohydrates, especially sugar, consumed.

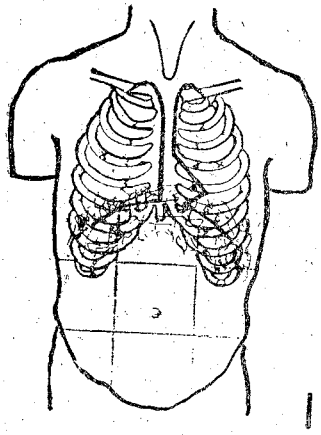
2. In addition to possible toxicity of sugar itself, there is the indirect action due to its decomposition and synthetization products. Sugar ingested may not all be absorbed but leave a residue to ferment in the lower ileum, forming acetic, lactic, valeric, oxalic, and other acid substances. Arrival of the contents of ileum at the more complex chemical environment of the colon is followed by formation of still other substances, fluid and gaseous, of which a certain amount is absorbed and reach portal circulation. It is reasonable to assume that this factor plays a part in the development of hepatic cirrhosis.

Over ten years ago Anthony Bassler expressed himself on this phase of the subject as follows: "I have yet to see one instance where at operation, whether the individual was alcoholic or not and a portal cirrhosis was disclosed, there was not present and easily demonstrable a saccharo-butyric toxemia in the intestional canal." On a communication to the author, he reaffirmed the above statement as follows: "I remember the quotation well and have not changed my opinion to any extent, in fact, had there been any doubt, subsequent clinical experience could not have done other than bring me to the belief that moderate degrees of liver cirrhosis are not due to alcohol except as it enhances a saccharo-butyric toxemia."

3. Excessive consumption of sugar and other carbohydrates, in many cases, engenders serious deficiency in diet which may play a direct part in the development of cirrhosis of the liver, as has been suggested by C. L. Connor and others for alcoholic cirrhosis of the liver.

4. There is still another class of cases, small in number, who suffer from carbohydrates intolerance, irrespective of the kind ingested, who could not be called excessive consumers of carbohydrates; yet have the same signs and symptoms.

The number of my clinical record of the patients suggesting the causal relationship of sugar addiction to thoracico-abdominal venules had reached about 80 when I noticed that many of my cases



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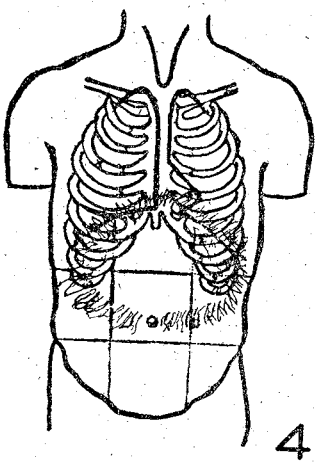

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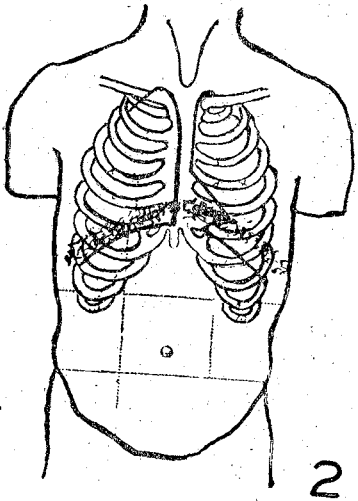

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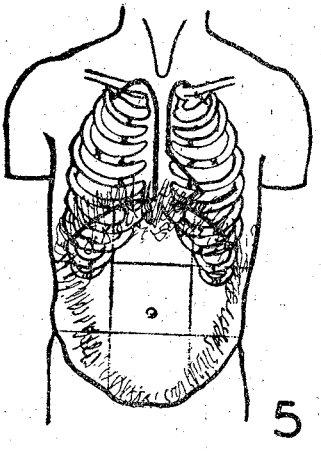

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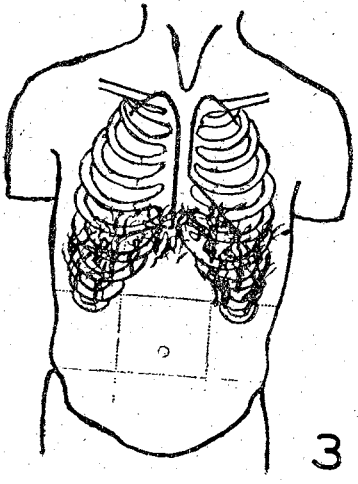

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had chronic non-active or healed tuberculous areas in their lungs. In subsequent cases with careful examination I found the presence of healed tuberculous areas to be more frequent than I had at first believed to be the case. I have not since seen a well marked case of thoracico-abdominal venules without the pulmonary lesions.

Under the treatment directed toward the relief of portal embarrassment, the general improvement in the patient's condition was more prompt than would have been possible in a case of clinical tuberculosis. To my definite knowledge only one of my cases developed into clinical tuberculosis. I have come to regard as benign or healed the tuberculous lesions of the lungs co-existent with well marked thoracico-abdominal venules.

Thoracico-abdominal venules mean: A person, with comparatively small liver, who ingests excessive amounts of sugar or other carbohydrates, with a variable degree of cirrhosis of the liver, with healed tuberculosis of the lungs, chronic catarrh of the mucus membrane with the history of frequent exacerbation because of sugar debauch and bad weather. In addition there may be excess of urobilin in the urine, loss of weight, tenderness to percussion on the liver, general depression, inertia, toxemia, feeling of weight in the hepatic region, continuous gaseous distention, brown rings around the eyes, persistent bad breath, bilious attacks, headaches after meals, etc.

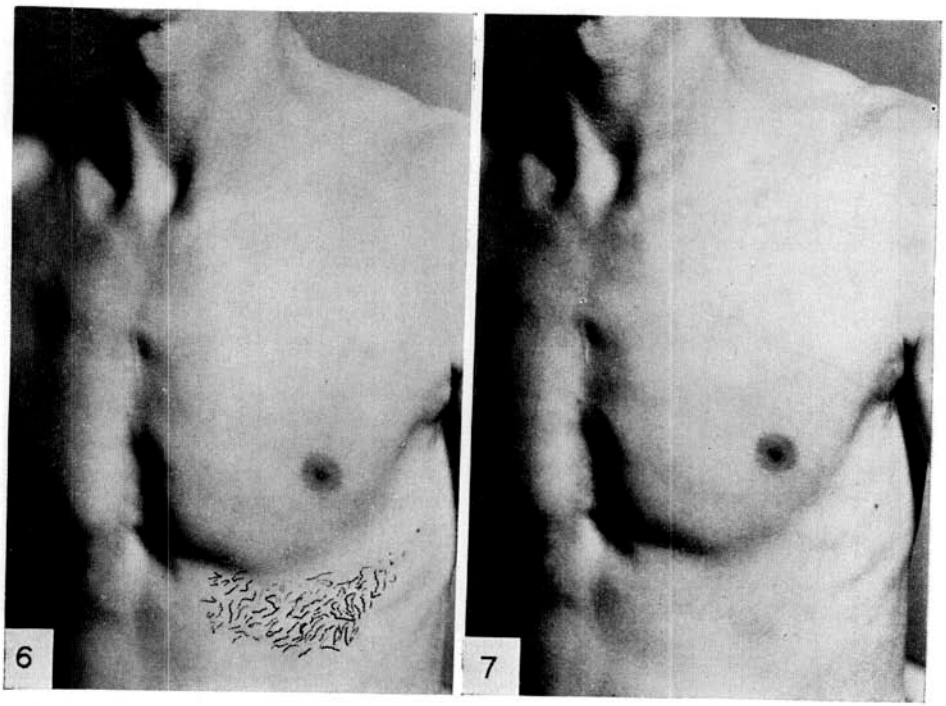
Ninety-four of my cases have been studied with special care, 104 somewhat casually. I have seen several hundred more cases during the last 15 years, but in their records only a brief mention of the special syndrome was made, unless the case presented unusual features.

With the portal obstruction compensated by new routes to the caval system, the patient is relieved of his symptoms and his life is not shortened nor health depreciated materially on condition that he live on a balanced diet, and limited liquids, at about the minimum physiological requirements. Salines, bile salts, calomel and sod salicyiate are useful drugs in the treatment of this class of cases.

I have seen the venules in various stages of their development. I have been lead to think that excessive consumption and intolerance of carbohydrates, especially sugar, is one of the factors in the development of cirrhoses of the class I have described.

Were it not for the accumulating evidence of the carbohydrate toxicity and the favorable results obtained from the application of the theory put forth in this paper, this report would not have been made.

S. T., farmer, 62 years old, who consulted me Oct. 12, 1932, has felt tired all his life. For years he had lived mainly on carbohydrates, including sugar. He has small liver, low blood pressure, and irritable colon. Never constipated, he has



Note to above: Figs. 6 and 7 are from the same photograph, with venules traced in ink on fig. 6 for this reproduction.

colicky attacks with frequent loose stools. He has an old tuberculous lesion in the left apex to the third rib anteriorly. He has the venules in lower anterior chest and on the right side they extend to the right ramus of the pubis forming about ½ of an ellipse.

Aug. 26, 1934. C. A. L., farmer, 56 years old, has a complete ellipse of the venules, the longer diameter reaching from the lower thorax to the pubis. This is one of the two unbroken ellipses among over 500 cases of venules I have seen. He has been an extreme type of sugar addict all his life, heavily sugaring everything he ate. Urine was loaded with sugar, blood sugar 165 milligram per cent, three or four sour and loose stools a day. He feels worse if bowels move less often. Liver atrophic and tender to moderate percussion.

Nov. 16, 1934. Mrs. E. N., sugar addict. There are ample evidences of chronic tuberculous lesions in her lungs with slight or no activity. Liver dullness two inches; venules over lower anterior chest where the diaphragm fuses into the internal surface of the chest wall are arranged compactly in a narrow strip resembling Hebrew or Arabic script.

June 24, 1935. H. P., 15 years old. At the age of seven he had an attack of nausea, vomiting, fever, tenderness under right costal arch and grayish stools for several days. He had a similar and severer attack the following year. Since then he has had the attacks at more frequent and irregular intervals. There are non-active tuberculous lesions in the upper chest. He is a sugar addict, liver dullness 1½ inches. The venules are found in lower anterior thorax, being more marked on the left side.

1935. J. B. McG. Age 44. Teacher, who had no history of sugar addiction, yet he had a well developed arch of venules in lower anterior thoracic region and signs and symptoms that accompany it. I considered this a case of carbohydrate intolerance, balanced his diet, reducing his carbohydrate intake. He felt better within a short time and after several months no venules could be made out in his lower anterior thoracic region.

May 6, 1938. A. C. A. 68 years. Retired, had chronic catarrh for many years. His nose and naso-pharynx filled up with catarrhal exudate, so that he had to syringe them with normal saline or alkaline antiseptic four or five times a day to be able to breathe through his nose. He has thoracico-abdominal venules and confessed to using sugar in almost every food he ate. His diet was balanced, sugar being greatly restricted. In a short time he improved considerably. About one month ago he reported that he is getting along comfortably with one treatment a day.

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