

minum bronze. The whole apparatus is then fitted to the foot before tempering; the bars along the inner and outer side and the anterior cross-bar permit a good deal of actual support to be given locally where it is needed, as in cases where it is desirable to support the anterior arch. After fitting, the apparatus is given a spring temper and is covered with leather, a thin pad of felt being fixed between the leather and the pressure plate, if necessary.

The apparatus is not intended for flat-foot or for feet with any appreciable bony change, nor for heavy patients. There are also cases of uncomplicated pronation which may for a time need a rigid support; for such cases, of whatever class, a plate on the lines of Whitman's, but furnishing two points of support externally, would seem to meet all requirements; often a simple arched steel plate without external support will suffice.

For the ordinary pronated foot, however, the apparatus here presented seems, so far as we have used it practically, sufficiently light and comfortable, checks vicious standing and tends constantly to correct pronation, while to some extent permitting the normal muscular use of the front foot, and allowing the rotation of the foot incident to its normal use. In this way it favors a normal gait, and facilitates the active use of the foot, which is necessary if we are to attain a cure, not merely the alleviation, of the pronated foot.

In closing, it may be said that this paper, in so far as it relates to treatment by apparatus, is a preliminary one only.

EPIDEMIC JAUNDICE.¹

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"JAUNDICE is strictly never an individual disease, it is merely an affection or a symptom of disease." These words of Austin Flint are as obvious and axiomatic to-day as when they were uttered; but as it is a symptom dependent upon so many and various conditions, it is impossible to wholly discontinue it from pathological nomenclature.

The object of this paper is to place upon record a few facts pertaining to an epidemic of jaundice which prevailed in Calumet and vicinity during the summer and fall of 1897. Jaundice in this case and in this paper will be referred to as meaning catarrhal cholangitis without the presence and irritation of biliary calculi.

The jaundice produced is what is known as hepatogenous jaundice. Professor Fitz, in the "American Text Book of the Theory and Practice of Medicine," mentions the opposite views of Ponsick and Stadelmann. In this, Ponsick elaborately demonstrates how hematogenous jaundice occurs, and Stadelmann demonstrates very conclusively that hemotagenous jaundice never occurs. These speculations are foreign to the purpose of this paper, and are mentioned merely in passing for the benefit of those who may be disposed to enter more elaborately into possibilities concerning the etiology.

For the purpose of this paper, hematogenous jaundice as it appears in typhoid fever, pyemia, cancer, aneurism, etc., will not be referred to except to exclude it from consideration.

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Although the term "epidemic jaundice" seems to be well recognized in literature, yet it was entirely a new term to all the physicians in Calumet. We have mentioned the epidemic to a number of professional men visiting us and to a number whom we have visited, and almost invariably the term has seemed to strike the auditor as something unusual if not entirely new. Strümpell says, in his well-known work: "Experience has repeatedly shown that often, particularly in spring and fall, this disease, catarrhal jaundice, comes like an epidemic," and in almost every volume of Sajous' "Annual" is a record of an epidemic of jaundice occurring somewhere. Professor Graham, of Toronto, in the recent "American System of Medicine," by Loomis and Thompson, says: "Jaundice occurs frequently in epidemic form," and mentions records of such epidemics confined to one or two villages, or to certain garrisons.

In looking through the literature of jaundice, simple catarrhal cholangitis, either sporadic or epidemic, is attributed by the authors, almost unanimously, to a gastro-duodenal catarrh. This catarrhal condition of the mucous membrane of the duodenum causes a catarrhal inflammation and thickening of the duodenal end of the common bile duct. This inflammation and thickening of the end of the duct produces a partial stenosis and a plugging of the lumen with thickened mucus — this, in turn, causing a damming back of the bile, and a reabsorption of the bile pigments with, perhaps, some of the more toxic elements of bile. It is not the purpose of this paper to contradict this theory of the etiology of jaundice. It is, however, noticeably strange that a disease depending so entirely upon gastro-duodenal catarrh for its origin should be so uniformly free from any symptoms of gastro-duodenal catarrh. This seems to be recognized by all of the authors when reaching the subject of treatment in their articles upon jaundice — all, almost without exception, advising a treatment to excite the activity of the stomach and duodenum, and the incidental activity of the liver and hepatic and cystic ducts and membranes.

Speculation in the etiology of jaundice is always excited when one attempts to comprehend either the immediate or the remote cause of the discoloration of the tissues. This speculation is especially excited by study of the epidemic which it is my purpose to record. If the cause of catarrhal cholangitis be always an extension of inflammation from a catarrhal duodenum into the mouth of the hepatic duct, as is claimed by some, or whether it be due to a toxin in the bile causing an irritation of the absorbent tissues, as is claimed by others, whether it be a transformation of hemoglobin into bilirubin, as is claimed by Semmola and Goeffredi, or whether it be a simple cholemia from the absorption of coloring matter from the bile, there are still features in the etiology which are hard to understand, and the epidemic to which I refer, to me increases the number of questions to be asked about the etiology, and to add but little that will throw light upon the answers to those questions. This is from the fact that the epidemic, although quite extensive, was uniformly mild, without a death, and with very few grave symptoms or noteworthy sequelæ.

My own opinion — and this opinion is shared by most, if not all, of the physicians in Calumet — is that the cause is a specific infection, to a considerable

degree contagious, and dependent to some extent upon the altitude of locality, and little if at all influenced by temperature; and, in the Calumet epidemic, polluted water, as a factor, either for drinking or culinary purposes, could well-nigh be excluded.

The infectious agent seemed to pollute the air of the room to a marked degree, as evidenced by the increased severity of the disease in the cases of those members of the family who were among the last to contract the disease. For example: a child exposed to the disease would become ill, and would be kept at home, and successively five, six or seven other members of the family would contract the disease, and invariably the last one in the family affected would manifest the severest symptoms. The next neighbor appearing to obtain the disease from association with this family would show the same progression from mildness to greater distress from the first one in the family to the last, and the presence of the disease in the house appeared to concentrate the infectious element to such a degree as to cause more pronounced symptoms in those the longest exposed to the contagium before contracting the disease. I cannot think the jaundice in the Calumet epidemic could have been caused simply by a mechanical occlusion of the ends of the bile ducts containing normal bile, but rather that some toxic product of the infectious element rendered either the bile abnormally absorbable or the tissues abnormally absorbent. Whether or not epidemic jaundice be always due to the same infection is somewhat doubtful. It is difficult to believe that the epidemic in Calumet, in Chasselay, and the one in Paris, where, as in Calumet, there was not one death, could be the same as the one in Essen, where the mortality was very high, or the one in Martinique, where there were 20 deaths in 30 cases. This is perhaps not less explicable, however, than the mortality rates in various epidemics of diphtheria.

This outbreak commenced in June, 1897. It prevailed in Calumet, and by Calumet I refer not only to the Calumet and Hecla mining location but also to the adjacent locations and villages: Tamarack, Osceola, Centennial, Wolverine, Kearsarge, Allouez, Red Jacket and Laurium, containing in all a population of 30,000. The elevation of land is about 700 feet above the level of Lake Superior. It is on the top of the ridge in the middle of Keewenaw Point, about five miles from Lake Superior, both on the east and west, and at the southeast lies Torch Lake, which is an arm of Portage Lake, which lies twelve miles to the south.

One peculiar circumstance in connection with the epidemic is, that although the communication is very active and free between the people living at the mines and the people living at the mills, five miles distant, at Lake Linden the epidemic did not exist along the lake either at Lake Linden or at Portage Lake (Houghton or Hancock), or at the Lake Superior water-works to the west of the mining location.

Between June, 1897, and January, 1898, there were about 675 cases of jaundice. The most of these cases were in children under eight years of age; the youngest was three months of age. In the whole number of cases there were approximately 30 cases among adults. In the epidemics cited by Professor Graham it is stated that the younger soldiers were most frequently afflicted in the garrisons, and children were the most frequently afflicted in other epidemics

of this disease. The mortality varies greatly. In Essen the mortality was very high, while in the epidemic at Chasselay and the one at Paris there was not one death. In an epidemic at Martinique there were 20 deaths in 30 cases, most of these being pregnant women. In Calumet the cases were about evenly divided between males and females.

It is the impression of all of the physicians attending these cases (in the Calumet epidemic), that the onset of the disease in adults was attended with much more pain and distress than with the children.

This impression may be the result of imperfect observation by the physicians and by the parents of the children. Generally the first symptom exciting the alarm of the parents was the discoloration, and then the physician was called. This discoloration among the adults did not occur until from two to four or five days following the onset of the disease. Presumably this was the case with the children to some extent, although we cannot believe that the attacks generally were ushered in with as much pain and distress in those cases attacking the children as in those attacking adults. In every case attacking an adult the pain was very much the same as a classical attack of colic from the passage of gall-stones, although generally somewhat milder. During this epidemic it was noticed that those patients who had for some years been subject to an occasional attack of cholelithiasis seemed to have attacks during the height of this epidemic. Within the space of seven weeks, at the height of this epidemic, there were more attacks of cholelithiasis, confirmed by the recovery of gall-stones from the dejecta, than were ever known in this community, in the same length of time, before or since. During one day I personally attended four women in attacks of gall-stone colic, and the gall-stones were found in the stools of three of these patients within forty-eight hours of these attacks. In three of these cases there was marked jaundice, but very evanescent in character, lasting not more than from one to three days.

The exciting cause of the jaundice cannot be traced to any pollution of drinking-water. The majority of those afflicted used habitually pure water drawn from Lake Superior; the others used, some of them, water from the Tamarack Dam, which is water subjected to frequent examination and uniformly good; others used wells, and still others, filtered rain water.

The disease appeared to be contagious to the extent that it appeared to progress along neighborhood lines. There would be from one to seven cases in a family, but in no case did it attack every member of a family. In one family of eight people there was but one case. These eight people were all adults. The one case was a woman who has a number of grandchildren living in the community who habitually visit this house; 12 of these grandchildren were jaundiced, but only one of the six adults among the parents of these grandchildren. In another family in the next house, of four people, there was one case; in another house about 400 feet from these, in one end of the house there were seven people of whom five were jaundiced; in the other end of the same house there were five people among whom two were jaundiced. In one end of the next house to this there were four people, of whom three were jaundiced; in the other end of the house there were four people and none jaundiced. In the next house were eight people,

of whom six were jaundiced; in the other end of the same building there were four people, of whom two were jaundiced. In the next house were seven people and none jaundiced. Across the street from this were two families, six people in each family; and of these 12 people, three were jaundiced. In another house nearly adjoining these were 12 people, all adults, and only two were jaundiced.

Another instance appearing to show contagion: four of five boys who habitually played together were all attacked with the disease at about the same time. In each case these four boys were the first to have the disease in their families; 12 others in their families were subsequently attacked. The period of incubation appeared to be from three to fourteen days. The remaining one of the five boys mentioned did not contract the disease at all; and although there are six people in his family, of whom three are children, none of them contracted the disease.

The weather during this epidemic was not unusually severe, and there was no other epidemic prevailing in the community at the time. We made several efforts to isolate some positive etiological factor in the disease, but wholly without success. The course of the disease was extremely mild; there were no deaths and, consequently, no autopsies, and the symptoms were not sufficiently alarming to excite either the family or the physician to extraordinary acuteness of observation. In a great many cases we made very painstaking analyses of blood, urine, feces, sputum and stomach contents; but these all failed to give us any light about the etiology of the disease. The symptoms were, as previously stated, mild; this was especially so with the children. There would be generally a rise in the temperature of one or two degrees; sometimes languor, dulness, irritability, occasional vomiting; and when from the outset of the disease the typical discoloration was delayed beyond a week, it was noticeable that the case was more severe, although in the severest cases it was remarked that upon the appearance of jaundice there was immediate amelioration of the more distressing symptoms. This description of symptoms applies generally to those cases which were not the first to be taken sick in the family. The first symptom, in the majority of cases, to cause the physician to be called to the family, was the appearance of jaundice; the alarm was excited by the discoloration more than by the severity of the other symptoms. It was frequently possible at this time to get a history of previous illness on the part of the children, but which, before this discoloration, had not been considered sufficiently severe to warrant the parents in sending for a physician.

Another feature of the disease which perhaps might here again be remarked, is one noticed by a number of physicians, namely, that those children who were the last to be taken sick with the jaundice showed the severest symptoms.

The character of the jaundice was unusual in being of a paler yellow than is ordinarily found in catarrhal hepatitis, and of much shorter duration. While among the 675 people attacked during this epidemic, there were some in whom the jaundice persisted for three or four weeks, the overwhelming majority were not jaundiced more than three or four days. In some patients in whom the jaundice could be most confidently predicted from the prodromic symptoms, and in whom the jaun-

dice did appear, it was not well marked more than from thirty to forty hours.

In my first call to one of the cases I was accompanied by Professor Dock, of Ann Arbor. The patient was a woman, aged fifty, who had been ill but a few hours; it appeared a characteristic, rather mild case of cholelithiasis. I predicted that the woman would be jaundiced within twenty-four hours; twenty-four hours passed with no appearance of jaundice, but on the following day, forty-eight hours from the attack, the jaundice was well marked, but disappeared on the third day, and there were no other cases among four people in that family; but under the same roof in another family of seven people, there were five attacked with jaundice.

The sequelæ were unimportant; in not more than one per cent. was there any enlargement of the liver or spleen one month after the appearance of jaundice, and at the present time, April 20, 1898, I am unable to find any case showing any symptoms referable to the jaundice. The only exception I can make to this statement is that several of the children who had previously been thin and delicate showed marked improvement in nutrition after the jaundice. This was very marked in a great many cases.

The treatment followed was generally the treatment of ordinary endemic or sporadic catarrhal jaundice. In adults an anodyne was first administered, followed by a vigorous cathartic, either a mercurial or saline, and then the phosphate of soda given until the stools were of normal color and the bile acids had disappeared from the urine. We still consider phosphate of soda our best hepatic stimulant, and I believe we are justified by the best authorities. Not that the best authorities are agreed upon the action of soda upon the liver; apparently about half of the very best give it because it increases the secretion of bile, and in the bile ducts the increased amount of bile acts by flushing out the inspissated mucous plugs in the ends of the bile ducts, and thus cleansing the ducts and relieving the hepatic cells from increased absorption. The other half have proven very conclusively that the soda salts in small doses have no effect whatever upon the amount of bile secreted, and that large doses decrease the amount secreted. These, however, use the soda salts as an hepatic stimulant on the theory that they produce an alkalinity of blood which affects the bile secreted, and this bile in the ducts is rendered more solvent to the inspissated plugs, and relief is thus obtained.

Other treatment which was entirely symptomatic, yet not directed to the discoloration, was for the itching, the hebetude, the weakened heart's action, anorexia, etc. For the itching, probably the best remedy at our command, in this as in simple jaundice generally, is the daily subcutaneous injection of from one-twentieth to one-fourth of a grain of muriate of pilocarpine, though I doubt if Witkowski's claim, that a malignant diagnosis can with certainty be made where this treatment fails to give relief within eight or ten days, can be sustained, yet there was nothing in this epidemic to controvert any theory concerning malignant hepatic disease. In many of our cases, however, we did give a reasonable amount of attention to the daily amount of excretion of urea. When this is kept at a normal amount, the physician can expel, from his own mind, at least, any danger of the jaundice being due to malignant disease, which he may previously have suspected.