

important cell selection becomes in the normal functional activities.

Since our profession have been so assiduously studying the microorganisms which are about us, upon the supposition that our diseases come from without, we have forgotten that the microorganisms which dwell within, whose names are legion, are being neglected.

How important it is, therefore, to prevent accumulations of effete materials within the intestinal tract. Brunton attributes the "depression, lassitude and dullness after a full meal, in the full-fed, inactive man, to peptones in the blood." Ought we not likewise to attribute many forms of disease to constipation and the faulty elimination of poisonous material from the alimentary canal?

Our food consists of proteids, carbohydrates, hydrocarbons, salts, and water. In their preparation in the alimentary tract before absorption, certain chemical changes take place. This requires normal functionation, or especial selective acts. If all alimentary substances were taken up by the blood, death would undoubtedly ensue. Hence the importance of normal cell selection during secretion, absorption or elimination. Poisonous substances are formed, which, if retained in the tissues, would become a source of infection. The cells would poison themselves by the products of their own metabolism.

Should the pancreas fail, in its cell selection, to produce trypsin from the blood by reason of any interference, a modified product of digestion would be taken to the liver. Urea would appear in the circulation, and symptoms would arise, which, if not eliminated, would be followed by dangerous results.

Bouchard informs us that sufficient poisonous matters are formed in the intestines every day to produce death should they be absorbed into the circulation.

Many of the so-called "diseases of the nervous system," arise solely from auto-infection, and require nothing but corrected digestion and proper elimination. Thus many febrile diseases during the months of autumn become grave only because of retained fecal infection.

We can truthfully say, therefore, that constipation and faulty elimination are important factors in disease.

CIRRHOSIS OF THE LIVER.

CLINICAL AND PATHOLOGICAL DIFFERENTIAL DIAGNOSIS, WITH CASE.

Read before the Chicago Pathological Society, Feb. 11, 1895.

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CHICAGO, ILL.

Patient was a Norwegian, married, 48 years old and a laborer. Under observation thirty-four days. Previous diseases: measles, colds, pneumonia four years ago. Family history negative. Personal history: in Chicago twenty-three years; married twenty-two years; three healthy children; smokes and chews; drinks moderately; venereal history denied. Present disease: ill for three weeks beginning with pain in epigastrium and hypochondria, which is at times absent, now dull, now shooting into both shoulders; dizziness on walking; weakness with pain in lumbar regions; no pain on inspiration; has not vomited; no hematemesis; eats less frequently

because of pain produced in epigastrium; during past month has emaciated rapidly; bowels regular; appetite good; never jaundiced; occasional eructations of gas; coughs a little in the morning.

Physical examination: considerable emaciation; muscles flabby; mind clear; eyes negative; tongue clean; mouth negative; lungs negative. Heart: systolic blowing, apical murmur, diagnosed accidental. No marked atheroma. Pulse somewhat quick but rhythmic; frequency varies from 80 to 104. The morning temperature averaged 99 degrees, the evening 101 degrees, preserving this type with fair constancy. Four evening elevations of 101.8, 102, 102.2, and 102.2 were observed. Urine negative; not bile stained. Feces normally pigmented. Stools irregular in frequency, constipation alternating with slight diarrhea. Nervous system negative.

The liver conformed to the normal contour above, moving freely on respiration; it extended three inches below the usual inferior limit, the enlargement being symmetrical. The edge, not readily palpable because of tenderness, seemed even. The hepatic tenderness and epigastric pain were constant. There was no conspicuous tympany. The splenic dullness was always uncertain, as the organ was never palpated and constipation was frequent. Lastly, the skin of the patient was dry and muddy, and his appearance cachectic. No icterus was observed. Neither hydrochloric nor lactic acid was present in the gastric juice.

The blood count gave 4,104,000 red blood corpuscles, and the red corpuscles sustained to the whites a ratio of 1 to 174. Later counts showed gradual reduction in the number of red corpuscles to 3,800,000, while the leucocytes were to the erythrocytes as 1 to 125.

The patient for several days prior to death was delirious and vomited. The treatment was directed chiefly to his cough and nausea. Antisyphilitic treatment was given. The diagnosis would have been Laennec's cirrhosis in the first stage, had there been collateral circulation, a caput medusæ, ascites, splenic tumor or any gastro-intestinal hemorrhage. Hypertrophic cirrhosis was considered but rejected because no spleen could be found and icterus was absent, although that form of biliary cirrhosis, known among French authors as hypertrophic cirrhosis without icterus, was thought of and discussed in the clinical conferences. The rapid emaciation suggested neoplasm and the leucocytosis looked toward some suppurating focus, although no confirming facts were established.

The autopsy showed little outside of the liver. The aorta and the peripheral vessels were atheromatous. Heart negative; lungs, marginal emphysema, few adhesions and ancient healed tubercular foci. The liver weighed 2,730 gm; there were a few perihepatic adhesions; the organ measured 29 x 19 x 20 x 8.5 x 5.5 cm. The capsule was here and there thickened, but the edges and surface were perfectly smooth. On section the lobules appeared quite large, some measuring 1 cm. across. The color was mottled yellow and red. The organ was firm and had a waxy appearance simulating amyloid. The spleen weighed 420 gm. and measured 15 x 12 x 6 cm. The kidneys weighed 530 gm., and measured 14 x 8 x 4 cm. The capsule peeled very readily, and the markings were fairly distinct. The cut surface was dark and very vascular, the kidney of passive congestion. On microscopic exam-

ination is found an increase in the inter- and intra-lobular connective tissue of the liver. Round cells are found in every part of the lobule and in some places the lobule is wholly replaced by them. The liver cells are now larger than normal, now atrophic, never fatty nor pigmented. The bile ducts are increased. Occasionally a very large lobule is found.

The most interesting pathologic feature in this case is the status of the cirrhosis, whether it is the first stage of a Laennec's cirrhosis, an hypertrophic alcoholic cirrhosis, a biliary cirrhosis or a mixed cirrhosis. It is certainly not a typical Laennec's cirrhosis.

A consideration of the possibility that the vulgar hepatic cirrhosis may be preceded by hypertrophy, is pre-requisite. Todd¹ claims that there is never preliminary enlargement in Laennec's cirrhosis and he was unable to find in literature a reliable instance of a hypertrophic passing into an atrophic cirrhosis. Stricker (Traube's clinic) observed livers shrink to half their former length in one month. Therefore subacute and acute hepatitis must be excluded. Rosenstein² and Labadie Lagrave³ agree with Todd. Bright,⁴ Budd,⁵ Saunders and Frerichs⁶ have said that contraction is occasionally antedated by a stage of enlargement. Murchison's⁷ experience taught that in a considerable proportion of cases of cirrhosis, the liver is still very much enlarged, often from fat, after symptoms of portal obstruction have set in and that patients often die in this stage with jaundice, hemorrhage and symptoms of blood poisoning. Leudet,⁸ from a pathologic standpoint comes to the conclusion that increase in liver volume is not always an index of a recent lesion, of an acute process or a curable one. Murchison says it is questionable whether such livers would shrink if the patient lived longer, yet he states that increase and decrease in liver volume are probably different stages of the same process. Ollivier⁹ believed that cirrhotic atrophy and cirrhotic hypertrophy were different states. Gilbert and Hanot believe in the preliminary enlargement of cirrhosis atrophica. Semmola and Klebs have studied the disease in its incipency and found that the organ is firm, smooth and dark, with dilated portal radicles and periportal extravasation of round cells (Tissier). Keussner¹⁰ Rokitansky, Stadelmann,¹¹ Litten,¹² Schapiro,¹³ Mangelsdorf,¹⁴ Niemeyer,¹⁵ Stricker, Strümpell,¹⁶ Hamilton,¹⁷ Orth and Zeigler describe enlargement antecedent to atrophy. Bamberger¹⁸ considers the decrease in size of an enlarged liver almost pathognomonic for atrophic cirrhosis. Rosenstein applies two tests, palpation of the liver's edge, continued clinical observation and necropsy—ascites, obstipation or tympany render percussion most uncertain. With these criteria Rosenstein has never seen atrophy following hypertrophy.

Definition of term "hypertrophic." — Hamilton¹⁷ takes exception to the term "hypertrophy," arguing that an enlarged liver may be very atrophic. Liebermeister considers that the terms, "biliary" and "hypertrophic" are not identical, nor are venous and atrophic cirrhotoses one. The portal vein form usually shrinks and the biliary form usually enlarges, but exceptions occur as, 1, portal vein forms remaining large till death; or 2, biliary types atrophying to a certain degree.

Most writers, *e. g.*, Strümpell, regard "biliary" and "hypertrophic" as synonymous, and "venous" and

"atrophic" as the same. The hypertrophic alcoholic cirrhosis of Hanot and Gilbert¹⁸ is characterized by edges less sharp than normal; red or brown color; surface furrowed by uneven areas, varying in size, the unevenness being less than in the atrophic form; by granulations on the surface; by enlarged spleen, ascites, collateral circulation, etc. They assert there are many transitional forms between it and the atrophic alcoholic cirrhosis, so that great importance does not attach to size alone. Recovery occurs in this more than in any other form.

Laennec seems to have known but one type of cirrhosis. Hanot, supported by Hayem, Cornil and Charcot, insisted even more than did Todd upon the divorce of hypertrophic and atrophic cirrhotoses. This separation of the two forms is the constant theme of the French school, but they do not seem concerned as to whether an enlarged liver may become small.

Cornil¹⁹ was the first to discover in the hypertrophic form, a very rich network of bile vessels and upon this Hanot based his clinical division of cirrhotoses into, 1, the venous; and 2, the biliary. Litten and Mangelsdorf sought to show that biliary was but the first stage of the vulgar cirrhosis plus icterus. In Germany, the different cirrhotoses are largely held to be mere variations of a single fundamental cirrhosis. Stadelmann¹¹ voices this opinion when he can not find a difference between the two forms for the following reasons: 1, there is a hypertrophic cirrhosis without icterus; 2, hypertrophic cirrhosis can later atrophy; 3, icterus can be very marked in Laennec's cirrhosis.

Rosenstein suggests three classes of cirrhosis: 1, the genuine contracted liver; the cirrhosis of Laennec, an analogue of the genuine contracted kidney.

2. The hypertrophic icteric cirrhosis, an analogue of the parenchymatous nephritis.

3. The hypertrophic mixed form, in which hypertrophy and atrophy combine, an analogue of the secondarily contracted kidney.

Ackermann distinguishes an atrophic cirrhosis (Laennec's) in which the cells die first and are replaced by connective tissue. According to him, Laennec's cirrhosis and one variety of large cirrhotic liver are the same, for in certain livers some parts are atrophic and others again are hypertrophic. Ackermann admits the identity of the French hypertrophic variety which has, he states, nothing in common with the atrophic cirrhosis except the connective tissue proliferation. It is apparently a primary connective tissue hypertrophy with consequent atrophy of liver cells, a hypertrophy occurring *around* the interacinous blood vessels, *never* leading to characteristic granulations, producing *moderate or no stasis, i. e., scanty ascites and insignificant splenic tumor*, and occurring also in horses, fowls and cattle, while the atrophic is seen in man only. The difference between this form and Charcot's description is obvious.

Charcot drew such sharp lines between the different forms that, as Rosenstein has said, one would conclude that a glance through the microscope would readily differentiate them. Charcot described:

1. *Cirrhose d'origine biliare.*

2. *Cirrhose d'origine veineuse.*

3. *Cirrhose monocellulaire*, characterized by the presence of connective between individual cells within the lobule, seen especially in hereditary syphilis.

The French school postulates the following differentia between the two types of cirrhosis: in the

venous variety, the connective tissue and inflammation extend around several lobules, sending in connective tissue processes late, if at all, into the lobule, hence a peripylephlebitis; in the biliary type, the connective tissue begins in the lobule, around the smaller bile radicles, with increase in the biliary vessels, hence a primary angiocholitis or periangiocholitis. In the venous form, the connective tissue exerts compression upon the lobule *en masse*, hence called cirrhose multilobulaire s. annulaire; in biliary cirrhosis, the compression is in the lobule, hence denominated cirrhose insulaire s. monolobulaire. In the vulgar cirrhosis the process is extralobular; in biliary, it is extra- and intralobular. In the venous or portal type, the liver is small, ascites is present, there is no icterus and the liver cells are degenerated; in the biliary form, the liver is large, ascites and other expressions of stasis are absent, icterus is the cardinal symptom and the liver cells are intact, a pathognomonic criterion.

To my mind the differential diagnosis demands elucidation of the following points: 1, distribution of the connective tissue; 2, status of the bile ducts; 3, character of the new-formed connective tissue; 4, condition of the liver cells; 5, blood vessels; 6, size of the liver; 7, bile stasis; 8, venous stasis; 9, classification.

1. *Distribution of Connective Tissue.*—This point is the most confusing of any connected with the differential pathology. Rosenstein rejects Charcot's and Hanot's terms, "intra- and extralobular," "multi- and monolobular," and asserts that there is no cirrhosis in which there are not now some small lobules surrounded by connective tissue and again many acini surrounded by connective tissue. Both distributions may occur in a single liver. In the annular and insular forms Rosenstein finds an actual histologic differential point, and the concentric ensnaring of smaller or larger groups of lobules in typical cases of Laennec's cirrhosis are to him and Ackermann truly distinctive. In typical hypertrophic cirrhosis, the connective tissue sends out processes into and around the lobules, now surrounding an entire lobule, now only certain cell groups. Rosenstein, Kelsch and Wannebroecq certainly overdraw the situation in saying that nearly all cirrhoses are mixed types (Type mixte of Dieulafoy²⁰ and Guiter.²¹ See also references^{22, 23, 24}). Orth²⁵ refuses to divide cirrhosis into different types upon the distribution of connective tissue in regard to the lobule. When said tissue seems to surround lobules, the appearance is usually accidental. The same liver may show multilobular islets, large and small monolobular islets. Regarding the relation between parenchyma and interstitium, the same specimen may show here a sharp distinction and elsewhere the connective tissue running into the lobules. Orth regards all cirrhosis as essentially the same process. Hamilton also thinks Charcot's and Gombault's views extreme.

Brieger²⁶ and Sabourin²⁷ affirm that the connective tissue begins in the hepatic vein zone. French authors find the connective tissue around the bile vessels, the veins being involved later and to a less extent. Hamilton says the secondary connective tissue bands into the lobule are more numerous in biliary than in atrophic cirrhosis.

2. *The Bile Ducts.*—In general, the French authors describe a hyperplasia of the biliary passages in biliary cirrhosis. Brieger finds in every circumscribed

connective tissue formation in the liver an increase in the bile vessels, in tuberculosis, carcinoma, adenoma, gregarinae, distomata, and even in corset liver. Rosenstein states that he has found them (perhaps by accident) more frequently in atrophic than in biliary cirrhosis and can not, therefore, consider them pathognomonic of biliary cirrhosis; also that they sustain no causal relation to icterus. The ducts are described as filled with degenerated and desquamated epithelium and less frequently with pigment masses. Rosenstein affirms the existence of this condition in the hypertrophic cirrhosis without icterus. Ackermann²⁸ thinks the new formed bile vessels communicate with the smallest biliary vessels on the one hand, and with the main duct on the other, and gives as proof the results of artificial phosphorous cirrhosis. Orth considers the canal system as due, chiefly at least, to atrophic liver cells arranged in rows, although he admits they may in part be neoplastic. They can be infected from the biliary system and are absent in no type of cirrhosis. (Also the view of Kelsch, Kiener, Sabourin.)

Friedländer²⁹ was the first to discover this reversion of atrophic liver cells to their embryonal duct-like condition. The ducts disappear where the connective tissue is densest. According to Price³⁰ the ducts are of two kinds: 1, true bile ducts; 2, duct-like structures continuous with and imbedded in large tracts of fibro-nucleated tissue, being transformed into fibrous tissue. Small nodules on the surface are occasionally due to biliary polyadenomata.³¹ Ziegler describes a bile vessel proliferation in all cirrhoses.

Character of the new-formed Connective Tissue.—Ackermann calls attention to the fact that the new-formed connective tissue in biliary cirrhosis remains uncontracted, hence the names elephantiasis hepatis (Eichhorst) and L'hypermégalie (Schachmann). In atrophic cirrhosis it is fibrous; in hypertrophic, embryonal in character (Rosenstein). Stadelmann remarks that the biliary cirrhotic liver never contracts. Orth admits that a biliary form exists, characterized by its clinical course and lack of atrophy, with the qualifying phrase that the future only can assign to biliary cirrhosis its exact status. Strümpell thinks the lack of contraction has been overestimated and that the organ would contract, were life protracted. Hamilton describes the connective tissue bands as finer in hypertrophic than in atrophic cirrhosis.

4. *Condition of the Liver Cells.*—That the liver cells preserve their form is characteristic of biliary cirrhosis. They are at most flattened only at the periphery of the lobule and may disappear. Fatty infiltration and necrosis is said to be very rare. The nuclei are preserved. The cells are sometimes pigmented or atrophied but often both cell and nucleus hypertrophy and divide. In Laennec's form the cells are often degenerated, very frequently fatty, now in the center, now in the periphery of the lobule. Hamilton says fatty degeneration is rare even in Laennec's cirrhosis, while fatty infiltration and hypertrophy occur.

5. *Condition of the Blood Vessels.*—The interlobular blood vessels in the portal cirrhosis are diseased, while free in the biliary type (Rosenstein). Ackermann's view has already been quoted (v. s.). The integrity of the hepatic or sublobular system is, according to Jaccoud,³² quite exceptional.

6. *Bile Stasis (Icterus).*—Icterus dominates the clinical picture in the biliary variety. Most author-

ities admit that jaundice occurs more frequently in biliary cirrhosis, although it may be absent.³³ Howard found icterus in 70 per cent. of hypertrophic and 71 per cent. in atrophic cirrhosis. Mangelsdorf found icterus in thirty-eight out of forty-nine cases of hypertrophic cirrhosis. Fogge (quoted by Charcot) found it in 25 per cent. of atrophic cirrhosis (130 cases) and Rosenstein in 15 per cent. Icterus is absent in the cirrhose hypertrophique graisseuse (Sabourin). In Laennec's cirrhosis, it is incomplete, caused by compression of bile ducts by contracting connective tissue. Its frequency is variously given; seldom (Murchison, Oppolzer and Charcot); frequent (Bamberger, Leyden, Fürbringer); necessary to the diagnosis (Leyden³⁴); slight in degree (Frerichs, Keussner); coming on before severe symptoms (Bright). In hypertrophic biliary cirrhosis some consider the icterus due to polycholia. (Rosenstein, Labadie Lagrave).

In atrophic cirrhosis, jaundice is a genuine complication, due to catarrh, glands, or a diffuse cirrhotic process (Andral, Clin. Med., II), yet few pass through the disease without a muddy yellow areola under the eyes. In hypertrophic cirrhosis the stools remain yellow, an important point (Fürbringer, S. 121 ref. 2), yet sometimes they are acholic (Liebermeister 2). Stadelmann believes many of Charcot's cases of biliary cirrhosis were only instances of retention icterus (from calculi, cicatrices, etc.). Rosenstein has observed catarrhal icterus lasting between one and two years and has seen three cases of amyloid with icterus. In biliary cirrhosis the icterus varies directly with the fever curve and the size of the liver (Jaccoud).

7. *Venous Stasis*—is absent or inconsiderable in biliary cirrhosis, being terminal or complicating. It is present in the mixed forms (biliary plus venous). In advanced cases of atrophic cirrhosis, ascites is rarely absent, yet the patient may die before ascites develops, *e. g.*, from hemorrhage, for hemorrhage is dangerous when there is no ascites (Leyden). An extensive though slowly developing collateral circulation may permanently relieve ascites. Ascites is no infallible sequence of atrophic cirrhosis (Lecorché, Hanot). It may disappear after hemorrhage (Fauvel), diarrhea (Linac) or carcinoma of esophagus (Lecorché, Telamon). It usually antedates edema of the legs, although anasarca may appear first from: 1, renal or cardiac complications; 2, compression of inferior vena cava; 3, perihepatitis involving cava; 4, cachexia; 5, thrombosis of iliac veins. Splenic tumor is seen in both forms. Atrophic cirrhosis is accompanied by mechanical gastrointestinal stasis with hypostatic hemorrhage. In biliary cirrhosis, hemorrhage in other situations, *e. g.*, epistaxis, is more frequent. Fürbringer has, however, observed in biliary cirrhosis, hemorrhagic gastritis and enteritis. The metabolic disturbances are the same in both forms, except that alimentary glycosuria is more frequent in the atrophic. In Laennec's type, constipation is the rule; in biliary, diarrhea. In both, dilatation of the right ventricle, leucocytosis, remittent or intermittent temperature occur. Tubercular peritonitis and granular kidneys are common in the atrophic form, while rare in the biliary. In biliary cirrhosis, albuminuria is infrequent while a high pulse rate and a terminal choleric condition are usual.

CLASSIFICATION.

I suggest the following classification, less as an arbitrary or infallible scheme than as an attempted tentative reconciliation of conflicting clinical and pathological data awaiting stricter future analysis:

Cirrhosis hepatis.	I. Capsular.	{ a. Chronic perihepatitis. b. Portal vein syphilis.
	II. Vascular	a. Hepatic vein. 1. Stasis cirrhosis (cyanotic induration). 2. Cirrhosis (in Laennec's cirrhosis also, Brieger and Sabourin).
		b. Portal vein. 1. Laennec's cirrhosis or atrophic cirrhosis. { 1st Stage. Pseudo-hypertrophy. 2d Stage. Atrophy. 2. Hypertrophic alcoholic cirrhosis, like Laennec's, only remaining large.
	III. Biliary.	{ a. Obstruction—"Retentions-icterus" and cirrhosis. b. Biliary or hypertrophic, { 1. With icterus. in French sense. Ha- { 2. Without icterus. not's cirrhosis. { (Vascular and biliary cirrhosis.)
	IV. Mixed.	

BIBLIOGRAPHY.

- 1 Todd: Clinical Lectures on Urinary Disease and Dropsy, 1859, p. 113; Med. Times and Gaz., 1857, p. 591.
- 2 Rosenstein: verhandlungen L. Congress für Innere Medicin, 1892, Bd. XI, p. 65.
- 3 Labadie Lagrave: Maladies du bois, p. 557.
- 4 Bright: Guy's Hosp. Rep., 1st Ser., Vol. I, p. 612.
- 5 Budd: Diseases of the Liver.
- 6 Frerichs: Klinik der Leberkrankheiten.
- 7 Murchison: Diseases of the Liver, 1855, p. 145.
- 8 Leudet: Clin. Méd., Paris 1874, p. 541.
- 9 Ollivier: L'Union Méd., Sept., 1871, pp. 361, 400, 449.
- 10 Keussner: Volkmann's Klin. Vorträge, 141, s. 1192.
- 11 Stadelmann: Verhandl. d. Congress f. Inn. Med., 1892, s. 90.
- 12 Litten: Charité Annalen, 1880, s. 173.
- 13 Schapiro: St. Petersburg, Med., March, 1891.
- 14 Mangelsdorf: Deut. Arch. f. Klin. Med., Bd. 31, s. 578.
- 15 Niemeyer: Handb. d. Spec. Path. u. Therap., p. 787.
- 16 Strümpell: Handb. d. Spec. Path. u. Therap.
- 17 Hamilton: Text-book Pathology, Vol. II, pt. 1, p. 216.
- 18 Hanot et Gilbert: De la cirrh. atroph. hepat., Soc. Méd. des Hôpit., Mai 23, 1890.
- 19 Cornill: Archiv. de Physiologie, 1875, p. 265.
- 20 Dieulafoy: Gaz. hebdomadaire, Sept.-Oct., 1881, Nos. 39, 40, 41 and 43.
- 21 Guiter: De cirrhoses mixtes, Paris, 1881.
- 22 Sacharjin: Klinische Vorträge, Bd. III, Fall 7 and 8 (Russian).
- 23 Goluboff: Zeitsch. f. Klin. Med., Bd. XXIV, H. 3-4, s. 350.
- 24 Jakowleff: Deut. Med., March, 1894, s. 851.
- 25 Orth: Handb. der Spec. Pathologie.
- 26 Brieger: Arch. f. Path. Anat. LXXV, 1879, p. 94.
- 27 Sabourin: Rev. de Méd., 1882 and 1883.
- 28 Wegner: Virch. Archiv., 55, p. 11, 1872.
- 29 Friedländer: Ueber Epithelwucherung und Krebs, 1877.
- 30 Price: Guy's Hosp. Report., XLII, 1884, p. 313.
- 31 Sabourin: La Glande biliaire, 1888.
- 32 Jaccoud: Lecons de Clin. Méd., Paris, 1885, p. 103.
- 33 Hayem: Arch. de Physiol. Normale et Patholog., 1874.
- 34 Leyden: Beiträge zur Pathol. d. Icterus, Berlin, 1866.

HYPERTROPHIC CIRRHOSIS OF THE LIVER WITH JAUNDICE.

Read before the Chicago Pathological Society, Feb. 11, 1895.

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The patient who died of this affection at the County Hospital last January, was a white woman of Irish extraction, 40 years old, married, having several living healthy children, and an exceptionally good family history. She had malaria and rheumatism several years ago but neither of these confined her to her bed, and she considered that she had had unusually good health up to one year before she came into the hospital, when she first noticed a dull heavy pain in her right side, about the border of her ribs, and became jaundiced. But all the symptoms of this attack passed away without any treatment, and she felt fairly well except for occasional vomiting spells, until toward the end of November, when she was again attacked with the same dull pain in the region of the liver and became yellow.

December 21, she came to the hospital and stated that she had been sick for five weeks; she complained of pain and tenderness in her right side, but was most concerned about the yellowness of her skin and a tumor of which she had lately become conscious in