A case of chronic jaundice and great enlargement of the liver, due to primary carcinoma of the extrahepatic bile ducts, commencing at the junction of the hepatic ducts / by F. Parkes Weber and E. Michels.

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The patient, George W., aged 35 years, was admitted to the German Hospital, July 24, 1904, for deep jaundice and enlarged liver. The jaundice commenced early in July, without pain or any gastric disturbance. Dr. Leonard Williams, who saw the man in the Out-Patient Department two weeks or so before his admission, says that at first the liver appeared not to be enlarged. There was no history of gall-stones or previous jaundice, and the general health was said to have been good. There was no evidence of syphilis, alcoholism, or hereditary tendency to disease of the liver.

On admission the jaundice was extremely deep. The liver was greatly and uniformly enlarged, extending downward on the right side of the abdomen to the anterior superior spine of the ilium. It seemed hard, and its surface was smooth. The spleen was also somewhat, but not greatly, enlarged. The feces were invariably colorless, and the urine deep brown from the presence of bile. It was free from sugar and practically free from albumin, but, as in most cases of deep chronic jaundice, it showed a marked "cloud" of nucleo-albumin on adding acetic acid, either in the cold or after boiling. It likewise contained the usual golden orange pigmented cells, and a few hyaline tube casts, as urines of severe jaundice always do.[1]

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[1] It is perhaps only in the most extreme degrees of jaundice, with chronic obstruction in the common bile duct, that the urine gives the very marked reaction for a mucinoid substance, or nucleo-albumin, to which one of us alluded in Trans. Path. Soc., London, 1900, vol. li, p. 176. On the other hand, in all, or nearly all, cases of jaundice with complete obstruction to the escape of bile (even when of relatively short duration), casts can be found in the urine,

Apart from the jaundice, the patient seemed to be in fairly good health. There was never any xanthoma, practically no prurigo, and no bleeding from the gums or tendency to cutaneous hemorrhages. The only peculiarity, apart from the disease for which he was admitted, was the very tall or "turriform" shape of his head (German, "Turmschädel"), which, however, has no bearing upon the subject of our paper. Nothing abnormal was noted in the thoracic organs. There was no enlargement of any of the accessible lymphatic glands. Examination of the blood (September, 1904) gave the hemoglobin value as 80 per cent. of the normal. The red cells were 3,880,000 in the cubic millimeter, and the white cells 7000.

Owing to the possibility of the obstruction to the outflow of bile being due to a syphilitic lesion at the hilum of the liver, prolonged treatment by potassium iodid was tried, but with no obvious result. Olive oil given by the mouth likewise had no effect.

On October 7, 1904, Dr. Michels performed an exploratory laparotomy, with the object of discovering the cause of the biliary obstruction, and remedying it if possible. At the operation the liver was found very large and rather hard; it had a dark, congested appearance; its surface was not granular. No biliary calculus was felt after full exposure of the bile ducts, nor could Dr. Michels make out any swelling or tumor in the pancreas, or at the hilum of the liver, which might be compressing the extrahepatic bile ducts. There were no adhesions about the gall-bladder, which was small, not much distended, and so far behind the edge of the liver that cholecystostomy could not have been safely undertaken, even had it

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if carefully searched for, though albumin be absent. These casts may contain pigment granules and pigmented cells. Nothnagel (Deut. Arch. für klin. Medicin, 1874, vol. xii, p. 326) thought that in every marked case of jaundice, whatever the cause of the jaundice, casts appear in the urine. (Cf. Dr. P. S. Wallerstein, Ueber reine Cylindrurie bei künstlich erzeugter Gallenstauung, Berliner klin. Wochenschrift, 1902 ,No. 14, p. 310.) Zeri (Il Morgagni, October, 1904), from observations of 30 cases of jaundice, concluded that renal epithelial cells and casts formed from degenerated renal epithelium could frequently be found in bilious urine, even in the absence of albumin. He thought that the epithelial cells and casts in the urine of icteric patients signified damage to the renal tubules, whilst albuminuria, when it occurred in such cases, pointed to damage of the glomeruli. In the present case we were able to note the disappearance (except for the ordinary faint trace) of the nucleo-albumin from the urine when the biliary retention was relieved. It is possible that the nucleo-albumin of bilious urines may, like the bile-pigments, be derived from the bile-passages.

been otherwise desired. Neither the common bile duct, nor any of the bile ducts which could be felt and seen outside the liver were distended.

The patient did not appear to suffer from the operation, though for a time the jaundice seemed still more intense. The wound healed fairly rapidly, and he was soon able to get about again. The jaundice and his general condition were apparently about the same on November 5, 1904, when he left the hospital, as they were on admission.

He was readmitted on November 24, 1904, with the idea that an operation might be performed to drain off the bile externally. The complete and constant absence of bile from the bowel and the history of the case made it practically certain that the condition of the liver was due not to a form of Hanot's cirrhosis nor to any form of cholelithiasis, but that it was caused by complete obstruction in the large bile ducts from a cause which could not be ascertained at the exploratory operation, in spite of careful examination of the region of the transverse fissure. (The result showed that the tumor causing the jaundice must at the time of the first operation have been very small and situated deep in the transverse fissure at the junction of the two hepatic ducts.) It was finally decided, after consultation with Dr. zum Busch, to establish a biliary fistula by the method suggested by M. Hirschberg[2] and J. Rotter.[3]

This operation was performed by Dr. Michels on December 13, 1904.[4] He made a small incision above the umbilical level, to the left of the scar of the first operation. The liver was found to be partly, but not altogether, adherent, and as attempts to stitch the liver to the abdominal wall led to bleeding from the liver, he shut off the peritoneal cavity with tampons. He then bored a hole into the liver with a Paquelin's cautery about 3 centimeters deep, and on probing this a great deal of serous-looking fluid suddenly welled up from the opening, and the probe then passed down for 6½ centimeters from the surface, as if one of the large bile ducts, or a cyst

[2] Die Behandlung schweren Lebererkrankungen durch die Anlegung einer Leber-Gallengangsfistul, Berliner Klinik, October, 1902, Heft 172.

[3] H. Scheuer, Casuistisches zur Chirurgie der Gallenwege, Berliner klin. Woch., 1902, No. 7, p. 138.

[4] Calcium chlorid was, as usual, employed before this operation, and likewise before the previous operation, in order to lessen the tendency to hemorrhage.

filled with serous-like fluid, had been entered. A drainage tube was left in the opening.

Some of the fluid which escaped at the operation was examined, and found to contain very little albumin; in fact, not more than could be accounted for by admixture with blood. No hydatid hooklets were seen.

The patient rapidly recovered from the effects of the anesthetic, and seemed quite comfortable. In the evening following the operation his temperature was 100° F. The next morning (December 15) it was 99° F., rising in the evening to 100.4° F., after which it remained below 100° F. The amount of fluid discharged from the liver was enormous, and on December 16 it was all allowed to run through a long drainage tube from the wound into a bottle beneath the bed, where it was collected and measured every day. On December 16 it was clear orange-brown in color, and not tinged with blood; the specific gravity was 1006; it was of neutral or slightly alkaline reaction, and yielded a very considerable "cloud" of nucleo-albumin on the addition of acetic acid in the cold. As the precipitate, after boiling and acidifying, was scarcely greater than after adding acetic acid in the cold, it was concluded that the fluid contained hardly any serum albumin. Though it yielded an intense Gmelin's reaction, no Pettenkofer's reaction for bile salts could at first be obtained. It still contained some red blood-cells.

The discharge of the bilious fluid continued, but the daily amount greatly diminished, and it became darker in color. On December 26, 1904, we noted that the fluid was clear, limpid, of dark color from the presence of bile pigments, of specific gravity 1010, and of neutral reaction. A drop or two of glacial acetic acid, added in the cold, produced a considerable precipitate of nucleo-albumin, which was redissolved in an excess of the acid. On boiling without the addition of acid only a very faint "cloud" appeared, but on adding acetic acid the same precipitate was produced as by the acetic acid in the cold: a very faint "cloud," however, remained after adding excess of acid to the boiling fluid, and this very faint "cloud" may have been due to a trace of serum-albumin in addition to the nucleo-albumin. Sediment obtained by the centrifuge showed a few red blood-corpuscles, but no cholesterin crystals. Some of the fluid, which had been allowed to become concentrated by slow evaporation, showed the presence of leaf-shaped crystals and of

"rosettes" and "horse-comb" clusters similar to clusters of uric acid crystals, sometimes observed in artificially concentrated urines. On some occasions octahedral crystals, like those of oxalate of lime, were noted, as well as cholesterin plates.

Some of the fluid from the liver, on December 22, 1904, was sent to the Clinical Research Association to be examined for bile acids. The report then was that a positive Pettenkofer's reaction was obtained, and that, after treatment by Dr. Tyson's method, the fluid also answered to Oliver's test (peptone solution), and to Pettenkofer's test, so that there could be no doubt that bile salts were present.

The specific gravity of the fluid varied slightly. On one occasion, as already mentioned, it was 1010, but on December 31 it was again only 1006. On January 5 the daily quantity of fluid from the biliary fistula was only 200 cubic centimeters, and as it escaped at the sides of the long drainage tube the latter was discontinued after that date, the fluid afterward being allowed to collect in the dressings, which had to be changed twice daily.

The following table shows the daily quantity of the fluid from the biliary fistula, as long as it was collected in a vessel; that is, until January 5. The daily amount of urine is likewise given.

Date.	Daily Amount of Fluid from Biliary Fistula.	Daily Amount of Urine.
December 17, 1904	2300 c.c.	.....
December 18, 1904	2000 c.c.	.....
December 19, 1904	2400 c.c.	.....
December 20, 1904	2000 c.c.	1000 c.c.
December 21, 1904	1650 c.c.	1100 c.c.
December 22, 1904	700 c.c.	750 c.c.
December 23, 1904	.....	2000 c.c.
December 24, 1904	.....	.....
December 25, 1904	400 c.c.	1700 c.c.
December 26, 1904	750 c.c.	700 c.c.
December 27, 1904	250 c.c.	750 c.c.
December 28, 1904	400 c.c.	1500 c.c.
December 29, 1904	.....	1000 c.c.
December 30, 1904	300 c.c.	1100 c.c.
December 31, 1904	200 c.c.	1300 c.c.
January 1, 1905	250 c.c.	1250 c.c.
January 2, 1905	.....	1750 c.c.
January 3, 1905	400 c.c.	1750 c.c.
January 4, 1905	.....	1700 c.c.
January 5, 1905	200 c.c.	1400 c.c.

We must now describe the patient's condition after the operation, an operation which might be termed an "intrahepatic cholan-

giostomy," or "lepatostomy," that is, the artificial production of a hepatic fistula. Though he was free from fever, and quite cheerful, and took his food fairly well, and though the jaundice diminished, and the liver became smaller, he steadily became more emaciated. The feces, of course, remained perfectly colorless, but the urine became less bilious. Occasionally there were attacks of vomiting, and on one occasion the vomit apparently contained altered blood. At the commencement of January there was much purulent expectoration, and impairment of resonance, with crepitation, in the right infrascapular region.

In the middle of January, 1905, the diminution of the jaundice was very striking, and on February 2 the icteric tinge had practically disappeared from his skin and sclerotics. About this time the liver had decreased so much in size that the lower border, in the right nipple line, was about the level of the umbilicus: in fact, the lower border had risen as much as the attachment of the front of the liver to the abdominal wall in the neighborhood of the biliary fistula would allow. The urine on February 2, 1905, gave no distinct Gmelin's reaction, though it turned red on the addition of nitric acid. It was clear, of medium color, acid reaction, and free from albumin, and—what we believe to be a very noteworthy point—free from nucleo-albumin, the addition of acetic acid in the cold giving no precipitate or turbidity.

By the middle of January the patient had lost about 14 kilograms (31 pounds) in weight since the operation, but after that he began gradually to increase in weight, gaining 5 kilograms (11 pounds) by the end of the month.

Early in February he commenced to have irregular fever. On February 7 slight edema and purpura of the legs was noted after he had sat up in the ward. On February 13, there was much edema of the loins, and the patient was manifestly losing strength. The slight purpuric eruption on the legs was still present. There was impairment of resonance in the left infrascapular region. The urine was of specific gravity 1020, acid, clear, of medium color, giving no Gmelin's reaction, and free from albumin and excess of nucleo-albumin. Increased feebleness was followed by death on the morning of February 16.

The accompanying chart (Fig. 1) shows the temperature, pulse, and daily amount of urine during the last weeks of the patient's life.

The *necropsy,* performed by Dr. Weber, February 16, 1905, showed the body to be much emaciated, and the front of the chest somewhat sunken on the right side. Examination of the head and brain showed nothing special, excepting the "turriform" shape of the skull, already alluded to, which was found to be associated with synostosis of the frontal and parietal bones, with complete obliteration of the sagittal and coronal sutures. The brain weighed 48 ounces (1450 grams). The heart was rather small, weighing only 8 ounces (240 grams). Otherwise it showed nothing peculiar. The right lung was completely and very firmly adherent to the chest wall, and was not removed for careful examination. The left pleura contained some serous effusion. There were some nodules, which to the naked eye appeared like metastatic tumors, in the left pleura, and in the left lung itself. At the apex of the left lung was a little old

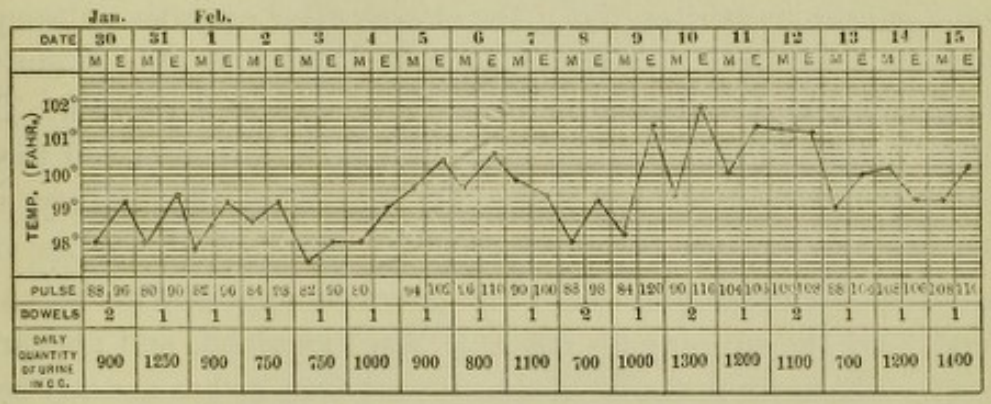

FIG. 1.—Chart showing temperature, pulse-rate, etc., during last weeks of the patient's life.

scarring, and some of the bronchial glands were enlarged, partly pigmented and partly caseated. The liver was firmly adherent to the abdominal wall in front. The fistulous opening on the front of the abdomen, for the production of which the second operation had been performed, was found to communicate by a very narrow channel with one of the intrahepatic bile ducts. Great contraction had evidently taken place since the time of the operation, but the track was still open. Evidently owing to this biliary fistula none of the bile ducts in the liver were dilated. The liver was apparently not enlarged, as when separated from the surrounding parts it weighed only about 50 ounces (1500 grams). Its substance was rather firm, and everywhere of a dark-green color. It contained no new growth of any kind or calculus. The gall-bladder was of moderate size, and filled with very sticky, inspissated blackish-green bile. The transverse fissure of the liver, including the whole region of the

junction of the cystic, common, and hepatic bile ducts, was occupied by a non-encapsuled, rather tough growth, resembling connective tissue. With probe and scissors it was possible to follow the channels of the various ducts most of the way through this growth. No calculus was found anywhere. The duodenal end of the common bile duct was pervious and apparently not in any way diseased. In the neighborhood of the hilum of the liver were some enlarged lymphatic glands. There was a good deal of serous peritoneal effusion. Scattered over the peritoneum were nodules, supposed at the time of the necropsy to be metastatic tumors. The omentum was collected into a tough mass, and seemed to the naked eye to be infiltrated with growth, and there was evidence of chronic peritonitis, with considerable thickening of the whole serous coat of the stomach,[5] such as is not rarely met with in cases of diffuse peritoneal cancer (the so-called "cancerous peritonitis"), sometimes after operative attempts to remove a primary growth. The spleen weighed only 4 ounces (120 grams), and contained one nodule of apparent growth. The kidneys weighed together 11 ounces (330 grams), and one of them, it may be remarked, showed a peculiar congenital abnormality, the pelvis and the ureter being attached to the ventral surface instead of in the usual position. The pancreas appeared to be quite normal. Of the suprarenals one only was examined, and appeared normal. The peritoneal coat of the stomach was, as has already been mentioned, considerably thickened. The intestines showed nothing peculiar (excepting the growths on their peritoneal surfaces, already mentioned), neither did the testicles. The left humerus was sawn through longitudinally, so that the bone-marrow of the shaft might be examined. This was of red color throughout, and of gelatinous consistence.

Microscopical Examination.—Sections of 24 different pieces from various parts were prepared. The tough connective tissue-like mass from the hilum of the liver filling up the transverse fissure was found on microscopic examination to consist of adeno-carcinomatous tissue (malignant adenoma). The microscopic sections (two pieces of the growth were examined) showed tubules (Fig. 2) lined by columnar-celled epithelium, cut across in various directions. The

[5] Superficially the stomach resembled an early stage of the so-called "leather-bottle stomach." *Vide* A. W. Nuthall and J. G. Emanuel, Diffuse Carcinomatosis of the Stomach and Intestines, Lancet, Jan. 17, 1903, p. 159.

FIG. 2.—The primary adenocarcinoma at the hilum of the liver, showing tubules of various sizes and shapes, one with its lumen filled with cells, or intracystic growth. ×110.

FIG. 3.—Portion of the growth, showing penetration of carcinomatous tubules into a nerve. ×110.

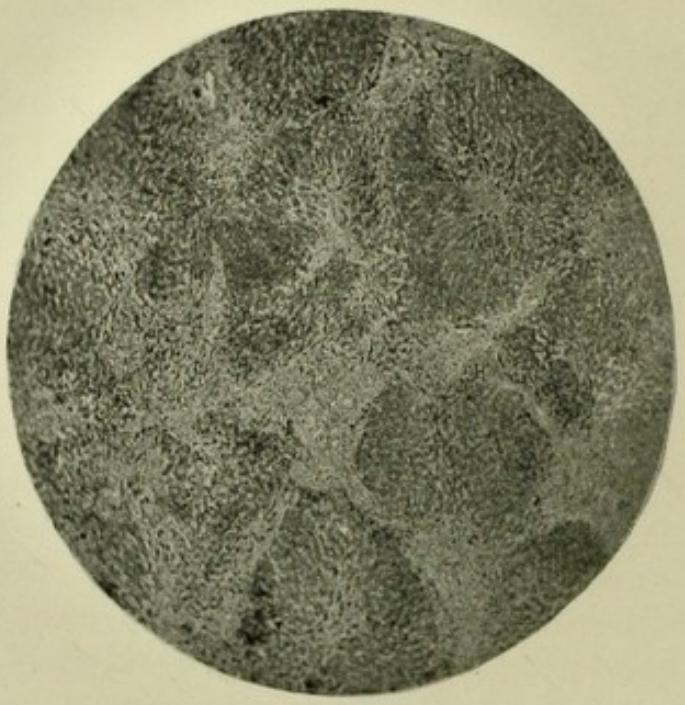

FIG. 4.—Section of the liver showing a certain amount of interacinous biliary cirrhosis. × 110.

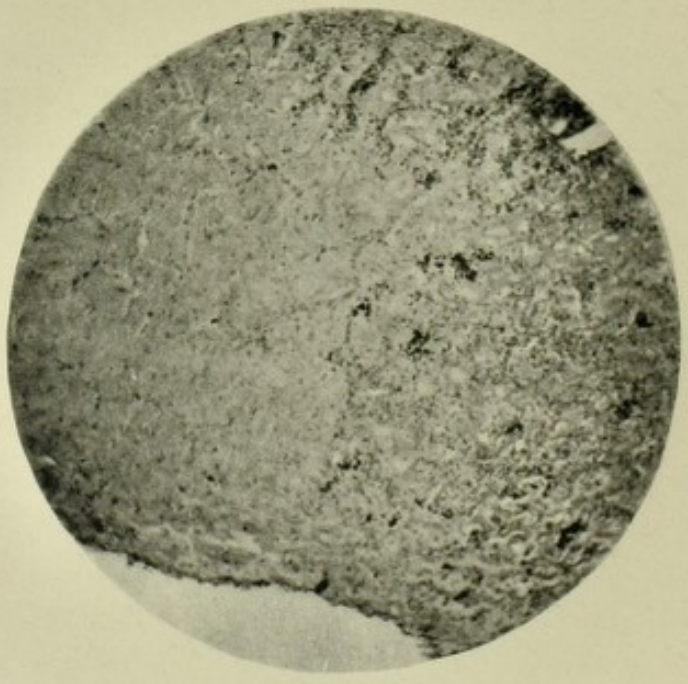

FIG. 5.—Section of the bone-marrow from the shaft of the left humerus, showing an erythroblastic reaction on one side and gelatinous degeneration on the other. × 120.

tubules were of various sizes and shapes, some of them being large enough to appear in transverse section as cysts (lined by columnar epithelium). In some parts tubules were to be seen inside larger tubules (intracystic growth), and a few of the tubules were lined by less distinctly columnar (more spheroidal-like) epithelium, and were filled up with cells so as to show no lumen. The tubules were separated from each other by a variable amount of connective tissue; in some parts they were close together, as in adenomas, but in other parts they were widely separated by connective tissue. Though the tubules were mostly lined by only a single layer of cylindrical epithelium, and in their regularity reminded one of adenomatous growths,[6] they were found to have invaded the structures at the hilum of the liver, penetrating between, and, in one or two places, into the nerves (Fig. 3). The growth was not encapsuled, and was evidently malignant, although, as we shall presently have to point out, no metastasis had as yet occurred, at least none that could be recognized. It was, therefore, a primary adeno-carcinoma or malignant adenoma, which, owing to its position, doubtless originated from the extrahepatic bile ducts at the hilum of the liver. We shall return to this subject later on.

Examination of the liver (Fig. 4) itself showed a certain amount of fibrosis between the acini, with increase (at least apparent increase) in the number of the biliary canaliculi: in fact, the appearances found in cases of biliary cirrhosis due, directly or indirectly, to obstruction in the large bile ducts.[7] One or two circular spots of cellular infiltration suggested commencing tubercle formation, but a section was specially stained for tubercle bacilli, with a negative result.

The supposed metastatic growths from the lungs and spleen and from the peritoneal surfaces of the jejunum and ileum, as well as the thickened omentum, the thickened outer wall of the stomach and the lymphatic glands from near the hilum of the liver, were found by microscopic examination to show the typical changes of tuberculosis with giant-cell formation, more or less caseation, etc. Sec-

[6] The tubules of adenocarcinoma of the rectum may, of course, also be well-formed and lined by a single layer of epithelium.

[7] F. Parkes Weber, Biliary Cirrhosis of the Liver, with or without Cholelithiasis, INTERNATIONAL CLINICS, 13th Series, 1903, vol. iii, p. 57; Transactions of Pathological Society of London, 1903, vol. liv, p. 103.

tions stained for tubercle bacilli showed their presence in the thickened wall of the stomach, in the omentum, and in nodules from the lung, spleen and jejunum.

Sections of the bone-marrow from the shaft of the left humerus showed typical "gelatinous degeneration." The degenerated tissue consisted of an almost homogeneous substance containing only a few erythroblasts and other cells, and the remains of fat vesicles. At one part of the field shown in Fig. 5 there is, however, a decided erythroblastic reaction. The tissue at this part is rich in cells, almost all of them being erythroblasts, with typical deeply-stained homogeneous-looking nuclei, some of them with two nuclei or a lobed nucleus, probably in process of division. Here and there large numbers of erythroblasts are grouped together in dense clumps.

The question of diagnosis claims our attention. At one time, especially after the negative result of the first operation (exploratory laparotomy), we almost regarded the case as one of Hanot's disease, that is to say, hypertrophic cirrhosis of the liver with chronic jaundice. The points against this diagnosis were: The absence of great enlargement of the spleen, the comparatively recent and sudden development of the jaundice, and of the hepatic affection altogether, and the complete absence of biliary pigments from the feces. It was this latter point which finally made us come to the conclusion that the cause of the jaundice and hepatic enlargement was some mechanical obstruction (probably not a calculus)[8] at the hilum of the liver, and owing to the absence of distention of the common bile duct and gall-bladder Dr. Michels thought that this obstruction was situated proximal to the junction of the cystic and common ducts. We, therefore, concluded that the bile ought to be drained off externally, and Dr. Michels succeeded in effecting this by the establishment of a hepatic fistula in the way described. In fact, the jaundice practically completely disappeared, the urine became free from biliary pigments, and the liver shrank, so as to weigh about the normal amount at the time of death. It is probable that the spleen also became smaller with the relief of the jaundice,

[8] There was no history of biliary colic or of previous jaundice or of attacks of fever such as are frequently met with in cholelithiasis. Moreover, it must be remembered that gall-stones in the hepatic and common bile ducts nearly always allow some bile to pass now and then.

as though it had been apparently enlarged by physical examination in the earlier part of the illness, it was found to weigh rather less than normal at the necropsy.

There is not much to be said in regard to the tuberculosis which developed. Minute carcinomatous metastases on the peritoneum and pleura often have the appearance of tubercles when examined by the naked eye at the time of a necropsy, or during an operation, but in the present case it was the tuberculous growths which at the necropsy appeared to us to be carcinomatous metastases. A striking example of the macroscopic resemblance of tuberculous growths to carcinoma has recently been recorded in Germany by Dr. E. Ruge.[9] His patient presented all the signs of carcinoma of the pylorus: the typical tumor was felt, and there was absence of hydrochloric acid and of lactic acid from the gastric contents. The diagnosis of cancer of the pylorus was confirmed by laparotomy, and even at the post-mortem examination the disease was supposed to be carcinoma of the pylorus, with numerous metastases. It was only the microscopic examination which showed that not only the supposed primary tumor, but all the metastases, were in reality of tuberculous inflammatory origin. Ruge compared the carcinoma-like tuberculous nodules to Perlsucht in cattle.

The explanation of the development of the tubercle in our case is not quite obvious. The presence of some partially caseous bronchial glands and remains of pulmonary (apical) tuberculosis show that the patient had previously had old quiescent tubercle in his body. The outbreak may, perhaps, be regarded as a reawakening of quiescent and latent tuberculosis, facilitated by the patient's cachexia and diminished power of resistance. Secondary infection from without appears extremely improbable, but it is just possible that at the first operation tuberculous glands in some part of the abdomen or thorax may have been damaged. On the other hand, no bad signs were noted until after the second operation, and at this operation it is exceedingly unlikely that anything of the kind can have taken place, as the organs were not moved about or pressed upon. The type of the tuberculosis was remarkable, as we have already noted, especially in respect to the tumor-like growths on the peritoneal surface of the intestines and the diffuse tuberculous thickening of the outer coat of the stomach.

[9] Beiträge zur Klinik der Tuberkulose, 1905, vol. iii.

Primary carcinoma of the bile ducts at the hilum of the liver, without metastases and without the liver itself being invaded by the new growth, is not a common cause of chronic jaundice. H. D. Rolleston [10] recently has pointed out that some cases described as primary cancer of the liver might possibly in reality have been cases of single large adenoma of bile ducts. In our case, however, the growth was a typical cylindrical-celled adenocarcinoma or malignant adenoma,[11] infiltrating the tissues at the hilum of the liver, and even penetrating into nerves.

J. Orth [12] says that primary cancer of the bile ducts, which is generally a cylindrical-celled adenocarcinoma, is a rare disease. "Its most usual situation is at the junction of the hepatic ducts or at the orifice of the choledochus." In our present case there can be practically no doubt that the growth originated at the junction of the two hepatic ducts to form the common hepatic duct, and that at the time of the first operation (exploratory laparotomy, October 7, 1904) the cystic and common bile ducts were not yet involved. At that time, although the obstruction to the exit of bile from the liver was complete, Dr. Michels found no dilatation of the gall-bladder or of the common bile duct, and was able to express some of the bile from the gall-bladder into the common duct. Doubtless the cancer could not be felt at the operation because at that time it was extremely small and situated deep in the transverse fissure of the liver, though at the time of death (February 16, 1905) it had already involved the junction of the cystic and common bile ducts.

We have already alluded to the fact that the present case confirms the view that chronic obstruction of the larger bile ducts leads gradually to more or less interstitial hepatic fibrosis, in the same way that chronic obstruction of a ureter, with hydronephrosis, leads to chronic fibrosis of the affected kidney.[13] We lay stress on this point,

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[10] Diseases of the Liver, Saunders & Co., Philadelphia and London, 1905, p. 455.

[11] Rolleston (loc. cit., p. 688) thinks that spheroidal-celled carcinoma of an extrahepatic bile duct may possibly be derived from mucous glands in its wall.

[12] Lehrbuch der spec. path. Anatomie, Berlin, 1887, vol. i, p. 988.

[13] *Vide* E. P. Weber, Trans. Path. Soc. London, loc. cit. In regard to this subject, our colleague, Dr. Fürth, has kindly allowed us to allude to the following striking case which was under his care at the German Hospital. The patient, a married woman, aged 66 years, commenced to suffer from jaundice

as it has been disputed by many. Amongst other writers on the subject, Scagliori [14] has quite recently drawn attention to biliary cirrhosis as a result of obstruction by columnar-celled carcinoma of the large bile ducts.

As far as it went, the operation of hepatostomy (biliary drainage by intrahepatic cholangiostomy) was successful. It was only intended to relieve the jaundice and the retention of bile in the liver. The jaundice practically disappeared; the liver, though it could not, owing to adhesions, return to its normal position, returned to its normal dimensions (as judged by its weight), and the urine became practically free from bile and nucleo-albumin.[15] But the tuberculous complication prevented the result from being really satisfactory, and we must also admit the possibility of the hepatic fistula closing up spontaneously had the patient lived longer, for the fistulous channel at the time of death had become extremely narrow.

The surgical aspect of the case requires but little comment. Where the situation of the obstruction is proximal to the junction of the hepatic and cystic ducts it would, of course, be futile to attempt to drain the gall-bladder or to make a cholecystenterostomy. Drainage of the intrahepatic ducts means a permanent biliary fistula (at least as long as the fistulous channel can be kept open), except in those cases in which, from the nature of the obstruction, a reopening of the natural biliary channels may be expected, as in the case pub-

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for the first time in March, 1904. Apparently no bile passed into the bowel, and jaundice, emaciation, and weakness became extreme. She was tapped four times for ascites. The liver, which was at first greatly enlarged, with a somewhat uneven edge, diminished so much in size that just before her death (October, 1904) the edge could only just be felt below the costal margin. At the necropsy the gall-bladder, full of calculi, was surrounded by malignant growth, but the point which specially concerns us is that all the bile ducts, intrahepatic as well as extrahepatic, were so dilated as to remind one of the appearance seen in a hydronephrotic kidney. Microscopically, the minute biliary ducts were seen to be full of inspissated bile (by naked eye examination the substance of the organ was of a deep-green color as usual), and there was a certain amount of biliary cirrhosis. Yet so much had the liver diminished in size before death that it weighed only about 40 ounces (1200 grams). The heart was also very small, weighing only 5 ounces (150 grams).

[14] Il Policlinico, November, 1904.

[15] As if the excess of nucleo-albumin, which constitutes a striking feature of some bilious urines, were, like the bile-pigment, absorbed from the biliary passages into the blood, and then (again like the bile-pigment) excreted with the urine.

lished by Rotter.[16] With a view to avoid a biliary fistula Kehr excised a piece of the edge of the liver, 6 centimeters long and 3 centimeters broad, made a corresponding opening into a loop of small intestine and stitched the edges of the intestinal opening to the margin of the wound in the liver. Kehr terms the operation "hepato-cholangio-enterostomy."[17] In his case the jaundice was soon relieved, the bile passing from the exposed intrahepatic ducts straight into the intestine, but the patient survived only four weeks. It seems a somewhat hazardous proceeding to bring the wound of the liver into contact with the contents of the bowel. The establishment of a biliary fistula is certainly the safer proceeding, and the operation should be performed on the left side of the liver, as the chance of reaching a large bile duct is supposed to be better on the left side than on the right. The operation can be done with a trocar (Hirschberg) or preferably (as in the present case) with the thermocautery.

We will only add a few words on the change in the bone-marrow. This consisted in a mixed erythroblastic reaction and "gelatinous degeneration," the latter predominating. It is well to ascertain as far as possible the bone-marrow changes associated with various clinical conditions. According to Roger[18] and Muir,[19] extreme inanition and emaciation seem to be the chief causes of gelatinous change in the bone-marrow, and great emaciation was certainly present in our case.

In conclusion, we have to thank the residents at the German Hospital, Dr. Schuh, Dr. Mülberger, Dr. Schenck, and Dr. Daser, for their many and excellent microscopic preparations from the case, and we are also greatly indebted to Dr. H. D. Rolleston and Mr. S. G. Shattock for their kindness in looking through the sections with us.

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[16] Loc. cit.

[17] Centralblatt für Chirurgie, 1904, No. 7, p. 185. See also Enderlen and Zumstein, "Ein Beitrag zur Hepato-Cholangio-Enterostomie," Mitteilungen aus den Grenzgebieten der Med. und Chir., Jena, 1904, vol. xiv, p. 104.

[18] Roger and Josué, Comptes rendus de la Soc. de Biologie, Paris, 1900, p. 417.

[19] R. Muir, Trans. Pathological Soc., London, 1902, vol. liii, p. 392.