

leaving of a liter of this solution in the cavity is earnestly urged by many.

The use of hydrogen peroxid, as recommended by Morris, has little to recommend it beside the fact that it spreads the infectious material the same as does the use of normal salt solution, and so allows nature to combat the infectious process at many points instead of at one situation.

The bactericidal property of the nascent oxygen can not be great, for if it were sufficient to kill the germs, such as the colon bacillus or streptococci, it would certainly at the same time destroy the sensitive endothelial cells lining the peritoneal sac.

The postural treatment, with elevation of the head, as recommended by J. G. Clark,⁴⁶ so aiding in the drainage of the infectious material through the diaphragm and into the mediastinal lymph glands, has many enthusiastic advocates, as has the method with elevation of the foot of the bed, as proposed by G. Ryerson Fowler,⁴⁷ which allows the deleterious substances to come in contact with the pelvic peritoneum, which, for some reason, seems more resistant to infection than is that peritoneum at the diaphragmatic portion of the cavity.

The continuous irrigation with the warm normal salt solution for acute general septic peritonitis, was suggested by Laplace⁴⁸ in 1889, at which time he said: "The method has not to my knowledge been used before." And at the St. Paul meeting he again refers to it and mentions cases which have recovered following its use.

We find an article by T. J. Maxwell⁴¹ of Keokuk, Iowa, in which he reports two cases of post-operative septic peritonitis treated by free irrigation of the peritoneal cavity as long as the pulse and temperature demanded it.

CONCLUSIONS.

1. The previous attempts at an early and accurate diagnosis of perforation of the gastrointestinal tract without opening the abdominal wall have not been adopted.

2. There has been practically no improvement in the method of diagnosing such conditions during the past century.

3. The treatment of the perforation *per se* during the last century has progressed to a state bordering on perfection when compared with the older methods.

4. The diagnosis of gastrointestinal perforations is one of the most important unsolved questions in the domain of abdominal surgery.

5. There is no pathognomonic sign or symptom or group of signs or symptoms of perforation.

6. The only positive method of arriving at a diagnosis to-day is to either perform an exploratory laparotomy or await the development of a peritonitis.

7. To await the development of a peritonitis will reveal the diagnosis too late for the most effectual treatment.

8. Exploratory laparotomy as a routine measure will of necessity result in some cases being needlessly exposed to the many dangers of a major operation.

9. Exploratory laparotomy is not a harmless procedure.

10. The consensus of opinion is in favor of exploratory laparotomy as a choice between two evils.

11. The fact that even the most expert clinicians fail to diagnose perforation in all cases and even diagnose such a condition when it does not exist shows the great need of improvement in diagnostic methods.

12. The diagnosis should be arrived at before the treatment of the perforation is complicated by the presence of a peritonitis.

13. The injection of air or normal salt solution into the peritoneal cavity and withdrawal of the same for examination will do no harm.

14. Such a procedure will, in many instances, reveal a perforation before any sign or symptom of peritonitis exists.

15. The treatment of the perforation *per se* is simple and satisfactory.

16. The treatment of the complicating peritonitis is multiple and unsatisfactory. The best treatment is prophylactic, i. e., early diagnosis and repair of perforation before peritonitis has become established.

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PRIMARY ENDOTHELIOMA OF THE GALL BLADDER.*

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NARRATION OF CASE.

History.—Mrs. R., age 65, referred by Dr. W. F. Malone. Usual diseases of children. Appendicitis three years ago. Full recovery. Thirteen months ago loss of appetite and loss in weight. Family history negative.

Present State.—Weight 52 kilo. Emaciation and weakness very marked. Almost complete anorexia. Dull continuous pain over gastric region; slight tenderness over region of the gall bladder. This is only elicited at intervals. Still's sign absent. Abdomen is distended to the utmost in both the gastric and umbilical regions. Ten c.m. below the umbilicus the resonance of percussion takes on a lower pitch. (Percussion from above downward.) No stool the last ten days. Patient

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vomits continually, which consists of a foamy, brownish, mal-odorous mass, made up of fermenting food remnants. Total absence of HCl free or combined. Presence of an immense number of sarcinae. No Oppler-Boas bacilli. Stomach is emptied by means of the stomach tube; 1½ liters of fermenting stomach contents are brought forth. Gas evolved by fermentation is CO₂. No lactic acid. After evacuation of the stomach the same is washed with salt solution. Distension over gastric region has now disappeared. The stomach is artificially inflated with air; lower border is found 10½ c.m. below the umbilicus. The partly inflated stomach containing water which is introduced for diagnostic purposes shows the lower border of the stomach 8 c.m. below the umbilicus. Determination by gurgling sound on introduction of air. An attempt to outline the colon by inflation proves too painful and is abandoned. Colonic treatment results in copious stools. Patient feels comfortable. This treatment is continued and the stomach contents are tested daily. After seventh treatment free and combined HCl are evident. Appetite improves and stool every two or three days. All stools contain reduced bile pigments; no blood at any time; never any blood in stomach contents. (Examination according to Weber's method.) In spite of moderately large meals patient continually loses in strength and weight. Percentage of urea ranges from 20 to 28 grams in 24 hours. As soon as a day is allowed to pass without gastric lavage there is rapid accumulation of gas with consequent painful distension of the stomach. This is particularly noticeable when the patient assumes an upright position, or sitting posture. On the seventh day of observation the lower border of the liver is felt for the first time, and a distinct hardening can be felt at the site of the gall bladder. The hardening appears to be in the liver substance. Pressure on this nodular hardening, for instance, on attempting to palpate the gall bladder from below, elicits moderate pain. On distension of the stomach this hardening can not be felt. Never any icterus. Spleen moderately large. The highest total gastric acidity reached was 25, of which 7 were free HCl. After this amount had been reached, the quantity gradually decreased until about two months later it disappeared completely and remained absent until death. Boas' gallstone sign, on pressure pain in region to the right of tenth dorsal vertebrae could not be elicited.

Diagnosis Intra Vitam.—Carcinoma of the gall bladder; kinking of the duodenum; enormous dilatation of the stomach.

AUTOPSY.

In the report of the necropsy only such findings will be mentioned as are considered relevant.

Necropsy was performed six hours after death. Protocol, Dr. M. A. Kleinhans. Abdomen tensely inflated. On opening the peritoneal cavity an enormously dilated stomach tensely filled with gas is evident, which had pushed the colon down into the large pelvis. Only a moderate degree of gastropnoia. On turning the fundus and greater curvature of the stomach upward the gas contained in the stomach can be expelled into the duodenum. The pylorus and duodenum are firmly adherent to the dorsal surface of the liver. The pancreas is, to some extent, enveloped in these gastric adhesions. No gall bladder is visible or tangible. In its stead is a circumscribed firm, slightly elastic hardening in the liver substance. Stomach, liver, pancreas and spleen—which latter is not enlarged—are taken out together. Weights and measures of the organs are all within normal limits except the measurement of the stomach. Serial incisions into the liver reveal an ovoid tumor of white color at the site of the gall bladder. This tumor is directly continuous with the serosa of the stomach and duodenum, and also with the pancreas by means of a slightly elastic tissue bridge. Liver substance shows distinct lobulation, increased periportal connective tissue, especially around the larger bile ducts.

Stomach.—Stomach contains about a half liter of undigested food. The mucosa shows no rugae. The usual petechia on posterior wall; disseminated plaques of smooth, grayish mucosae, which are diagnosed and proven by microscopy to be

atrophic areas. No bile discoloration of the stomach. No thickening of the mucosa anywhere suggested carcinoma; no ulcers, deep or superficial. Pyloric orifice patent. In the posterior wall of the antrum pylori a hard thickening is seen and felt. It contains crepitating material. The highest point of this elevation is devoid of mucosa. The mucosa is replaced by connective tissue. The thickening is incised, and two gallstones, one the size of a filbert, the other the size of a small pea, are found therein. The gallstones were only slightly faceted and medium hard. The floor of this cavity which contained the stones is formed by the substance of the above mentioned tumor in the liver. The tumor formation had evidently forced the gallstones into their present position. No cystic duct could be made out. Hepatic duct is uninterruptedly continuous with the common duct, and the whole duct is patent. Diffuse fibrosis of the mucosa of this duct. No scar or other mark which might designate the entrance of the former cystic duct. Pancreatic duct is patent and enters the duodenum at 1 c.m. distance from common bile duct. Pancreas moderately sclerosed.

Now, all the length of the intestinal canal is explored, together with the genitalia, uropoietic organs, likewise the contents of the thorax, but no evidence of carcinoma is found.

Heart.—The right ventricle of the heart contained a large marantic thrombus, which had occluded the sinus venosus and branched into the pulmonary artery.

It being a private case, the privilege of opening the cranium was denied.

Anatomic Diagnosis at Time of Autopsy.—Primary carcinoma of the gall bladder; adhesions with pylorus of the stomach, duodenum, and pancreas. Enormous dilatation of the stomach. Cause of death, probably occlusion of the sinus venosus by marantic thrombus. (Patient died with symptoms of apnea.)

After examination microscopically the diagnosis is changed into primary endothelioma of the gall bladder.

DESCRIPTION OF ILLUSTRATIONS.

Pathology.—Figure 1 shows the gross appearance of the tumor located in the lower dorsal aspect of the liver at *a*. The white tumor substance merges gradually into that of the darker liver substance; *b* represents the pylorus and duodenum. The serosa of these two last named organs is seen to be nodularly swollen; *c* shows the increase in the portal connective tissue.

Figure 2 shows the liver and stomach, in order to demonstrate their relative size. It is plainly seen that there is no obstruction of the pylorus, but simply adhesions by means of the serosa. The comparison between the sizes of the two organs, stomach and liver, will at once convey the excessive degree of dilatation of the stomach (*a*). Rosenheim (*Krankheiten des Verdauungs-Apparates*) states in a differential diagnosis between liver and gastric cancers, that "*Gallenblasen-Carcinome bedingen meist keine Dyspepsie und niemals Ectasie.*" A number of authors, including American and French, have, I think, thoughtlessly, repeated this statement. I have seen five cases of carcinoma of the gall bladder during the last year, four of which showed an immense ectasia of the stomach. Figure 3 represents a transverse section through the whole liver, together with an immensely dilated stomach for comparison. In this case, as in the case of endothelioma (Fig. 2), there was no involvement of the mucosa of the stomach whatsoever, and only a slight, easily separable adhesion of the stomach and the under portion of the gall bladder. In one more case of the four that showed signs of dilatation of the stomach during life, I have performed the autopsy which revealed similar conditions as seen in Figure 3. One case is still alive, but since an exploratory laparotomy was performed I had an opportunity to see the dilated stomach in that case. Rosenheim's statement, therefore, can-

not be considered of any value in differential diagnosis between carcinoma of the stomach and gall bladder.

Histology.—A large number of specimens of tissues of all regions were examined. The liver tissue remote from the tumor shows a moderate degree of increase of the periportal connective tissue with new formation of bile ducts. This suggests interstitial hepatitis. The connective tissue, however, does nowhere invade the lobules proper. Study of the individual liver cells shows them to be fairly normal. Epithelium of the old and new formed bile ducts shows no tendency to atypical proliferation anywhere. The connective tissue immediately

plasm of which takes a deep eosin stain. In some areas these cells are seen to rise from oblong spaces or narrow slits, which I take to be lymph spaces. Where there is a large number of these cells, those marginally situated seem to undergo gradual fibrillation, and it is then extremely hard to make out the dividing line between the connective tissue proper and the tumor cells.

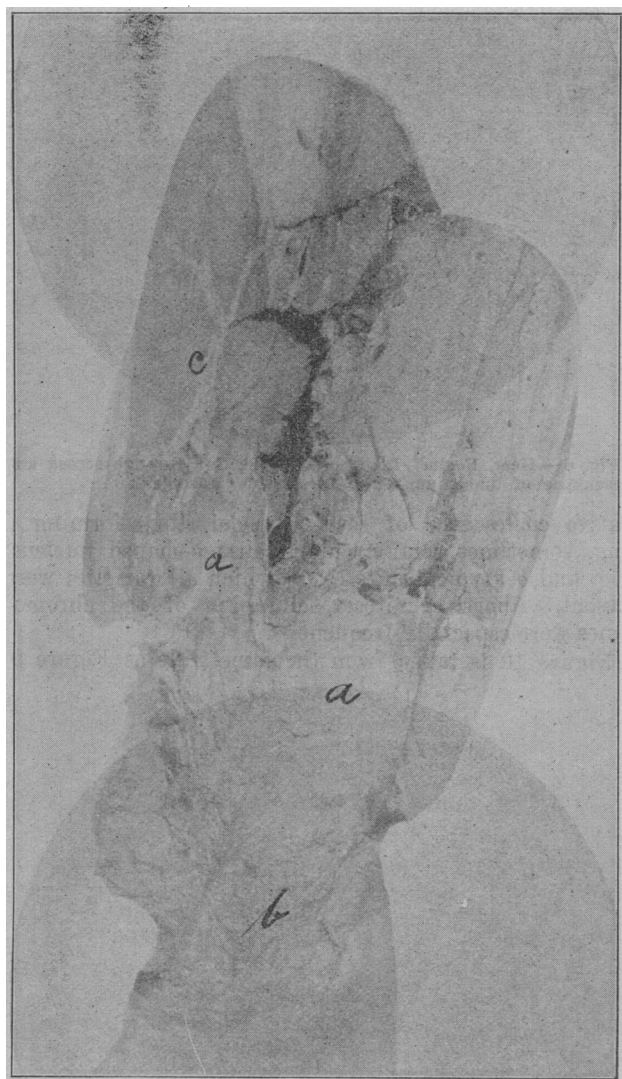


Fig. 1.—Vertical section through liver. *a.* Endothelioma. *b.* Pylorus. *c.* Pericholangitis.

surrounding them is in active proliferation (pericholangitis).

In Figure 4 the margin of the tumor is made up of connective tissue containing foci of round cell proliferation (*a*). The individual liver cells surrounded by this connective tissue formation (*b*) are entirely normal. The blood capillaries of this liver tissue are slightly over-distended. This congestion is thought to be due to prolonged agony.

Figure 5, from a section taken about 1½ c.m. away from the margin toward the center of the tumor, also shows mostly connective tissue, young and old, but containing strings of polymorphous large cells, the cyto-

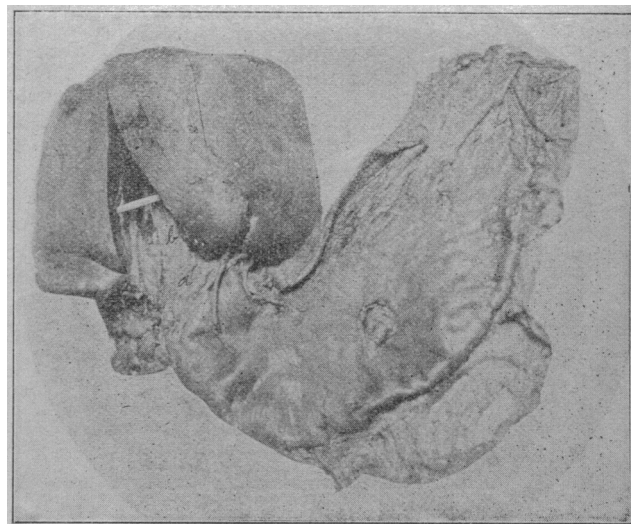


Fig. 2.—Endothelioma of the gall bladder, with dilatation of the stomach. *a.* Pylorus. *b.* Endothelioma.

The cells contained in the center of the larger aggregations undergo hyaline degeneration. No karyorrhexis; no vacuolization. In other areas the centrally located cells of the cluster seem to lose their cytoplasm and then resemble common small lymphocytes. The change of the cells, or decrease in the size of the cells from the margin toward the center of the cluster, is extremely gradual. Necrosis described by Adler in his paper on "Endothelioma of the Pleura" was not found in this case.

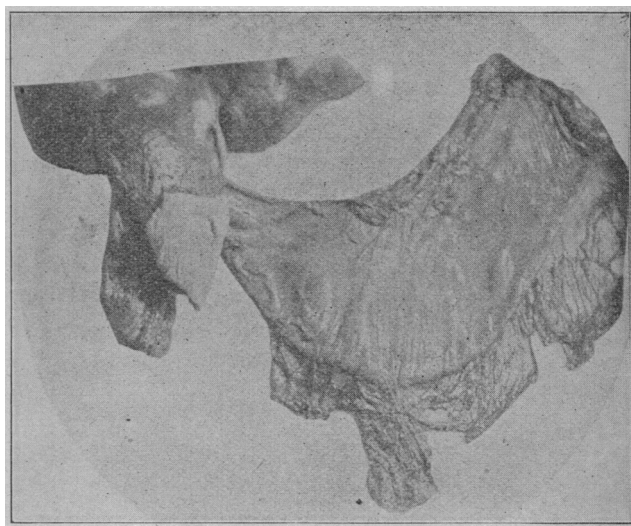


Fig. 3.—Cancer of the gall bladder, with enormous dilatation of the stomach. Adhesion to skin.

The serosa of the stomach and duodenum affords a better opportunity to study these cells as following the course of the lymph spaces. Extensive serial sections were made.

Figure 6 shows a section through the serosa and muscularis of the pylorus (low power). The thickened serosa extends from *a* to *a*. Within it can be seen strings

of the above described polymorphous cells surrounded by a rather voluminous connective tissue stroma. This connective tissue stroma contains numerous foci of round cell proliferation.

Figure 7 shows an area of the serosa under higher power; *a* represents a string of endothelial cells cut

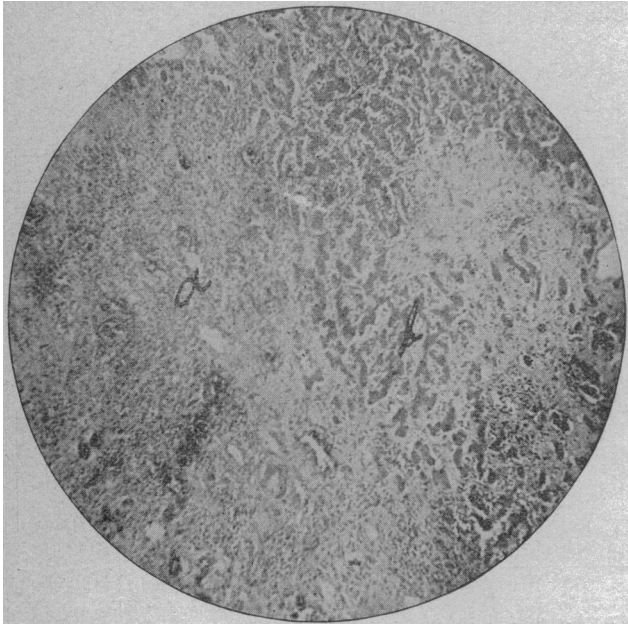


Fig. 4.—Hem. Eosin. Leitz, obj. 3. Section from margin of primary tumor. *a*. Connective tissue (inflammatory). *b*. Liver cells.

longitudinally; *b*, a similar string cut transversely; *c*, an irregularly branching cluster of endothelioma cells. The stroma is seen to contain numerous young connective tissue cells.

Figure 8 is a section prepared from the above-mentioned tissue bridge between the gall bladder and the

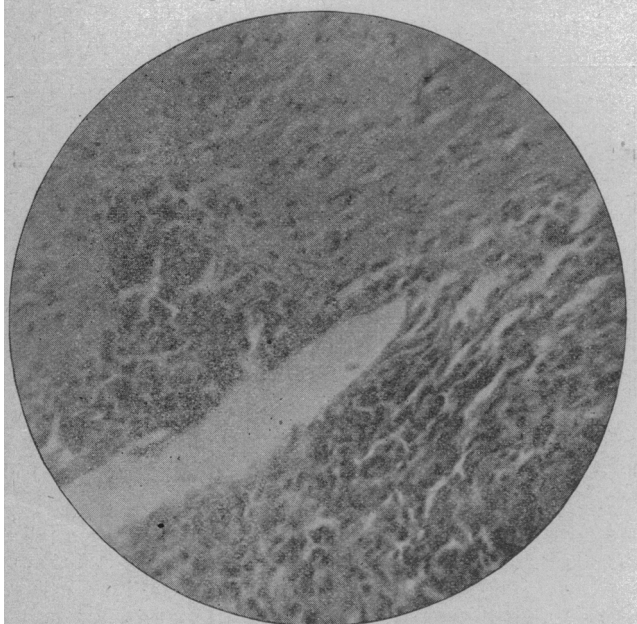


Fig. 5.—Hem. Eosin. Leitz, obj. 6. Section of tumor about 1½ c.m. inwardly from margin of primary tumor. Shows fibrillation of older tumor cells.

pylorus and duodenum. It shows exactly the same structure as does the center of the primary tumor; *a* represents the center of a large endothelial cell cluster. Distinct fibrillation of the endothelial cells at the margin of the cluster.

Figure 9 shows the cluster of tumor cells in serosa of the duodenum. The nuclei of the cells seem to be swollen (vesicular), the chromatin filaments stain distinctly; at *c* is cell with giant nuclei. Numbers of these giant cells, and also polynuclear giant cells were observed. There are a great number of cells which showed degen-

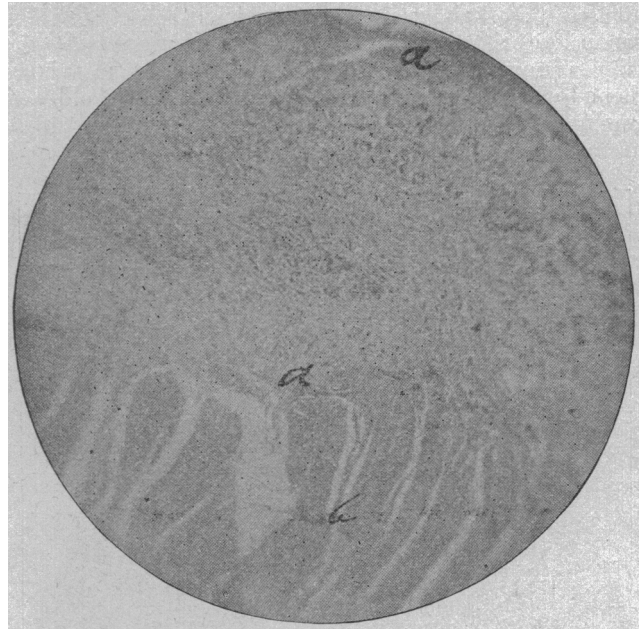


Fig. 6.—Hem. Eosin. Leitz, obj. 3. Section through serosa and muscularis of duodenum. *a-a*. Serosa. *b*. Muscularis.

erative coalescence of several nuclei, thus forming a long, sometimes semi-circular, sausage-shaped nucleus. At *a* and *b* atypical mitoses, of which all varieties were present. Abortive mitoses, clumping of the chromosomes were especially frequent.

Figure 10 is taken from the same slide as Figure 9.

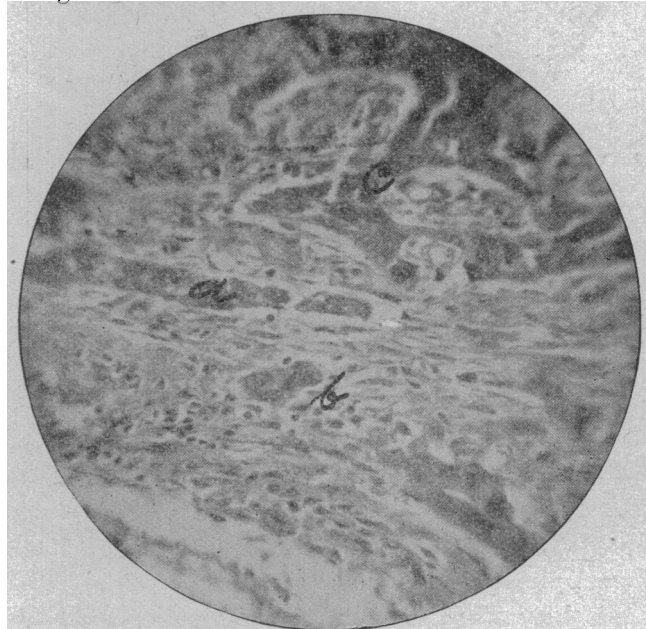


Fig. 7.—Hem. Eosin. Leitz, obj. 7. Area from Fig. 6 under higher power. *a-b-c*. Strings of endothelial cells.

In the center a cell is visible with a giant nucleus; in the cytoplasm a dead polymorphonuclear leucocyte. The nucleus of this leucocyte is undergoing karyolysis, the cytoplasm of the leucocyte is represented by a light ring around the fading semi-circular nucleus. Many in-

clusions similar to this one, with all gradations of degeneration, were observed. (Russell-Plimmer-Leyden-Schueller's Bodies.*)

In the lymph spaces, dilated excessively, the polymorphous "tumor cells" seemed to be suspended in the serous fluid (Fig. 8, *a*). An attempt was made to determine the chemical nature of this fluid in the fresh condition, but, owing to the small quantity obtainable, the attempt was unsuccessful. This much, however, was ascertained: The fluid did not contain any mucin; it may have contained pseudomucin, such as is found in numerous connective tissue tumors, but it was not viscid enough to consist entirely of this substance, or contain a great deal thereof. It did not ferment when brought in contact with yeast cells. It gave the reaction of albumin. The presence of small, round cells, scattered promiscuously through the polymorphous elements, suggested the interpretation of the fluid as lymph. (?)

DETERMINATION OF THE CHARACTER OF THE GROWTH.

These microscopic findings being incompatible with the diagnosis of carcinoma, and agreeing in every detail

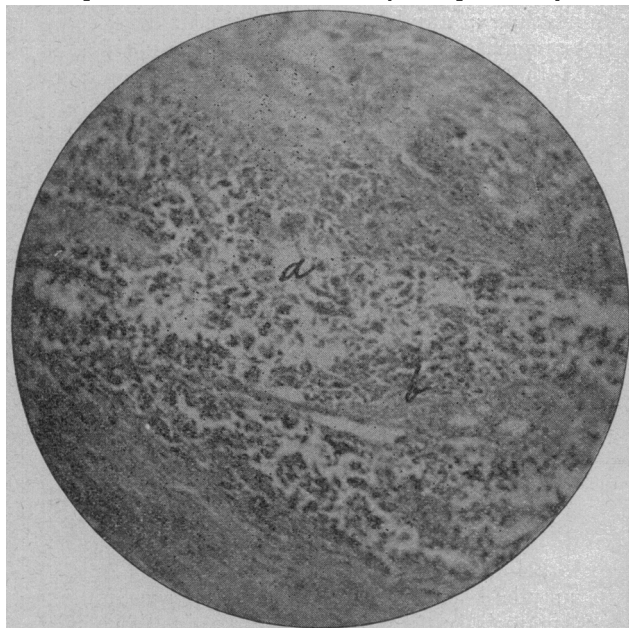


Fig. 8.—Hem. Eosin. Leitz, obj. 3.—Section of tissue bridge between liver and duodenum and stomach. *a*. Center of dilated lymph space filled with endothelial cells. *b*. Fibrillation of peripheral cells.

with the histopathology of endothelioma, the latter diagnosis was held as the correct one. Endotheliomata have been described by a great number of authors, and under a multitude of names, endothelial cancer, perithelioma, alveolar sarcoma, sarco-carcinoma, etc. I am not inclined to enter into the nomenclature of the subject, since that would lead to an almost endless discussion. A group of investigators believing the nature of endothelioma to be entirely or partly inflammatory, have proposed names like lymphangitis proliferans, and to convey also the neoplastic idea, lymphangitis carcinomatoides. One thing is certain, that a large number of sarcomata of organoid structure have been described as endotheliomata, which were common angiosarcomata, peritheliomata or hemangioendotheliomata. I do not see the necessity of classifying these tumors as endotheliomata, since we know that the majority of sarcomata originate from the perithelium or endothelium of the blood vessels anyway.

* With apologies to those discoverers whose names have not been mentioned.

Morphologic peculiarities are no criterion for the classification of tumors. This is shown by the confusion brought into the subject of endothelioma by the morphologic classification suggested by Hanseman.

If we, however, reserve the name of endothelioma exclusively to those neoplasms which are the result of an

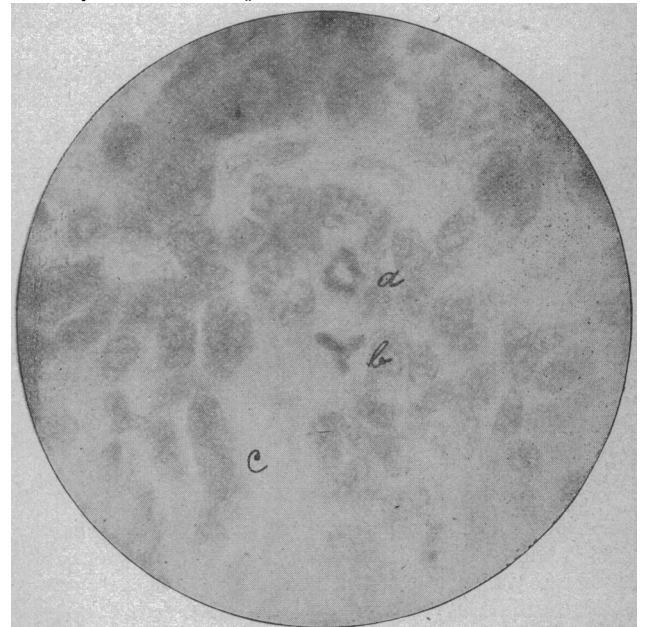


Fig. 9.—From center of alveolus in serosa of duodenum. *a*. Asymmetrical mitosis. *b*. Atypical, tripolar mitosis. *c*. Endothelial cells with giant nucleus. Saffranin. Leitz, 1/12.

abnormal proliferation of the endothelium lining the lymph spaces and serous cavities, we have well-characterized, clear-cut tumors.

That the name of lymphangitis proliferans is also justifiable will be shown below, for I think the inflammatory character of the tumor which I have described

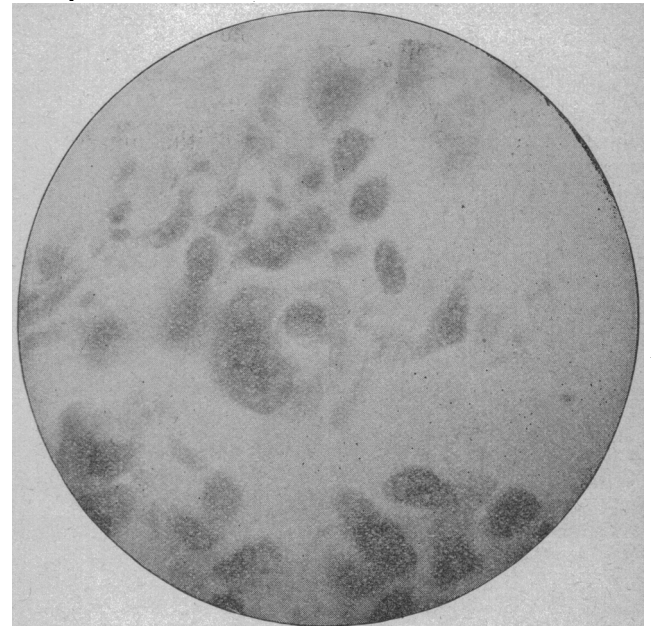


Fig. 10.—Polymorphous endothelial cells. In center of field cell with giant nucleus containing a leucocyte. Saffranin. Leitz, 1/12.

can not be questioned, at least the *primum movens* of the proliferation of the endothelial cells is of a nature the same as that found in chronic inflammations.

In a number of endotheliomata which I had the opportunity to study, I have never seen any endothelial

proliferation in the lymph spaces unless the surrounding tissue was also in a state of inflammatory hypertrophy. This observation is adduced by Neelsen as an argument in favor of the inflammatory character of the new growth. In addition to this one can easily see that the filling up with cells of the lymph spaces is not the result of an invasion of cells, as we see it in carcinoma and sarcoma, where the elements first get into places not their legitimate habitat, proliferate, and then destroy the surrounding tissue. In the true endothelioma there is first a swelling of the resident cells due to irritation (Reiz Anschwellung, Pollman), most likely of chemical nature, perhaps a cellular poison as described by Ehrlich. Thereafter the proliferation again of these same resident cells takes place in answer to the same formative irritation; dilatation of the lymph space follows. It is evident that the differentiation of the endothelial cells will be lost, or will at least suffer through this onslaught, and anaplasia will result. But this anaplasia may run through "physiologic" gradations. The highly differentiated mesodermic endothelial cells will change into connective tissue cells of a lower type, and will share the fate of all common connective tissue cells. They will fibrillate. This is shown strikingly in Figure 5. The older cells undergo fibrillation first, namely, those seen along the margin of the dilated spaces. Being remote from the lymph current they are deprived of an opportunity to perform their function, even if they were able to perform it; as a result, the cells resign themselves to the quiescent state of fibrillation. This is incompatible with anaplastic, blastomatous carcinoma or sarcoma cells, but is known as the routine process in inflammatory scar tissue formation, i. e., the cells do not maintain the blastomatous character of malignant cells.

The regional expansion of the process may be explained thus: The primary formative irritant was furnished by the presence of the gallstones in the gall bladder, perhaps with an addition of micro-organisms (which were not found). Or there may have been at first an inflammatory occlusion of the cystic duct. The lymphatics of the gall bladder had to remove the altered contents of the same. The change in the chemism of the substance carried by the lymph spaces caused the inflammatory changes of the lining epithelium, as described above.

The cells thus vitiated secreted in turn a vitiated substance which acted as the formative irritant to the adjoining still normal endothelial cells. In this method a circulus vitiosus is established. Experimental pathology and physiology may be able to prove these ideas some day.

Other retrogressive metamorphoses described in connection with endotheliomata, as myxomatous, and especially hyaline degeneration, are likewise frequently found in other common chronic inflammation, viz., in certain forms of peritonitides, zuckerguss exudates, etc., and in a great number of so-called benign tumors.

Furthermore, there is not an area in the tumor where there is not common inflammatory tissue surrounding the tumor cells proper, no matter how few they may be in number. Even in the extreme ends of the tumor where the endothelial cells are simply swollen and not multiplied, there is lively inflammation in the surrounding connective tissue. This, also, is unlike the peripheral growth of both carcinoma and sarcoma. We see a round cell sarcoma invade the normal muscular structures, a carcinoma metastasis in a normal lymph node, etc. Specimens stained with Sudan 3 (Formalin-freezing sections) showed numerous endothelial cells containing

fat droplets. These are most likely the "vacuoles" found and described in hardened specimens of endothelioma by Adler and others. The significance of these fat drops is not easily explained. Thoma has found that the covering endothelium of the serous surfaces absorbs albumin contained therein into fat. It may be that such a process went on in this endothelioma. The cells certainly do not show any fatty degeneration, but that there is an energetic metabolism going on in the cells is shown by the presence of the serous exudate filling and dilating the affected lymph space (Fig. 8, a). I can not believe that this liquid is a simple transudate since there was only an inconsiderable quantity of fluid in the free peritoneal cavity.

METASTASES.

In the tumor here described there was no metastasis; in two more which came under my observation, one of the peritoneum (Dr. E. F. Fish's case), and another of the skin, they were likewise absent. The cases reported in the literature of the last twenty years show about 50 per cent. with metastasis, but I have noticed in the illustrations that a number of these appear like common angio-sarcomata, i. e., the tumor cells are not found restricted to the lymph spaces. Neelsen, who described a number of endotheliomata, lays especial stress on the fact that the cells proliferate *in loco* and do not immigrate. But he has metastases. Wagner could not find any secondary tumors in a woman of 69, where all the tumor cells were within the lymph spaces. In a great many cases the metastatic tumors were found in the vicinity of the primary one; in these instances it is doubtful whether there was not a contiguous growth, as not one report mentions reconstruction methods as a means of ascertaining true metastases. Age and sex do not seem to affect the frequency of occurrence of endothelioma.

Hereditary influence has not been proven in man. Eberth-Spude have seen endothelioma in several members of a family of white mice. Transplantation has been unsuccessful.

OTHER CONCLUSIONS.

Endothelioma, hemangio-endothelioma and perithelioma may be blastogenetically alike, but the first tumor differs materially from the last two in the mode of growth, histology and clinical aspect. One need but think of the functional difference of the normal elements to appreciate this fact.

Endotheliomata are only relatively malignant. A great number of tumors reported under this name are essentially inflammatory.

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THE OPERATIVE TREATMENT OF CYSTOCELE AND PROCIDENTIA UTERI.

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The evolution of the operative treatment of cystocele and rectocele with resultant procidentia includes the following essential factors:

1. Operations of superficial denudation and reefing of the anterior vaginal wall as practiced by Sims and Emmet.
2. Operations on the lateral vaginal walls with superficial denudation, as described by myself and others.
3. The recognition of the essential fact that no operation can succeed that does not include plastic work on the lateral and anterior vaginal fornices with the definite purpose of forcing the cervix uteri back to its normal location in the hollow of the sacrum.

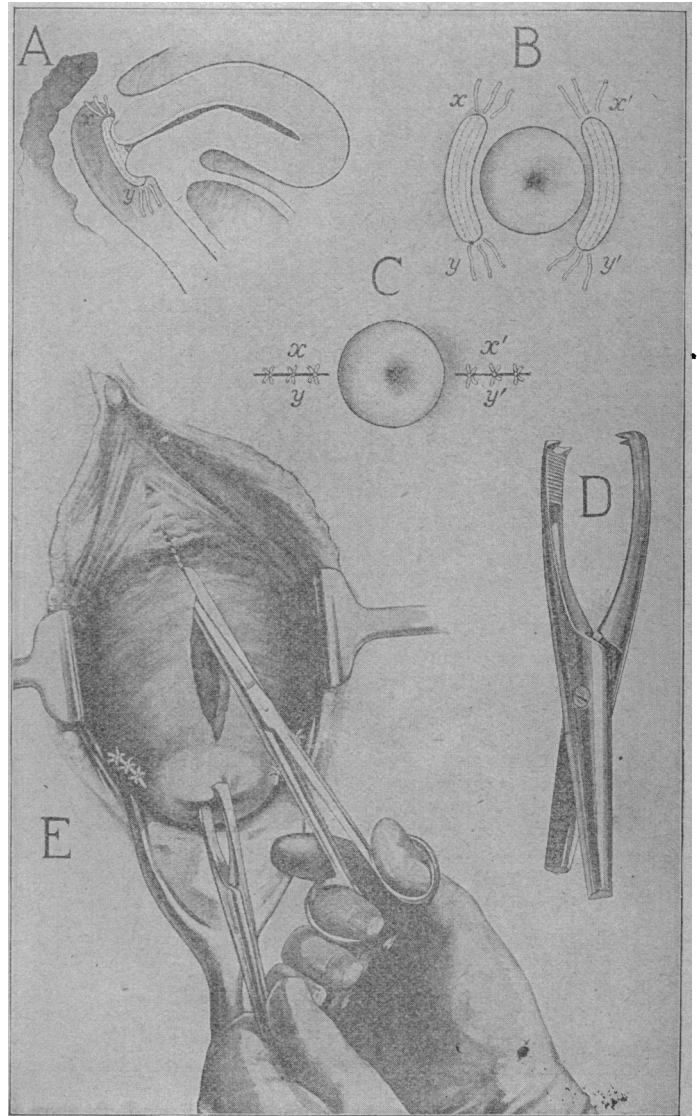
All these measures have been employed with varying success, the variation being dependent on the ability of the operator to force the cervix uteri back into the hollow of the sacrum and to make it stay there. This location for the cervix is the prime indication. With the cervix uteri in its normal posterior location, the corpus uteri must necessarily fall forward into its natural position of anteversion.

The methods above outlined, even though supplemented by hysterorrhaphy, have so frequently failed to give permanent results that many surgeons advocate the more radical alternative of hysterectomy. Of late, however, the essential principles and indications on which successful plastic work may be done are becoming more clearly defined. Dr. Edward Reynolds¹ of Boston sets forth two anatomic points which in the previous literature have not been very clearly brought out, but which necessarily underlie the success of this class of operations. They are: 1. That cystocele is hernia of the bladder and should not be treated by superficially denuding and reefing in the overstretched portion of the vaginal wall, but that this overstretched portion should be removed and the lateral margins of it should be united by interrupted sutures. In other words, this hernia should be treated like other similar conditions by reduction and the excision of the sac so as to expose and unite the strong, fixed facial margins of the wound. 2. A successful operation must recognize two fixed points of attachment of the anterior vaginal wall; the first at its lower extremity to the inner surface of the pubes; the second at its upper extremity to the facial structures

on either side of the uterus, which are in direct connection with the broad ligaments. Accordingly, I have made radical changes in my method of elytrorrhaphy for the correction of cystocele and procidentia uteri. The modified operation which I have performed several times with most gratifying results may be divided into two steps, and is briefly as follows:

1. A crescentic denudation and suturing in the vaginal wall on each side of the cervix uteri.
2. The removal of a portion of the anterior vaginal wall and suturing together of the wound thus made.

FIRST STEP.—The first part of the operation is performed preferably with the patient in Sims' latero-prone



position and the vagina exposed by means of Sims' speculum. The blade of the speculum should be perforated at the extreme end. Before the speculum is introduced the cervix should be attached to the end of the blade by means of a temporary suture which is passed through the posterior lip of the cervix and the perforation in the speculum and tied. This suture should remain in place throughout the first step of operation. The object of this temporary stitch is to hold the cervix uteri far back in the hollow of the sacrum while the sutures are being secured which shall fix it there. When the cervix is thus held back the space anterior to the cervix is so increased

1. An epoch making paper on this subject has recently appeared from the pen of Dr. Edward Reynolds, American Medicine, Aug. 2, 1902.