CHOLECYSTITIS

ETIOLOGY, DIAGNOSIS AND TREATMENT *

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The remarks which I shall make on this subject are based on the analysis of the histories of 350 gallbladder cases, which have recently been reviewed. Rather than give a statistical study involving percentages, which are often misleading, I propose to present the fundamental principles which have been elucidated. The application of these to pathologic conditions of the biliary tract I feel has aided in more accurate and, what is more important, earlier diagnoses, resulting in more rational treatment, with better end-results.

ETIOLOGY

In order to form our own opinions regarding this phase, all gallbladders and, when possible, all appendixes removed at operation were subjected to routine culture between July, 1916, and July, 1920. Recently we have not been content with aerobic cultures solely, but have used partial anaerobic culture methods. In the latter, the tissue has been macerated by grinding in a mortar with fine sand, and then planted in the depths of agar medium. Miss Wessels, working in the department of bacteriology under the supervision of Detweiler, has recently most carefully carried out this part of our investigation. In the earlier cases, it was possible to isolate a streptococcus from the wall of the gallbladder in a certain number of instances. In many of these, cultures of an identical strain were also taken from the wall of the appendix; the majority of the cases, however, were sterile, or grew *Bacillus coli,* which we considered was incidental rather than pathogenic. The number of instances of associated streptococci in the appendix and gallbladder led us to believe that organisms from a distant focus were the source of the trouble. The question then arose: Did the gallbladder and appendix become involved simultaneously, or was the appendix the initial lesion, with the gallbladder secondarily infected through the portal circulation? The latter we believe to be most common.

Between July, 1919, and February, 1920, however, our results as far as cultures are concerned have given us grave cause for thought. This series representing fifty-two cases gave cultures in eight instances;

* From the surgical clinic of Dr. F. N. G. Starr.

one pure colon; one colon and streptococcus; two *Staphylococcus aureus;* one diphtheroid; three pure streptococci; all the rest being sterile. One interesting feature in this series was again called to mind. namely, that as yet we have not recovered *Staphylococcus aureus* on culture in any instance in which the patient did not give a clinical history of mucous colitis in varying degrees of severity. That we have not removed normal gallbladders is proved by the subsequent clinical history and the microscopic examination, which is made in every instance. The microscopic changes which have been found are those characteristic of reaction to injury and were present in all gradations.

Thus, we are forced to believe that the symptoms resulting from a diseased gallbladder for which patients seek relief are not due to the immediate presence of organisms in the lumen or wall of the gallbladder. The corollary, then, is that they are the result of damage to the tract which remains after the infecting organism has been effectively dealt with. That the infecting organism is killed one cannot say, but, if present, it is so susceptible to changes of environment that only rarely can one recover it by culture.

We are quite in accord with Naumyn's hypothesis that changes seen in the biliary tract are the result of the reaction to bacterial injury. Graham [1] recently, however, has thrown some light on the significance of colon bacilli found with cultures. He believes that they are not incidental or natural habitants of the biliary tract, but are pathogenic factors, the latter belief being based on the fact that the serum from such individuals agglutinates the strain of colon isolated in the particular culture. In one case, agglutination occurred with a serum dilution of 1 in 80. That this phenomenon is sufficient evidence on which to base a conclusion is open to doubt.

DIAGNOSIS

The diagnosis of the gallbladder disease presents difficulties of varying degrees, dependent upon the damage which has remained in the biliary system. In order that we may classify the various groups of biliary symptoms, the scheme [2] formerly suggested seems adequate: (1) the type in which all the symptoms are gastric with nothing definite to direct one's attention to the right upper abdomen. Here the diagnosis has to be made very largely by a process of elimination. (2) That in which symptoms are referred to the right upper abdomen. These are the result of subacute inflammatory changes, or to the movement of calculi, or both. (3) Acute emergencies—such as empyema, gangrene and common duct obstruction, not forgetting malignancy.

1. Graham, E. A.: Hepatitis, Surg., Gynec. & Obst. **26**:521 (May) 1918.
2. Starr and Graham: Cholecystitis, Ann. Surg. **68**:188 (Aug.) 1918.

The first two types present the greatest difficulty. The reason for this is to a large extent due to the fact that for such a long time gallbladder lesions meant gallstones. Until we can forget that such foreign bodies form, or at least until we consider them only as an incident rather than as essential, there is slight hope for improvement in the surgical therapy of biliary lesions. To wait for jaundice and colic in order to make a diagnosis is like waiting for metastasis before diagnosing a malignant new growth; also nausea and vomiting are no more essential for diagnosis than is jaundice. The diversity of symptoms relieved by surgical intervention in diseases of the biliary tract is too widespread to be explained by contiguity inflammation, originating in the gallbladder and ducts. Moreover, such an explanation can gain no support from the gross pathology observed during operative procedures. The fact remains, however, that patients are relieved of widely divergent and varied symptoms by dealing surgically with the local pathologic condition of the biliary tract.

In a recent article, Goldie [3] has laid stress on the proper interpretation of gastro-intestinal symptoms, pointing out clearly that in the large percentage of instances in which the patient seeks relief from "stomach trouble" the etiologic factor is found in other portions of the gastro-intestinal tract, with no gross lesion of the stomach. It is in the proper interpretation of such symptoms as fulness in the epigastrium, belching, loss of appetite, etc., that we can hope for most in the accurate diagnosis of noncalculous cholecystitis.

An important fact to remember is that the gastro-intestinal tract has a double nerve supply: first, the vagus and the nervus irrigens from the bulbar and sacral segments; and secondly, the thoracolumbar autonomic system, or the sympathetic nervous system of the old nomenclature. The thoracolumbar autonomic system controls the sphincter system of the gastro-intestinal tract; that is, it has control of the pylorus and ileocecal sphincters, and possibly the sphincter of Oddi, at the entrance of the ampulla of Vater into the duodenum.

The association between the ileocecal sphincter and the pylorus was proved by Gaskell [4] more than thirty years previous to its clinical application, in the treatment of so-called appendicular dyspepsia, by the removal of a chronically diseased appendix. This association has now come to be so well recognized clinically that it needs only to be mentioned. A confirmation of this close association was recently reported,[5]

3. Goldie, W.: Interpretation of Gastro-Intestinal Symptoms, Canad. M. A. J. **11**:5 (Jan.) 1921.

4. Gaskell, W. H.: The Involuntary Nervous System, New York, Longmans, Green & Co., 1910.

5. Ileocecal Sphincter, Editorial, Brit. M. J., Aug. 19, 1919.

in which as a result of a cecostomy for dysentery, the ileocecal valve became visible in the wound; thus the contraction of the sphincter could be visualized and palpated, and the close relationship between its contraction and gastric intake was demonstrated. Is it then inconceivable to believe that the same relationship exists between ileocecal valve, pylorus and sphincter of Oddi, which controls the outflow of the common bile duct? Meltzer's [6] opinion would seem to substantiate this.

Thus, we see that in addition to the so-called "appendicular dyspepsias," we must add the "biliary dyspepsias," and be on our guard lest gastric symptoms mask the pathologic condition of the biliary tract. We are forced to believe that in nearly 90 per cent. of instances in which the symptoms are gastric, the pathologic condition is remote from the stomach, and the clinical picture the result of reflex irritation arising from other portions of the gastro-intestinal tract. Such reflex irritation is exemplified by pylorospasm. In one instance, during a gastro-intestinal roentgen-ray examination with an opaque meal, a gastric spasm was noted and examined at intervals for a period of half an hour, and a series of plates were made. This spasm was so constant and unchanged that a roentgenographic diagnosis of hour-glass stomach was made. At operation, however, the stomach was found to be normal, and the patient was relieved of all symptoms after the pathologic condition of the biliary tract had been dealt with and the appendix had been removed. Pylorospasm is very real, and can temporarily give symptoms which would lead even careful observers to diagnose a primary stomach lesion. Often during the course of operations one can visualize spasm in the pyloric region of the stomach, occasionally of the ring type, but most often localized areas become contracted, giving a gross appearance not unlike that which would develop from the application of a hot iron to the serous surface. Such areas are blanched, raised above the serous surface of the stomach, feel firm to the touch; in fact they are very like the base of an ulcer, and could easily be mistaken for such unless one were experienced in their gross appearance, or waited for from twenty to thirty seconds and watched them disappear, leaving a normal stomach area. Indeed, it has been our misfortune to have to deal with cases in which, as the result of an erroneous interpretation of these areas, a gastro-enterostomy had been performed with, of course, no relief to the patient.

A further interesting observation has been made during operation on patients suffering from pylorospasm due to cholecystitis. An area of the stomach, from 2½ to 3 inches proximal to the pylorus, appeared erythematous, not unlike the appearance of a first degree burn. The

6. Meltzer, S. J.: The Disturbance of the Law of Contrary Innervation as a Pathogenetic Factor in Diseases of Bile Ducts and Gallbladder, Am. J. M. Sc. **153**:469 (April) 1917.

superficial vessels were engorged, tortuous and dilated; the pyloric vein varicose, and the vessels in the gastrohepatic omentum markedly varicose. May this be a factor in the production of a gastric or duodenal ulcer, which is not infrequently found in association with a chronically diseased gallbladder?

One realizes also that nearly all abdominal pain, crampy or colicky in character, is the result of muscular spasm in, or distention of, a hollow viscus with its own contents, or those of a mildly inflammatory character. Hence a spasm can be initiated by a diseased gallbladder, and give crampy pain of varying degrees of severity. Also a spasm occurring out of time with the normal peristaltic waves could cause increased intragastric pressure and a distention of a segment of the stomach, with the same result. That this is no mere hypothesis is shown by the fact that such occurrences are daily visualized in the roentgen-ray laboratory.

I cannot agree with Stewart and Barker [7] that there is a constant hypermotility in chronic disease of the gallbladder, for we have seen 40 per cent. gastric residue ten hours after a barium meal, and at operation found a normal stomach and duodenum. Thus, it would appear that the essential thing is to differentiate the lesions that will initiate pylorospasm.

The recent tendency has been to rely so much on roentgen-ray diagnosis that it will be considered last. The really important part of the investigation of such a patient is a carefully taken history. One has only seriously to attempt this for a few months to realize how difficult and formidable the task becomes. We are dealing with patients who have a history of gastro-intestinal disturbances extending over many years, and have tried to rise above it for so long that unconsciously they will give misleading answers to inquiries, unless one approaches their disabilities from many angles. An inquiry as to the previous health of such patients will usually reveal a history of gastro-intestinal upsets or bilious attacks beginning when quite young and continuing with remission until seen at consultation.

One can divide the life cycle into various stages: first, the prepubescent period; second, puberty; third, from puberty until 25 years of age; fourth, from 25 to 40 years of age; fifth, from 40 years of age till death. In these various stages we have three main factors to consider: physical trauma; mental and physiologic strain, and infections. In the prepubescent period there are the gastro-intestinal upsets due to dietetic errors, and the infections so common in childhood. In the

7. Stewart, G. D., and Barber, W. H.: Hypermotility of the Stomach in Gallbladder Disease, Duodenal Ulcer and Appendicitis, J. A. M. A. **73**:1817 (Dec. 13) 1919.

second period, there are the physiologic phenomena of puberty at a time when with our educational system there is tremendous mental strain. In the third, from puberty till 25 years, there is the greatest freedom from all these stimuli. In the fourth, there are business responsibilities as a mental strain in men, and child-bearing in women. In the fifth, there are the inevitable degenerations coupled with the menopause in females.

In gallbladder disease, it is noted that the periods of exacerbation correspond very accurately with these disturbances, and this fact is strong evidence of such a lesion. In other words, there is a close association between nervous, physical and physiologic strain, and the occurrence of symptoms. One of the important physical factors to consider is, of course, not only the acute infections from which the patient has suffered, but also the possible chronic ones from which he may still be suffering, such as, infected teeth, tonsils, or accessory sinuses.

Goldie [3] has also pointed out the very great importance of noting the time of occurrence of gastric distress, and also of analyzing in detail the various symptoms. He cites appetite as an instance of the necessity for qualifying statements, because in the atonic stomach the appetite is lost, with no distress, whereas in the hypertonic stomach there is loss of appetite with distress. It is common to hear statements made that there is a loss of appetite in chronic gallbladder disease; but if this is pursued further one will find that the appetite is good, but readily satisfied. It is not the loss of appetite which one finds in cases of gastric ulcer, in which the patient has a fear of the consequences of taking food. The gallbladder patient, however, in addition to being satiated by a small volume of food, complains of an indefinite fulness, distress or uncomfortable feeling in the epigastrium about half an hour later, rarely complaining of actual pain. There may be belching of gas, but this will not occur for about half an hour after taking food. This must not be confused with the belching which occurs immediately after a ravenously eaten meal, which is due to the fact that the cardiac sphincter has not yet closed. Whether the patient suffers from belching of gas is dependent upon whether the peristaltic wave is going to force the pylorospasm or the cardiac sphincter. This discrepancy in the contracting of various parts of the stomach is evidenced by clinical symptoms of belching, pyrosis, or actual nausea and vomiting, depending on the degree of muscle tonus.

At this stage of gastro-intestinal metabolism, bile is normally poured into the duodenum. If the biliary lesion has rendered the nervous control of the sphincter hypersensitive, can we not have spasm of the ampulla and of the pylorus occurring at the same time? That the flow of bile is interfered with is borne out by the fact that these patients

volunteer the information that they suffer more discomfort when eating food fried in grease, or that containing an excess of fat. Archibald and Brow [8] and Mann [9] have shown clearly that the sphincter of Oddi is real, and when contracted will withstand a pressure varying from 100 to 500 mms. of water.

Sick headaches or bilious attacks are often the real cause of a patient's appearance in the consulting room. Here one has to rule out headache and nausea of extragastro-intestinal origin. The commonest probably is a refractive error, in which the headache precedes any gastro-intestinal discomfort. This emphasizes the fact that in patients whose headache precedes any abdominal discomfort, the etiologic factor is usually extragastro-intestinal. In chronic appendicitis, we have the nausea, but headache is a rare accompaniment; but both occur subsequent to abdominal distress. In gallbladder disease, there is an epigastric distress previous to the appearance of a headache or nausea. In atonic lesion of the cecum, with incompetency of the ileocecal valve, there is a cyclic occurrence in which the headache is worse in the morning, with the period of greatest well-being between 4 and 6 p. m., which time corresponds to the reemptying of the ileum. If the patient complains of headache and nausea of recent occurrence, with no history dating over a long period, one can reasonably exclude gallbladder disease.

Having thus elicited the subjective history, one proceeds to the objective, by means of physical examination. Babcock [10] has drawn attention to the fact that one must be on the alert to avoid an erroneous diagnosis of a primary cardiac lesion, when a patient presents a cardiac murmur, associated with an enlarged liver and gastric symptoms. One should not lose sight of the fact that the liver and bile ducts may be the seat of the primary pathologic condition. One patient under observation presented extreme arrythmia, enlarged liver, and jaundice, the whole condition clearing up when the pathologic condition of the biliary tract was dealt with. In this instance there is additional interest because the source of the infection was apparently from an ischiorectal abscess.

On abdominal palpation one too often elicits nothing but negative information in gallbladder disease. If the gastric distress is due to an appendicular lesion, one can often elicit a tender area in the right iliac

8. Archibald and Brow: Experimental Production of Pancreatitis in Animals, As Result of Resistance of Common Duct Sphincter, Surg. Gynec. & Obst. **28**:529 (June) 1919.

9. Mann, F. C.: A Study of the Tonicity of the Sphincter at Duodenal End of Common Bile Duct, J. Lab. & Clin. Med. **5**:107 (Nov.) 1919.

10. Babcock, R. H.: The Diagnosis of Chronic Cholecystitis Complicating Cardiac Lesions, J. A. M. A. **73**:1929 (Dec. 27) 1919.

fossa, with pain referred to the epigastrium; or if due to an atonic cecum, one can roll the full boggy cecum under the fingers. In the noncalculous variety of gallbladder disease, the tenderness under the right costal margin is indefinite, but is found occasionally. When pressure in this area produces pain radiating to the epigastrium, it is fairly good evidence of a biliary lesion. Several deep inspirations following one another in quick succession often produce a tired sensation in the right upper quadrant of the abdomen. Another tender point often noted is just above and to the left of the umbilicus. This has been ascribed by some observers to spasm of the colon, but in view of the constant location of the tender point and the extreme variation in the site of the colon, it seems highly improbable that this is the proper interpretation. It is felt that it is due to pancreatic involvement. In a former communication the close association between pancreatic lesions and gallbladder disease has been emphasized.[11]

However, tenderness along the course of the whole large bowel, together with the presence of mucus in the stools, has been noted so often in connection with biliary disease, that it cannot be regarded as a mere coincidence. In fact, it is felt that biliary lesions are responsible for mucous colitis to such a degree that we have explored the liver and bile ducts on little more than this evidence in a few instances, and have been rewarded by finding pathologic conditions which when dealt with surgically have relieved the patients of their disability. The convalescence in such a clinical condition is nearly always in direct ratio to the amount of mucus present, and the chronicity of the lesion; that is, the more severe the colitis, and the longer its presence, the more prolonged will be the convalescence.

Another important factor in determining the site of the lesion is the alteration of the superficial sensory and motor reflexes. Goldie [3] lays emphasis on this, and states:

> There are apparently three sources from which the sensory nerve fibers are derived, all the nerve cells of which remain within the central nervous system, including the posterior root ganglia. First, those fibrils which accompany the original ingrowth; second, those which accompany the blood vessels and are associated with the thoracolumbar autonomic system; third, those derived from the somatic areas, which provide supporting structures to the specialized and fully developed secondary outgrowths of the gastro-intestinal tract.
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> The primary ingrowths develop from the anterior and posterior appendages, without any intimate relationship with the somatic areas, and join each other in the region of the tenth dorsal somatic area. Before the secondary development takes place, the great growth in length gives rise to an "S"-like curve in the anterior ingrowth, the upper part of which passes to the left in the region of the fifth somatic area, crosses the midline to the right about the seventh to

11. Starr Clinic, Canad. M. A. J., March, 1921.

eighth dorsal, and recrosses the middle line from right to left in the region of the seventh to eighth dorsal, terminating at this point in the cecum. The stomach develops mainly on the left from the sixth to eighth segments. The outgrowth, which becomes the gallbladder and liver, develops dorsally, and grows out to the right, so that the liver obtains its supporting structures from the eighth to ninth dorsal segments. The gallbladder, less extensive in its growth, develops near the middle line, obtaining its structural support just to the right of the midline of the ninth to tenth somatic areas.

The clinical application of this is that in chronic gallbladder disease one finds that on lightly stroking the abdomen there is increased hypersensitiveness and hypermotility in the right upper quadrant of the abdomen, and also, as pointed out by Ewald, just below the tip of the right scapula. Also one finds tenderness on pressure over the posterior branches of the *right* ninth and tenth intercostal nerves, whereas if the lesion were in the cecum or appendix, there would be hypersensitiveness of the *left* upper abdomen, and tenderness on the posterior branches of the *left* eighth, ninth and tenth intercostal nerve; and if the stomach itself were at fault, it would be tender over the posterior branch of the left sixth and seventh intercostal nerves.

Thus, we see the extreme importance of a careful history, taken with a wide mental perspective, and a careful physical examination based on embryology, if we are to deal effectively with gallbladder disease at a stage when the mortality is very low, rather than waiting for definite localizing signs, when the involvement is such as to give an operative mortality of from 15 to 25 per cent., with a much smaller percentage of complete cures in the survivors.

Having examined a patient with a chronic gastro-intestinal disorder in this manner, one can then refer him to the roentgen-ray laboratory with a provisional clinical diagnosis. The most one can hope for from the roentgen-ray examination of patients suffering from early noncalculous cholecystitis is the exclusion of other pathologic conditions. A roentgen-ray expert can exclude ulcer of the stomach or duodenum, and atony of the cecum with incompetency of the ileocecal valve. Whether one should accept a roentgen-ray diagnosis of chronic appendicitis is doubtful. Chronic appendicitis, as the sole lesion, in the absence of a previous acute attack, is a very rare condition, and a diagnosis that should not be lightly given, if the number of persons one sees with beautiful scars following appendectomy in such cases, but no relief from symptoms, is any criterion. It would thus appear unwise to accept the evidence of retention of barium in an appendix as sufficient evidence in itself to justify a diagnosis of chronic appendicitis.

COMPLICATIONS

It is not proposed to enumerate the complications which may occur for they are well known to everyone, but to state that all complications have been encountered in this series. Some are worthy of note. Two patients with empyema had an associated acute appendicitis in which both organs were covered with fibrin. Such an occurrence serves to put one on guard during operations for these lesions. Two instances of focal necrosis in the liver have been encountered, and we can give no satisfactory explanation. In both cases, the liver presented white areas which looked not unlike localized tubercles in one instance, and in the other, closely resembled new growth. In each case, however, microscopic examination proved that they were the result of the inflammatory process. Both patients have replied to the questionnaire and are at present perfectly well.

One often hears it stated that rupture of a gangrenous gallbladder is of common occurrence. In our series it has occurred only twice. Neither case gave the grave clinical picture that one would expect from such a serious lesion.

Pancreatitis has been encountered in all stages from a mild fibrosis to the acute hemorrhagic variety. Of the latter type, one case occurred recently, and while stones were present in the gallbladder, the common duct was patent. Can spasm of the ampulla explain the regurgitation of bile into the pancreatic duct with the resultant lesion? Archibald [8] has shown that this is very likely. Professor Mackenzie [12] suggests as a further explanation that there is a stenosis at the ampulla due to long continued inflammation and fibroblastic proliferation, causing some bile to be constantly forced into the pancreas.

Lastly, as a complication let us not forget the possibility of carcinoma. Stones were present in every case of carcinoma encountered. We know the disastrous clinical results of such a lesion. This in itself should be a great stimulus to an attempted early diagnosis previous to the formation of calculi.

TREATMENT

That a diseased gallbladder will eventually require surgical treatment for relief of symptoms is now fairly well established. That the end-results of surgical intervention show a higher percentage of cures than formerly is a fact. That still better results can be obtained is not without the realm of possibilities. Our ambition in treatment is thus to find the guiding principles for our operative procedure which will give the maximum percentage of cures.

The greatest principle underlying such an idea is to realize that we must enlarge our mental horizon and visual field regarding gallbladder

12. Mackenzie, J.: Personal communication.

disease and the pathologic condition associated with it. One can but rarely diagnose with certainty all existing pathologic conditions prior to operation. Unless one appreciates the dissemination of the lesions which may be present, one will many times fail at operation to add to the preoperative findings. Hence adequate and appropriate exposure is absolutely essential not only from the standpoint of safety, but to enable one to grasp in panorama the whole pathologic condition.

When the abdomen is opened, a definite routine of investigation is followed before deciding upon the particular procedure necessary in any case. In other words, we adapt the operation to the pathologic condition; we do not make the pathologic condition suit a stereotyped operation.

The plan of investigation is this: The cecum is delivered into the wound, and its tonicity is noted. The terminal ileum is examined for kinking. The presence or absence of pericecal or pericolic bands is noted. The glands of the mesentery are palpated, and here one is often rewarded by finding calcareous areas, tombstones of a previous tuberculous lesion which may be invaluable in future treatment and prognosis. The appendix is removed; the competency or incompetency of the ileocecal valve is noted. Attention is next directed to the stomach and duodenum for the presence of ulcer or spasm. The pancreas is then examined by palpation and if it suggests a pathologic condition, can often be visualized through the anterior layer of the lesser sac above the stomach. Having determined the presence or absence of a pathologic condition in these areas, the gallbladder and ducts are examined. To determine at operation whether a gallbladder is pathologic or not in the early stage of the disease requires careful attention to detail and accurate observation.

It is now well established that a gallbladder which in the gross shows little evidence of a pathologic condition can cause a diversity of clinical symptoms, which are relieved by its removal. Recently, we have observed that the liver presents gross pathology in a considerable number of cases. The changes are principally two: First, in the instances of a calculous cholecystitis, the liver is larger and more friable; secondly, areas of localized scarring or cirrhosis either in the region of the gallbladder or occasionally scattered diffusely throughout the liver. As to the nature and pathogenesis of this condition, I cannot state definitely; but in the few cases in which we have excised areas of liver and examined the tissue microscopically, we have found evidence of an inflammatory reaction around the peripheral bile capillaries. I feel that this is the result of a pathologic condition of the biliary tract and will often give a clue to the localization of the lesion in an otherwise

obscure intra-abdominal condition. The ducts and their associated lymph glands are palpated in their entirety and as accurately as possible any abnormality is made out.

As far as pericholecystic adhesions are concerned, I believe that with the exception of the obvious instances in which they result from contiguous inflammatory processes, they are an indication of a pre-existing inflammatory process, originating in the wall of the gallbladder. That such adhesions can cause definite symptoms I do not doubt. In 1,000 cases reported by Smithies,[13] only 4.2 per cent. gave no symptoms of gallbladder disease. That the mere separating of these adhesions will secure the desired result, I do not believe. In a later communication, it is hoped to prove that abdominal adhesions are the result and not the cause of intra-abdominal pathologic conditions, the latter being responsible for the clinical symptoms, except in the few instances in which they cause intestinal obstruction.

As to the question of drainage versus excision of the gallbladder, I believe that the condition of the patient is the determining factor, except in case of irreparable common duct obstruction, in which it is used in performing cholecystenterostomy. One reason for this stand is that I agree with Judd [14] that the common duct dilates after cholecystectomy. Thus the sphincter of Oddi is no longer effective, and continuous drainage of bile into the duodenum is allowed, and thus we remove the source of our reflex irritation. This is borne out by Meltzer.[6]

The real question we have to decide is: Shall we or shall we not drain the biliary tract? There are four instances in which I feel drainage of the biliary tract is demanded in addition to the removal of the gallbladder: (1) in cases of subacute pancreatitis with swelling of the pancreas; (2) in cases in which the gallbladder is filled with fine sand; (3) in those in which the patients' condition permits a cholecystectomy but in which the patients are undernourished and anemic, the result of a long illness, and (4) in cases of long-continued noncalculous jaundice of biliary origin.

To put a tube in the gallbladder I feel is not in the least efficacious in draining the biliary tract. The average volume of bile collected from a cholecystostomy is but a small percentage of the volume secreted.

Direct drainage of the common duct is not without its disadvantages. In order to do this, the duct has to be incised and it must heal by fibroblastic proliferation which may cause a stenosis, if not obstruction.

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13. Smithies, Frank: Pericholecystitic Adhesions, J. A. M. A. **71**:1804 (Nov. 30) 1918.

14. Judd, E. S.: The Effect of Removal of the Gallbladder, Surg., Gynec. & Obst. **24**:437 (April) 1917.

I have chosen to pass a catheter through the stump of the cystic duct and thence into the duodenum. Such a procedure has several advantages. In the first place, the entrance is gained to the common duct with no trauma to that structure; the catheter passing through the sphincter of Oddi at the ampulla causes the sphincter to relax and the bile drains continuously into the duodenum, around the catheter; thus, only the small volume which drains through the cystic duct is lost to the patient's metabolism. This volume is drained to the outside by a second tube which is placed close to the stump of the cystic duct, the general peritoneal cavity being walled off by means of strip gauze.

Still other advantages are the therapeutic possibilities of a tube being in direct communication with the duodenum. This has recently been pointed out by McWhorter.[15] The use of this tube in conveying nourishment directly into the duodenum was first described by F. N. G. Starr [16] in 1899. We put in now varying strengths of glucose for nourishment, and if the requirement is simply to supplement the body fluids, the normal saline can be used. This is particularly valuable in the type of case in which the patient is undernourished and suffering from a secondary anemia. When the patient has reached the stage in which cathartics are required, they too can be given by means of the tube. Since adopting this practice in emaciated patients, the postoperative convalescence has been much less anxious, and the patients have recovered much more rapidly. As to the length of time that the tube should be left in situ, I believe that with the exception of the pancreatic cases the condition of the patient is the index of when it should be removed. In the former, I believe that it should be left in situ for at least two, if not several, weeks, depending upon the degree of pancreatic involvement.

Another factor which I believe is of great importance is the prevention of postoperative adhesions. This to a large degree can be accomplished by the restoration of the peritoneal continuity of the under surface of the liver. A running suture of catgut is used to unite the reflected margins of the peritoneal coat of the gallbladder. In some instances, this is not possible, particularly if there has been a large gallbladder or an acute process, with a resulting friable liver tissue. In such a condition, a free omental graft is laid over the fissure and fastened by interrupted catgut sutures. This in addition is very effective in controlling the oozing which is often troublesome in this type of case.

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15. McWhorter, G. L.: The Common Bile Duct Sphincter, Surg., Gynec. & Obst. **32**:124 (Feb.) 1921.

16. Starr: Canad. J. M. & S., 1899.

As to the advisability of always using drainage in these cases, I believe that it is safer. Why it should be necessary to drain a simple, straightforward cystectomy when we can feel practically certain that no bile will leak from the cystic duct, I cannot explain. However, in cases in which no drain was left, I felt that the postoperative convalescence was not so smooth.

PROGNOSIS AND RESULTS

The result of surgical interference in biliary lesions is disappointing to a certain percentage of the profession. This conclusion to a large extent is due to the fact that biliary colic was the signal for operation in the past, and surgery could immediately relieve this. Today we are attempting earlier treatment and at a time in the course of the disease when surgery can deal only with the exciting cause, having to depend on nature and the lapse of time to repair the damage done to the nervous mechanism and thus abolish the reflex disturbances. This improvement may in a fair percentage of cases be spectacular; but in the type of case in which there is a definite relationship between the attack and mental and nervous exhaustion, the improvement will be definite but gradual over a period of from ten to twelve months.

In our experience, the stone cases have a certain chance of complete cure from drainage, but in replies to our questionnaires we find that in the earlier acalculous cholecystitis cases in which drainage was used there is a persistence of a certain degree of the gastric distress.

CONCLUSIONS

1. The primary etiologic factor in gallbladder disease is bacterial infection. If, however, the patient seeks relief at this time, it will be most often because of an acute inflammatory lesion of the gallbladder.

2. The chronically diseased gallbladders produce symptoms of a reflex nature as a result of tissue changes, resulting from the presence of an avirulent organism.

3. The vast majority of gallbladders removed for reflex symptoms are sterile on culture.

4. The diagnosis of a diseased gallbladder rests largely on a well-taken history, keeping in mind the innervation from an embryologic standpoint, and thus making possible a correct interpretation of clinical symptoms.

5. The increased efficiency of gallbladder surgery lies in the avoidance of stereotyped operations, the recognition of the whole pathologic condition present, and then suitably dealing with it.

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