

BILIARY DYSKINESIA FROM THE SURGICAL VIEWPOINT

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THE CLINIC

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FUNCTIONAL CHANGES causing disease are in general poorly understood and frequently go undetected because of the lack of demonstrable pathologic changes at the operating table and in postmortem examinations. Dyskinesia or disorders in the motility of the extrahepatic biliary passages is an outstanding example.

Few surgeons, of wide experience in gallbladder surgery, have escaped disappointing results following cholecystectomy. This may be the case even when there is definite evidence of cholecystitis, with or without cholelithiasis. It is more particularly so when, at operation, no definite pathologic condition is found, and the gallbladder is removed because the history suggests trouble or the gallbladder visualization test suggested that the organ was not functioning properly. After the abdomen is opened it is extremely difficult, at times, to weigh all the evidence accurately that has been accumulated pre-operatively with that obtained by visual and manual examination, in determining whether or not to remove the gallbladder or explore the bile ducts. I can recall being unable to detect small stones in a normal looking gallbladder by palpation even after all the bile had been aspirated, and their presence was only demonstrated after the gallbladder was widely opened. At another time the gallbladder was removed without gross evidence of disease because of the characteristic history and because there was a small, questionable filling defect in the visualized gallbladder. On opening the removed organ a very small pedunculated adenoma was found that had undoubtedly intermittently obstructed the cystic duct. Removing the gallbladder relieved the patient of her symptoms. If doubt exists at times as to the advisability of removing the gallbladder how much more frequently the question arises as to the advisability of exploring the common duct. After the duct is opened one can never be sure that all the stones have been removed, even since the use of visualization of the biliary ducts by opaque media during the course of the operation. If symptoms of biliary colic recur following operation, doubt usually still exists as to whether or not symptoms are due to a stone having been overlooked, as to whether or not one is dealing with a functional type of biliary passage involvement, or whether or not some other organ or structure is at the bottom of the trouble.

We are familiar with the teachings of Lahey and with his insistence on exploring many more common ducts than have been explored in the past. This, in the hands of men qualified to do this type of surgery, has undoubtedly resulted in fewer instances of postoperative disappointment. It

has not, however, solved all of the problems that arise in this field of surgery.

That symptoms of gallbladder colic can occur and no stones can be demonstrated at operation, either in the extrahepatic biliary ducts or in the gallbladder, is a fact well known, and considerably written about. The first individual apparently to call attention to this was Krukenberg,¹ in 1903, and Borghi,² in 1913. In 1909, the pathologists Aschoff and Bacmeister³ contributed further to the subject by describing the "stasis gallbladder occurring without inflammation or stones." Schmieden,⁴ in 1920, wrote of the individual with gallbladder distress in whom at operation no stone or inflammation could be found and yet who showed a markedly distended gallbladder. He attributed these symptoms and findings to an anatomic defect involving the cystic duct. Berg,⁵ in 1922, suggested that biliary stasis might be the result of a functional disorder of the sphincter muscle of the ampulla of Vater, because he was able to demonstrate hypertrophy of this muscle in an individual with biliary symptoms and no other findings. Numerous investigators have interested themselves in the question of functional disorders of the biliary system. The concept that biliary distress and pain may result from motor dysfunction of the extrahepatic ducts presupposes that spasm of the sphincter mechanism about the ampulla of Vater, or perhaps elsewhere along the ducts, raises the intraductal pressure to the point where pain is produced. Distention of organs causes pain. Severe and excruciating pain occurs in Dietl's crisis when the pelvis of the kidney is suddenly distended; plugging of the cystic and common duct by stone results in severe colicky pain; distention of the intestine following obstruction gives similar symptoms. That the sphincter muscle about the ampulla of Vater is capable of resisting high intraductal pressure has been demonstrated experimentally and was strikingly evident in one of the cases which will be cited later.

Ivy⁶ has shown experimentally in dogs that the gallbladder may contract with a maximum of force of about 30 cm. of bile pressure, which is also about the secretory pressure of bile, whereas the common duct sphincter may contract and exert a resistance of as much as 80 cm. of bile pressure. Thus, it is evident in the dog, at least, that a spastic choledochoduodenal sphincter mechanism may cause an intrabiliary passage pressure sufficient to block the flow of bile either from the gallbladder or liver. Further evidence was elicited by Ivy, and his coworkers,⁷ in humans, which permitted them to conclude that "pain may be elicited from noninflamed biliary passages by the maximum pressure that may occur in their lumen provided the pressure is raised rather rapidly, especially in the presence of a tonic musculature."

To further quote Ivy, and coworkers: "This concept also provides a rational basis for gallstone colic without gallstones, for so-called 'cholecystitis,' with a normal gallbladder at operation, and for so-called 'hepatic neuralgia.' It is further possible that the presence of an irritable or hype-

trophied sphincter of the common duct may explain the recurrence of symptoms in some patients following cholecystectomy. The early relief would be due to the temporary paralysis of the sphincter that always follows cholecystectomy. But, after recovery from the temporary paralysis, the irritable or hypertrophic sphincter may again produce symptoms of biliary tract distention. The recognition of this possibility should emphasize the importance of the medical rather than the surgical management of these patients, although it must be recognized that some dyskinesia patients are benefited by cholecystectomy and some patients with residue after cholecystectomy are benefited by choledochoduodenostomy. In this connection, Aschoff³ reports a series of 215 cases in which 25 stone-free gallbladders were removed; of the 25, seven had residual symptoms accountable for only by dyskinetic processes. It is likely that the number of discouraging results may be fewer when the idea of a functional disturbance is better understood and managed therapeutically by the surgeon and internist. We would state, however, that we do not believe the diagnosis of dyskinesia to be simple. The presence of pain in the gallbladder region with a normal 14-hour cholecystogram, and absence of clinical evidence of an inflammatory process, may lead one to suspect but not diagnose a dyskinesia. In this communication we have desired only to emphasize that the possibility of the occurrence of biliary dyskinesia should be borne in mind and requires consideration in the etiologic analysis, diagnosis and therapy of biliary tract diseases."

McGowan, and coworkers,⁸ in 1936, interested themselves in the direct measurement of changes in physiologic function of the common bile ducts of human beings who had disease of the biliary tract. Apparently, they were the first to carry on such investigations in the human being. Individuals into whose common duct T-tubes had been placed for prolonged biliary drainage were used for their investigations; eight individuals were used and 15 observations were made. They found that giving $\frac{1}{6}$ gr. morphine subcutaneously produced an increase in intraductal pressure on 14 occasions, pressure began to rise from two and one-half to four minutes after the injection, and reached a plateau in from 10 to 15 minutes. Rise in pressure was associated with constant pain in one case. The pain was situated in the right upper abdominal quadrant and extended around the right subcostal region and to beneath the right scapula. Five attacks of pain occurred during the course of study, and in each instance was associated with a rise in intraductal pressure. The duration and severity of the pain corresponded in each case with the height of the pressure curve. On one occasion the pressure was as high as 160 mm. of water. This was the same type of pain from which the patient had suffered since removal of her gallbladder one year previously.

From their studies, the effects of morphine on the biliary system made this evidence available: "(1) Fluid can be made to flow from the common

bile duct into the duodenum after administration of morphine only by increasing the pressure. In other words, the perfusion pressure is increased. (2) Roentgenograms made before administration of morphine give evidence of rapid emptying of the common duct; the opaque medium usually is found in the duodenum. Roentgenograms of the same patients after administration of morphine give evidence of distention of the common duct. Opaque substances remain in the hepatic ducts and smaller branches of the biliary tree and the lower end of the common duct tapers to a sharp point, suggesting muscular spasm; the picture is not unlike that of the esophagus in the presence of cardiospasm. The drug that produced complete disappearance of pressure and absolute relief of pain was amyl nitrite. A few whiffs of this drug, almost at once, brought the pressure down to zero, where it remained for a few minutes and slowly returned, after about 15 minutes, to the level at which it had been after administration of the morphine. At the same time that the pressure fell, the patient was completely relieved of pain."

Strauss,⁹ and associates, in 1933, reported 29 cases of chronic biliary stasis without stones, which they believed were due to a triangular infection of the duodenum, pancreas and common duct. They thought the infection probably began as a duodenitis and ascended the common duct producing infiltration and swelling of the ampulla of Vater and lower part of the common duct, thereby causing spasm and obstruction of the papillary outlet of varying intensity. In some of these cases attacks simulating gallstone colic were produced, and in others jaundice simulating that caused by obstruction of the common duct from carcinoma of the head of the pancreas. Satisfactory results in most instances followed choledochoduodenostomy plus gastro-enterostomy. Undoubtedly, most of these cases could be classified in the category most frequently spoken of as dyskinesia of the biliary passages.

From this citation of the evidence accumulated by the above investigators and clinicians, it is evident that a typical picture of gallstone colic can be produced when no stones are present, either in the gallbladder or biliary ducts. Those are the cases in which the surgeon at the time of operation may be very much in doubt as to what course to pursue. His decision usually takes one of three directions: (1) He removes the gallbladder—perhaps explores the common duct. (2) He decides that a mistake in the diagnosis has been made, looks around for something else to account for the symptoms and, generally, winds up by taking out the appendix—meanwhile, offering up a prayer to the effect that he hopes the trouble has been found and eradicated. (3) He closes the incision and admits defeat.

Whatever the course taken, it is surprising how many people remain free of further symptoms. An indefinite percentage of the cases continue to complain of symptoms the same as previous to celiotomy, and are generally then classified as being neurotic. Again, it may happen that sufficient trouble is found at operation, usually cholecystitis with cholelithiasis, even

stones in the common duct, to account for the patient's symptoms, and yet after proper correction the symptoms persist. The conclusion usually reached, and the condition usually found, if the patient is reoperated upon, is that a stone was left or had reformed in the common duct.

Such an example stands out vividly in my memory, even though it occurred a number of years ago. A woman with typical gallstone colic was operated upon. The gallbladder, containing numerous stones, was removed, the common duct was opened and numerous stones were removed from it. None remained, so far as could be ascertained. Shortly after discharge from the hospital the patient experienced severe abdominal pain suggestive of gallstone colic. Attacks recurred with increasing severity but the patient steadfastly refused further operative interferences and died suddenly in the height of a paroxysm of pain. Autopsy revealed a small stone, a few millimeters in diameter, in the ampulla of Vater.

Such a train of events may take place, however, and no stones or other pathologic condition be found to account for the symptoms. It is in such a case that one must bear in mind the possibility of a functional abnormality—so-called dyskinesia of the biliary passages. The two following case reports are illustrative, in my opinion, of such a condition:

Case 1.—Mrs. A., Queen's Hospital No. 152060, first came under my care, November 24, 1928, because of pain in the region of the gallbladder, suggesting gallbladder colic. She stated that for the past 15 to 20 years she had suffered intermittently from such attacks of pain. There had never been fever, chills or jaundice, in spite of recurrent severe attacks of pain requiring morphine for relief. She did not submit to operation until July 23, 1932. The gallbladder contained one large stone, several smaller ones, and débris. The common duct was palpated but not explored. The gallbladder was removed. Twelve days after operation the same type of abdominal pain recurred, requiring morphine gr. $\frac{1}{2}$ for relief. These attacks occurred repeatedly until she was operated upon for the second time on May 21, 1935. It was our conclusion that a stone in the common duct had been overlooked. At operation, the common duct was found to be somewhat larger than normal, but not markedly so. The extrahepatic ducts were thoroughly explored including probing, scooping and irrigating, and no stone could be found. A rubber ureteral bougie could be passed well down into the duodenum and, after freeing the descending duodenum and rotating it to the left, the common duct in its entirety could be palpated, but nothing abnormal could be detected. We were, again, much chagrined but equally in doubt as to the cause of our patient's disability. Symptoms were relieved for a while but soon recurred the same as before.

These attacks were unrelieved by amyl nitrite or nitroglycerin. She at no time developed clinical jaundice though the icterus index was 25 on November 10, 1930, and on May 9, 1940, it was 11 and the van den Bergh 3.25 mg. per 1,000 cc. All types of examinations were made, including gastro-intestinal, urinary, blood tests for lues, *etc.*, to explain the attacks of pain, but were all negative. The pain was so typical of biliary trouble—epigastric and right upper quadrant, radiating to back, and of such increasing severity, we felt justified in again exploring the common duct. This was done February 9, 1942. Needless to say, the procedure was difficult because of many adhesions from the two previous operations. The common duct, which was somewhat larger than normal, was again opened and explored, as well as the hepatic ducts, and

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no stones could be found. A rubber ureteral bougie, similar to the one which had been passed into the duodenum at the previous operation, could not be made to pass the ampulla of Vater, and neither would saline injected into the duct. In order not to overlook a stone that might be lodged in the distal end of the duct, transduodenal exploration of the ampulla of Vater was decided upon. The descending part of the duodenum was freed along its lateral side, allowing mobilization of this part of the intestine. A longitudinal incision was made in the anterior wall of the duodenum over the region of the opening of the common duct (Fig. 1a). Though the interior of the duodenum contained a considerable quantity of bile, the opening of the duct could not be located. Saline was then injected into the common duct above by inserting

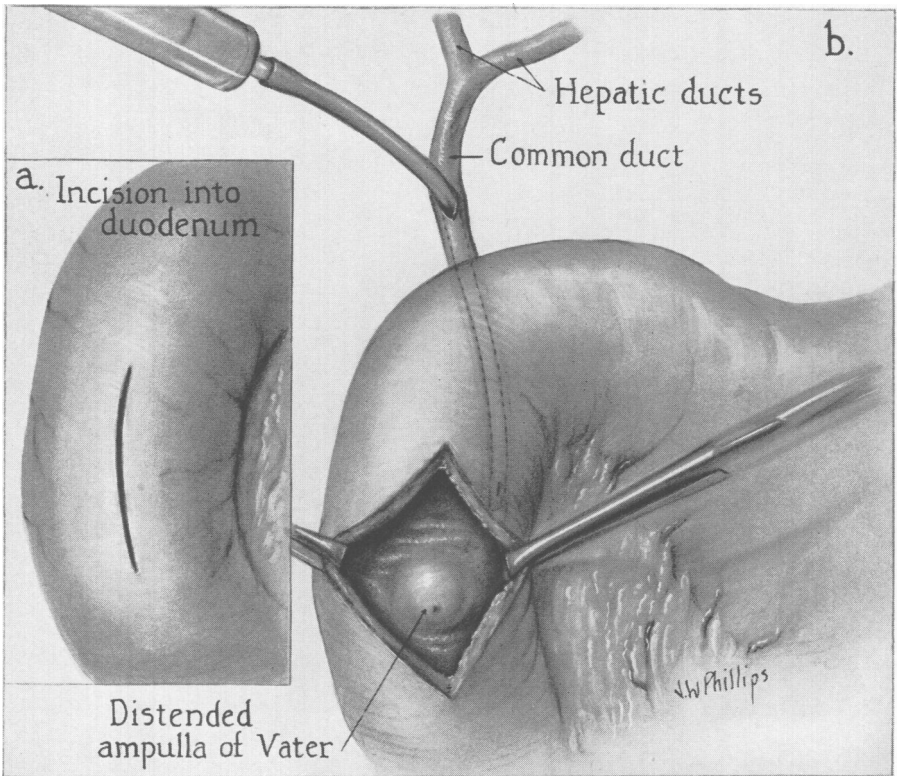


FIG. 1.—A. Shows site of incision in the duodenum to expose the opening of the common duct. B. Showing site of insertion of catheter to irrigate the common duct.

a catheter, as shown in Figure 1b, and though the region of the ampulla of Vater blew up like a balloon not a drop of saline escaped. Repeated efforts brought the same results. Since the patient had never been clinically jaundiced and there was bile present in the duodenum, it was concluded that we were dealing with a spasm (dyskinesia) of the sphincter of Oddi.

An incision was made, approximately one-half inch in length, into the ballooned-out area (Fig. 2c), and this gave a free opening into the lower end of the common duct. So far as I could determine there did not appear to be an unusual amount of hypertrophy of the sphincter muscle. However, lack of familiarity with the thickness of the normal sphincter, plus difficulty of getting a clear view because of blood and bile, made this observation of doubtful value. The common duct was now easily irrigated out, probed and scooped, and no evidence of stone could be found. The opening into the

ampulla of Vater was held open by interrupted fine chromic catgut sutures (Fig. 2d). The opening in the duodenum was sewn transversely, using a row of chromic catgut and an outer row of silk; the line of suture being covered over with a patch of omentum (Fig. 2e). The opening in the common duct was closed tight with interrupted chromic sutures. One rubber tissue drain was inserted into Morison's pouch and the incision was closed in layers. The patient made an uneventful recovery and to date (October 1, 1942) has been entirely free of gallbladder symptoms.

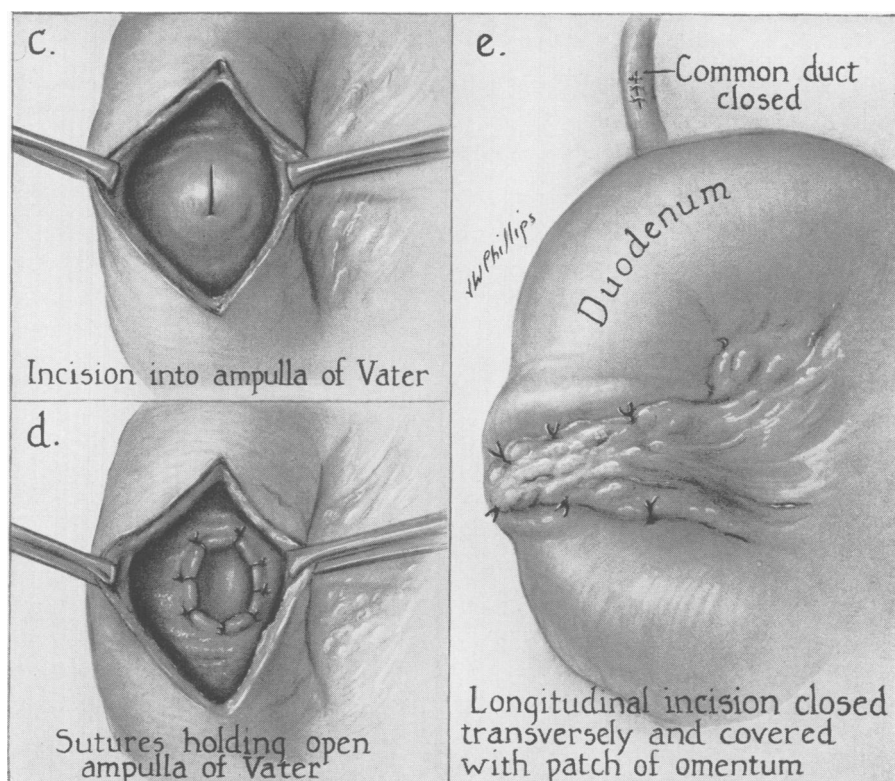


FIG. 2.—C. Showing the incision into the ampulla of Vater. D. Showing the technic of procedure of holding open the ampulla of Vater. E. Showing the method of protection of the incision into the duodenum.

Case 2.—A female, age 48, had had, for the past 20 years, recurrent severe attacks of epigastric pain. After the December 7, 1941, "blitz" these attacks became much more frequent and more severe. Roentgenologic examination following gallbladder visualization substantiated the diagnosis of gallbladder disease. At operation, June 1, 1942, a gallbladder containing numerous stones was removed. The common duct was explored by palpation only, since neither the history nor local findings suggested common duct stones. The second day following operation the patient stated that the same pain recurred, though it may have been that she could not differentiate this from postoperative distress. At least, very soon following operation she developed excruciating attacks of pain similar, but even more severe, than previous to operation. These attacks required repeated injections of large amounts of morphine for their relief. At no time was clinical jaundice evident. Because stone in the common duct is the most frequent cause of such symptoms, it was felt that we were probably dealing with this condition,

though dyskinesia of the sphincter of Oddi was considered largely because of our recent experience.

Exploration of the common duct was undertaken, June 2, 1942, with practically the same findings as in Case 1. The duct was not noticeably enlarged. No stones could be found. Fluid could not be made to pass into the duodenum when injected into the common duct. A soft lead probe was gently passed down the duct as far as possible in order to help identify the proper place to open the duodenum in our search for the ampulla of Vater. The ampulla was not readily identified, but with a finger in the duodenum, as a guide, the probe was gradually worked through the common duct opening. Rubber catheters of increasing size were attached to the probe, and to one another, and drawn back up the common duct. No stones were extracted and none could be washed out by irrigation. An incision was made through the ampulla to include the sphincter of Oddi which, again, did not seem unduly hypertrophied. No sutures were placed in this incision. The duodenal incision, the opening into common duct, and abdominal wall were closed as described in Case 1. The patient made a good recovery. She left the hospital on the twelfth postoperative day, and has been symptom-free to date, October 1, 1942, of all symptoms suggesting biliary colic.

In the treatment of biliary dyskinesia and other conditions attributed to an abnormality in the region of the ampulla of Vater, in which no organic lesions were demonstrable, various surgical means of correction have been advised. Removal of the gallbladder with prolonged drainage of the common duct by means of a T-tube has its advocates. Dilating the ampulla of Vater by using sounds of increasing size has been done, apparently, with success but not without unfortunate and even fatal results, due to injury of the duct, followed by retroperitoneal infection. Strauss,⁹ in the cases previously referred to, preferred choledochoduodenostomy followed by gastro-enterostomy was added to the primary operation to side-track the stream of food and lessen intraduodenal pressure, in order to minimize the danger of food regurgitating up the biliary passages. It would seem to me controversial whether the danger from a gastrojejunal ulcer developing following gastro-enterostomy might not outweigh the dangers from bile regurgitating up the biliary passages.

Colp and Doubilet¹⁰ have found that physiologic disturbances of the sphincter of Oddi bear an important relationship to certain types of acute pancreatitis, gallbladder disease, the post cholecystectomy syndrome, the dyskinesia of the biliary tract, and some forms of transient jaundice. To correct the spasm of the sphincter of Oddi they have devised an instrument (sphinctertome) which they pass down the common duct into the duodenum. It is then withdrawn until it contacts the intestinal wall. A piece is then bitten out which is supposed to include a portion of the sphincter muscle. The procedure corresponds to the blind-punch operation for prostatic hypertrophy, which preceded our present methods of visual precision. While the method worked satisfactorily in the hands of the inventors it would seem to be fraught with considerable danger due to the blindness of the maneuver.

Anastomosing the gallbladder to the stomach or duodenum may be done, and is the procedure usually carried out in the presence of jaundice thought

to be due to malignancy of the head of the pancreas. Usually the gallbladder has been removed before it becomes apparent that one is dealing with a physiologic disturbance of the sphincter of Oddi. In fact, it is a diagnosis arrived at largely by the process of exclusion.

The operation of transduodenal exploration of the ampulla of Vater and subsequent division of the sphincter of Oddi, the method employed in our two cases, is the only procedure that lends itself to arriving at a correct diagnosis. It permits of a direct visual attack on the seat of trouble and if a stone is present this can be removed and unnecessary and undesirable side-tracking operations prevented. It is not an operation to be considered lightly, for it has potential possibilities of serious complications. However, with due amount of care and caution the risks can be kept at a minimum.

SUMMARY AND CONCLUSIONS

1. Symptoms of biliary colic can and do occur in the absence of gallstones. Biliary dyskinesia must be thought of when no stones are found at exploration.

2. Dyskinesia of the biliary passage is a disease entity with which the medical profession needs to become more familiar.

Two cases of this condition are cited, with the method employed in their correction, and a short discussion of the subject in general is indulged in.

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