tion, the infant making an uneventful recovery. The cyst was present and appeared as a calcified ring on roentgenograms during the eighth month of fetal life. The differential diagnosis is discussed and the cyst is concluded to be a calcified meconium abscess. The subject of meconium peritonitis is reviewed.

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Congenital Absence of the Gallbladder

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CONGENITAL ABSENCE of the gallbladder is a rare anomaly of the biliary tract. It is the purpose of this communication to report an additional case and to briefly review the literature. The approximate incidence of this anomaly, derived from autopsy statistics of several reported series, is given in Table 1. It is felt that this is at best an approximation. There are a number of individual case reports that cannot be considered in estimating the incidence, since the total numbers of patients from which these are drawn is unknown. The true incidence can only be derived from autopsy statistics, since absence of a gallbladder shadow in roentgen films, following ingestion of dye, is more likely to be due to a nonfunctioning gallbladder than to absence of the organ.

The overall incidence based on the data in Table 1 is .09 per cent. However, it will be noted that the reported incidence in the pediatric age group (0.37 per cent in data from the Hospital for Sick Children) is considerably higher than the overall incidence. This is probably in keeping with the higher incidence of all congenital anomalies in this age group, since mild deformities which are compatible with life may go unrecognized for many years into adult life, while the more severely incapacitating anomalies usually will be recognized during the early years. Exclusive of the data on the pediatric group, the incidence in the remainder of the combined data in the Table is 0.04 per cent, which is probably more nearly the actual incidence of this anomaly in adults. A more accurate figure could only be obtained by the compilation of a large number of autopsy statistics, since the chance for error with a rare condition and a relatively small sample is quite high.

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The signs and symptoms in patients with congenital absence of the gallbladder who have disease of the common duct are apparently little different from those in patients with a complete biliary tract and disease in either the gallbladder or the common duct, or both. In autopsy material the anomaly is observed about equally often in both sexes. Clinically, however, it is observed about twice as often in females as in males, which accords with the usual sex ratio in surgical disease of the biliary tract.[5] That is, the anomaly apparently occurs as often in men as in women but disease of the common duct requiring operation is twice as frequent in women.[5]

The effect of the absence of the gallbladder on biliary function is not known, as no laboratory studies to determine the effect have been reported. Mouzas and Wilson[8] expressed belief that there is no alteration in function. This opinion is not shared by Caylor[1] who was of the opinion that this anomaly is fairly frequently associated with disease of the remaining intact portions of the biliary tree, especially the common duct. Reports in the literature would seem to bear out this latter opinion. Thus, in a series of 60 cases of congenital absence of the gallbladder reported by Dixon and Lichtman,[2] 58 per cent of the patients had symptoms of cholecystic disease. Of this 58 per cent, 48 per cent had jaundice

TABLE 1.—*Incidence of Congenital Absence of the Gallbladder (Data from Reports of Autopsy Series).*

Data supplied by	No. Cases	No. Autopsies	Incidence (Per Cent)
‡Knox[4]	2	2000	
‡Mentzer[7] and Nagel[9]	1	1600	
‡Lloyd[6]	1	5000	0.04
‡London Hospital*	9	21631	
†Hospital for Sick Children*	20	5395	0.37
Total	33	35626	0.09

*Quoted without reference in Mouzas and Wilson.[8]
‡Presumably series of adult subjects.
†Pediatric data.

and 26.6 per cent had choledocholithiasis. In the same series, 73 per cent of patients past the age of 45 had symptoms of cholecystic disease. Of the cases reported in the literature almost half were discovered at operation that was done because of signs and symptoms of biliary tract disease. The others were observed incidentally, either at operation for unrelated conditions or at autopsy. This incidence is far higher than the incidence of gallbladder disease in the population at large and is suggestive of the possibility that even with an intact intrahepatic and extrahepatic ductile system, the absence of the gallbladder in some way predisposes to disease of the common duct. It should be emphasized, however, that this is an impression and that statistical proof is lacking.

The cause of this anomaly is now generally held to be a defect in the embryological development of the biliary system, although at one time it was considered possibly an acquired state most likely due to various infectious diseases, including syphilis. On the basis of a defect in embryological development, two theories have been proposed to explain absence of the gallbladder.[3] The first involves a failure of the gallbladder bud to develop from the hepatic diverticulum. However, such a failure, if complete, would also result in an absence of the cystic duct. The second theory supposes failure of the gallbladder bud to resolve from its solid embryonic stage. This would not necessarily involve the cystic duct, and the explanation may be a more likely one. The biliary system is frequently the site of development anomalies. This was the subject of an excellent review by Gross[3] in 1936.

Following is a report of a case of congenital absence of the gallbladder in a middle-aged man.

REPORT OF A CASE

A 46-year-old white man, was first observed in January, 1955, because of "bloating," epigastric pain and "upset stomach." The patient was well developed and well nourished and in no acute distress. Epigastric tenderness was the only abnormality noted at physical examination. X-ray examination of the gallbladder and upper gastrointestinal tract was reported as showing a nonfunctioning gallbladder, moderate duodenal stasis, nephrolithiasis on the right, minimal hypertrophic arthritis of the lumbar spine and generalized arteriosclerosis. A low fat diet and the use of antispasmodics were prescribed. The patient was next observed in April, 1955, after several episodes of abdominal distress which were thought to be due to gallbladder disease. Cholecystectomy was scheduled but the patient died quite suddenly shortly before he was to go to the operating room.

Autopsy revealed the cause of death to be pulmonary atelectasis due to mucus plugging of the right main bronchus and, to a lesser extent, the bronchi of the left lung. Other findings of interest besides those in the biliary tree were moderately severe arteriosclerosis of both coronary arteries with narrowing of the lumen, and a small focus of myocardial scarring in the interventricular septum. The liver was grossly and microscopically normal. The gallbladder was congenitally absent. The hepatic duct was 1.0 cm. in diameter. The ductile system was probed from the ampulla of Vater to the hepatic parenchyma. The ducts contained dark green bile but no calculi or other obstruction. The hepatic ducts were traced into the liver parenchyma and no other abnormality was noted.

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