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Hepatic Arterial Ligation Associated with Gastrectomy. Report of Six Cases with a Review of 36 Cases

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Accidental ligation of the hepatic artery or a major branch is considered to be rare in gastric resections. However, there is a certain risk when the dissection of the hepatoduodenal ligament is difficult, as when it is invaded by a carcinoma, or transformed into a scar tissue. Division of the lesser omentum or the left gastric artery also involves the risk of severing an aberrant hepatic artery, since the left gastric gives off a hepatic branch in more than 15% of dissected cadavers¹⁾⁴⁸⁾⁵⁵⁾. On the other hand, few cases have been reported of intentional ligation in radical gastrectomy⁴⁾. The majority of surgeons still accept the traditional concept, first put forth by HABERER²⁷⁾, and modified by NARATH⁶⁰⁾, that ligation of the common hepatic artery proximal to the gastroduodenal artery may be undertaken, but that of the proper hepatic artery distal to the right gastric artery or one of its main branches should not be performed for fear of fatal necrosis of the liver. However, the consequences of hepatic arterial ligation in human beings are less understood because the number of reported cases is still very small, and moreover, they vary greatly with the type of operation, underlying disease and treatment.

In the present paper, six cases of hepatic arterial ligation, intentional or accidental, during radical gastrectomies are reported. Their postoperative courses were variable with different clinical manifestations, but all survived without lasting sequelae. In addition, 36 similar cases are collected from the literature. The factors which influenced the outcome are analysed, and some features in man are discussed.

Report of cases

CASE 1. A 62-year-old woman had subtotal gastrectomy for an advanced cancer of the stomach causing pyloric obstruction. During dissection of the hepatoduodenal ligament the proper hepatic artery was mistaken for the right gastric artery, and was severed between the ligatures. No pulsation was felt in the hepatoduodenal ligament. 600,000 units of penicillin and 1 gm of chloramphenicol were injected. In a subsequent attempt to reconstruct the artery, the right gastric and gastroduodenal arteries were divided, and the common hepatic artery was mobilized toward the porta hepatis, so as to approximate the proximal arterial stump to the distal one. However, repeated efforts at vascular suture were futile because of atheromatous changes in the vessel, resulting in further damage to both stumps, which were finally ligated; the distal ligature was placed beyond the bifurcation of the artery, so that the right and

left hepatic arteries were occluded simultaneously. During this procedure there was back flow from the distal stump of the artery; the liver became slightly pale and its capsule wrinkled, but no cyanosis or congestion was noted. The gastrectomy procedure was then resumed, with dissection of the lesser omentum up to the esophagocardiac junction and division of the left gastric artery at its origin. No further change in the gross appearance of the liver was seen before closure of the peritoneum. The general condition of the patient remained good throughout the operation.

After operation she was kept in an oxygen tent, fasted for five days, and given heavy doses of broad-spectrum antibiotics, including cephaloridine, aminobenzyl penicillin and kanamycin. For the first postoperative week she had pronounced dyspnea and extreme weakness which were aggravated by sitting up in bed or even assuming Fowler's position. She also complained of orthostatic dizziness and could not stand by the bed until the 13th postoperative day. Other symptoms which appeared in the postoperative course included petechiae of the arms, diarrhea, anorexia and increased desquamation of the skin. However, the patient slowly improved and was discharged two months after operation. The SGOT on the 1st, 3rd, 7th and 14th postoperative days was 40, 106, 40 and 6 units, and the SGPT 18, 26, 40 and 8 units, respectively. Other liver function studies, including serum protein fractions, bilirubin, alkaline phosphatase and colloidal reactions, remained normal. The bleeding time, partial thromboplastin time and thrombocyte count were within normal limits on the fourth postoperative day when petechiae were noted on the arms. The electrocardiogram showed depressed ST segments and inverted T waves in V_1 through V_4 .

CASE 2. A 42-year-old woman underwent subtotal gastrectomy for gastric carcinoma. During the resection of the stomach the lesser omentum was found to contain an aberrant left hepatic artery, 1.5 mm in diameter, arising from the left gastric artery. Since temporary clamping of this artery produced no gross change in the liver, it was divided, together with the lesser omentum, and the left gastric artery was also severed close to the celiac axis. The postoperative recovery was uneventful. Unfortunately, liver function studies were not done in the immediate postoperative period.

CASE 3. A celiotomy was performed on a 62-year-old lean woman who had a non-tender mass in the left upper quadrant. At operation, the upper half of the lesser curvature of the stomach was fixed to the left lobe of the liver, forming a firm tumor. There were enlarged lymph nodes along the celiac axis and its branches. Subtotal gastrectomy was performed along with regional lymphadenectomy and partial resection of the left hepatic lobe. Because of continuous oozing of blood from the liver stump the left hepatic artery was ligated at the bifurcation, and the liver stump was compressed with a gauze pack which was removed seven days after surgery. The peripheral area of the left lobe was cyanotic. Examination of the resected specimen revealed an inflammatory tumor with small abscess formation. During the first postoperative week she complained of general malaise and occasional chest pain with tachycardia and tachypnea. The electrocardiograms showed a transient depression of the ST segments with inverted T waves in Leads V_4 and V_5 . The

SGOT and SGPT were 28 and 42 units on the 5th postoperative day, and 19 and 4 units on the 9th postoperative day. She developed serum hepatitis after discharge, but the liver function tests were completely normal six months after operation. Obliteration of the left hepatic artery was confirmed by postoperative celiac angiography (Fig 1).

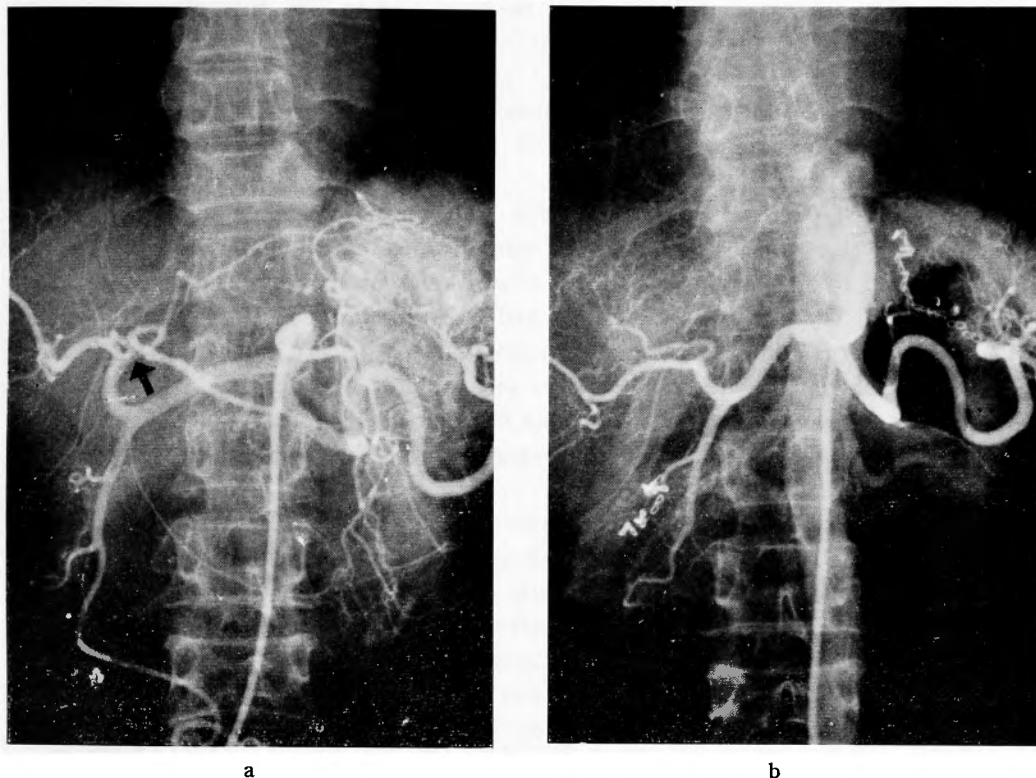


Fig. 1. Celiac angiography (a) before and (b) 36 days after operation. Arrow indicates the point of ligation. Postoperatively, the left hepatic artery is obliterated, but the left hepatic area is still opacified in the arterial phase.

CASE 4. A 68-year-old woman underwent total gastrectomy for gastric carcinoma and cholecystectomy for cholelithiasis. During the dissection of the lesser omentum, an aberrant left hepatic artery 2 mm in diameter was found to originate from the left gastric artery. Both arteries were severed, and the inferior phrenic artery was also divided in the course of esophageal mobilization; the triangular ligament was preserved. There were areas of cyanosis and congestion in the anterior portion of the left hepatic lobe. Tachycardia and tachypnea developed during operation and continued postoperatively. On the second postoperative day her pulse rate increased to 130 and her respirations to 42 per minute. There was cyanosis of the nail beds, and the central venous pressure was 120 mm of saline. The patient complained of a heavy sensation in the chest. Chest films were negative. Electrocardiograms revealed depressed ST segments and inverted T waves in Leads V_1 through V_4 . With digitalization, chemotherapy and other supportive measures, these signs and symptoms disappeared within five days, except for the electrocardiographic findings which did

not improve until more than a month later. The SGOT and SGPT values which were 216 and 150 units on the first postoperative day fell to 115 and 77 on the 3rd day, but rose again to 270 and 114 units on the 5th day. Thereafter, they decreased gradually and were 33 and 13 units on the 25th day. During the first postoperative month the LDH activity remained in the range of 23.3 to 27.5 Hill units; the LDH₅ fraction was greater than the LDH₄ fraction for the first week. The alkaline phosphatase, leucine aminopeptidase and cholesterol levels were slightly elevated throughout her stay in the hospital, although serum bilirubin remained normal. Postoperative celiac angiography revealed the absence of the left branch of the proper hepatic artery.

CASE 5. A 51-year-old man underwent subtotal gastrectomy for carcinoma of the stomach. When the lesser omentum was divided close to the liver, a ligature slipped off and brisk bleeding occurred from the hepatic stump of the omentum. The bleeding artery was immediately identified and religated. It was an aberrant left hepatic artery, 2 mm in diameter, arising from the left gastric artery. There was no gross change in the liver, and the operation was concluded with the usual technique. His postoperative recovery was uneventful. Except for a relative increase in LDH₅ on the first postoperative day, repeated liver function studies revealed no abnormalities.

CASE 6. A total gastrectomy was performed along with splenectomy and distal pancreatectomy in a 68-year-old man with carcinoma of the stomach. During mobilization of the stomach an anomalous hepatic artery 2 mm in diameter arising from the left gastric artery was divided; the latter was severed at its origin. During the operation the systolic blood pressure fluctuated between 60 and 140 mm Hg.. For the first two postoperative days the patient was agitated and disorientated, but made a smooth recovery thereafter. There was an increase in the LDH₅ fraction during the first two days. The maximal values of SGOT and SGPT were 75 and 42 units, respectively, on the second postoperative day.

Additional data on these six cases are shown in Table 1.

Comment

The six patients reported here had hepatic arterial ligation, accidental or intentional, during radical resection of the stomach. All of them survived without lasting sequelae, although three had cardiopulmonary complications. In Case 1, efforts at arterial reconstruction led to the most extensive dearterization in this series, entailing division of all visible arteries in the hepatoduodenal ligament, i. e., the gastroduodenal, right gastric and proper hepatic arteries, both branches of the latter artery being ligated also. This error, followed by subtotal gastrectomy, resulted in almost total obstruction of the hepatic arterial inflow through the celiac and superior mesenteric arteries. The remaining five cases underwent interruption of a left hepatic artery; One (Case 3) had ligation of the main left branch of the proper hepatic artery for bleeding from the liver, and four were subjected to deliberate division of the aberrant left hepatic artery, measuring more than 1.5 mm in diameter, which constitutes the sole or, at least, main source of arterial blood supply to the left lobe of the liver. In Case 4, not only the replaced left hepatic, but also the inferior

Table 1 ANALYSIS OF SIX CASES

Case No.	Age Sex	Diseases	Operation (Anesthesia)	Artery	Blood loss Transfusion Fluid	Intraoperative complications	Postoperative complications (Outcome)	Treatment (Antibiotics)
1	62 F	Gastric carcinoma with Pyloric obstruction, anemia, hypoproteinemia	Subtotal gastrectomy (GOF)	PH, RH, LH, LG, GD	639 gm 1000 cc 1000 cc	Liver capsule slightly shrunken and anemic	Dyspnea, orthostatic dizziness, petechia, general malaise, diarrhea, anorexia, skin desquamation, increased transaminase, abnormal ECG (Recovery)	Oxygen tent; dexamethasone (CP, PC, KM, CER)
2	42 F	Gastric carcinoma with pyloric obstruction, anemia, hypoproteinemia	Subtotal gastrectomy (GOE)	aLH, LG	507 gm 800 cc 1000 cc	None	None (Recovery)	(PC, SM)
3	62 F	Inflammatory tumor of left hepatic lobe penetrating to stomach, hypoalbuminemia	Subtotal gastrectomy, partial resection of left hepatic lobe (GOF)	LH, LG	1030 gm 600 cc 1500 cc	Partial cyanosis of left hepatic lobe	General malaise, chest pain, tachycardia, tachypnea, excessive perspiration, abnormal ECG (Recovery)	Oxygen tent; (CER, KM, OM, TC)
4	68 F	Gastric carcinoma, cholelithiasis, anemia	Total gastrectomy, cholecystotomy (GOE)	aLH, IP, LG	464 gm None 1500 cc	Partial cyanosis of left hepatic lobe, tachycardia, perspiration	Tachycardia, elevated venous pressure, tachypnea, cyanosis, chest discomfort, increased transaminase, LDH ₅ and BUN, abnormal ECG (Recovery)	Oxygen tent, digitalization (KDM, AB-PC, CP)
5	51 M	Gastric carcinoma	Subtotal gastrectomy (GOE)	aLH, LG	284 gm None 1500 cc	None	Increased LDH ₅ (Recovery)	(PC, SM)
6	68 M	Gastric carcinoma	Total gastrectomy, distal pancreatectomy, splenectomy (GOF)	aLH, LG S	1066 gm 800 cc 1000 cc	Fluctuation of blood pressure	Agitation, disorientation, increased LDH ₅ (Recovery)	(SM, CP, AB-PC, LCM)

PH, proper hepatic; RH, right hepatic; LH, left hepatic; aLH, aberrant left hepatic; IP, inferior phrenic; GD, gastroduodenal; LG, left gastric; S, splenic; CP, chloramphenicol; PC, penicillin; KM, kanamycin; CER, cephaloridine; SM, streptomycin; OM, oleandomycin; TC, tetracyclin; KDM, kanendomycin; ABPC, aminobenzyl penicillin; LCM, lincomycin; GOF (or GOE), endotracheal anesthesia with nitrous oxide, oxygen and fluothane (or ether).

phrenic artery was severed without grave effect, although the latter artery is regarded as the crucial collateral route^{27) 62)}.

Review of clinical cases (Table 2). A review of the literature dealing with hepatic arterial interruption in man was compiled by NARATH⁶⁰⁾ (6 cases, 1906); his nephew, NARATH⁶¹⁾ (20 cases, 1916); RITTER⁶⁵⁾ (26 cases, 1922); GRAHAM and CANNELL²³⁾ (28 cases, 1933); and more recently by BRITAIN et al.⁸⁾ (20 cases after 1933, 1964). The early authors, especially RITTER⁶⁵⁾ attached great importance to the site of ligation as a determining factor for the final outcome, and due consideration was not given to other factors such as types of operation, underlying diseases and intraoperative or postoperative complications. The importance of the latter was stressed later by BRITAIN⁸⁾ and others^{17) 52)}. When cases collected by RITTER⁶⁵⁾ in 1922 are reanalyzed, it is found that of eight patients subjected to accidental ligation of the proper hepatic artery or one of its branches during gastric surgery all died except one whose survival was ascribed to the presence of an intact accessory artery. On the other hand, four out of six patients survived hepatic arterial ligation during cholecystectomy. This higher mortality associated with gastric resection may be due partly to more extensive division of collateral routes in the dissection of the stomach. However, it must be noted that accidental ligation in gastrectomy is unusual except in a very difficult dissection which is often accompanied by other operative complications such as bleeding (three out of seven fatalities). As pointed out by BRITAIN⁸⁾, such an associated complication may be the primary cause of death. In 1921 HOFMEISTER³⁰⁾ first reported a case of recovery following ligation of the proper hepatic artery during gastric resection for ulcer. Since 1950 there have been an increasing number of survivals in spite of more extensive surgery for gastric cancer (EDGEcombe and GARDNER¹⁷⁾, ANDREASSEN et al.⁴⁾, KAJITANI and YAMADA³⁸⁾, and MIYAZAKI and NAKA⁵⁷⁾). ANDREASSEN⁴⁾ resected the celiac axis from its origin to the pancreaticoduodenal artery and both branches of the proper hepatic artery during total gastrectomy, splenectomy and distal pancreatectomy for gastric carcinoma. The patient recovered without lasting damage to the hepatic parenchyma. Recently, more extensive dearterization of the liver, not combined with other operative interventions, was attained by ALMERSJÖ et al.³⁾ on seven patients with metastatic cancers of the liver. They divided not only the proper hepatic artery, but also the falciform and triangular ligaments as well as the lesser omentum and the peritoneal attachment of the right lobe; there was only one postoperative death. This improvement in survival rate obtained by recent authors and by us can most likely be ascribed to 1) the use of antibiotics, 2) advances in techniques of anesthesia and respiratory care, and 3) adequate correction of fluid and blood deficits.

However, there have still been a number of fatal cases despite the great progress in the management of surgical patients. Death has often occurred either in poor-risk patients or after extensive surgery. As evidenced by four cases of MONAFO et al.⁵⁸⁾ and two cases of KARASEWICH and BOWDEN³⁹⁾, death is the almost invariable result in pancreatoduodenal resection, whether the proper hepatic, one of its branches or an aberrant hepatic artery is ligated. FRIESEN²¹⁾ divided the replaced left hepatic artery in an esophagectomy through a thoracoabdominal incision for esophageal carcinoma.

The patient died in two days, and autopsy revealed infarction of the left lobe of the liver. A similar case of fatal necrosis of the left lobe was reported by LURIE⁴⁷⁾ who ligated an accessory left hepatic artery 1.5 mm in diameter during gastrectomy through a thoracoabdominal approach. APPLEBY⁵⁾ resected the celiac axis and its main branches, preserving the gastroduodenal artery, in en bloc dissection for early cancer of the stomach. All of his 13 patients survived this operation. The same operation was done on two patients through a thoracoabdominal incision by CLARKE¹¹⁾; both died and had focal necrosis of the liver at autopsy, together with necrosis of the gastric remnant in one patient and multiple perforations of the first duodenal portion in the other. The preservation of the gastroduodenal artery does not always serve as a safeguard against liver infarction. This is evidenced by the cases reported by GRAHAM and CANNELL²³⁾, ROSENBAUM and EGBERT⁶⁷⁾, and TSAGAREISHVILI⁷⁶⁾. GRAHAM²³⁾ ligated the common hepatic artery in a patient with pyloric carcinoma and noted irregular areas of necrosis in the left hepatic lobe at autopsy. ROSENBAUM⁶⁶⁾ reported hepatic infarction in a patient with liver cirrhosis who died after deliberate ligation of the common hepatic artery in association with gastrectomy and vagotomy for duodenal ulcer and esophageal varices. A fatal case of liver cirrhosis was also reported by TAKAHASHI⁷³⁾, who ligated the hepatic and splenic arteries in a patient who developed ascites after gastrectomy. Even without gastrectomy or other operations, MADDEN⁴⁹⁾ found four fatalities in eight cases of liver cirrhosis, seven of which had common hepatic artery interruption with or without simultaneous ligation of the splenic artery. BERMAN⁷⁾ and RIENHOFF⁶⁴⁾ also reported a mortality rate of about 30%, following hepatic arterial ligation in their series of 23 cases of cirrhosis of the liver. This analysis reveals that the results of hepatic arterial ligation are very variable and cannot be predicted from the site of ligation alone. Rather, the prognosis seems to be much influenced by the preoperative condition, the extent of operation and many other factors.

Factors influencing the outcome. Since the careful experiments of HABERER²⁷⁾ in 1905 the majority of authors⁶⁾⁹⁾³⁴⁾⁶¹⁾ had supported the view that, provided no anatomical variations exist, the interruption of hepatic arterial inflow from the common hepatic artery as well as retrograde flow through the right gastric and gastroduodenal arteries almost always produces extensive necrosis of the liver as a direct consequence, with an invariably fatal outcome; Occasional survivals had been explained by the anatomical presence of collateral arteries. Some authors⁴³⁾⁴⁶⁾⁷⁸⁾, however, doubted that interruption of the oxygen supply from the arterial source alone could cause necrosis of liver cells, which are nourished by two blood sources, and the importance of superimposed factors was emphasized among the causes of liver necrosis.

It has long been known, probably since COHNHEIM and LITTEN's report¹³⁾ that hepatic arterial occlusion results in vascular lesions of the liver, not confined to its arterial system. The circulatory disturbance thus produced was regarded by some authors³²⁾⁴³⁾⁴⁶⁾ as an essential factor for parenchymal damage while others⁶⁰⁾⁶¹⁾ maintained it was secondary to liver cell necrosis. LOEFFLER⁴⁶⁾ and earlier workers⁴³⁾, with the anatomical point of view that intrahepatic bile ducts and blood vessels were

Table 2 CASES OF HEPATIC ARTERIAL INTERRUPTION DURING OR AFTER GASTRIC SURGERY

Author Date	Age Sex	Disease	Operation	Artery	Complications	Outcome	Note and autopsy findings
Socin 1883**	38 M	Gastric cancer	Gastrectomy, pancreatectomy	CH, GD	Circulatory collapse	Death in 3 hrs.	Operative bleeding
Eiselberg 1889**	39 M	Gastric cancer	Gastrojejun- stomy	PH	Circulatory collapse	Death in 7 hrs.	Bleeding during gastric dissection. Flabby liver with clear markings, no macroscopic necrosis.
Narath 1904**	46 F	Gastric ulcer	Gastrectomy	LH	Tachycardia, hyper- pyrexia, anastomotic disruption	Death in 6 days	Extensive necrosis of left and caudate lobes and contiguous areas. Localized peritonitis
Wendel 1911*	NR NR	Gastric cancer	Gastrectomy	PH	NR	Death in 36 hrs.	Total necrosis of liver and gall bladder
Hirschel(Wilm) 1911**	47 M	Gastric cancer	Gastrectomy	LH ?	Postoperative bleeding due to LH injury	Death in 16 days	Extensive necrosis of left lobe
Ritter 1917**	NR M	Gastric ulcer	Gastrectomy	RH	Weakness, bronchitis, fever, abdominal pain, jaundice, dyspnea	Death in 11 days	Necrosis of right Lobe. Localized peritonitis. Endarteritis. Anatomical anomaly of hepatic artery
Holst 1920*	66 M	Gastric cancer	Gastrectomy	PH	Sudden death	Death in 8 days	Left lobe shrunken and partly necrotic. Thrombus in hepatic artery
Ritter 1921**	58 F	Gastric ulcer	Gastrectomy	CH, GD	Uneventful	Recovery	An accessory common hepatic artery was present
Hofmeister 1921**	56 F	Gastric ulcer	Gastrectomy	PH	Uneventful	Recovery	
Graham 1930**	49 M	Gastric cancer	Gastrectomy	CH	Fever, tachycardia, tachypnea, broncho- pneumonia, cardiac fai- lure, pulmonary edema	Death in 7 days	Partial necrosis of left lobe. Bilateral bronchopneumonia, pulmonary edema
Koga 1947*	NR NR	Gastric cancer	Gastrectomy	CH	NR	Recovery	
Koga 1947*	NR NR	Gastric cancer	Gastrectomy	CH, PH, GD	NR	Death	NR

Table 2 -continued

Author Date	Age Sex	Disease	Operation	Artery	Complications	Outcome	Note and autopsy findings
Sato, Kitamura 1949*	47 NR	Gastric cancer	Gastrectomy	PH	Oliguria, coronary insufficiency	Death in 3 days	Total necrosis of liver. Glomerular atrophy, tubular degeneration. Small necrotic focus in cardiac apex
Appleby 1949**	63 M	Gastric cancer	Total gastrectomy, distal pancreatectomy, splenectomy	CA, CH	NR	Recovery	A total of 13 patients were operated on by the same procedure without mortality or untoward primary incident with respect to liver
Edgecombe 1950**	50 M	Gastric cancer	Gastrectomy	PH	Ileus, atelectasis, wound dehiscence	Recovery	Operative bleeding
Rosenbaum 1951**	40 M	Duodenal ulcer, liver cirrhosis with varices	Gastrectomy, vagotomy, CH ligation	CH	Hypotension, blood oozing, jaundice, fever, lethargy	Death in 2 days	Beginning bronchopneumonia and minimal edema. Splenomegaly, hepatomegaly with portal cirrhosis. Hemorrhagic and ischemic infarcts of liver
Tsagareishvili 1951**	48 F	Gastric cancer	Total gastrectomy	aLH	Severe dyspnea, jaundice, peritonitis, bronchopneumonia, left-sided pleuritis	Death in 4 days	Yellowish fluid in peritoneal and left pleural cavity. Fibrinopurulent membrane over left liver lobe and diaphragm. Multiple necrotic foci in left lobe. Pneumonia. Thrombotic lesion? in mitral and aortic valves
Kajitani 1953**	52 M	Gastric cancer	Gastrectomy. Later, three operations	GD, PH CH	Pancreatic drainage, anastomotic leakage. Bleeding from GD and CH	Recovery	PH and CH were ligated 21 days after gastrectomy for bleeding from GD stump
Clarke 1953**	58 M	Gastric cancer	Esophagogastrectomy, splenectomy, distal pancreatectomy, left adrenalectomy	CA, CH	Hematemesis, jaundice, azotemia, acidosis, weakness, dependent edema, basal pneumonitis	Death in 24 days	Bronchopneumonia, Necrosis of gastric remnant with ulceration. Two areas of infarction in right and left lobes with liver cell necrosis
Clarke 1953**	52 M	Gastric cancer	Total gastrectomy, splenectomy, distal pancreatectomy	CA, CH	Vomiting, abdominal pain, vascular collapse, peritonitis	Death in 8 days	Infarction of duodenum with multiple perforations. Generalized peritonitis. Portal embolism, microscopic liver infarction

Table 2 -continued

Author Date	Age Sex	Disease	Operation	Artery	Complications	Outcome	Note and autopsy findings
Takahashi 1954*	NR NR	Gastric ulcer, liver cirrhosis	Gastrectomy. PH and S ligation	PH, S	Oliguria, jaundice, fever, leucocytosis	Death in 7 days	PH and S were ligated 24 days after gastrectomy for ascites
Melinikov 1954*	NR NR	NR	Gastrectomy	aLH	Bile peritonitis	Death	Focal necrosis of liver. Bile peritonitis
Friesen 1954**	58 M	Esophageal cancer, hypertension	Esophagocardiac resection	aLH	Hypotension, oliguria, increased tracheobronchial secretion, lower abdominal pain, fever, azotemia, restlessness	Death in 2 days	Infarction of left lobe with necrosis. Questionable infarction of gastric fundus. Chronic pyelonephritis
Levin 1957*	NR NR	Gastric ulcer	Gastrectomy	aLH?	Bile peritonitis	Death	Focal necrosis of liver. Bile peritonitis
Tsagareishvili 1958**	38 F	Gastric cancer	Gastrectomy	aLH?	Peritonitis	Death in 3 days	Focal necrosis of left lobe. Bile peritonitis
Tsagareishvili 1959**	48 F	Gastric cancer	Gastrectomy	CH	Bile peritonitis	Death in 4 days	Small, necrotic foci of liver. Bile peritonitis. LG originated from CH
Kato 1961**	60 NR	Gastric cancer	Gastrectomy	aLH	NR	Recovery	
Andressen 1962*	53 M	Gastric cancer	Total gastrectomy, splenectomy, distal pancreatectomy, cholecystectomy	CA, RH, LH, PD	Intestinal fistula, impaired liver function	Recovery	CA with its three branches was resected
Lurie 1962**	47 M	Gastric cancer	Total gastrectomy	aLH	Rapid, feeble pulse, dyspnea	Death in 2 days	Necrosis of left lobe. Thrombosis in hepatic lobar vein
Mori 1963**	60 M	Gastric cancer, gall stone	Total gastrectomy, splenectomy, partial resection of left hepatic lobe and pancreas, cholecystectomy	CH, SM	Sudden apnea followed by dyspnea and coma	Death in 30 hrs.	CH was anastomosed to SM; Both completely obstructed. Intestinal and hepatic necrosis

Table 2 -continued

Author Date	Age Sex	Disease	Operation	Artery	Complications	Outcome	Note and autopsy findings
Postlethwait 1964*	38 M	Duodenal ulcer	Gastrectomy	GD,PH	Biliary and pancreatic drainage	Recovery	Reoperation 6 weeks later : Spontaneous obliteration of PH. Liver slightly enlarged, red and soft. Necrosis of gall bladder and common duct
Monafo 1965*	61 F	Gastric cancer	Gastrectomy	PH	Shock, alternating hypotension and hypertension, jaundice, azotemia	Death in 5 days	Irregular areas of necrosis in both lobes
Karasewich 1965**	56 M	Duodenal ulcer	Gastrectomy	PH	Uneventful	Recovery	Artery was immediately reconstructed
Miyazaki 1968*	56 M	Gastric cancer	Total gastrectomy	PH	Bile drainage. Gall bladder perforated?	Recovery	
Akovbiantz 1968*	61 NR	Ulcer	Gastrectomy, choledochostomy. Later, hepatoduodenostomy	GD,PH	Impaired liver functions, liver abscess, biliary fistula, cholangitis	Recovery	Common duct was also injured. PH was anastomosed immediately, but was found to be occluded 5 years later.
Akovbiantz 1968*	58 NR	Ulcer	Total gastrectomy. splenectomy. Relaparotomy	CH,S	Impaired liver functions, adhesion ileus	Recovery	CH was anastomosed immediately, but was found to be occluded 4 years later

* Date of publication ; ** date of operation; CH, common hepatic; GD, gastroduodenal; PH, proper hepatic; LH, left hepatic; RH, right hepatic; CA, celiac axis; S, splenic; aLH, aberrant left hepatic; PD, pancreaticoduodenal; SM, superior mesenteric; NR, not recorded.

nourished by the hepatic artery, believed that occlusion of the portal branches or hepatic veins due to necrosis of their walls or thrombosis was the real cause of hepatic necrosis. In some instances³⁴⁾⁴⁵⁾ pathological changes of the gall bladder or biliary canal with consequent bile leakage were a possible cause of death.

With the advent of antibiotics the concept of the sequence of hepatic dearterization underwent a remarkable change. In 1949 MARKOWITZ et al.⁵⁰⁾ found that, contrary to the previous belief, dogs survived hepatic arterial ligation when treated with penicillin. This was explained by the prevention of the intrahepatic proliferation of anaerobic organisms which had been blamed for the fatal autolysis of the liver¹⁹⁾⁸⁰⁾. The dramatic effect of antibiotics and the importance of bacterial proliferation have been confirmed by many investigators¹⁰⁾¹²⁾²⁰⁾²⁵⁾³³⁾³⁶⁾⁶²⁾⁷¹⁾⁷⁴⁾. Some of them³³⁾⁷⁴⁾ believed that collateral circulation was of little importance while some other workers²⁰⁾²⁵⁾³⁶⁾⁶²⁾ found that its complete interruption was invariably fatal despite antibiotic treatment. Almost all of them except SHATTEN⁷¹⁾ were of the opinion that clostridial infection was responsible for the pathological changes in the liver; TANTURI et al.⁷⁴⁾ ascribed the effect of ligation to clostridial toxin. Release of toxic substance from the ischemic or autolyzed liver was also suggested by others¹⁵⁾¹⁶⁾²⁴⁾³⁵⁾. Since the human liver is usually aseptic²²⁾⁶⁶⁾ unlike carnivores' livers which harbor anaerobes, the role of bacterial infection in man is not fully understood. Its possibility, however, can not be ruled out, since intestinal flora may gain access to the liver via the portal vein³⁹⁾. It is also difficult to evaluate the relative importance of other factors in man which have been obtained from experimental studies.

Some features in man. A survey of human cases subjected to hepatic arterial ligation during various operations reveals that extensive liver necrosis at autopsy is not so common in man as in experimental animals (dog, cat and rabbit). In some fatal cases there was no recognizable necrosis⁸⁾⁴⁹⁾⁶⁴⁾⁷⁷⁾ while in a few survivors the necrotic liver tissue was found to be sequestered⁴⁰⁾⁷⁰⁾. Thus, even before the antibiotic era, liver necrosis was not always the cause of death. On the other hand, various complications were seen postoperatively or at autopsy, including pneumonia⁸⁾¹¹⁾²³⁾³⁹⁾⁶⁵⁾⁶⁷⁾, cardiac infarction⁴¹⁾⁴⁹⁾, glomerular or tubular damage³⁹⁾⁴⁹⁾⁶⁹⁾, focal necrosis of the gastrointestinal canal¹¹⁾²¹⁾²⁶⁾ abdominal abscess and such symptoms as dyspnea, tachycardia, oliguria, azotemia and nervous disorders. It is likely that the authors regarded many of them as either terminal consequences or as coincidental events so that not so much attention was paid to them as to hepatic lesions. However, the exact sequence of events, as well as their causal relationship to each other, cannot be determined by the autopsy finding alone. Moreover, there is clinical evidence to indicate that some complications, though etiologically related to hepatic arterial ligation, precede and may contribute to the aggravation of the hepatocellular damage found at autopsy. In this respect it is noteworthy that in three of the present six cases, the postoperative course was complicated by cardiac or pulmonary manifestations such as dyspnea, tachycardia, chest pain or cyanosis, while the slight or moderate increase in transaminase values and the relative increase in the LDH₅ fraction were the only findings indicating liver cell damage. The electrocardiograms of all these cases showed depressed ST segments and inverted T waves in precordial leads,

although there was no preceeding episode of hypotension, hypoxia or significant blood loss. KITAMURA⁴¹⁾ also noted electrocardiographic changes following accidental hepatic arterial ligation in radical gastrectomy and found a microscopic focus of myocardial necrosis at autopsy together with extensive necrosis of the liver. MADDEN⁴⁹⁾ reported coronary thrombosis causing sudden cardiac arrest at the end of the operation which consisted of hepatic artery ligation for cirrhosis of the liver; No liver necrosis was encountered at autopsy. It is tempting to postulate that hepatic dearterization may indeed cause systemic changes involving not only the liver but also other organs in which circulation is impaired because of vasospasm, thrombosis, embolism or some other unknown mechanism. It is possible that some of the unexpected complications such as cardiopulmonary disorders, renal insufficiency, gastrointestinal infarction etc. which surgeons occasionally encounter after gastrectomy may result from inadvertent ligation of the hepatic artery, especially that of an aberrant left hepatic artery. The protean manifestations of hepatic arterial ligation are exemplified in the following case in which evidence indicates that, in all likelihood, an aberrant left hepatic was ligated during the previous gastrectomy, leading to atrophy of the left hepatic lobe.

CASE 7. A 61-year-old man was admitted to this hospital because of epigastric pain, backache, vomiting and chills. Nineteen months earlier, he had had a subtotal gastrectomy for an early gastric carcinoma in another hospital. On the fifth post-operative day he had developed a high fever which had lasted for four weeks with no demonstrable focus of infection. During this time pleural effusion was noted on the left side. After discharge he had experienced occasional episodes of backache and high fever of one week's duration. Similar symptoms developed on the day of the present admission along with tachycardia, hypotension, disorientation and oliguria. Blood culture grew *Klebsiella*. Laboratory studies revealed a leucocytosis with neutrophilia, thrombocytopenia and a transient increase in the LDH₅ fraction; other liver function tests were within normal limits. The amylase values were also normal. The patient responded satisfactorily to intensive chemotherapy with amino-deoxy-kanamycin (Kanendomycin) and other antibiotics. After all the symptoms had disappeared, an exploratory laparotomy was performed through the same mid-line incision as before. However, the abdominal cavity was not entered until the falciform ligament had been divided longitudinally because its hepatic attachment was deviated 3 cm to the left of the mid-line. This was due to postoperative atrophy of the left hepatic lobe which was reduced to one-third of the normal size. This lobe was friable, and firmly adherent to the gastric remnant and surrounding tissues from which dilated capillaries entered the liver. No other abnormalities were encountered.

Aberrant hepatic artery from the left gastric artery. Since HALLER's designation of the left gastric artery as "the left gastrohepatic artery" in 1745,²⁸⁾ it has been known that this artery may give off a large branch to the liver. In some instances this branch may be the sole source of arterial blood supply to the left hepatic lobe, replacing the left branch of the proper hepatic artery. According to MICHELS⁵⁵⁾ the latter variation occurred in 11.5% of 200 cadavers. From the works of ADACHI¹⁾ (250 cadavers), LURJE⁴⁸⁾ (194 cadavers) and MELLIÈRE⁵⁴⁾ (200 cases) the incidence of this variation is roughly estimated at 12%, 7%, and 11%, respectively, excluding a few

ambiguous cases with ill-defined distribution of the middle hepatic artery. COUINAUD¹⁴⁾, who observed intrahepatic arterial distribution in his 96 injection-corrosion preparations, found that in 16 instances an aberrant left hepatic from the left gastric artery supplied either the total area of the left lobe or more extensive areas of the liver. MIKHAILOV⁵⁶⁾, using a similar method, gave a figure of 22% in his 100 preparations.

As compared with these figures obtained from the studies of cadavers, the frequency with which this artery was encountered and preserved at operation is rather low. During the past three and a half years we have had 434 cases of either total or subtotal gastrectomy for cancer (16 deaths within one month). An aberrant left hepatic was preserved in only nine instances (2%). FRIESEN²¹⁾ preserved it in five out of about 100 patients operated for esophageal or gastric diseases. LURIE⁴⁷⁾ could preserve it in only seven (1.4%) of his 536 operations for cardiac cancer of the stomach. Each of these authors reported a fatal case of liver necrosis resulting from accidental division of the artery. Fatal necrosis of the left hepatic lobe was also observed by MELNIKOV⁵³⁾, LEVIN⁴⁴⁾ and TSAGAREISHVILI^{75) 76)}. These authors and many others^{37) 68)} stressed the importance of the preservation of this artery. However, all of our four patients have survived deliberate severance of an aberrant left hepatic, and in none of the fatal cases in our gastrectomy series was hepatic necrosis or insufficiency evident, although in some fatalities the cause of death remained obscure. Assuming that the replaced left hepatic arises from the left gastric artery in 10%, these results indicate that in no less than 5% this artery may be overlooked and divided during operation, but serious complications are rare.

Moreover, as compared with en bloc resection of the entire lesser omentum and left gastric artery along with accompanying lymph nodes, the practice of preserving the left gastric arterial trunk and its hepatic branch may decrease the radicality of the operation because connective tissue and lymph nodes along the course of these arteries are frequently involved by cancerous infiltration and metastasis. Considering these points, we believe that an aberrant left hepatic may be sacrificed in good-risk patients, especially when there is possible involvement of the lesser omentum or gastropancreatic fold. Prior to its division, however, the presence of the hepatic artery in the hepatoduodenal ligament must be confirmed, and the lesser omentum should be temporarily grasped with vascular clamps so as to observe the resulting changes in the liver. It is preferable to divide the lesser omentum before it is ligated or clamped, because this enables one to evaluate the actual hepatic blood supply via the lesser omentum.

In conclusion, the prognosis of hepatic arterial ligation is generally more favorable than was previously believed. Its consequences, however, are very variable and cannot be predicted from the site of ligation or the anatomical pattern of hepatic blood supply alone. Rather, the patient's condition, associated operative intervention and intraoperative as well as postoperative management play important roles in the outcome of the ligation. Therefore, even ligation of the common hepatic artery, celiac axis, or an accessory hepatic artery may occasionally be followed by death in poor-risk patients with potential impairment of peripheral perfusion, such as those

with azotemia, diabetes, liver cirrhosis, dehydration and cardiovascular diseases.

Summary

Six cases of hepatic arterial ligation during radical gastrectomies are reported with an additional case of probable ligation. All of them have survived. In addition, 36 reported cases of hepatic arterial ligation associated with gastric surgery are reviewed. The present study suggests that hepatic dearterization may produce a variety of clinical manifestations and pathological changes which are not confined to the liver. The prognosis is more favorable than was previously assumed. It depends not only on the site of ligation or the anatomical presence of collaterals but, to a great extent, on the patient's condition, associated operation, management and many other factors. The problem of the division of an aberrant left hepatic artery is also discussed in detail.

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Addendum

After this paper was received for publication, 34 additional cases of APPLEBY operation were found in the literature, including 19 additional cases reported later by APPLEBY (J. Int. Coll. Surg., 34:143, 1960), one by KAJITANI (Sogoh Igaku, 14:309, 1957) and 14 by WADA (J. Jap. Surg. Soc., 71: 1248, 1970). Recently, we have performed a modified operation in a 73-year-old man in which the cardia of the stomach has been spared.

和 文 抄 録

胃切除に伴う肝動脈結紮. 6自験例と36文献例について

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胃切除中に肝動脈または肝動脈枝を結紮した6例を報告した。また再開腹により、最初の胃切除の際肝動脈枝を結紮したと推定される1例を合わせて報告した。全例回復した。3例では心肺合併症が最も著明な症状であり、最後の1例では高熱と胸水が主症状であった。乳酸脱水素の Isozyme を調べた最近の4例では、LDH₅ 分層の一過性増加の方がTrans-aminase 値の上昇よりも明らかであった。

文献から胃切除に関連して肝動脈の遮断された36例を検討した。また過去の実験成績や自験例に基づいて肝動脈結紮の死因や臨床像の再検討を行った。その結果、肝動脈結紮による変化は肝臓に限らず、同時に心、肺等全身的な病変を生じ、多種多様な臨床像を呈

することが少ないことが分った。

その予後は結紮の位置や、解剖学的な副血行路の有無で決まるものではなく、手術の種類や程度、患者の状態や管理方法等で大きく左右される。しかし過去に考えられたよりは一般に良好である。

胃の全摘や亜全摘または噴門部切除の際、左胃動脈から出る肝動脈枝を発見して保存する頻度は、解剖学的に証明される頻度より少い、つまりこの動脈枝を見落して切断することも稀ではないが、大部分は支障なく経過すると思われる。またこれを切断して胃癌の根治性を高める方がよい場合もある。しかし稀には胃切除後の予期せぬ合併症がこの肝動脈枝の切断に起因することもある。