THE SURGICAL TREATMENT OF CANCER OF THE HEPATIC DUCT CONFLUENCE

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The surgical treatment of cancer of the hepatic duct confluence

A clinical, anatomical and experimental study and literature survey

PROEFSCHRIFT

TER VERKRIJGING VAN DE GRAAD VAN DOCTOR IN DE GENEESKUNDE
AAN DE KATHOLIEKE UNIVERSITEIT TE NIJMEGEN
OP GEZAG VAN DE RECTOR MAGNIFICUS PROF DR J G H I GIESBERS
VOLGENS HET BESLUIT VAN HET COLLEGE VAN DEKANEN
IN HET OPENBAAR TE VERDEDIGEN
OP VRIJDAG 6 MEI 1983 TE 14 00 UUR PRECIES

DOOR

EGGE JAN BOERMA

GEBOREN TE RENKUM

H VEENMAN & ZONEN B V - WAGENINGEN - 1983

Of all the tumors I know of no other that can be quite as challenging as these small localized lesions in the upper portion of the hilus.

W. P. Longmire Jr., 1975

ISBN 90-9000450-5

aan Jannie, Djamila, Jasper en Sanjé

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Contents

Introduction

Fortunately carcinoma at the confluence of the hepatic ducts is rare. A tumour of the extrahepatic bile duct is found in one out of every 1,000 autopsies (33, 88, 273, 321, 433, 485) and in one out of every 100 bile duct operations (41, 218, 273, 348, 413, 440, 609). One percent of all malignant tumours originates from the extrahepatic bile ducts, peri-ampullary and gallbladder cancers excluded (60, 348, 458, 505), and half of them is localised in the confluence of the hepatic ducts (5, 9, 19, 44, 178, 258, 273, 343, 477, 538, 564). Probably this represents an underestimation of the true picture, now they have become so well known and notorious (Klatskin et al.(65)) and since the assessment of obstructive jaundice recently has been improved so much (79, 80, 208, 292, 371, 412, 484, 564). Tumours of the hepatic duct confluence have slight predominance in men over women. The average age of the patients is 60 to 65 years but nearly all series contain a few patients between the age of 30 and 40.

Aetiology

Different pathogenic factors may play a role but the real cause of a bile duct cancer is not known. Chronic inflammation of the ducts with stasis of bile may finally lead to cancer (501), as in sclerosing cholangitis (123, 320), typhoid carrier state (603), chronic suppurative cholangitis with intrahepatic gallstones (513) and bile duct parasites (31, 45, 274, 553). Gallstones occur in patients with bile duct cancers as often as in those without and so are not causally related (88, 433, 437). Various substances can induce bile duct cancers in laboratory animals (190, 292, 501, 532, 553). Spontaneous bile duct cancers that may have been caused by carcinogens are reported in animals (153, 162). Labourers in the rubber industry are said to have an increased risk (316).

Malignant degeneration of benign bile duct adenomas, papillomas (280, 288) or bile duct cysts (301, 569) is well recorded. Concomitant colitis ulcerosa occurs in 0 to 14% of the patients (7, 44, 107, 223, 470, 548, 579) and a second primary tumour is present in 3 to 16% (88, 172, 223,235, 343).

Symptoms, signs and course of the disease

The symptoms of a bile duct tumour are non-specific and are almost entirely due to bile duct obstruction which results in a progressive jaundice (12, 33, 187, 286, 309, 596, 606). Non-specific prodromal complaints may be present about two months before the outset of the jaundice and consist of loss of appetite, emaciation, abdominal pains, itch or diarrhoea (13, 43, 394, 542, 564). Jaundice usually comes into being only when the bile duct obstruction is virtually complete (55, 484). It may be intermittent at first (17, 88, 111, 113, 145, 321, 477),

especially when caused by a papillary tumour (372), and is often attended with pruritis and weight loss. Half of the patients have upper abdominal pain (88, 292, 477, 489). Portal hypertension (217), ascites (171, 321, 365) and gastrointestinal disturbances are also reported.

In half of the patients (29-78%) the diagnosis has not been made at a previous laparotomy (7, 13, 195, 208, 594, 606, 609). Fever and cholangitis rarely complicate the natural course of bile duct tumours (113, 321, 489) but infection is common after surgical intervention (285, 351).
The average duration from the start of the jaundice to the surgical treatment amounts to four months (7, 171, 208, 258, 273, 286, 409, 462, 485, 542, 581).

Physical examination of a patient with a proximal bile duct cancer usually reveals jaundice and an enlarged liver. Laboratory examination of the blood just confirms the obstructive jaundice (17). A selectively raised alkaline phosphatase level, although non-specific (73) and not significant (579), may indicate a bile duct tumour in the pre-icteric stage (41, 63, 77, 235, 252, 596, 606). The typical findings at surgical exploration are a large, firm, congested liver and a collapsed gallbladder and common bile duct.

Without treatment the patient usually dies from hepatobiliary insufficiency within three months of the diagnosis being made (319), but a much longer survival is reported incidentally (442). The treated patient also dies usually from the consequences of biliary obstruction and not from the malignant properties of the tumour. Cholangitis, septicaemia and liver abscesses as well as liver insufficiency or biliary cirrhosis are by far the most important causes of death (12, 13, 14, 60, 174, 223, 277, 285, 286, 420, 477, 485, 507, 564, 581). At autopsy only a small, undisseminated tumour is found in nearly half of the patients (396).

Diagnosis of the cause of jaundice

Since there is no reason for any diagnostic delay, a patient with obstructive jaundice should be prepared for surgery by the second week of being jaundiced (489). For a rapid, efficient and appropriate diagnosis of jaundice, the many modern diagnostic tools have to be used selectively (35, 36, 54, 63, 251, 272, 621). Anamnesis and physical and laboratory examinations usually make it possible to differentiate between medical and surgical jaundice (554). Ultrasound investigation demonstrates bile duct dilatation with 95% certainty (55, 63, 388, 577), provides information about the size of the liver lobes (583) and may indicate liver metastases (560).
Dilated ducts necessitate an intervention (286). Cholangiographic identification and, if possible, cytologic verification of the tumour (177) form the final assessment of the diagnosis.

Therapy

Restoration of the bile flow to the bowel is the first aim of

the therapy since the bile duct obstruction determines both mortality and morbidity. A lasting bilio-enteric reconstruction is only rarely attained and consequently the mean post-operative survival is only one to 1½ years. Resection of the tumour offers the best therapeutic possibilities and moreover has the potential to remove the malignancy, however tiny this chance seems to be in practice. Palliative internal bile drainage either paratumoral or transtumoral is considered if resection is impossible or not pursued.

In patients with high operative risks percutaneously or endoscopically inserted drains may offer the best solution. The benefit of adjuvant radiotherapy remains unclear but irradiation may be administered. Finally it is important 'to distinguish between the need for operations that may prolong the act of dying and for those that may extend a tolerable but limited span of life' (5).

Aims and purposes of the thesis

Renshaw (462) wrote in 1922:'The surgical treatment of malignant conditions of the biliary tract has not advanced proportionately with that of other parts of the upper abdomen. Alas, this still applies to the cancers of the hepatic duct confluence, as it does to gallbladder cancers, in spite of the fact that many of the confluence tumours are small, circumscript and not yet disseminated at the time they are discovered (309, 396, 537a).

The present understanding of a tumour of the hepatic duct confluence and of the many pathological factors which play a role in this disease is ill-defined and superficial. Progress of the knowledge of confluence tumours requires a thorough prospective investigation based on knowledge obtained in the past. The aims of this study are threefold:

1 to provide a comprehensive review of the data already known, from which a prospective study can be initiated;
2 to record the present-day treatment methods and their suitability for the individual patient; and
3 last but not least, to investigate the causes of the common therapeutic failure and to develop a better treatment.

For that purpose the anatomy of the liver hilus, the characteristics of these tumours and the various types of treatment are reviewed. A retrospective survey of 51 patients with confluence tumours and a prospective study of 24 patients with confluence tumours involving the portal vein are presented. Based on an analysis of the poor results the required extent of a radical resection is defined and discussed together with the practicability of such a resection. Subsequently a technically feasible operation is described as developed in an anatomical study. The clinical applicability of such an operation seems reasonable as demonstrated in animal experiments.

1. Anatomy

1.1. Anatomy of the liver

The liver has a twofold segmental structure (27, 28, 142, 219, 247, 267). One segmentation is governed by the structures which enter and leave at the porta hepatis i.e. the portal vein, hepatic artery, hepatic ducts, lymphatic vessels and nerves. These portal or hilar structures and their intrahepatic branches stay together, the bile ducts usually in ventral and the arteries in dorsal position to the accompanying portal vein branches (466). Also inside the liver they are enveloped in a fibrous sheath which is the intrahepatic continuation of the perihepatic capsule of Glisson (142). This capsule condenses in the porta hepatis to a fibrous hilar plate (plaque hilaire) and from there ramifies, following the intrahepatic divisions of the portal structures. Eight liver segments can be discerned based on the intrahepatic division of these portal structures. The portal structures of these segments have no mutual communications other than by the segmental main branches, they are numbered from I to VIII according to Couinaud (142). The other segmentation is determined by the three suprahepatic veins which leave the liver posterocranially, each of them draining its own suprahepatic segment.

The portal and suprahepatic structures are intertwined in such a way that the main branches of the suprahepatic veins run in the plains of separation (scissures) between the portal segmental structures and vice versa (Fig. 1).

The portal segments II and III correspond with the left suprahepatic segment and form the classic left liver lobe, situated at the left side of the umbilical fissure, ligamentum teres hepatis, ligamentum falciforme and sulcus Arantii. The segments IV up to and including VIII correspond with the middle and right suprahepatic segments and form the classic right liver lobe. The quadrate lobe consists of the anterior part of segment IV. The caudate lobe of Spigel, also indicated as portal segment I or dorsal liver segment, forms a separate segment and is situated at the cranio-dorsal side of the liver, straddling the left anterior side of the inferior caval vein beside the right adrenal gland.

The plane of separation between segment IV and segments V and VIII is called principal scissure and contains the middle suprahepatic vein. This scissure divides the liver according to its portal structures in two main parts (387), a right and a left hemi-liver. Along this principal scissure a right or left hemipatectomy is carried out. The plain of separation be-

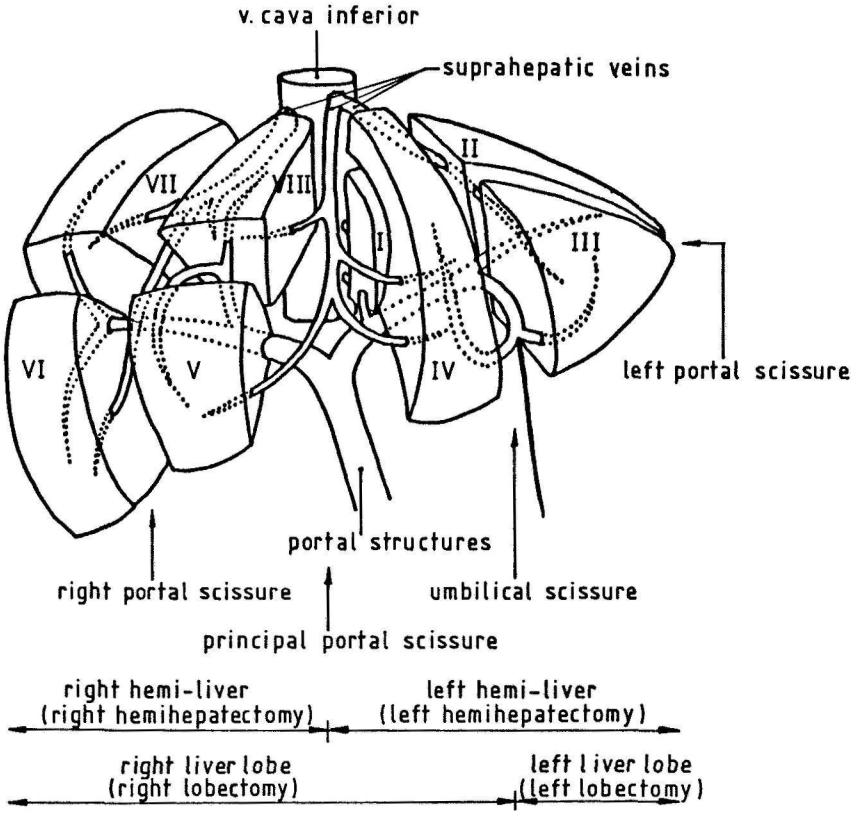

Fig.1. Segmental anatomy of the liver.

tween segment IV and segments II and III is called umbilical scissure and corresponds with the left suprahepatic intersegmental plane. This scissure divides the liver in its classical right and left lobes and resections along this plane are therefore best called right and left lobectomy (49, 565).

1.2. Anatomy of the hepatoduodenal ligament and hilus of the liver

The hepatoduodenal ligament forms the free right margin of the lesser omentum and contains the structures which enter or leave the porta hepatis (Chapter 6.3.; Fig. 10).

Vena portae

The portal vein arises behind the pancreas and before the inferior caval vein from the confluence of the splenic vein, which runs from cranial-left to caudal-right along the posterior surface of the pancreas, and the superior mesenteric vein. The portal vein is 8 cm in length and runs along the left-posterior side of the bile duct and right-posterior to the hepatic artery in the hepatoduodenal ligament to the porta hepatis. It receives the v. coronaria, v. pylorica, v. pancreaticoduodenalis superior and v. gastroepiploica dextra, enters the right corner of the porta hepatis and there widens and bifurcates posterior to and in contact with the left side of the hepatic duct confluence (268). The right main branch measures 2-3 cm and divides into the right lateral and right paramedian branches.

The left main branch is smaller than the right one but still as wide as the splenic vein. Considering the many variations of the hilar anatomy, the course of the left portal vein branch is remarkably constant. It always runs extrahepatically and transversally in the porta hepatis in cranial-left direction, measures 4-5 cm and gives off some side branches to the caudate and quadrate lobes. In the left edge of the porta hepatis the left portal vein branch bends anteriorly and widens to its umbilical part, also called the recessus of Rex. This recessus of Rex is situated in the posterior side of the umbilical fissure and runs in anterior direction.
From its base the portal vein branch to segment II originates and further downstream the branches to segment III and to the left side of segment IV emerge. Finally the recessus of Rex terminates in the closed umbilical vein in the ligamentum teres (142, 157, 206, 346, 502) (Figs 2 and 3).

Variations of the anatomy of the portal vein system are rare. Rests of valves are only found in small side branches of stomach and omentum veins of neonates. They atrophy and vanish quickly (157, 615), therefore the direction of the blood flow in the portal system is determined by the existing pressure gradient only (479).

Arteria hepatica

The common hepatic artery arises from the truncus coeliacus, runs in right-anterior direction along the cranial border of the head of the pancreas, crosses the portal vein left-anteriorly, gives off the gastroduodenal artery and the right gastric artery and then runs as proper hepatic artery left-anterior to the portal vein and along the left side of the bile duct to the porta hepatis.

The gastroduodenal artery runs anterior to the portal vein in postero-caudal direction, at the left side of the common bile duct and posterior to the duodenum. There it divides into the a. gastroepiploica dextra and the a. pancreaticoduodenalis superior which anastomose with the corresponding left and inferior arteries arising from the splenic and the superior mesenteric artery respectively (18, 401, 415, 519).

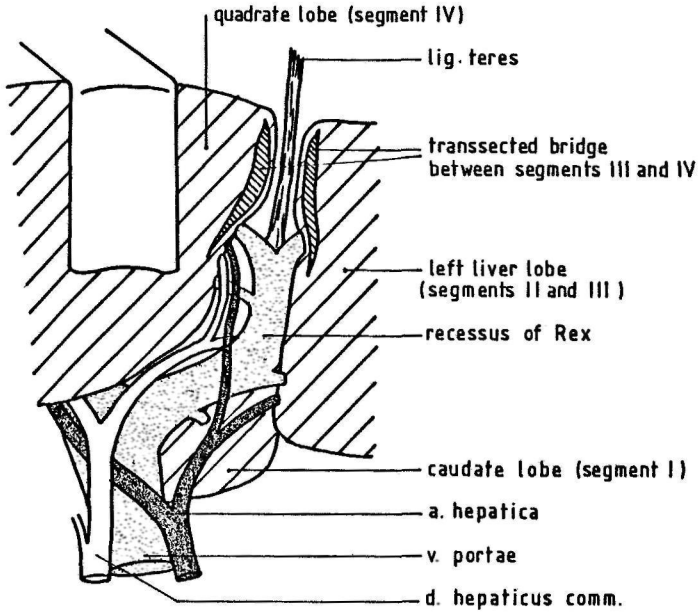

Fig. 2. The left extrahepatic portal structures. (seen from ventrocaudal direction).

The proper hepatic artery runs along the left side of the bile duct and left-anterior to the portal vein to the porta hepatis and splits somewhat earlier than the portal vein into a right and left branch. The right branch runs between the portal vein and the common hepatic duct to the right, launches the cystic artery, then runs right-anterior to the right portal vein branch and divides into the right lateral and right paramedian branches. The left branch runs left-caudal to the left portal vein branch to the left liver lobe. It gives off a middle hepatic artery which runs on the right side of the umbilical fissure to the quadrate lobe.

Deviations of this pattern exist in 45% of the people. The most important anomalies are a left hepatic artery orginating from the left gastric artery (22%) and a right hepatic artery sprouting from the superior mesenteric artery (12%) (397).

Bile ducts

The ductus hepatocholedochus runs in the anterior free margin of the hepatoduodenal ligament, right-anterior to the portal vein and right to the hepatic artery. Following the 8 cm long duct upstream from its entrance in the duodenum, at first the cystic duct and gallbladder are given off. At the right edge of the porta hepatis the 3 cm long common hepatic duct divides into the right and left hepatic ducts.

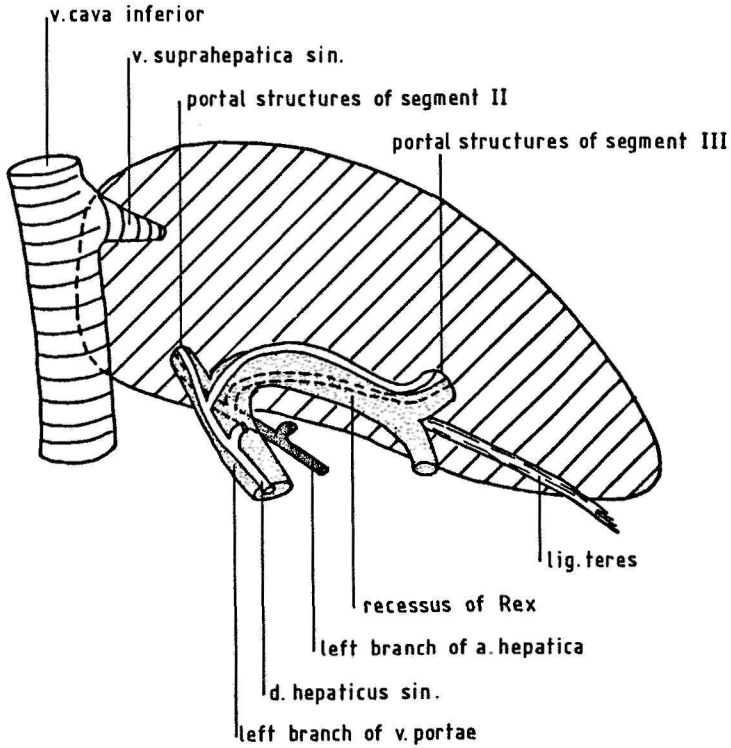

Fig. 3. The left edge of the porta hepatis and the subhilus of the left liver lobe (seen from right-lateral direction after removal of the right lobe). The branching of the segment II structures has been drawn outside the parenchyma.

This bifurcation, more correctly called 'confluence' with respect to the direction of the bile flow, is always situated extrahepatically, as is the left hepatic duct (58). The distance between the bile duct and the portal vein becomes even smaller as both structures approach the liver and in the porta hepatis they are in contact with each other. The hepatic duct confluence lies directly on the portal vein bifurcation or on the base of its right main branch (268). The anterior side of the confluence and the beginning of the left hepatic duct are covered by the hilar plate (142, 254).

The right hepatic duct measures 1-2 cm, runs left-anterior to the right portal vein branch and splits intrahepatically into the right lateral and right paramedian ducts. The 4-5 cm

long left hepatic duct runs transversally in the porta hepatis to the left and gives off one to three side branches to segment IV. Some small branches to the caudate lobe originate from the posterior side of the left hepatic duct and sometimes from the hepatic duct confluence too.

Following the left hepatic duct in upstream direction, the duct is at first in cranio-anterior position to the left portal vein branch but at the left edge of the porta hepatis it is cranial to the base of the Recessus Rex.
There the left hepatic duct divides in general inside the left liver lobe into the duct to segment II and the duct to segment III. The segment III duct runs at the left antero-cranial side of the corresponding portal vein branch to segment III (121, 142, 247) (Fig. 3).

Variations of the extrahepatic biliary anatomy are frequently seen. Only 56-72% of the people have a hepatic duct confluence as described. In 28-44% one or more segmental ducts arise in an unusual way from a hepatic duct (142, 484). The course of the left hepatic duct is fairly constant, although the second order confluence, in which the segmental ducts II and III come together, is located to the right of the umbilical fissure in 22-42% of the people (scission horizontale) (142, 247).

The vascularization of the extrahepatic bile ducts consists of a peribiliary arterial and venous plexus. The arterial network arises from branches of the proper hepatic and gastroduodenal arteries. The venous plexus, together with a similar plexus around the gallbladder, is reported to drain its blood partially to small venules which enter the adjacent liver parenchyma of the quadrate lobe and gallbladder bed. These venules would divide into capillaries, that finally re-unite in the suprahepatic venous system (3, 181, 405, 445).

Lymphatic system

Lymph vessels in the hepatoduodenal ligament are numerous. The lymph drainage of the liver hilus follows two main chains: one along the portal vein and the bile duct and the second along the hepatic artery. The periarterial chain mainly collects lymph from the left liver lobe and carries it via one or two lymph nodes near the origin of the left hepatic artery branch and nodes along the proper or common hepatic artery to the coeliac and para-aortal nodes.

The periportal and peribiliary chains drain the lymph of the extrahepatic biliary tract and the right liver lobe. There is only recently agreement about the presence of lymph nodes in the liver hilus as former reports were contradictory (181). Obstructive jaundice caused by compression of the hepatic duct confluence by a tuberculous lymph node is described (377) and finally Hardy and coworkers (240) demonstrated that hilar lymph nodes do exist and are situated posterior to the portal vein bifurcation and the beginning of the left portal vein branch. These nodes receive lymph from adjacent regions and drain on the lymphatic network around the portal vein. This periportal chain is connected with the peribiliary lymphatic

system and with the periarterial chain by lymph vessels posteriorly in the hepatoduodenal ligament.

The peribiliary lymph vessels run via the cystic node and the hiatal node to the superior pancreatoduodenal nodes beside the distal end of the common bile duct and beside the duodenum and pancreas. From there the lymph flow heads for the coeliac nodes and via the posterior pancreatoduodenal nodes for the nodes near the origin of the superior mesenteric artery (159, 181, 240, 415, 377, 466).

Some peribiliary lymph vessels run straight to the pancreatoduodenal nodes (159, 415). During obstruction of the lymph drainage a retrograde flow of lymph is possible (181, 449). Because of a biliolymphatic permeability part of the bile may be drained by the lymphatic system during biliary obstruction (159, 213).

2. Review of the literature on carcinoma of the hepatic duct confluence

The main problem in reviewing the literature is the absolute lack of a systematic classification of the patients and their tumours. The reported frequencies of tumour characteristics and metastases depend strongly on the systematic thoroughness of the investigation and on the moment this is done. Detailed information on these factors is seldom provided. Often exact information about tumour properties and therapies is missing or concealed in a subjective personal classification. Therefore the patients to be discussed in this Chapter form a very heterogeneous population and the mutual comparability of the various components leaves much to be desired.

In many papers tumours of the hepatic duct confluence are discussed in conjunction with tumours of other parts of the bile duct without proper specification. The results of treatment of confluence tumours are worse than those of more distally localised tumours (421, 489). After resection the malignancy is histologically confirmed, but in patient studies on other types of treatment histological confirmation is unclear or only partially provided which makes the results of these treatments susceptible to criticism. Operative mortality usually is defined as death in the first month after the operation, but in some publications it means death in the first two months thus influencing the average postoperative survival. Some authors understand by duration of survival, the time of life from the outset of the jaundice but the major part starts to count as from the main operation. In most reports one part of the patients has died while the other part is alive. Consequently the average survival is determined partly by the death of the patients and partly by the time of writing. This last imperfection is relative as it applies to all patients.

The present review includes as much as possible only histologically proven primary tumours of the hepatic duct confluence. In this context 'bile duct tumour' means only primary tumours of the left, right and common hepatic ducts and supraduodenal portion of the common bile duct and exclude tumours of the gallbladder and periampullary area. Duplication and overlap of series or updated reviews on the same patients I have tried to avoid. Operative mortality is defined as death within the first month after operation. The duration of survival is counted as from the day of the main operation. In this period of survival those patients are excluded who died in the first postoperative month, all other patients are included in this study whether dead or alive at the time of writing. Liver resection along the principal scissure is called hemihepatectomy, and resection along the umbilical scissure lobectomy. Heterogeneous as it remains, no effort was made to apply statistics to this patient material.

2.1. Pathological anatomy of cancers of the hepatic duct confluence

Macroscopic features

The main macroscopic types of bile duct tumours can be discerned as nodular or annular and as papillary or villous types. The macroscopic type is mainly determined by the stroma reaction around the tumour (433). The stroma of tumours situated at the liver hilus usually consists of strongly fibrotic connective tissue which produces a scirrhous nodular or annular mass (602). A papillary type is found in 6% of patients with a bile duct tumour (77, 88, 309, 372, 384, 441, 484,564).

A bile duct tumour is found to be multiple or diffusely spread for a long distance of the bile duct including the confluence in 9% of the patients. In these cases the extrahepatic bile duct may become a rigid tube (linite biliare (112), cancer en tuyau (433), Röhrenform (437)), while intrahepatically the tumour may follow the biliary bile ducts fanwise (cancer en éventail (33, 433)) (Table I).

Table I. Incidence of the multicentric or diffuse type of primary bile duct tumours.

Authors	bile duct tumours	diffuse or multicentric tumours
Adson and Farnell (5)	221	11
Andersson et al. (17)	53	6
Benhamou et al. (33)	17	8
Bertrand et al. (43)	16	4
Bessot and Boissel (44)	36	2
Brown et al. (88)	37	5
Ham and Mackenzie (235)	32	2
Hepp and Mercadier (255)	9	1
LeNeel et al. (348)	20	2
Mourgue-Molines et al. (409)	16	4
Neibling et al. (413)	90	4
Saubier et al. (491)	45	3
Tompkins et al. (564)	96	7
Warren et al. (596)	77	13
Total no. of patients	765	72 (9.4%)

In general the macroscopic and palpable size of a proximal bile duct tumour is small (309, 396). Sometimes the tumour forms a large, hard mass (480) in which the site of the primary lesion hardly can be traced. In these cases the place of the largest thickening or the centre of the bile duct obstruction may be regarded as origin of the tumour (171).

The tumours may preferably arise from the left side of the confluence or the left hepatic duct (458, 596) although tumours arising from the right side are also described. There is a possibility that right-sided tumours are more often missed or booked as intrahepatic tumours as they are more

difficult to reach than left-sided tumours (357).

There are three possibilities for a bile duct obstruction in the hepatic duct confluence (43, 48): 1 partial or complete obstruction of a duct; 2 unilateral or bilateral obstruction; and 3 obstruction confined to the main confluence or extended to secondary confluences.

Microscopic features

Virtually all primary bile duct tumours are adenocarcinomas. The mucin produced by tumour cells becomes blue-stained by Alcian blue, contrary to the mucin of benign cells (602).Other types of epithelial tumours are found occasionally, non-epithelial tumours are extremely rare (171, 210).

Well-differentiated tumours are predominant (13, 171, 172, 309, 485, 542, 548, 564, 596) although in some series most tumours are poorly differentiated (343, 477).

In proximal bile duct tumours the connective tissue around the tumour tissue is in general densely fibrotic, sclerotic and scirrhous (13, 77, 171, 210, 290, 413, 433, 484, 523, 548,602). The fibrotic reaction around the tumour may even be so abundant that hardly any tumour cells can be found (477). Virtually, it can be impossible to discern desmoplastic and proliferative changes of bile duct tissue from sclerosing carcinoma (602). For these reasons a representative biopsy may be difficult to obtain for the surgeon and difficult to interpret for the pathologist. Moreover a periductal fibrosis may unjustly be considered as a carcinomatous expansion (602). These factors should be taken into account when judging the results of the various treatments.

There is no clear relation between the histological type and the macroscopic aspect (433, 542). In general tumours at the confluence of the hepatic ducts are small, well differentiated, scirrhous, annular adenocarcinomata (309, 396, 515, 602, 606).

Infiltration and local spread

Submucosal spread in the wall of the bile duct is the most common way of local tumour expansion and may occur for a wide distance (77, 88, 145, 229, 292, 433, 480, 556, 572). Therefore a bile duct carcinoma has to be regarded as a regional rather than as a local lesion of the bile duct (433). It is not clear as to how far involvement of the gallbladder, which may occur in up to 13% of the patients (321) is due to local spread, separate dissemination or multifocality of the primary tumour.

The wall of the bile duct only consists of an epithelial layer and a thin layer of connective tissue with some smooth muscle fibres. The outside of the bile duct is supplied richly with nerves, lymph vessels and blood vessels. The epithelial layer forms glands bulging from the exterior of the duct. Hence penetrating malignant cells immediately reach the periductal tissues (172, 229, 556, 561, 579, 619). Consequently spread of the tumour outside the bile duct is found in 70% of the patients

(171, 172, 216, 542, 579).

Spread in and along the periductal tissues occurs by way of invasion of

- the perineural lymph clefts, reported in 39-64% of the patients (77, 88, 172, 210, 292, 413, 433)(Chapter 3;Fig.6); such spread can progress far from the origin of the tumour (594);
- the periductal venules and arterioles, reported in 18 of 22 patients (82%) (433);
- the adjacent liver parenchyma of segments IV and I;
- the abundant lymphatic network;
- the portal vein and the hepatic artery. Penetration of a bile duct tumour into the portal vein (217, 442) may occur often and early (77, 268, 542, 556, 557), according to others only occasionally (60, 171) or even rarely (88, 172, 216, 433).

Since the confluence of the hepatic ducts is in direct contact with the right side of the underlying bifurcation of the portal vein, an early invasion seems plausible (268). Involvement of the portal vein can be caused by the tumour itself as well as by fibrotic tissue that surrounds the tumour (424, 557).

Regional dissemination

Involvement of the lymphatic system occurs early (8, 409). A staged specification of the lymph nodes involved is not reported in the literature. Likewise no suggestion could be found which nodes should be considered as the limit of regional lymph spread. In this context the area of regional lymph spread is defined to consist in the complete hepatoduodenal ligament up to and including the pancreatoduodenal nodes but excluding the mesenteric and coeliac nodes.

Table II. Incidence of regional lymphatic metastases demonstrated in patients with primary bile duct tumours.

Authors	bile duct tumours	regional lymph node metastases
Benhamou et al. (33)	17	5
Bessot and Boissel (44)	18	6
Black et al. (60)	80	47
Brown et al. (88)	62	32
El Domeiri et al. (172)	24	13
Glenn and Hays (211)	10	7
Ham and Mackenzie (235)	32	9
Kuwayti et al. (321)	63	21
Ross et al. (477)	103	15
Thorbjarnarson (557)	31	7
Tompkins et al. (564)	96	14
Warren et al. (596)	77	25
Total no. of patients	613	201 (33%)

Regional lymphatic metastases so defined are described in 33% (15-70%) of the patients with bile duct tumours (Table II). In patients with bile duct cancers localised in the hepatic duct confluence the reported incidence is 21% (0-44%) (Table III). Involved coeliac or para-aortal nodes are observed in 13% of the patients with a bile duct tumour (88, 321, 416, 596).

Table III. Incidence of regional lymphatic metastases demonstrated in patients with primary tumours of the hepatic duct confluence.

Authors	confluence tumours	regional lymph node metastases
Altemeier et al. (13)	18	8
Bertrand et al. (43)	16	1
Edmondson (171)	11	4
Ingis and Farmer (290)	14	0
Klatskin (309)	13	1
Launois et al. (330)	11	3
Van der Velde and Leer (581)	8	2
Total no. of patients	91	19 (21%)

Liver metastases are reported in 30% (0-50%) of the patients with a bile duct tumour (Table IV). Localisation or extent of metastases is rarely mentioned (365). Hepatic metastases may occur more often as the tumours are situated closer to the liver (421). Since small deposits will be easily missed, hepatic metastases probably occur in about half of the patients with tumours of the hepatic duct confluence (8, 51).

Lymphatic and hepatic metastases may occur simultaneously in one patient. In about half of the patients with a bile duct cancer one or both types of metastases are reported (Table V). As this incidence is probably too low rather than too high, it may be estimated that lymphatic or hepatic metastases exist at the time of treatment in about 1/2 to 2/3 of patients with bile duct tumours. Nevertheless, in many patients no metastases are found after previous operations or even after death, as is illustrated in 86 of 165 patients (52%) from six combined series in whom metastases were explicitly searched for during autopsy (60, 109, 236, 290, 396, 556).

Dissemination at distance

Metastases outside the regional lymph nodes or the liver are seen in about 15% of the patients (0-30%) (33, 60, 88, 171,172, 235, 273, 290, 309, 321, 409, 433, 564). In general they are located in the peritoneum of the lesser omentum, but other

Table IV. Incidence of hepatic metastases demonstrated in patients with primary bile duct tumours.

Authors	bile duct tumours	hepatic metastases
Benhamou et al. (33)	17	7
Bertrand et al. (43)	16	1
Bessot and Boissel (44)	18	9
Black et al. (60)	80	28
Brown et al. (88)	54	21
El Domeiri et al. (172)	54	25
Ham and Mackenzie (235)	32	7
Ingis and Farmer (290)	14	0
Iwasaki et al. (294)	23	8
Klatskin (309)	13	2
Kuwayti et al. (321)	63	21
Neibling et al. (413)	90	30
Neugebauer et al. (416)	54	15
Thorbjarnarson (557)	31	7
Tompkins et al. (564)	96	17
Van der Velde and Leer (581)	8	2
Total no. of patients	663	200 (30%)

Table V. Incidence of regional lymphatic or hepatic metastases or both, demonstrated in patients with primary bile duct tumours

Authors	patients (percentage)
Andersson et al. (17)	47
Bismuth and Grebert (51)	40
Black et al. (60)	45
Braasch et al. (77)	33
Goinard and Pélissier (216)	70
Kirshbaum and Kozoll (308)	77
Krain (316)	33
Kune (319)	33 to 66
Lees et al. (343)	23
Neibling et al. (413)	63
Olivier (430)	75
Rodgers et al. (471)	46
Saegesser et al. (484)	63
Stewart et al. (533)	46
Van Heerden et al. (579)	71
Van der Velde and Leer (581)	52
Warren et al. (596)	45
Whelton et al. (606)	55

peritoneal locations are also possible, as are incidental metastases in pancreas, ovaries, spleen and stomach. Dissemination outside the abdominal cavity is rare (although reported in 25% of one series (235)) and occurs mainly in the lungs.

Rapidity of growth and mutual relation of tumour properties

The growth of a bile duct cancer in general and of a confluence cancer in particular is called fast by some authors (77, 441) and slow by others (12, 13, 294, 309, 409, 562). There is no systematic investigation into the mutual relations of the various tumour characteristics.

The poorer the differentiation of the tumour, the worse is the prognosis of the patient (30, 433) but according to other authors this makes no difference (556). Patients without metastases show a longer survival than patients with metastases (343, 471). The histological type of the tumour would not correlate with the occurrence of metastases (606) and a relation between infiltrative growth and the macroscopic type of the tumour is denied (542). However,a papillary tumour would be less inclined to infiltrative growth (561) but more to a slow intraductal growth and hence should have a better prognosis than the usual sclerosing carcinoma (372, 523, 561, 563). When a scirrhous tumour has progressed so far that it blocks the lumen of the bile duct, the tumour cells have already invaded the surrounding tissues. Therefore tumour cells may remain in or between the many hilar structures after a supposedly complete resection of the tumour (314, 561, 579).

2.2. Pathophysiology and surgical considerations

Bile duct obstruction

Bile can pass through a very narrow lumen (55). Only when the bile duct is almost completely blocked the upstream biliary system becomes dilated, caused by the raised biliary pressure. Many intrahepatic bile ducts may widen up to 1½ or 2 cm in cross section (13, 344). When the maximal hepatic secretion pressure of 30 cm water is reached the bile secretion stops (216, 513). The portal blood flow may be hampered by the strongly dilated ducts, as both ducts and vessels are enclosed by the stiff, fibrous Glissonian sheaths (22, 85, 390). The liver tissue of which the biliary drainage is blocked, at first becomes congested and swollen and then atrophies. If another part of the liver keeps its normal biliary drainage then in that part a compensatory hypertrophy develops simultaneously (78, 236, 245, 359, 390, 494, 495, 496). If the complete biliary tree is blocked and the drainage of only a part is restored, then in that part a compensatory hypertrophy will occur, provided that the liver parenchyma is not seriously damaged by long-standing biliary obstruction (55, 69, 147, 473).

Jaundice only occurs in case the biliary drainage of the entire liver is almost completely blocked (484). Dilatation and jaundice are absent as long as the hepatocholedochal duct

is partially obstructed (63). A tumour of the hepatic duct confluence which has completely blocked one hepatic duct does not cause jaundice until the other hepatic duct is also seriously obstructed. Meanwhile the completely obstructed part of the liver has atrophied considerably. Dilatation of the bile ducts precedes jaundice. Delay of the operation until the bile ducts are appropriately dilated is for that reason useless (484).

The intestinal flora is disturbed by the absence of bile (63) and in particular fats and fat-soluble vitamins are malabsorbed (285). The resulting alimentary derangement is further deteriorated by anorexia. After two to three months of total biliary obstruction a secondary biliary cirrhosis develops with portal hypertension and impaired liver function, bringing about hypalbuminaemia and coagulation disorders (36, 63, 430). Often these patients are operated on more than once with concomitant infections of the bile ducts and imminent renal insufficiency (11).

The treatment of choice

The kind of operation is determined by the palliation and potential cure to be expected, coupled to the lowest possible postoperative morbidity and mortality (243). In making this choice the condition of the patient and his tumour is just as determining as the attitude and skill of the attending surgeon (458). An operation in which no bile drainage is achieved may only speed up the fatal course of the disease. Without therapy the patient dies jaundiced from liver failure, but after inadequate restoration of the biliary drainage he dies jaundiced from cholangitis. Unless chosen as terminal palliation for patients with intractable pruritis, external bile drainage only adds a biliary fistula to the disease (430).

Surgical intervention mainly consists of resection, paratumoral bile drainage and transtumoral bile drainage.

Resection - Both for duration and quality of survival a well-functioning internal bile drainage forms the main goal of therapy. Resection of the tumour followed by the reconstruction of a proper bilio-enteric anastomosis offers the best and longest survival (8, 66, 178, 333, 471, 489)(Chapter 2.5; Fig. 4), and the only chance to cure the patient of his malignancy, however small this chance in practice may be. In most patients a recurrent tumour growth develops after resection, so that even excision of the tumour can be considered as some sort of palliation (137, 223). No patient could be traced with a clearly demonstrated curative resection although at least nine patients with a five-years survival after resection have been reported (74, 80, 109, 146, 333, 517, 561). Sometimes, however, the tumours grow so slowly that even after palliative drainage procedures five years survivals have been described in patients with proven cancers of the hepatic duct confluence (13, 286, 343). Even after an incomplete or palliative local resection with or without resection of the quadrate lobe, the quality of

the remaining life is better than after any other drainage procedure, because it provides the possibility of a good bilateral bile drainage (8, 66, 259, 558).

When comparing the various possibilities also the risks have to be considered (302, 472). A higher risk to perform a resection seems justified when considering the generally shorter time and inferior quality of life after drainage procedures (487, 488). Moreover the operative mortality is not higher after resection than after drainage operations, although the comparability of the patients remains questionable. Radiotherapy may theoretically eradicate the tumour but is in practice disappointing.

In conclusion: If possible a resection of the tumour should be performed and from a palliative point of view even an incomplete resection is better than non-resective drainage. Only if resection has been excluded para- or transtumoral drainage can be considered (66, 330, 458, 487, 488, 489, 492, 601). Therefore the aim of the pre-operative examination is to trace or to eliminate the contra-indications for a resection and to determine precisely the biliary and vascular anatomy.

Pre-operatively the patient has to be brought in an optimal condition for a possible liver resection. Therefore it may be beneficial to operate in two sessions as performed by Rohner (473). In a first operation the internal biliary drainage of the liver segments, to be preserved after the actual resection, is restored by a paratumoral biliodigestive connection using a dilated intrahepatic duct for the crucial anastomosis. This enables the liver to recover and to subside which makes the hilus better accessible (48). If the communication between the left and the right hemi-livers is blocked, this unilateral biliary drainage at the same time provokes a compensatory hypertrophy of the liver part to be maintained. In a second session resection of the tumour and the adjacent part of the liver is carried out, leaving the hypertrophied liver part with the crucial biliodigestive anastomosis which has already been made (473).

The disadvantages of a staged procedure are the inevitable formation of adhesions which impede a later resection and the meanwhile continuing tumour growth. Consequently if feasible, the complete operation should be performed in one session (484). A preceding biliary decompression without operation can be obtained by percutaneous transhepatic drainage (392, 423).

Para- and transtumoral bile drainages - If resection is impossible and palliation is considered to be the treatment of choice, the quality of the remaining life is even more important than the duration. In that case postoperative treatment or control has to be avoided as much as possible (57). A comparison between a paratumoral biliodigestive bypass and a transtumoral drainage encounters the doubtful matching of the treated patients. If a good biliary bypass can be constructed this may offer the best possible and reliable palliation in

one session. The construction of an intrahepatic bilio-enteric anastomosis is undeniably a more extensive, longer and more difficult operation than the insertion of a transtumoral tube. A paratumoral bypass has a higher operative mortality and the postoperative survival is about the same after both procedures. Therefore transtumoral intubation has strong advocates (128, 547) as well as opponents (243, 339). In transtumoral drainage the tumour is stretched and torn (516) but no reports could be found indicating an increased tumour growth or dissemination. Transtumoral drains may be blocked or may dislocate, external extensions of the drains may cause local and biliary infection as well as psychological problems (Chapter 2.4.3; Table XVIII).

Application of either method where indicated is most appropriate (285, 286). A paratumoral bypass is especially indicated in a young patient in a good general condition with dilated intrahepatic ducts and a tumour too large to resect. Contra-indications against a formal intrahepatic bilio-enteric anastomosis are: undilated ducts, atrophy of the liver segments concerned, tumour involvement of the secondary confluences of the bile ducts concerned, portal hypertension, ascites, liver or other metastases and a poor general condition (50, 285, 286). If transtumoral drainage is chosen, then a sunken drain (285) or a transhepatic drain with a subcutaneously placed end (65) are to be preferred from a palliative point of view. U-tubes which leave the abdomen may also give satisfactory palliation (545).

The aim of a palliative procedure is a good bile drainage of as much liver tissue as possible for as long as possible. An adequate bile drainage of 30% of the liver is enough to clear obstructive jaundice (55, 285, 315, 371, 436). If the communication between the left and right bile duct system is blocked by the tumour, then restoration of the bile drainage on one side is sufficient to make the jaundice disappear (48, 147, 285, 309, 359, 371, 396, 457, 495, 500, 504, 545, 556, 572). An end-to-side cholangiojejunostomy with a dilated bile duct of segment III as described by Soupault and Couinaud (521) is a suitable method (48, 258).

Theoretically the right hemi-liver should be preferred for a unilateral drainage as the right hemi-liver is larger than the left one (16, 111) but a good right-sided cholangiojejunostomy is much more difficult to construct than a left one (48, 50). In this respect transtumoral drainage is suitable, the more so since the right hepatic duct is approximately in line with the common hepatic duct. The right hepatic duct measures 1-2 cm and a tumour of the hepatic duct confluence may quickly reach the secondary confluence of the right lateral and right paramedian duct. Therefore unilateral right-sided drainage carries the risk of soon being confined to drainage of only half of the right lobe by progress of the tumour.

The main disadvantage of unilateral drainage is the risk of infection and abscess formation in the undrained liver segments. Purulent cholangitis and liver abscesses are likely to develop in an obstructed bile duct system contaminated by

bacterias during surgical manipulation or percutaneous punction (124, 277, 359, 438, 573, 609). Unless it is expected that an adequate bile drainage can be achieved, it may be better for a completely blocked but not infected part of the liver to be left alone and to atrophy, provided a sufficient mass of the remaining liver is well drained and functions well (128, 277, 359).

For these reasons many authors prefer a bilateral bilio-enteric reconstruction (107, 124, 137, 285, 328, 330, 430,438, 457, 460, 480, 484, 500, 503, 572). Exact information about the site and extent of the tumour and about the complete biliary anatomy is required to be able to achieve an optimal bile drainage.

If after dissection of a tumour, up to that moment thought to be completely resectable, it becomes obvious that only incomplete resection is possible, this should be carried out and followed by a bilateral hepaticojejunostomy (63). If after exploration even incomplete resection appears impossible, a paratumoral bilio-enteric bypass is the best solution, if possible bilateral but otherwise in combination with a transtumoral drainage of the contralateral side (137, 489). If it is clear immediately after opening of the abdomen that resection is impossible or useless, one has to choose between paratumoral and transtumoral drainage in agreement with the local situation and the life expectancy of the patient (137, 286, 489). Only in case the possibility of a resection has been ruled out and the risk of a palliative operation is too high, a percutaneous percutaneous transhepatic transtumoral drainage is indicated as main treatment. It offers an elegant but usually brief palliation. Combined with local radiotherapy by temporary insertion of radioactive material a period of survival comparable to that of operative drainage procedures may even be achieved (135, 188).

Intrahepatic bilio-enteric anastomosis

The reconstruction of a good bilio-enteric biliary drainage is the crucial part of the operation and often also its most difficult part. A careful apposition of the bile duct mucosa and the intestinal mucosa is thought to be essential (39, 50, 63, 106, 147, 243, 256, 258, 330, 353, 371, 457, 484, 498, 514, 600). Use of a healthy, well-vascularised bile duct without any tension on the sutures and a scrupulous technique are imperative. At the enteral side a 60 cm long Roux-Y jejunal loop offers the best possibilities (8, 39, 52, 66, 256, 266, 328, 330, 458, 514, 600, 606). Dissection has to be confined to the minimum in order to prevent an impaired vascularization of the bile duct wall (243, 303). The anastomosis should be as wide as possible and should preferably be contructed laterally on the bile duct (256, 486, 498).

A carefully sutured anastomosis with interrupted stitches provides the best anastomosis. It is necessary to place all or part of the stitches at first and only then to tie them according to a strict schedule in order to keep a close view on the anastomosis during reconstruction (457, 498, 588).

If a regular anastomosis is impossible,the mucosal graft technique may offer a solution. In this technique a funnel of dissected jejunal mucosa from an area near the top of a jejunal loop, is tied to a transhepatic drain and blindly drawn into the bile duct together with the drain (150, 515). The fixation and the width of the resulting bilio-enteric connection are inferior to a sutured anastomosis and the intestinal mucosa drawn into the bile duct may block biliary side branches (57, 66).

Although hepato-enterostomy does not produce intermucosal contact it may give good results as well, provided that it is performed on a level that has healthy and wide bile ducts.It is a relatively simple procedure and the desirable intermucosal contact still develops since the intestinal mucosa overgrows the cut surface of the liver and joins the biliary mucosa (124, 207, 243, 303, 323, 325, 328, 385, 386, 438, 539, 572).

In case the hepatic ducts are cut beyond their secondary confluences, several duct orifices stay behind to be dealt with. Mutual connection of these ducts may provide one or two large biliary channels suitable for a direct anastomosis with a jejunal loop (8). Theoretically, postoperative regeneration of parenchyma between the various ducts may push the ducts aside and disrupt the anastomoses. Therefore a hepato-enterostomy may be preferred if duct orifices are separated from each other by intervening liver tissue.

2.3. Preparations for treatment

Pre-operative assessment

Percutaneous transhepatic cholangiography (PTC) is the favourate investigation after an ultrasound examination has suggested a proximal biliary obstruction. PTC provides accurate information about place and upstream expansion of the tumour and about the biliary anatomy, the bile duct dilatation and atrophy or hypertrophy of liver segments. During investigation bile can be collected for cytological and bacterial examinations. If required, temporary or permanent external bile drainage can be added. A complete, good quality cholangiogram is essential as both choice and performance of the operation depend on it.

In the differential diagnosis all other obstructing processes either inside or outside the bile duct have to be considered but all may need laparotomy for both diagnosis and treatment. Caroli (111) stated that a primary total block of the hepatic duct confluence is always caused by a malignant tumour and that a partial block at the hilar level is nearly always caused by a malignant tumour. In four out of 75 consecutive patients operated by Blumgart (68) for a typical tumour of the hepatic duct confluence, no malignant cells could be found.

Unless a resection is already abandoned, the pre-operative investigation at this stage is directed towards the assessment of the downstream and periductal expansion of the tumour and the existence of metastases. Endoscopic retrograde cholangiography (ERC) (62, 63), CT scan (76, 223, 289, 439) and angio-

graphy (72, 260, 270, 463, 537, 616) are performed and laparoscopy may provide additional information (8, 64). The vascular anatomy of the liver and the hepatoduodenal ligament is defined and the presence of segmental atrophy or hypertrophy assessed. Since the portal vein bifurcation is situated close to the hepatic duct confluence, the possibility of tumour involvement of the vein has to be examined carefully. The venous phase of a coeliac arteriogram can be sufficient, but a direct splenoportogram (616) or percutaneous transhepatic portogram (270, 537) may be necessary. Angiographically demonstrated involvement of the portal vein by the tumour or by fibrotic reaction surrounding the tumour is always confirmed at operation (270, 616), but a negative angiogram does not exclude vascular impairment (270, 330).

If possible, tumour tissue should be obtained prior to operation. Operative dissection of the fibrotic hilus of the enlarged, firm liver is difficult and risky (277, 473). Biopsies often turn out to contain fibrotic tissue only (609). Histological diagnosis from a biopsy can be notoriously difficult (13, 88, 208, 246, 277, 430, 477, 485, 553), especially if it has to be done by frozen section examination. Besides an open biopsy is undesirable from an oncological point of view. Aspiration of bile rarely yields tumour cells (609) but percutaneous transhepatic brush-scraping is described to be successful in up to 42% of the patients (131, 173, 177, 178, 393).

Pre-operative measures

Every jaundiced patient requires immediate medical attention and the bad general condition of the patient after a long-standing bile duct obstruction should be prevented. If the general condition and the liver function are poor - often because a previous operation failed - they must where possible be improved pre-operatively. This can be done during the pre-operative investigations. The common postoperative problems that have to be anticipated are septicaemia, hypalbuminaemia, coagulation disorders and renal insufficiency. An operative field disturbed by previous operations and an age over 60 years are unfavourable factors (38).

Rehydration of the patient and correction of fluid and electrolyte balance are essential. Anaemia, hypalbuminaemia, coagulation and nutrition disorders have to be corrected, if necessary by parenteral nutrition (36, 64, 450). Antibiotics are necessary to treat septic bile ducts but success may only be expected in a well-drained biliary tree.

Pre-operative percutaneous transhepatic biliary drainage (PTBD) may be required (42). Habitual use of pre-operative PTBD in patients with obstructive jaundice, however, seems unjustified (109, 423, 491). The benefits of pre-operative PTBD consist of an improved condition of the patient and his liver, a gain of time for diagnostic assessment and a possibility of repeated brush biopsies. Disadvantages are the complications of drain insertion (bile leakage, haemorrhage, haemobilia, pneumothorax, dislocation or breaking of the drain) in about 5 to

10% of the patients (156, 161, 169, 482, 575), the risk of conveyance of tumour cells (429), the shrinkage of the dilated bile ducts (55, 587, 597) and in particular the risk of contamination of a blocked but previously sterile biliary system (61, 239, 277, 392, 574).

The operative mortality of extensive pancreatobiliary operations in icteric patients does not need to be high and additional pre-operative PTBD carries the considerable risk of the introduction of infection (427). Right hepatic lobectomy has been successfully performed in jaundiced patients (432, 573, 574) and the operative mortality in hepatic surgery can be related to the septic complications of pre-operative PTBD (66). If the operative risk is increased by an already existing obstructive cholangitis or liver insufficiency, then pre-operative PTBD can be of value (61, 63, 161, 392, 450, 572).

Peroperative assessment

A good peroperative assessment, especially regarding the upstream expansion (5) and the vascular tumour involvement, is essential for the operation to be carried out. However, lymphatic and peritoneal metastases as well as small liver metastases elude pre-operative discovery; the invasion of the tumour into the caudate and quadrate lobes is also difficult to demonstrate. Pre-operative assessment of resectability of liver tumours appears at laparotomy to be wrong in 25% of the cases (307). Therefore the definitive establishment of surgical possibilities and resectability is only possible by the combination of röntgenological information and surgical exploration (9, 60, 66, 243,330, 357, 394, 420, 430, 433, 484, 489). If during pre-operative examination no contra-indication has been demonstrated against resection, surgical exploration and dissection is indicated. The only possibility to determine the resectability is to go for the tumour with the intention to remove it (489). Even then it may turn out only at the end of the dissection when the rear side of the tumour has been reached, that a potentially curative resection is impossible (489). In that case palliative resection still is the best solution and makes the dissection not futile.

Unless the pre-operative cholangiogram provides all biliary information desired, a peroperative cholangiogram by transhepatic punction is indicated (8, 39, 277, 286, 357, 394, 474, 477). If a choledochoscope can be placed in the collapsed, narrow main bile duct the tumour can be inspected and biopsied (609). Multicentric occurrence of the tumour can be examined in the same way (548, 562, 563). As long as a paratumoral bypass is one of the possibilities, the gallbladder should not be removed as it can serve this purpose (606).

An extensive mobilisation of the duodenum and head of the pancreas is required to assess the extension and possible dissemination of the tumour. This is followed by a careful inspection and palpation of the liver, hepatoduodenal ligament, gallbladder and duodenopancreatic area. Tumour involvement of the portal vein can be judged by opening the peritoneum of the

right free margin of the hepatoduodenal ligament and to dissect the portal vein from the bile duct towards the liver (5). As the common bile duct does no longer function it can be divided distally and dissected upwards from the underlying portal vein towards the liver. This is a simpler procedure and provides a better exposure (66, 294, 330, 513).

Histological confirmation of the tumour is necessary, before proceeding to the actual resection (178) as in case of a benign bile duct obstruction a limited resection or adequate bilioenteric bypass is sufficient. If at that time no representative tumour tissue has been obtained this has to be done by a direct approach to the tumour. Puncture may yield appropriate tissue but often the only way for a proper biopsy is a local resection of the tumour (66).

The hilus of the liver and the hepatic duct confluence can be explored by 1 lifting of the hilar plate, 2 liver split along the principle scissure, and 3 resection of the quadrate lobe.

- Lifting of the hilar plate (Hepp and Couinaud, 1956 (254)) (décollement de la plaque hilaire (119, 121, 142, 144, 257)).

The hilar plate is transversally incised at its transition to the capsule of the quadrate lobe and dissected in caudal direction from the hepatic duct confluence and left hepatic duct. This is the essential manoeuvre in the treatment of benign strictures of the confluence (257). The large, firm liver and the fibrosis around the tumour render this approach less suitable for a hilar bile duct cancer.

- Liver split along the principal scissure (Champeau and Pineau, 1955 (117, 215, 544, 590).

Transection of the liver along the portal avascular plane between the left and right hemi-liver which runs from the gallbladder fossa to the left side of the inferior caval vein, provides a wide exposure of the right side of the hilus including the confluence of the hepatic ducts. Also the upstream extension of the tumour can be well assessed by this approach (39, 63, 64, 100, 486, 488).

- Mobilisation or resection of the quadrate lobe (Champeau, 1967 (120, 122)).

The left corner of the hilus is exposed by transection of the bridge of liver tissue which connects the segments III and IV in front of the recessus of Rex (118). Then the quadrate lobe is mobilised or resected by incising the liver in the umbilical and principle scissures and to connect these two incisions by a transverse incision cranially through the parenchyma of segment IV. Now the complete hilus is completely exposed (8, 9, 39, 49, 66, 330, 489). After exposure of the tumour area both distally and proximally, the nature, extent and resectability of the tumour can be determined.

2.4. Therapy

2.4.1. Resection

Extent of the resection

To achieve a curative resection of a tumour without demonstrable metastases the areas of potential invasion and initial dissemination have to be removed en bloc with the tumour. The presence of the portal vein and the hepatic artery in the liver hilus so far rules out a radical block resection of a cancer of the hepatic duct confluence (5, 374, 599). Leaving these vessels out of consideration a resection which intends to be as curative as possible has to include the following areas of potential invasion: 1 the biliary tract downstream of the tumour; 2 the biliary tract upstream of the tumour; 3 the adjacent liver tissue (segments IV and I); and 4 the lymphatic tissue in the hepatoduodenal ligament.

1 the biliary tract downstream of the tumour

Considering the characteristics of the tumour it is preferable always to remove the gallbladder (39, 128, 330, 557) and to transect the common bile duct as distally as possible at the margin of the duodenum (8, 39, 51, 66, 330, 538). Frozen section examination of the transected duct is essential (330) but not completely reliable because of its difficult interpretation (572).

2 the biliary tract upstream of the tumour

From a curative point of view it is preferable to keep away from the tumour as far as possible. This means that the bile ducts, especially on the right side, have to be transected beyond the secondary confluences (39, 243, 294, 489). If the tumour extends far in one of the hepatic ducts or affects a secondary confluence, a hemihepatectomy at the side involved is necessary (9, 294, 572). Also a unilateral periductal invasion of one of the main branches of the portal vein or hepatic artery requires a resection of the hemi-liver concerned (330), as does a solitary metastasis (458).

3 the adjacent liver tissue, consisting in the quadrate lobe (9, 39, 243, 330) and the caudate lobe (410).

4 the lymphatic tissue in the hepatoduodenal ligament

Removal as complete as possible of all the regional lymphatic nodes and vessels in the hepatoduodenal ligament and of the posterior duodenopancreatic nodes (480) has to be added to the resection, leaving only the bare portal vein and hepatic artery (curage du pédicule hépatique) (39, 58, 330, 480, 458, 489).

To cope with these four requirements the resection theoretically has to be as extensive as possible. A wide resection of the bile ducts requires a concomitant partial hepatectomy and a hepatico-enterostomy for biliary reconstruction. The addition of a liver resection may increase the operative mortality of tumour resection (66, 168, 243, 330). Therefore many types of resection are practiced, varying from local resection of the hepatic duct confluence with its tumour to extended liver resections and vascular reconstructions.

The various liver resections eligible for the treatment of a tumour of the hepatic duct confluence are described in detail in the manuals on liver surgery (49, 56, 121, 142, 206, 565, 582). Seen from the principal scissure the resection may be extended to the right as well as to the left according to the portal or suprahepatic intersegmental planes. Wedge resections disregarding the intrahepatic anatomy, are objectionable because of the risk of bile leakage, necrosis and abscess formation (430). Resection of the pre-hilar quadrate lobe is the smallest hepatectomy practiced concomitantly with excision of a confluence tumour. Besides, left and right hemihepatectomy and right lobectomy are the most commonly applied liver resections to remove a confluence tumour.

Resectability

The current local criteria of potentially curative resectability are (294):

- main stems of portal vein and hepatic artery free of tumour
- main branches of portal vein and hepatic artery contralateral to a tumour extension in one of the hepatic ducts free of tumour
- distal extension of the tumour leaves a long part of the distal common bile duct free of tumour
- proximal extension of the tumour respects at least unilaterally the secondary duct confluences

The tumour is generally considered as not curatively resectable in the following situations:

- invasion of tumour of the main stem of the portal vein or hepatic artery (7, 178, 294, 330, 354, 489)
- invasion by tumour of the main brain branches of the portal vein or hepatic artery bilaterally or contralaterally to a tumour extension in one of the hepatic ducts (66)
- bilateral invasion of the secondary confluences of the hepatic ducts (137, 289, 572)
- liver metastases (137)(ipsilateral or solitary liver metastases may be resectable) (458)
- metastases at distance (7, 137, 178), poor general condition or short life expectancy of the patient

The concept of resectability, expressed as a percentage of all possible treatments performed, only makes sense of it relates to surgeons who have the preconceived intention and are able to resect the tumour if possible as the best way of treatment. The word 'resectability' has no meaning if it merely indicates the amount of resections performed, however incidentally these may have been. The resectability rate of the combined series of 13 surgeons who strive to resect cancers of the hepatic duct confluence is 33% (15-61%) (Table VI). These series include resections postoperatively considered as curative as well as incomplete resections since both types can seldom be distinguished from in each other in the reports (74).

Table VI. Resectability of tumours of the hepatic duct confluence.

Authors	patients	treated by resection	
Balasegaram (25)	39	15	
Benjamin (37)/Blumgart (68)	78	17	
Cameron et al. (109)	36	10	
Chitwood et al. (131)	24	5	
Evander (178)	34	16	
Iwasaki et al. (294)	23	9	
Launois et al. (330)	18	11	
Skoog and Thorén (512,558)	17	8	
Smith (517)	33	5	
Starzl (529)	16	8	
Tompkins et al. (564)	47	22	
Tsuchiya (570)	31	9	
Warren et al. (596)	35	9	
Total no. of patients	431	144	(33%)

Results and types of resections

250 Patients were found in whom a tumour of the hepatic duct confluence was resected. The results of these resections are listed in Table VII. The mean postoperative mortality is 14.4% and the mean postoperative survival 19.5 months. Many of the operations presented concern incidental reports of a successful operation. The results in series of at least three or at least five resections, have a striking resemblance to the results of all the resections together. The morbidity after resection consists of bile leakage, bile fistulae, cholangitis, haemorrhage, stress ulcers and subphrenic abscesses. The complication incidence is poorly reported but may be estimated together at 25 to 50%.

Table VII. Results of the total series of resection of primary tumours of the hepatic duct confluence (Appendix I gives the results of these series in full).

	patients	operative mortality	mean survival (months)	alive after (yrs) (%)				
				½	1	1½	2	3
total all series	250	36 (14.4%)	19.5	91	69	43	33	21
total series of at least 3 patients	201	27 (13.4%)	18.9	92	67	43	37	22
total series of at least 5 patients	159	21 (13.2%)	18.3	87	67	41	36	22

The various types of resections can be divided according to the concomitant liver resection into 1 resection confined to the hepatic duct confluence (or combined with cholecystectomy); 2 resection of the hepatic duct confluence and the quadrate lobe; 3 resection of the hepatic duct confluence and left hemihepatectomy; 4 resection of the hepatic duct confluence and right hemipatectomy; 5 resection of the hepatic duct confluence and right lobectomy; and 6 resection of the hepatic duct confluence with major liver resection and resection of the portal vein or the hepatic artery.

1 resection confined to the hepatic duct confluence (or combined with cholecystectomy)

The smallest form of resection is a curettage of a stalked papillary tumour (115). Local excision limited to the confluence itself is the most frequently performed resection. This resection was originally used to excise a tumour of the common hepatic duct (426) and was applied to a confluence tumour only in 1954 by Brown and Myers (87) Although this operation may be the least burdening for the patient, its radicality has to be doubted in view of the tumour characteristics.

The reconstruction of the bile drainage to the intestines after a local resection of the hepatic duct confluence can be restored in the following ways:

- hepatico-enterostomy with a Roux-Y jejunal limb; hepaticoduodenostomy is also possible (208, 545, 589);
- end-to-end anastomosis between the remaining parts of the bile ducts (315, 384, 596):
  - connection of the left and right hepatic duct to one united orifice, which is end-to-end anastomosed with the common hepatic duct
  - end-to-side anastomosis of one hepatic duct with the other, followed by end-to-end anastomosis of the last duct whith the common hepatic duct (384)
  - end-to-end anastomosis between the left hepatic duct and the common hepatic duct and between the right hepatic duct and the spared cystic duct (385, 446)
  - ligation of one of the hepatic ducts and end-to-end anastomosis of the other with the common hepatic duct (315, 571), if required combined with percutaneous drainage of the blocked part (270)
  - interposition of the gallbladder (208, 255, 606);
- hepato-enterostomy if no bile ducts of a size sufficient for an anastomosis are available (110, 273, 303).

The results of 75 tumour resections confined to the hepatic duct confluence are listed in Table VIII.

The mean postoperative mortality is 11% and the mean postoperative survival 19 months. Where reported, the cause of death of the patients who survived the operation was mainly due to a local recurrence of the tumour (39, 195, 208, 270, 273, 315, 569).

Table VIII. Results of local confluence resection for primary tumour of the hepatic duct confluence.

Authors	patients	operative mortality	mean survival (months)
Bérard et al. (39)	1	0	10
Brown and Myers (87)	2	1	8
Cameron et al. (109)	10	0	21
Camprodon et al. (110)	1	0	16
Chitwood et al. (131)	5	0	17
Crismer et al. (145)	1	1	
Finney (184)	1	0	16
Fortner et al. (195)	1	0	17
George et al. (208)	3	0	12
Heully et al. (263)	1	0	?
Hoevels and Ihse (270)	1	0	7
Hollender et al. (273)	1	0	11
Karakousis and Douglass (303)	2	0	15
Kozaka et al. (315)	2	0	20
Launois et al. (330, 333)	5	1	33
McDermott and Peinert (384)	4	0	10
Nishimura et al. (420)	1	0	?
Philippakis et al. (446)	1	0	14
Quattlebaum (458)	1	0	12
Rodgers et al. (471)	1	0	28
Salembier (489)	4	1	20
Saubier et al. (491)	3	0	?
Skoog and Thorén (512,558)	4	1	8
Smith (513)	3	0	31
Terblanche et al. (545)	1	0	?
Tsuchiya (570)	4	1	8
Van Vroonhoven and Malt(589)	1	1	
Warren et al. (596)	7	0	27
Whelton et al. (606)	2	1	20
White (609)	1	0	23
Total no. of patients	75	8 (11%)	19

2 resection of the hepatic duct confluence and the quadrate lobe

This operation is considered to be the ideal resection of a confluence tumour by many authors (9, 32, 39, 243, 330, 361, 420, 531). An intestinal loop is required for the biliary reconstruction and is anastomosed with both hepatic duct remnants separately. If the secondary duct confluences are resected, the remaining duct remnants can be united on each side to form combined ducts (8, 39) or have to be anastomosed separately with the intestinal loop. Hepatojejunostomy is also reported to function properly (386). The results of 12 such operations are listed in Table IX. Local recurrence of the tumour is the usual cause of death in these patients too.

Table IX. Results of resection of the hepatic duct confluence and the quadrate lobe for primary tumours of the hepatic duct confluence.

Authors	patients	operative mortality	mean survival (months)
Alexandre et al. (8,9)	1	0	25
Bérard et al. (39)	2	0	18
Bismuth et al. (56)	1	0	78[a]
Hart and White (243)	3	0	11
McDermott (386)	1	0	36
Nishimura et al. (420)	1	0	?
Stephen (531)	2	1	26
Total no. of patients	12	1 (8%)	20

[a] not included in mean survival

3 resection of the hepatic duct confluence and left hemihepatectomy

By a hemihepatectomy the resection of the proximal bile ducts is enlarged maximally at one side and a large area of potential hepatic dessemination is also removed. Moreover it makes the operative view ideal (7). The bilio-enteric reconstruction is performed mostly by a hepaticojejunostomy with a Roux-Y loop. Good results are also described of periductal hepatojejunostomy (7, 304, 572). The results of 41 resections are listed in Table X. The operative mortality is 7% and the mean survival 21 months. Again the usual cause of death in patients who survived the operation is local tumour recurrence.

4 resection of the hepatic duct confluence and right hemihepatectomy

Nine patients who underwent this operation could be traced, two without any further information(235, 310). Two patients died in the postoperative period, at least one from liver insufficiency (432, 458). Postoperative survival is reported in three of the five remaining patients, being seven months in two patients (300, 489) and more than four years in the last one (270).

5 resection of the hepatic duct confluence and right lobectomy

The results of this operation in 15 patients are listed in Table XI. The postoperative mortality was caused by liver insufficiency in three patients (330, 432, 476), by portal vein thrombosis in one (435) and by uncontrolable bleeding from a stress ulcer in one (476). In one of the survivors the postoperative course was laborious because of marginal liver function (330). The problem of the small liver remnant may be obviated by constructing - in a separate operation before the actual resection - a cholangiojejunostomy with the liver part to remain after the resection, as described on page 19 (473). Also in this group of patients the reported cause of death was due to tumour recurrence (435, 475).

Table X. Results of resection of the hepatic duct confluence and left hemihepatectomy for primary tumour of the hepatic duct confluence.

Authors	patients	operative mortality	mean survival (months)
Akwari and Kelly (7)	4	0	33
Alexandre et al. (8,9)	2	0	8
Bérard et al. (39)	1	0	7
Engler (175)	1	0	10
Fortner et al. (195)	2	0	21
Haynes et al. (246)	1	0	9
Hoevels and Ihse (270)	1	1	
Hollender et al. (273)	1	0	4
Kelly (304)	1	0	19
Launois et al. (330, 333)	3	0	28
Longmire et al. (357)	1	1	
Mann (373)	1	0	13
McDermott and Peinert (384)	3	0	?
Mistilis and Altemeier (402,14)	1	0	30
Nicoli et al. (418)	1	0	15
Nishimura et al. (420)	1	0	14
Quattlebaum (458)	1	0	12
Skoog and Thorén (512, 558)	4	1	38
Tsuchiya (570)	1	0	13
Tsuzuki and Uekusa (572)	?		18 (2 patients)
Tsuzuki et al. (574)	8	0	?
Warren et al. (596)	2	0	±24
Total no. of patients	41	3 (7%)	21

6 resection of the hepatic duct confluence with major liver resection and resection of the portal vein or the hepatic artery

Seven patients whose confluence tumour was treated in this way are to be discussed in Chapter 4. Three patients died in the first postoperative month. The mean postoperative survival of the other patients is 15 months (range 12-18 months). Three of the four survivors died from local recurrence (361, 574) (Chapter 4; Table XXX).

Table XI. Results of resection of the hepatic duct confluence and right lobectomy for primary tumour of the hepatic duct confluence.

Authors	patients	operative mortality	mean survival (months)
Bird et al. (46)	1	0	12
Blumgart et al. (63, 64) [a]	1	0	12
Launois et al. (330)	2	1	8
Nicoli et al. (418)	1	0	3
Ong and Lee (432)	1	1	
den Otter (435)	3	1	13
Rohner (476)	4	2	36
Tsuzuki (574)	2	0	?
Total no. of patients	15	5 (33%)	17

[a] extended left hepatectomy

2.4.2. Paratumoral bilio-enteric bypass

Intrahepatic bilio-enteric diversion

An intrahepatic bilio-enteric anastomosis is best situated as far from the liver hilus as possible to prevent future involvement by the tumour. Moreover a hazardous and protracted exploration of the fibrous hilus is avoided, especially if this operative field is already scarred by previous operations. To find a bile duct as wide as possible, however, one prefers to keep somewhat near the hilus (467, 498). Both requirements are best met by the well-described use of the dilated duct of segment III according to Soupault and Couinaud (521). Various other possibilities of intrahepatic bile diversions have also been developed mainly by Franch surgeons, and are therefore especially practiced in France. A Roux-Y or omega jejunal loop is mostly used as enteric part of the anastomosis. The stomach may also serve this goal (133, 158, 438) and interposition of an isolated jejunal segment (318), dacron velours prosthesis (434) or silicone prosthesis (318) has also been described.

The hepatic bile ducts can be approached either unlaterally or bilaterally in the hilus or in the periphery of the liver (48, 50, 121, 142, 186, 216, 273, 322, 394, 430, 480):

1 Hepatic ducts in the hilus, if necessary after transection of the parenchymatous bridge between the segments III and IV (118), mobilisation (120) or resection (121) of segment IV or after opening of the principal scissure (117).

- left hepatic duct (Hepp and Coyinaud, 1956)(254)
  The left hepatic duct always runs extrahepatically (57) and so strictly speaking does not belong to the intrahepatic biliary system. Opening and lowering of the hilar plate reveals the duct.
- right hepatic duct
  In addition to the direct anastomosis between the right hepatic duct and a Roux-Y loop (349) there is the possibility of hepaticocholecystoduodenostomy: the base of the

gallbladder is anastomosed with the right hepatic duct and the gallbladder fundus with the duodenum (116, 484).

2 (A) Ducts in the periphery (left side)
- peripheral bile ducts of the segments II and III after partial excision of the left lobe
- anastomosis with jejunal loop (Longmire and Sanford, 1948) (139, 352)
- anastomosis with stomach (Dogliotti and Fogliati, 1956) (158);
- bile duct of segment III
- after partial excision of segment III (143, 253, 438)
- after hepatotomy to the left of the ligamentum teres insertion (Duboucher, 1954; Soupault and Couinaud, 1957) (164, 520, 521)
- after punction cholangiography and resection of a wedge of liver tissue up to the demonstrated bile duct, guided by the punction needle which is left in place (253, 460, 498, 503);
- subcapsular dilated bile ducts (101, 490)

2 (B) Ducts in the periphery (right side)
- peripheral ducts of segments V and VI after resection of the anterior edge of these segments (258, 438)
- duct of segment V
After resection of segment IV followed by a transverse hepatotomy into the left side of segment V (Prioton et al. 1968) (457)(48, 50, 69)
- duct of segment VI after transection of the liver along the right portal scissure between segments V and VI (Salembier, 1982) (489)
- ducts of segment V after transection or punction of the gallbladder bed (216, 230, 317, 322, 490, 513, 595, 618)
- peripheral duct dissected after cholangiographically guided wedge excision (253, 460, 498, 503)
- subcapsular dilated ducts (101, 490).

Several biliary diversions particularly on the left lobe are standardised. Most authors consider the use of the segmental duct III after Soupault and Couinaud (521) as the best, the safest and most efficient intrahepatic biliary diversion (43, 48, 128, 143, 164, 167, 207, 216, 233, 258, 266, 277, 328, 330, 371, 394, 409, 484, 489). Between this duct and the anterior liver surface there are no large calibre vessels. Needle aspiration, if required with cholangiography, may be useful to identify the duct.

Through a large hepatectomy 1 cm to the left of the ligamentum teres the duct is found, cranio-anterior to the accompanying portal vein branch of segment III, and anastomosed with a Roux-Y loop (521), jejunal omega loop (164), stomach (133) or gallbladder fundus (148). Transection of the accompanying portal vein branch is originally described (147, 521), but is not imperative (216). In strongly enlarged livers the hepatotomy can be performed rather more to the left (258, 457, 504).

Complete (141) or partial (253, 255) resection of segment

III provides the same bile duct but terminally transected. After subtotal (158) or partial (350, 352, 353) resection of the left lobe both segmental ducts II and III can be used.

Results

The results of paratumoral bypass operations in 400 patients with a tumour of the hepatic duct confluence are listed in Table XII. The operative mortality is 23.8% and the mean post-operative survival 11.2 months. Just like the results of the resections, there appears to be a great similarity between the results of large and small series. This group must be even more heterogeneous than the resection group. As after resection the postoperative morbidity consists of bile leakage and fistulas, cholangitis, haemorrhage, subphrenic abscesses and gastrointestinal ulcers. Probably these complications are more frequent after bypass operations than after resections. However, the incidence of these complications is seldom accurately reported.

Table XII. Results of the total series of paratumoral bilio-enteric bypass for primary tumour of the hepatic duct confluence (Appendix II gives the results of these series in full).

	patients	operative mortality	mean survival (months)	alive after (yrs) (%) ½	1	1½	2	3
total all series	400	95 (23.8%)	11.2	60	37	19	8	1
total series of at least 3 patients	382	93 (24.3%)	11.1					
total series of at least 5 patients	349	88 (25.2%)	11.1	65	38	22	12	2
total series of at least 10 patients	306	75 (24.5%)	11.2	65	37	24	11	2

Table XIII. Results of paratumoral bilio-enteric bypass on hilar level for primary tumour of the hepatic duct confluence.

Authors	patients	operative mortality	mean survival (months)
Bertrand et al. (43)	2	1	24
Levasseur et al. (349)	1	0	15
Longmire et al. (357, 359)	2	0	12
Mercadier et al. (394)	5	2	11
Salembier (489)	2	2	
Total no. of patients	12	6 (50%)	16

The results of 12 patients with a hilar paratumoral diversion on the left or right hepatic duct just beside the tumour are listed in Table XIII, while Table XIV shows the results of bilateral diversion in 10 patients. The results of 89 diversions after Soupault and Couinaud (521), 42 Dogliotti (158) and Longmire (352) procedures, and 18 peripheral diversions on the right hemi-liver are given in Tables XV, XVI and XVII respectively.

When comparing the results of the various operations, the bilateral and hilar diversions seem to be somewhat better than the results of the other types although the groups are too small and the patients too heterogeneous for significant conclusions.

Table XIV. Results of bilateral paratumoral bilio-enteric bypass for primary tumour of the hepatic duct confluence.

Authors	patients	operative mortality	mean survival (months)
Bertrand et al.(43)	2	0	18
Dalmas et al. (148)	2	0	12
Prioton et al. (457)	3	0	19
Ragins et al. (460)	1	0	24
Schutt (500)	1	0	26
Seigert et al.(503)	1	0	20
Total no. of patients	10	0	19

Table XV. Results of paratumoral bilio-enteric bypass using cholangio-jejunostomy on segmental duct III after Soupault and Couinaud for primary tumour of the hepatic duct confluence.

Authors		patients	operative mortality		mean survival (months)
Arianoff et al. (19)		2	1		6
Chigot et al. (128	(a)	20	7		9
Corlette and Bookwalter (137)	(b)	1	0		18
Couinaud (143)		8	4		5
Gautier et al. (207)		1	0		5
Huguet and Salem (277)	(c)	16	1		?
Huguet et al. (286)	(c)	13	4		17
Launois et al. (330)		13	4		6
Malt et al. (371)		4	0		12
Mercadier et al. (394)		7	3		19
Mourgue Molines et al. (407)		2	1		16
Salembier (489)		2	0		4
Total no. of patients		89	25	(28%)	10

(a) operative mortality as death in first two operative months

(b) combined with transtumoral drain in right hepatic duct and 4500 rad irrad.

(c) these series may have mutual overlap

Table XVI. Results of peripheral paratumoral hepato- or cholangio-enterostomy on the left liver lobe for primary tumour of the hepatic duct confluence.

Type of operation	authors	patients	operative mortality	mean survival (months)
Dogliotti	Hepp et al.(253)	2	0	9
	Salembier (489)	6	2	6
	subtotal	8	2 (25%)	7
Longmire or resection of segment III	Alvarez (16)	1	1	
	Arianoff et al.(19)	4	3	4
	Hepp et al. (253)	1	0	10
	Launois et al. (330)	1	1	
	Longmire (355, 357)	7	2	8 [a]
	Périssat et al.(443)	8	4	7
	Ragins et al. (460)	3	1	9
	Salembier (489)	5	1	3
	Sullivan et al.(539)	1	0	29
	White (609)	3	0	16
	subtotal	34	13 (38%)	9
	Total no. of patients	42	15 (36%)	9

[a] 1 patient excluded in mean survival (exceeds 4½ years)

Table XVII. Results of peripheral paratumoral bilio-enteric bypass on the right hemi-liver for primary tumour of the hepatic duct confluence.

Type of operation	authors	patients	operative mortality	mean survival (months)
cholangio-jejunostomy	Arianoff et al.(19)	2	0	30
	Bodner (69)	4	1	8
	Crismer et al. (145)	1	0	12
	Osborne et al. (434)	1	0	15
	Ragins et al. (460)	1	0	15
	Salembier (489)	1	1	
	Schamaun (498)	1	0	5
	subtotal	11	2	13
hepato-jejunostomy	Alvarez (16)	1	0	18
	Longmire et al.(357)	2	0	3
	Périssat et al.(443)	2	0	4
	subtotal	5	0	6
cholangio-cholecystectomy	Wagenaar (591)	1	0	?
	Wong (618)	1	0	12
	subtotal	2	0	
	Total no. of patients	18	2 (11%)	10

2.4.3. Transtumoral drainage

The internal bile drainage can be restored by pushing an intraluminal drain through the tumour. This can be done both operatively and non-operatively.

Operatively

The procedure consists of stretching of the tumour from inside by dilators or bougies, followed by intubation with a side-holed drain (12, 13, 34, 60, 128, 285, 286, 292, 312, 545, 546, 548, 564). The dilatation of the tumour is a blind procedure and has to be done carefully and in the right direction to prevent a false passage or portal vein damage (285, 360). Blockade of a lateral bile duct by the drain can be avoided by tubographic control (609). Silastic is the best drain material for long-term intubation (59, 83, 106, 208, 285, 455). If possible, bilateral drainage is to be preferred (107). In unilateral drainage, intubation of the hepatic duct of the larger right hemi-liver is advised (51, 208, 548). Operative transtumoral drainage can be achieved by three main types of intubation: 1 ascending, terminal drain; 2 transhepatic drain and 3 sunken drain and endoprosthesis.

1 ascending, terminal drain

Via a choledochotomy the tumour is dilated and the drain inserted. The choledochotomy is closed around the drain or connected to a jejunal loop and the rear end of the drain is brought outside the abdomen.

2 transhepatic drain (Goetze, 1951) (214)

After having dilated and passed the tumour, a bended probe is carefully carried up in the liver as long as possible inside the bile duct, but finally through the liver parenchyma to the anterior surface of the liver. A drain is connected to the probe and railroaded through the tumour and liver. The anterior end of the drain is brought out of the abdomen. The posterior end of the drain is left in the common bile duct or is led outside as a U-tube via the choledochotomy and the abdominal wall, if needed combined with a choledochojejunostomy (78, 95, 205, 214, 285, 453, 454, 455, 478, 493, 514, 545). In case an ascending insertion of the drain is impossible because of inaccessibility of the hilar bile ducts by tumour spread, then retrograde insertion via a transhepatically punctured biliary radicle may be tried (137, 285, 455).

Many variations of transhepatic drains exist such as terminal, T-, Y-, U-, O-, transversal and transanastomotic drains and their combinations (205, 455). A standard abdominal drain should be added to a transhepatic drain as bile leakage from the drain tract always occurs (106, 454). Replacement of the drain every three or four months is recommended (108, 455). If an extension of the drain outside the abdomen is present, daily irrigations with normal saline and if necessary with antibiotics (548) are advised.

3 sunken drain and endoprosthesis

A silastic drain of about 10 cm length is placed through the

dilated tumour and the choledochotomy is closed over it (208, 228, 285, 469). In the same way a plastic or metal endoprosthesis can be inserted (244, 419, 483).

There is little agreement about the best type of drain. Dislocation, obstruction and cholangitis are complications of all drains. Drains with external extensions have the advantages but also the disadvantages of a lasting access to the bile ducts and the opposite accounts for sunken drains (Table XVIII). All effects have to be considered in respect of the mostly short life expectancy of the patient. The disadvantage of an external extension is sometimes considerable and may be avoided by burying the extension in a subcutaneous pocket (65). The main disadvantage of a sunken drain is its inaccessability.

Obstruction and dislocation of a sunken drain occur in 9% and 4% of cases respectively (228, 285).

Table XVIII. Advantages and disadvantages of the various types of transtumoral drains. (The notation is relative and has only comparative value).

	sunken drain endoprosth.	terminal drain via common duct	T-drain via common duct	terminal transh.drain	transh. U-drain
Advantages					
visual assessment (röntgen, scopy)	-	+	+	+	++
bacteriol.examin. therapy	-	+	+	+	+
dislocation correction	-	+	+	+	++
relief of obstruction	-	++	+	++	+++
replacement of drain	-	++	+	++	+++
Disadvantages					
risk transhepatic passage	-	-	-	+	+
extent of operation	+	++	++	+++	++++
chance of dislocation	++	+++	+	+++	-
chance of cholangitis	+	++	++	++	+++
chance of obstruction	+	+	+	+	+
chance of subphrenic abscess	-	+	+	++	+++
chance of bile leakage	-	+	+	++	+++
chance of drain pinching	-	+	+	++	+++
pain abdominal outlet opening	-	+	+	+	++
inconvenience	-	+	+	+	++

Table XIX. Results of the total series of operative transtumoral drainage for primary tumour of the hepatic duct confluence (Appendix III gives the results of these series in full).

	patients	operative mortality	mean survival (months)	alive after (yrs) (%) ½	1	1½	2	3
total all series	216	33 (15.3%)	11.4	69	36	19	14	4
total series of at least 3 patients	200	33 (16.5%)	11.5					
total series of at least 5 patients	190	30 (15.8%)	11.4	71	35	17	14	4
total series of at least 10 patients	152	24 (16%)	12	71	34	17	15	4

The results of transtumoral drainage in 216 patients are listed in Table XIX. The postoperative mortality is 15.3% and the mean postoperative survival 11.4 months.

Non-operatively

The last decade a significant development has occurred with non-operative transtumoral drainage by percutaneous transhepatic or endoscopic transpapillar access. These techniques can be performed under local anaesthesia or sedation and form a welcome addition to the treatment of patients with high operative risk or short life expectancy (169).

Percutaneous transhepatic drainage (91, 161, 165, 169, 224, 241, 269, 271, 297, 299, 366, 411, 451, 468, 612, 613) is performed in addition to a PTC. After percutaneous dilatation of the tumour, preferably by a Grüntzig balloon catheter (93, 376) a transtumoral catheter or endoprosthesis with an internal diameter of 1-3 mm is inserted.

The complications of this procedure are cholangitis (7-27%), intrahepatic vascular lesions (9-16%) and dislocation or obstruction of the drain (5-33%) (91, 269, 271, 411). The more flexible and wider the drain, the smaller the risk of cholangitis (91). The average duration of survival of patients with malignant bile duct obstruction treated in this way, is three months (91, 224, 411, 451) and the mortality in the first month is 0-28% (91, 224, 612). Survival can be increased to 11 months by temporary irradiation with radioactive material placed inside the drain (188).

Transpapillary insertion of an transtumoral endoprosthesis can also provide terminal palliation with survival of 1½ to 4 months (140, 287).

2.4.4. Radiotherapy

Adenocarcinoma is relatively radioresistant (560) and a statistically proven beneficial effect of radiotherapy on bile duct cancer does not exist (564). Radiotherapy can be administered both externally and internally.

External radiotherapy

After operative marking of the tumour and construction of a

bile drainage, a usual dose of 5000 to 6000 rad (3000-7500) is given by a linear accelerator, betatron, or Cobalt 60 source (60, 63, 107, 109, 137, 217, 226, 275, 313, 314, 342, 343, 399, 449, 518, 572). A tumour dose of 4000-6000 rad is regarded as potentially curative (238, 342, 399, 449) and may be supplemented by an extra dosage of 4000 rad on the complete regional area (342). Peroperative irradiation of 3000 rad in 10 to 20 minutes is also applied (294, 560).

Irradiation by 3000 rad induces a light fibrosis, atrophy and degeneration of the hilar tumours (560) and 6000 rad causes a strong fibrosis (135, 217, 226). For that reason radiotherapy of the bile duct may require a permanent U-drain (208, 529). Radiotherapy with these doses is usually well tolerated (342, 395) but lethal radiohepatitis is reported (60).

The value of radiotherapy on the duration of survival is suggested by the the results in Table XX, but comparability of both groups is dubious. Radiotherapy may re-establish a bile drainage through a duct previously blocked by tumour, although it is not clear in how far this may be due to a concomitant surgical procedure (Table XXI). At all postmortem examinations of irradiated patients tumour tissue is still present in spite of the radiotherapy received (217, 226, 342, 560). The benefit of radiotherapy is for these reasons denied by some investigators (30, 290, 339, 606) and recommended by others (238, 289, 314, 342, 343, 518, 529, 581). Perhaps irradiation may play a role as adjuvant therapy after intended radical resection (238, 314).

Table XX. Mean duration of postoperative survival with and without radiotherapy for primary tumour of the hepatic duct confluence, irrespective the surgical therapy applied.

Authors	with radiotherapy		without radiotherapy	
	patients	mean survival (months)	patients	mean survival (months)
Black et al. (60)	12	9	?	6
Cameron et al. (107)	6	14	6	10
Hanna and Rider (238)[a]	14	12	3	1
Lees et al. (343)	9	17	9	4
Tsuzuki and Uekusa (572)	4	23	2	6
Whelton et al. (606)	5	16	8	16
Total no. of patients	50	14	28	9

(a) also includes other bile duct cancers

Internal radiotherapy

Local implantation of radioactive material has been proposed to administer a very high dose of irradiation to the tumour without damage to the surrounding tissues (149). Implantation of radioactive gold seeds made a histologically proven tumour

Table XXI. Results of radiotherapy for biliary tract cancer (bile ducts and gallbladder).

Authors	patients	place tumour	surg.drainage (type)	rad.dose (rad)	mean surv. (months)	palliat. effect
Green (226)	2	comm.hep.duct?	-	4600-6000	19	+
Green (226)	2	comm.hep.duct	transtumoral	4600-6000	10	+
Hudgins (275)	3	bile ducts	?	3250-5000	4	+
Iwasaki (294)	8	biliary tract	external	3000	1-11	?
Kopelson (313)	7	biliary tract	transtumoral	5000		+
Kopelson (313)	5	biliary tract	-	5000		+
Leer (342)	11	biliary tract	various types	6000+4000	1- 6	?
Pilepich (449)	11	biliary tract	transtumoral	4000-6000	10	+
Smoron (518)	8	bile ducts	?	4500	?	50%
Terblanche (546)	1	confluence	transtumoral	6000	12	+
Todoroki (561)	11	biliary tract	-	3000	10?	+

rest to disappear indeed (384). Insertion of Iridium^{192} (188, 262, 408) or radium (135) through a percutaneous transtumoral tube may offer new hope on the effects of radiotherapy. Quantities of 20,000 rad on the bile duct wall and the tumour and of 14,000 rad 1 cm around the duct can be reached in this way, resulting in necrosis of the tumour but also in an extensive necrosis of the periductal tissues (135). Hence late fibrotic strictures of the bile duct and perhaps even of the portal vein have to be feared, although the hilar vessels seem less to suffer from irradiation (511). Lower doses of radiation cause less fibrosis, but then again at autopsy it appears that the tumour has not been eradicated in patients who received 4400 rad (188). The duration of survival achieved by this method equals that of operative transtumoral drainage.

Finally a palliative effect has been reported of intravenous injection of rose bengale radioactivated with Iodine^{133} which is concentrated in the liver parenchyma and excreted in the bile (20).

2.4.5. Liver transplantation

Liver transplantation has past its experimental stage (53,170) but remains a tremendous enterprise with a 50% one year survival (105). Four patients could be traced who survived a liver transplantation for carcinoma of the hepatic duct confluence (104, 526, 295). One patient died from systemic candidiasis and brain abscess after three months (295), the other three died from tumour recurrence or liver metastases after 12, 25 and 54 months respectively (295), which gives a mean postoperative survival of 19 months. However scrupulous the pre- and peroperative examinations may be, micrometastases will always be missed (105) and their growth will be unrestricted because of the immunosuppression.

2.4.6. Chemotherapy

Adriamycin and 5-fluoro-uracil are the chemotherapeutic agents most commonly used in the treatment of bile duct cancers. Adriamycin has probably some effect on hepatocellular carcinoma (431) as suggested by an investigation to which I contributed as a referring district medical officer in Zambia (586). Adriamycin is excreted in the bile in a high concentration provided the excretory function of the liver is not hampered by an obstruction of the bile flow (24, 234, 467). The impact of chemotherapeutics on bile duct cancer, however, is small (7, 152, 234, 290, 489). In only one report a beneficial effect may be probable (343). Other reports only mention incidental remissions with a survival of 12 to 30 months (127, 152, 195, 234, 404, 471, 506, 579, 619). A combination of chemotherapy and radiotherapy has been advised (403, 567). In view of the doubtful benefit and the serious side effects the role of chemotherapeutics in the treatment of bile duct cancers is small.

2.4.7. External drainage

External drainage is only indicated as a terminal palliation to relieve the patient from pruritis and jaundice, since otherwise it just adds a biliary fistula to the disease (480). The former cutaneous hepatectomy (23) is replaced by an operatively or percutaneously inserted drain. The average survival after contruction of external drainage is three to five months (309, 572). This is as long as the survival after explorative laparotomy in which no form of drainage can be achieved (7, 286).

2.5. Conclusion

Prior to the diagnosis the clinical course of the disease consists of a non-specific obstructive jaundice. The postoperative course is rather monotonous as well and consists of an only temporary or partially relieved obstructive jaundice often with superadded iatrogenic cholangitis (351). An earlier diagnose is impossible but refraining from diagnostic or therapeutic loitering is imperative to preserve the possibility of a hepatectomy without an increased risk (168).

Proper bile drainage is the primary therapeutic goal, as patients die from recurrent bile obstruction. Even if adequate drainage can be achieved, the final results still will be determined by the continuing growth of the malignant tumour. Long survival is reported mainly after resection, but also after palliative procedures with and without radiotherapy. This may be more indicative of the slow growth of some of the tumours than of the value of the therapies applied (5) or may indicate the histological difficulty to discern malignant from benign tumours.

However different the various groups of patients may be, resection of the tumour at the earliest possible moment offers

Table XXII. Cumulative results of the various surgical treatments of primary tumour of the hepatic duct confluence.

Type of operation	patients	operative mortality (%)	mean survival (months)	alive after (yrs)(%)			
				½	1	2	3
resection all patients	250	14	20	91	69	33	21
paratumoral bypass all patients	400	24	11	60	37	8	1
transtumoral drain all patients	216	15	11	69	36	14	4
resection series ≥3 patients	201	13	19	92	67	37	22
paratumoral bypass series ≥5 patients	349	25	11	65	38	12	2
transtumoral drain series ≥5 patients	190	16	11	71	35	14	4
resection series ≥5 patients	159	13	18	87	67	36	22
paratumoral bypass series ≥10 patients	306	25	11	65	37	11	2
transtumoral drain series ≥10 patients	152	16	12	71	34	15	4

the best treatment. The combined results of therapy are displayed in Table XXII and Fig. 4.
The resection series of at least three patients and the paratumoral and transtumoral series of at least five patients are included in Table XXII and Fig. 4 as these series are the largest representatives.

Comparable to the treatment of gallbladder cancer (84, 179) and middle bile duct cancers (212, 310) extension of liver resection only incidentally provides a longer survival in patients with cancers of the hepatic duct confluence. However, many of the confluence cancers that have been fatal by bile obstruction, at autopsy still appear to have been resectable (13, 172, 396, 458, 559).

Early and efficient application of the diagnostic tools for accurate assessment of obstructive jaundice and an aggressive surgical approach are required to make such a treatment possible.

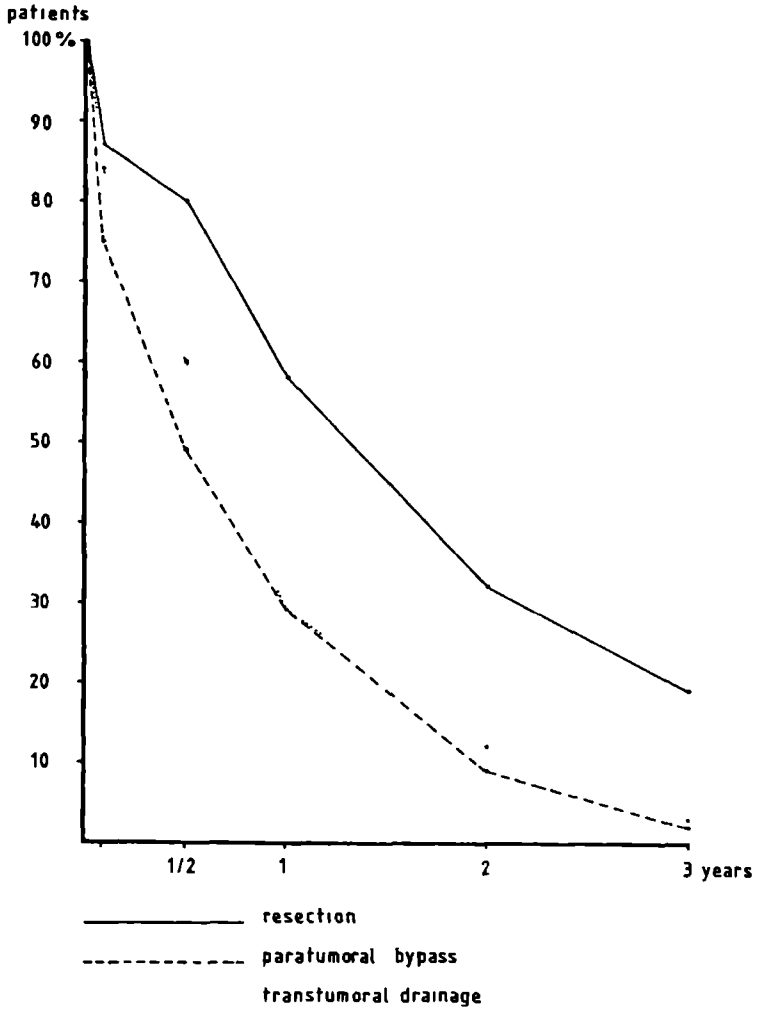

Fig. 4. Survival after surgical treatment of primary tumours of the hepatic duct confluence as from the moment of operation.

3. The surgical treatment of hilar bile duct cancer in The Netherlands

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Despite the general conception that cancers of the hepatic duct confluence grow slowly and disseminate late (309) the treatment remains unsatisfactory.

Many questions related to pathology and treatment are still not answered. The final solution regarding the best way to treat these tumours will have to wait for a prospective study which compares the various treatments (66). The value of such prospective studies seems doubtful until the moment that we are better informed about the pathology and the effects of treatment, which can only come from retrospective studies. For that reason a retrospective study was carried out to investigate the properties of these tumours and the results of treatment in 51 patients. Twenty one patients were treated in the Municipal Hospital of Arnhem and 30 patients in six university or teaching hospitals in The Netherlands.

The hospital files of 51 patients treated between 1960 and 1980, were studied. The group consisted of 29 men and 22 women (1.3:1) with a mean age of 61 years (range 28 to 86) at the time of operation. The patients were divided into three groups according to the greatest diameter of the tumour in order to assess a possible relation between the size of the tumour and the results of treatment:

Group I	(17 patients)	tumour diameter ≤ 2 cm
Group II	(19 patients)	tumour diameter 2-5 cm
Group III	(15 patients)	tumour diameter >5 cm

The mean age in Group I and Group II was 59 years (range 34 to 86 and 28 to 79 years respectively) and in Group III 67 years (range 52 to 84). In 49 patients an operative treatment was performed and in two patients a percutaneous transhepatic drainage was inserted.

3.1. Pathology and clinical course

In all but one patient jaundice was mentioned as the first symptom. Pain was recorded 16 times and itch 17 times. The complaints and symptoms began on an average four months prior to operation (range 1 to 14 months), there was no difference between the three groups.

The biochemical pattern was dominated by the obstructive jaundice type. The serum alkaline phosphatase level was analysed before the appearance of jaundice or rise of bilirubin level in five patients and was increased in all five cases. Where recorded, the specific pre-operative assessment

of the cause of obstructive jaundice was mainly based on percutaneous transhepatic cholangiography.

Twenty patients had been operated before for the same complaints and symptoms. Only on seven occasions the tumour was recognised at that operation and the patient was referred. In this study the term 'the operation' will be defined as the first operation during which the tumour was recognized and the patient was treated, if treatment was possible.

Tumour presentation

The whole series of 51 patients had a cancer of the hepatic duct confluence. As already indicated the diameter of the tumours did not exceed 2 cm in 17 patients (33%) and was 5 cm at most in 36 patients (71%; Group I and II). The macroscopic type of the tumour was recorded in 38 patients: firm and nodular in 32 patients and papillary in six.

Biopsies from the primary tumour were taken in 27 patients and proved to contain tumour in 22. Biopsies from lymph nodes or suspected liver areas were examined in 29 patients and proved to be tumour-positive in 20. In the other patients the diagnosis was confirmed either at autopsy or at a second operation. In two cases a negative result of a frozen section examination of the primary tumour turned out to be positive later on.

Local invasion and metastatic dissemination

In 20 of the 49 operated patients, palpable invasion of the adjacent liver tissue or dissemination elsewhere in the liver was recorded during operation. In Group I only one patient was reported to have liver metastases. Metastases in the regional lymph nodes were present in 15 patients. Intra-abdominal metastases outside the regional area were found in 10 patients during operation, all these metastases were situated in the peritoneum.

Local extension of the tumour to the portal vein or hepatic artery was seen in six patients, all with a tumour of less than 5 cm in diameter. The three patients with arterial involvement had metastases in the liver and regional lymph nodes. In two of the three patients with portal vein involvement no metastases were found at operation nor at autopsy after four months in one patient nor at re-operation after 26 months in the other. Ascites was mentioned in one of these patients. Operative mortality and survival did not differ from the patients without involvement of the hilar vessels.

In 17 patients an autopsy was performed 0 to 2 years after operation (mean 9 months). Local and regional spread was found at two autopsies one month after operation. Although not mentioned during operation this spread must have existed during the operation. Twice extra-abdominal metastases were recorded situated in the lungs, pleura, mediastinal nodes and spine. One of the two patients who were drained percutaneously and examined by autopsy one month later showed multiple metastases,

in the second patient none were present. In nine of the 14 autopsies in Group I and Group II 0 to 2 years (mean 10 months) after operation, no metastases were recorded.

No metastases were recorded in 12 patients of Group I, in seven of group II and in one of Group III at the time of operation (Table XXIII).

Table XXIII. Number of patients with or without macroscopic metastases recorded at the time of intervention.

Size of tumour (Ø)	patients	with liver metastases	with metastases in region.nodes	with metastases at distance	without metastases
Group I					
tumour ≤2 cm	17	1	4	2	12
Group II					
2 cm< tumour ≤5cm	19	10	7	1	7
Group I and II					
tumour ≤5 cm	36	11 (31%)	11 (31%)	3 (8%)	19 (53%)
Group III					
tumour >5 cm	15	12	6	8	1
Total no.	51	23 (45%)	17 (33%)	11 (22%)	20 (39%)

Histology

All tumours consisted of adenocarcinoma originating from the bile duct. Differentiation of tumour cells was reported in 33 cases: well differentiated in 21 and moderately or poorly differentiated in 12. The stroma was called highly fibrous or sclerotic in all 17 cases of the nodular tumours it was stated.

Postoperative course

Forty nine patients died within 32 months after intervention. The data after discharge from the hospital could not be traced in one patient. Only one patient is still alive at the time of writing (December 1982). The exceptional case history of this patient is described as it may show the possible value of radiotherapy:

> At the age of 49 years, this male patient (MHA 3) was operated on for obstructive jaundice caused by an obstruction of the hepatic duct confluence. At laparotomy a nodular tumour was found in the confluence with a diameter of 2 cm. This tumour consisted of a sclerosing adenocarcinoma of poor differentiation. Bile drainage was restored by transtumoral intubation with a transhepatic drain (Fig. 5). Postoperatively radiotherapy was administered which consisted of a total

dose of 6000 rad directed at the tumour. Four months later the drain was removed and a cholangiojejunostomy was constructed between a dilated duct of liver segment III and a Roux-Y jejunal loop (521) while the liver hilus was left untouched. Now, eight years later, the patient is in good health, has a normal alkaline phosphatase level, there is no evidence of tumour.

The histology of the tumour was reviewed (F.B. Bronkhorst, pathologist) since one of the criteria of malignancy, perineural invasion, is recently described in benign pancreatic tumours (138). The malignancy of this tumour, however, was incontestable (Fig. 6). The survival of this patient is not included in the general results as it is so disproportionally longer than the survival of the other patients.

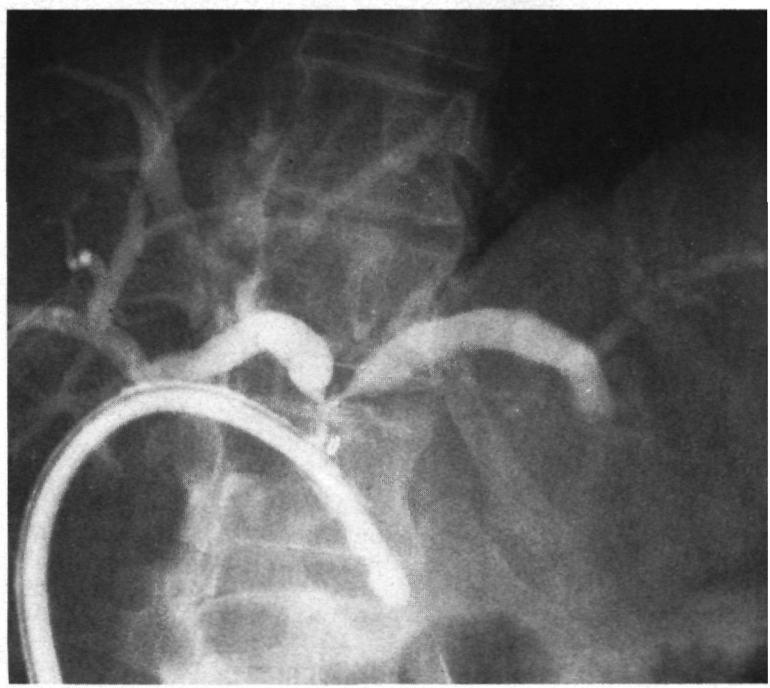

Fig. 5. Patient MHA 3: Tubogram via the initial transhepatic transtumoral drain, indicating the tumour of the hepatic duct confluence. A hemoclip marks the tumour radiologically. The drain has made a false passage and runs only partially through the tumour which resulted in an insufficient drainage. Therefore this drain was removed and replaced by a transtumoral bypass.

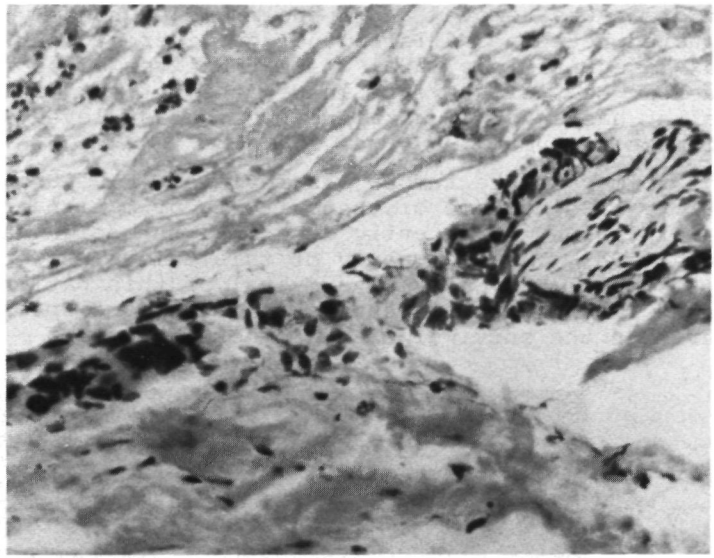

Fig. 6. Peripheral nerve with invasion of perineural lymph spaces by clearly malignant cells of a poorly differentiated adenocarcinoma (patient MHA 3) (HE x500).

Forty nine patients were operated on, 11 died in the first month after operation (22%). The mean postoperative survival of the others was nine months (range 1 to 36 months). No differences were seen in mortality and mean survival between Group I and Group II (14%, 10 months respectively). In Group III the operative mortality was 43% and the mean survival two months (range 1 to 7 months)(Table XXIV).

Two of the 18 patients with a tumour diameter of at most 5 cm without reported metastases died in the first postoperative month, the mean postoperative survival of the others was 14 months (range 4 to 32 months) (patient MHA 3 not included). An operation was carried out in 17 patients with equally sized tumours but with metastases. Four of these patients died within one month, the others had a mean postoperative survival of only six months (range 1 to 11). There was no difference in the cause of death of the patients with or without metastases.

From these data no relationship could be demonstrated be-

Table XXIV. Operative mortality (first month) and mean postoperative survival related to the size of the tumour.

Size of tumour (Ø)	patients	operative mortality	mean survival[a] (months)
Group I tumour ≤ 2 cm	16	2	11
Group II 2 cm < tumour ≤ 5 cm	19	3	10
Group I and II tumour ≤ 5 cm	35	5 (14%)	10
Group III tumour > 5 cm	14	6	2
Total no. of patients	49	11 (22%)	9

(a) One patient from the Municipal Hospital Arnhem (MHA 3) excluded

tween the macroscopic or microscopic type of the tumour on the one hand and the operative mortality, time of survival or the incidence of metastases on the other. This applies to the entire group of 49 operated patients as well as to Group I (16 patients), Group II (19 patients) and Group III (14 patients) separately.

Cause of death

Fifty of the 51 patients died, the cause of death could be traced in 47. Fourty-five patients (96%) died 0 to 36 months after the intervention from the consequences of biliary obstruction, being cholangitis or liver insufficiency and in most cases from a combination of the two. Only two patients could have died from conditions not related to the biliary obstruction. A gastro-intestinal bleed in the premortal phase was reported in fives cases and originated from gastroduodenal ulceration in the three patients who were examined at postmortem. Tumour cachexia was described in five patients, four times accompanied by jaundice. In these five patients carcinomatous peritonitis was seen at operation. No autopsies were conducted.

3.2. Therapy

The tumour was resected in four patients. Palliative reconstruction of an internal bile drainage was performed in 29 patients. Transtumoral drainage was carried out as the main treatment in 21 patients, operatively (19 patients), percutaneously (1 patient) or by a combination of the two (1 patient). In eight patients a paratumoral bilio-enteric bypass was performed, at the hilar level (2 patients) or intrahepatically (6 patients). In one patient an unsuccessful transtumoral drainage was changed to a temporary successful intrahepatic bypass. Twice external drainage was carried out, in one case percutaneously without operation.

In 16 of the 51 patients no more than an explorative laparotomy was done for lack of any therapeutic solution.

Radiotherapy was applied as additional treatment in 12 patients.

In six patients the reconstruction of the bile drainage appeared to fail postoperatively and so no bile drainage was achieved in 22 of the 51 patients (45% !).

Resection

All four resections were performed in patients with a tumour of at most 2 cm in diameter. Three times a local resection of the hepatic duct confluence was performed and bilio-enteric continuity restored by a hepaticojejunostomy between both left and right hepatic ducts and a Roux-Y loop. In one patient the resection consisted in a diathermic removal of a stemmed intraductal papillary carcinoma. In at least two patients the resection edges were not free of tumour. Both patients received 5000 rad postoperatively.

In all patients a well functioning bile drainage was obtained, but they died from recurrent bile duct obstruction 7 to 32 months after the operation (mean 22 months). Autopsy was done in two patients and revealed a local recurrence of the tumour and multiple metastases in the liver. One patient showed no signs of macroscopic metastases at operation 26 months after incomplete resection; six months later he died.

Non-resective restoration of internal bile drainage

Transtumoral drainage

Transtumoral drainage was accomplished operatively by a transhepatic drain in 10 patients, by a terminal, ascending drain or a T-drain in eight, by an endoprosthesis in one patient and in one patient the tumour was just dilated.

Four of these 20 patients died in the first month after the operation. The mean postoperative survival of the others was nine months (range 1 to 24 months). A satisfactory postoperative relieve of the bile obstruction was obtained in 13 of the 16 surviving patients (Table XXV). No differences could be demonstrated between the results in the patients of Group I and those of Group II, nor between the results obtained by the different types of transtumoral drains. The drains had to be changed once in four months because of obstruction.

Paratumoral bypass

In nine patients a paratumoral bypass was constructed, eight times as the primary or main procedure and once after three efforts to achieve transtumoral drainage had failed. Twice the bypass was made at hilar level by a hepaticojejunostomy between the left hepatic duct and a Roux-Y loop (254). Four times a dilated branch of the intrahepatic duct of liver segment III was used just to the left of the umbilical fissure (521). Twice a dilated duct behind the bed of the gallbladder in segment V was connected to the jejunum and once a hepatojejunostomy on segment VI was performed (50).

Two of the nine patients died in the first postoperative month from cholangitis or a leaking anastomosis. In five of the seven surviving patients a satisfactory relief of the bile obstruction was obtained postoperatively, the other two remained jaundiced. Change of a transanastomotic drain or correction of the anastomosis had to be done once in four months. The four patients with a proper follow-up showed a mean postoperative survival of six months (1 to 12 months). There was no demonstrable difference neither between the results of the hilar and peripheral bypasses nor between the results of Group I and Group II (Table XXV).

Radiotherapy

Twelve patients were postoperatively treated with external radiotherapy. In none of them the tumour diameter exceeded 5 cm. Six were treated by a rotative irradiation of 6000 to 7500 rad with $Cobalt^{60}$, administered in a split course of 30 daily sessions (D. Miete† and P. van der Made of the Department of Radiotherapy, Municipal Hospital, Arnhem). During the preceding operation the tumour had been three dimensionally marked with clips and a bile drainage had been constructed. The other six patients received 3000 to 7000 rad.

Patients whose bile drainage had been effectively restored could stand the irradiation better than those lacking proper relief of the obstruction. One of the patients is alive, eight years after treatment. The mean postoperative survival of the other 10 patients irradiated shortly after the operation (Group I: 5; Group II: 5) was 11 months (range 8 to 20 months), the same as of the 18 patients with an equally sized tumour who survived the operation but did not receive radiotherapy.

Table XXV. Results of surgical transtumoral and paratumoral bile drainage.[a]

Size of tumour (Ø)	patients	operative mortality	mean survival (months)	satisfactory drainage
Group I and II tumour ≤ 5 cm				
transtumoral	17	2	10	13/15
paratumoral	7	2	7	4/5
Group III tumour > 5 cm				
transtumoral	3	2	1	0/1
paratumoral	2	0	3	1/2
Total no. of patients				
transtumoral	20	4	9	13/16
paratumoral	9	2	6	5/7

[a] one patient has been excluded from the duration of survival of each group, because of disproportionate length of survival, re-operation after several transtumoral intubations or missing follow-up

Table XXVI. Results of postoperative external radiotherapy for primary tumour of the hepatic duct confluence in 12 patients.

Patient no.	tumour size	histology	surgical therapy	radiation dose (rad)	postop. survival (months)	autopsy tumour present	local ext. or metas.
1	II	poorly diff. nod.sclerosing	paratumoral bypass	6000	12	+, yes	yes
3	I	poorly diff. nod.sclerosing	paratumoral bypass	6000	96	alive	
4	II	poorly diff. nod.sclerosing	transtumoral drain	6000	8	-	
8	I	well diff.[a] nodular	transtumoral drain	6000	8	-	
9	I	well diff. nod.sclerosing	paratumoral bypass	6000	9	-	
10	II	poorly diff. nodular	transtumoral drain	6000	10	-	
36	II	nodular sclerosing	transtumoral drain	3000	14	-	
37	I	well diff. papillary	resection	5000	20	+, yes	yes
39	II[b]	unspecified	transtumoral drain	7000	24	+, yes	no
43	II	well diff.[a] nodular	exploratory laparotomy	5000	9	+, yes	yes
45	I	well diff. nodular	resection	5000	7	+, yes	yes
51	I	well diff.[a] nodular	transtumoral drain	5000	9	-	

(a) metastases present at the time of operation

(b) radiotherapy administered three months before death

There was also no difference in survival between the patients of Group I and Group II who had been irradiated. No demonstrable connection could be found between the results of irradiation and the histological type of the tumour or the kind of surgical therapy. The adenocarcinoma in the hepatic duct confluence still existed in all five irradiated patients who were examined postmortem, these patients died 3 to 20 months after the operation. In four of them no metastases had been recorded during the operation, three showed dissemination at autopsy (Table XXVI).

Survivors of more than one year

Nine patients lived for more than one year after operation. All had a tumour not exceeding 5 cm in diameter and in none of them metastases were reported at the time of operation. For the whole series this means 18% and for Group I and Group II 25%. Four patients were alive two years after the operation (8% and 11% respectively). There was no correlation between the duration of survival and factors as age of the patient, period of preceding jaundice, histological type of the tumour, size of the tumour as long as it did not exceed 5 cm diameter, kind of operation, administration of radiotherapy or cause of death.

3.3. Discussion

The tumour symptoms were aspecific. The only feature that may have aroused suspicion before the patient became jaundiced, was an isolated alkaline phosphatase level. Two thirds of the tumours were smaller than 5 cm in diameter at the time of operation. The larger the tumour, the more often local extension and metastases occurred in the liver. The size of the tumour was not related to lymphogenic dissemination or to local extension to the portal vein or hepatic artery. Involvement of the portal vein was not associated with an increased incidence of liver metastases or a shorter postoperative survival.

There was no demonstrable relationship between the macroscopic or microscopic type of the tumour and dissemination or local extension. In 20 of the 51 patients (30%) no metastases were recorded at the time of operation, a percentage that corresponds well with other investigations (60, 235, 290, 396, 556) In patients whose tumour diameter did not exceed 5 cm, this percentage rose to 53% and in patients with a maximal tumour diameter of 2 cm even to 71%.

The patients died from the consequences of unrelieved or recurrent bile duct obstruction before the malignant properties of the tumours became manifest. Of all the investigated parameters only the size of the tumour, the presence of metastases and the kind of therapy seemed to have an impact on the postoperative survival. Assessment of the various types of surgical therapy was hindered by the relative incomparability of the treated patients. Nevertheless the four resections resulted in a far better mean survival without a higher mortality than the other operations (Table XXVII).
Probably all these resections were incomplete regarding the scantiness of the resections performed, the tumour-positive edges in at least two patients and the cause of death in all four patients. Consequently the hepaticojejunostomies were constructed with tumour containing tissue. Still the patients had a mean postoperative survival of 22 months. This confirms the supposition that these tumours are growing slowly, and furthermore it indicates that resection should be preferred to all other modalities even as a palliative procedure. Theoretically a resection could have been possible in all 12 patients without metastases or local tumour extension of

Table XXVII. Comparative results of surgical treatment in 33 patients with carcinoma of the hepatic duct confluence.

Treatment	patients	operative mortality (1 month)	mean survival (months)	satisfactory drainage (patients)	patients alive (years)				
					½	1	1½	2	3
resection	4	0	22	4/4	4	3	3	2	0
transtumoral drainage	20	4	9	13/16	13	4	2	1	0
paratumoral bypass	9	2	6[a]	5/7	3	2	1	1	1

[a] patient MHA 3 excluded

group I and perhaps even in some of the seven patients without tumour spread of group II.

No differences were seen in operative mortality and duration of survival between transtumoral and paratumoral drainage (Table XXVII). The role of radiotherapy was limited. With the exception of one patient, the mean survival of patients who did receive radiotherapy equalled that of patients who did not. Moreover the tumours were not eradicated and had disseminated without hindrance. Nevertheless one patient (MHA3) is in good health eight years after a palliative operation and radiotherapy. Incidentally, long-term survivors are reported after palliative drainage procedures both with (285, 343, 548) and without (12, 78, 286) radiotherapy. Temporary local implantation of radioactive material may be promising (188). (Recently we treated a patient with a confluence tumour and metastases in the caudate lobe. The treatment consisted of transtumoral U-tube drainage and local radiotherapy after introduction of the irradiation source in the tube. This patient died after four months from thrombosis of the inferior caval vein and pulmonary and extensive hepatic disseminations).

Only when the tumour has almost completely blocked the bile outlet of the liver, jaundice develops and only then the patient seeks medical assistance. At that time, the stage of tumour growth, size and spread of the tumour, and the consequences of the already existing partial or unilateral bile duct obstruction are of major importance to the treatment. Jaundice can not be regarded as an early symptom since the mean duration of pre-operative jaundice is independent of tumour size. The tumour has already invaded the surrounding tissues at the stage when the bile duct lumen is almost completely blocked (561). An undelayed diagnosis can only prevent damage to all the liver segments by a complete bile obstruction.

The aim of therapy is twofold: 1 a lasting reconstruction of the bile drainage of as much as possible liver tissue and 2 removal of the malignancy. If resection is possible, it is the treatment of choice even in case the resection will be incomplete.

Although the resectability of a hilar bile duct cancer can be assumed by thorough pre-operative examination, the resectability can only be ascertained by scrupulous operative exploration (66, 332, 357). Palliative drainage should be considered only if the feasibility of resection has been excluded. Both a paratumoral bypass and a transtumoral intubation have its own indications and can be employed together (137). The two sides of the liver should be drained to prevent infection in obstructed segments (457).

Patients who are not suitable for surgery or in whom no operative drainage can be achieved, can be treated by percutaneous transhepatic or endoscopic transpapillary drainage (140, 224). In a patient whose life expectance is only a few months, the indication for palliation is mainly defined by the presence of itching. In the absence of pruritis the best solution for a terminal patient may be to withdraw from any treatment.

In 45% of the patients discussed in this study, no bile drainage could be accomplished. A thorough knowledge, skill and presence of all feasible ways of examination and treatment are essential to offer these patients proper treatment. The first operation is the patient's chance of a life time.

4. Characteristics and resection of cancer of the hepatic duct confluence involving the portal vein (Royal Postgraduate Medical School, Hammersmith Hospital, London)

Introduction

Portal vein involvement is generally considered to rule out resection of an otherwise localised hilar bile duct carcinoma. Ipsilateral affection of the main branches of the portal vein and hepatic artery still can be treated by removal of the tumour and vessels involved together with the hemi-liver of the affected side. Theoretically, even a tumour with contralateral involvement of the main branches of the hilar vessels may be resectable by removing the tumour and the affected vessel segments. This leaves the one hemi-liver with just an arterial blood supply and the other side with only a portal blood supply which should be tolerated by men as well as by dogs (604). Additional major hepatectomy would leave the remaining part of the liver with only one type of blood supply, which is a critical situation. Just as every other vessel the portal vein can be divided and resutured. Therefore portal vein involvement by a bile duct cancer may be treated by resection of the affected part of the vein in conjunction with the tumour, and followed by reconstruction of the vein.

The best treatment of tumours of the hepatic duct confluence is resection of the tumour. A prospective study was carried out in 24 patients with angiographically demonstrated venous involvement in the Surgical Hepatobiliary Unit (head Professor L.H. Blumgart) to assess the pathology and the possibilities of resection of confluence tumours affecting the portal vein.

4.1. Material and methods

From May 1979 to November 1982, 78 patients were treated for tumour of the hepatic duct confluence. Angiographic studies were performed as part of the pre-operative resectability examination in 53 patients. Portal vein involvement by the tumour was demonstrated in 24 of the 51 patients (45%). There were 13 women and 11 men. The mean age was 53 years (range 33 to 69).

In general the patients were referred from other hospitals, where the diagnosis was made by clinical and laboratory examination, ultrasonography and percutaneous transhepatic cholangiography (PTC). The mean duration of pre-operative jaundice was 2½ months (range 1 to 7 months). During the investigations into the resectability of the tumours (63) nutritional disorders were treated and in 17 patients the biliary tree was preoperatively decompressed by percutaneous transhepatic drainage (PTD) (392) for a mean time of 23 days (range 8 to 43 days). Two patients were not suitable for operation and were treated

by percutaneous transhepatic drainage. They died within two months. The other 22 patients received operative treatment.

The size and extension of the tumours were assessed by combining the cholangiographic, operative and histopathological findings. In all 24 patients a coeliac arteriography was performed and if necessary supplemented by a superior mesenteric arteriography. The venous phase of the arteriogram provided sufficient information about the portal vein and its branches in 19 patients. Four times a direct splenoportography was added and once a percutaneous transhepatic portography. The vascular involvement was determined by combining the angiographic data with the operative and histopathological findings. The presence of partial liver atrophy and of metastases was derived from the combined findings of ultrasonography, angiography, CT scan, operative investigation and histopathological examination.

4.2. Pathology

In all operated patients the tumour consisted of a nodular mass. In 17 patients the diameter or largest width of the tumour did not exceed 5 cm. The mean extension of the tumour in the left hepatic duct was 2 cm (range 0 to 5 cm) and in the right duct 1.5 cm (range 0 to 3 cm). Regarding the tumours where the width did not exceed 5 cm the mean extension on the left side was 1.5 cm (range 0 to 3 cm) and on the right side 1.3 cm (range 0 to 3 cm). All tumours consisted of adenocarcinoma of the bile duct with fibrous stroma. The differentiation of tumour cells was established in 15 patients: well differentiated in nine, moderately differentiated in four, poorly in one and mixed in one.

The portal vein of these 24 patients was involved at the origin of the left portal vein branch in 19 and of the right branch in nine. The bifurcation of the portal vein was involved in 11 patients, six times on the left only, once on the right side only and four times on both sides. Fourteen times the affected portal vein branch was occluded completely and in the other cases the lumen was narrowed. In 11 patients the hilus was dissected at operation. In four of these cases the venous involvement was more extensive than expected from the angiography. Once an encasement expected to be bilateral turned out to be single-sided only (Fig. 7 and 8).

Portal hypertension, diagnosed by measurement of a raised portal pressure, splenomegaly or the presence of wide and heavily bleeding splanchnic veins, was present in five patients (23%). In all patients the portal vein obstruction was serious: bilateral in three patients and unilaterally complete with bifurcation involvement or with displacement of the contralateral branch in two. In four of these five patients the left portal vein branch was completely occluded. In three patients ascites was noted which did not coincide with the portal hypertension. In eight patients the tumour was resected and careful histopathologic examination of the resected specimens was carried out (Professor K. Weinbren).

Although no systematic serial sections were made of the

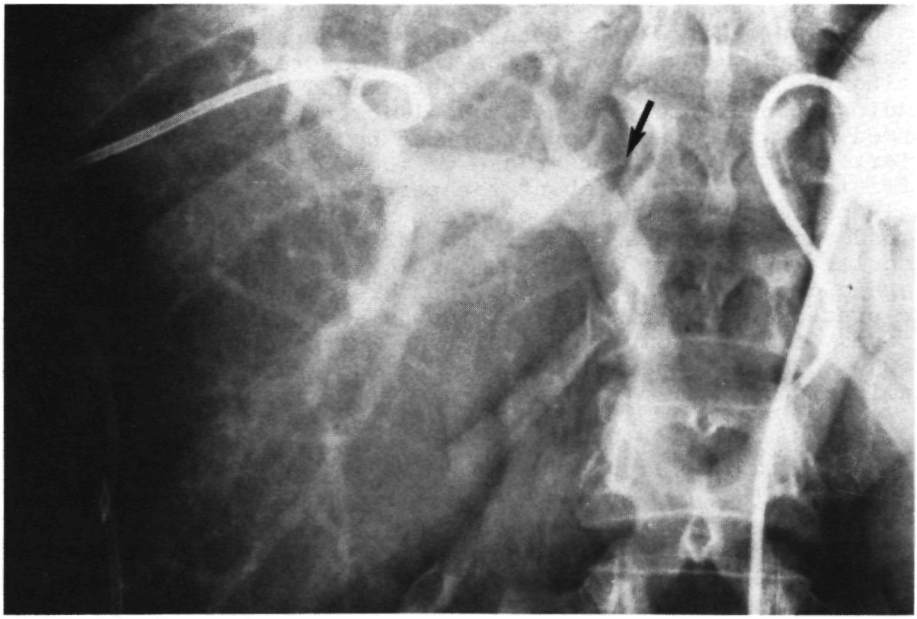

Fig. 7. Venous phase of coeliac axis arteriogram with missing left portal vein branch which is totally obstructed by a cancer of the hepatic duct confluence.

involved portal vein segments, a penetration of the tumour through the vessel wall into the lumen was never demonstrated. At most the tumour abutted on the wall and actual invasion into the wall was demonstrated in only one specimen.

Involvement of a main branch of the hepatic artery was seen in 10 of the 24 patients (42%), five times of the left and five times of the right branch (Fig. 9). In nine patients the occlusion was partial and in one total. A crossed vascular involvement which consisted in affection of the portal vein branch of the one side and of the artery branch of the other side, was present in three patients. Again penetration of the vessel wall by the tumour was not demonstrated.

Atrophy of a part of the liver was seen in eight patients (33%), six times of the left and twice of the right hemi-liver. In six of the eight cases of hemi-liver atrophy there was total occlusion of the ipsilateral portal vein branch and in two a partially occluded portal vein branch was accompanied by a stenosed hepatic artery branch.

Local extension of the tumour into the parenchyma of the caudate lobe was found in nine of the 22 patients operated on (43%). In 11 patients (50%) the tumour extended into the base

Fig. 8. Percutaneous transhepatic portogram. Involvement of the portal vein bifurcation by a cancer of the hepatic duct confluence, compromising the origins of both the right and left portal vein branches.

of the quadrate lobe. Concomitant tumour extension into the caudate and quadrate lobes was found in five patients (23%).

Liver metastases were found in five of the 22 patients operated on (23%). There was no correlation between the localisation of the metastases and the side of vascular involvement. Metastases in the regional lymph nodes were seen in nine patients (41%) and extended to the peripancreatic or paraaortal nodes in four. Distant metastases (peritoneum, lung, vertebra) were found at the time of operation in two patients.

4.3. Therapy

Twenty two patients were operated on. In eight patients the tumour was resected. A paratumoral bypass was constructed in seven patients and in five the treatment consisted of transtumoral drainage only. Twice just a laparotomy was performed. Radiotherapy was administered in one patient. Six of these patients died within the first month after operation (27%). The mean postoperative survival of the others was eight months (range 1 to 29 months). Six patients are alive at the time of

Fig. 9. Coeliac axis arteriogram.
Missing left hepatic artery which is totally obstructed by a cancer of the hepatic duct confluence.

writing (December 1982), on a mean of 10 months after operation (range 1 to 29 months).

One of the aims of this chapter is to deal with extensive resections and vascular reconstructions. Therefore the non-resective operations will be discussed briefly.

Non-resective operations

Paratumoral bypass

A paratumoral bypass was constructed in seven patients by anastomosing a Roux-Y jejunal loop with a dilated bile duct at the hepatic side of the tumour. In five patients the bile duct of segment III was used, in one the left hepatic duct and in one a hepatojejunostomy was performed on the left lobe. In three of the seven patients the contralateral hemi-liver was drained by a transhepatic transtumoral tube.

Two patients died from cholangitis and septicaemia or liver failure in the first postoperative month. The mean postoperative survival of the other five patients was seven months (range 1 to 11 months), one of them being alive seven months after operation. One patient received 5000 rad postoperative external irradiation and died 11 months after operation, autopsy was not done.

Transtumoral drainage

Dilatation of the tumour followed by transtumoral drainage of the right hepatic duct as a procedure complete in itself, was performed in five patients. No postoperative death was seen, the mean survival was seven months (range 1 to 10 months). One patient is alive 10 months after operation (Table XXVIII).

Death in the two non-resective groups was caused by either cholangitis with sepsis or recurrent bile obstruction with liver failure and usually by a combination of the two.

Table XXVIII. Results of surgical treatment of carcinoma of the hepatic duct confluence involving the portal vein.

Operation	patients	operative mortality	mean survival (months)	alive after (years)				
				½	1	1½	2	3
resection	8	3	11	3	2	1	1	0
paratumoral bypass	7	2	7	4	0	0	0	0
transtumoral drainage	5	0	7	3	0	0	0	0

Resections

In five patients the tumour was resected together with a right lobectomy and in three patients with a left hemihepatectomy. Three times a lateral segment of the portal vein bifurcation was included in the resection. The defect was repaired by direct suturing. Twice the complete portal vein bifurcation was removed, followed by end-to-end reconstruction. A tumour free resection margin was obtained in four of the six in whom an intended curative resection was performed.

Three of the eight patients died within the first postoperative months, one from liver necrosis and two from septicaemia. The mean survival of the other patients was 11 months (range 1 to 29 months) at the time of writing (December 1982)(Table XXIX). The case histories of six patients are discussed here. In all patients the biliary drainage was restored by an end-to-side hepaticojejunostomy between the remaining hepatic duct and a Roux-Y jejunal loop over a transanastomotic transhepatic U-tube.

4.4. Patients

1 This 59-year old man was referred for treatment of obstructive jaundice of 3½ months duration. Ultrasonography and PTC demonstrated an obstruction in the hepatic duct confluence. PTD was established. The venous phase of a coeliac arteriography demonstrated encasement of the origins of the left and right portal vein branches.

At operation a nodular tumour of 3 cm diameter was found at the right side of the hepatic duct confluence,no metastases were detected. The tumour was removed in conjunction with the

right liver lobe and a large part of the caudate lobe. Peroperative blood flow measurement (22a,378a) showed a 130% increase of the blood flow in the retained left lobe. The tumour consisted of a well-differentiated sclerosing adenocarcinoma which extended around the right branches of the portal vein and the hepatic artery and into the caudate lobe. A lymph node in the hilus adjacent to the tumour was tumour positive but the resection margins were free of tumour. The postoperative course was excellent and the patient is alive 29 months after operation.

COMMENT: Although the angiography suggested bilateral involvement of the portal vein branches, it appeared at operation that especially the right branch was affected and that the bifurcation and left branch could be freed from peritumoral fibrosis.

2 A 53-year old man was referred for treatment of obstructive jaundice of two months duration. Ultrasonography and PTC showed a lesion in the hepatic duct confluence. A PTD was inserted and cytological examination of the bile demonstrated adenocarcinoma cells. The venous phase of a coeliac arteriogram delineated a narrowed right portal vein branch. CT scan did not demonstrate metastases or liver atrophy.

At operation a 2x2x5 cm sized nodular tumour was found which extended along the right hepatic duct and compromised the origin of the right portal vein branch. No metastases could be detected. Resection of the tumour with excision of a lateral segment of the portal vein bifurcation and right lobectomy was performed. The tumour consisted of a well-differentiated sclerosing adenocarcinoma. The resection margins were free of tumour. Postoperative recovery was uncomplicated.

COMMENT: At the end of the dissection when the left hepatic duct had to be cut, a firm nodule was felt at that spot which was suggestive of tumorous extension. Frozen section revealed fibrous tissue only, which was postoperatively confirmed in the paraffin sections. Even at the end of a dissection, a tumour up to that moment considered to be resectable can be irresectable.

3 This 43-year old woman presented with a two months obstructive jaundice. Ultrasonography and PTC demonstrated an obstruction in the hepatic duct confluence which extended in the left hepatic duct, PTD was brought in. Coeliac arteriography showed encasement of the common hepatic artery and left portal vein, and a complete occlusion of the left hepatic artery. A CT scan showed left sided liver atrophy.

As the curative possibilities were little, she was operated with palliative intention. A 3 cm sized nodular tumour was found which consisted of a well differentiated sclerosing adenocarcinoma disseminated in the atrophic left lobe. The tumour was resected by a left hemihepatectomy in conjunction with the common hepatic artery and a lateral segment of the portal vein bifurcation. The portal vein was repaired by direct suture. The right hemi-liver was left with portal blood supply only. In the removed specimen two small tumour positive lymph nodes were found in the liver hilus. The cut edge of the right hepa-

tic duct contained tumour.

In spite of an initial postoperative recovery she had to be re-operated two weeks later because of partial liver necrosis, from which she died 29 days after the first operation.

COMMENT: Only after the final transection of the right hepatic duct it became clear that curative resection still could not be achieved. The right portal vein with a laterally sutured portal vein bifurcation provided the only afferent blood supply to the remaining right hemi-liver which was apparently not enough.

4 This 58-year old woman was referred for treatment of obstructive jaundice of two months duration which was caused by a localised obstruction of the hepatic duct confluence as shown by PTC. PTD was established. Angiography demonstrated a narrowed left hepatic artery and a totally obstructed left portal vein branch.

At operation a nodular mass of 3 cm diameter was found in the hepatic duct confluence nearly surrounding the origin of the left portal vein branch. The left liver lobe was somewhat atrophic. No metastases could be detected. The tumour was removed in conjunction with a left hemihepatectomy, excision of a lateral part of the portal vein bifurcation and ligation of the right hepatic artery branch, leaving the right hemi-liver with portal blood supply only. The tumour consisted of a moderately differentiated sclerosing adenocarcinoma. Tumour tissue extended to the resection surfaces of the right hepatic duct and portal vein branch.

Four days later the patient had to be re-operated because of necrosis of the cut surface of the retained right hemi-liver, including the biliary anastomosis.The hepaticojejunostomy was recreated. After one month dehiscence of the anastomosis reoccurred, but this time because strongly regenerated liver tissue had disrupted the biliodigestive connections. A hepatojejunostomy was successfully constructed and resulted in good palliation for one year. Then the jaundice recurred and metastases in the abdominal scar and in the lung became evident. An endoprosthesis was inserted percutaneously but the patient died four days later, 14 months after the initial operation. Autopsy was not performed.

COMMENT: At the first operation a slice of liver tissue was vascularised insufficiently but the rest of the right hemi-liver showed good regeneration though supplied by portal blood only. The regenerated liver tissue later on even caused disruption of the reconstructed biliodigestive anastomosis. This anastomosis consisted of a connection of the jejunal loop with four separate bile duct orifices, which were two by two connected with each other and formed two common bile channels. The disruption consisted in a division of the interconnected bile ducts, producing again the original four separate ducts. These were successfully covered by the jejunal loop in a hepatojejunostomy.

5 When 33 weeks pregnant this 33-year old woman became jaundiced. One week later she delivered a premature but healthy baby girl. The jaundice was obstructive in nature and was shown by ultrasonography and PTC to be caused by an obstruction of the hepatic duct confluence. The venous phase of a coeliac arteriogram showed total obstruction of the right portal vein branch.

At operation a 2x2x2 cm nodular tumour of the hepatic duct confluence was found. There were no metastases. After distal transection of the common bile duct the portal vein was dissected liverwards. The portal vein bifurcation appeared to be adherent to the tumour. The left corner of the porta hepatis near the base of the umbilical fissure was dissected from a caudal to a cranial direction. The left portal vein branch and left hepatic duct were transected. Then the main stem of the portal vein was cut just upstream of the bifurcation and reanastomosed with the distal side of the transected left portal vein branch. In this way the bifurcation and the right branch of the portal vein were excluded from the portal blood stream and the portal blood was led directly into the left hemi-liver. This required 15 minutes occlusion of the portal blood stream. Then the tumour was resected in conjunction with a right lobectomy and excision of the portal vein bifurcation.

The tumour consisted of a well differentiated sclerosing adenocarcinoma with initial spread in the pre- and retrohilar liver tissue. Two lymph nodes near the cystic duct entrance were tumour positive. The resection margin of the left hepatic duct and the adjacent liver tissue contained tumour as well.

After good progress in the early postoperative period, her condition began to deteriorate. A subphrenic collection was drained but 10 days after the operation she died from uncontrolable septic shock. Autopsy revealed no specific abnormalities and no septic source was found. The portal vein anastomosis and the hepaticojejunostomy were patent.

COMMENT: When approached from a caudal direction, the left part of the porta hepatis can be explored by opening the covering peritoneum and hilar plate. The first structure encountered is the left portal vein and cranial to that lies the left hepatic duct (page 91). Both structures can be dissected without prior transection of liver parenchyma and consequently the described diversion of the portal blood stream can be performed prior to the hepatectomy. A hepaticojejunostomy as it is, unhampered in its construction by a portal vein anastomosis, is imperfect in a quarter of the patients (178). As the left portal vein branch and the left hepatic duct lie close to each other, insertion of stay sutures before transection is beneficial to the subsequent reconstruction. Portal vein reconstruction is done before biliary reconstruction, in time as well as in place, and therefore the hepaticojejunostomy has to be carried out cranially of the portal vein anastomosis, which requires a scrupulous and strictly systematic technique (588).

The operation had an excellent course and resulted in a well vascularized and bile-drained left liver lobe with good ana-

stomosis. The cause of the final tragic outcome remains a mystery.

6 This 46-year old woman was referred for treatment of obstructive jaundice of six weeks duration. PTC and ERCP had shown an obstruction of the hepatic duct confluence. PTD was installed. Angiography showed encasement of the right hepatic artery and left portal vein branch. CT scan did not show metastases or atrophy.

At operation the gallbladder appeared to be attached to the left liver lobe on the left side of the ligamentum teres, no quadrate lobe could be identified. A 2x2x4 cm nodular tumour was found in the right side of the hepatic duct confluence, compromising the right hepatic artery and the portal vein bifurcation with the origins of its left and right branch. No metastases could be discovered.Because of the abnormal anatomy, at first transection of the liver along the anomalous gallbladder bed was necessary to reach the left portal vein. Then the tumour was resected in conjunction with the portal vein bifurcation and with an atypical extended right hepatectomy along the plane between the left sided gallbladder bed and the inferior caval vein which included the right part of the caudate lobe. The portal blood supply to the remaining left lobe was interrupted for 60 minutes and reconstructed by an end-to-end anastomosis between the portal vein and the left branch.

The tumour consisted of a well differentiated adenocarcinoma that abutted on the portal vein but did not invade the vein wall. There was initial invasion of the pre- and retrohilar liver tissue. The cystic lymph node contained tumour tissue but all resection margins were free of tumour.

Postoperative recovery was slow but without major problems. Four months after the operation, the patient is still recovering and in a good condition.

COMMENT: A left sided gallbladder in itself is a rare and harmless anomaly and is mostly detected accidently (231, 261, 370, 417). The abnormal anatomy in this patient, however, complicated the liver resection as an atypical plane of transection had to be used. This resulted in considerable blood loss from crossing suprahepatic veins which necessitated clamping of the portal vein for 60 minutes. This was well tolerated by the patient and did not cause haemodynamic disturbances. Man and monkey usually well tolerate acute occlusion of the portal circulation and even ligation of the portal vein is compatible with life (129, 381, 534). Moreover complete vascular isolation of the liver up to one hour has been shown hardly to interfere with the postoperative function and regeneration of the retained liver parenchyma (281).

Table XXIX. Resections of carcinoma of the hepatic duct confluence involving the portal vein in eight patients.

Patient	liver resection	management involvement v.porta bif./a.hepatica		resection margin	postop.survival (months)	alive/ cause of death
1	right lobectomy + caudate lobe	-	-	free of tumour	29	alive
2	right lobectomy	excision lat segment and direct repair	-	free of tumour	1	alive
3	left hemihepatectomy (palliative)	excision lat. segment and direct repair	excision a.hep.com. no repair	tumour positive	0	necrosis liver remnant
4	left hemihepatectomy	excision lat. segment and direct repair	ligation a.hep. dextra	tumour positive	14	tumour recurrence; sepsis
5	right lobectomy	excision bif. and end-to-end repair	-	tumour positive	0	cholangitis sepsis
6	right lobectomy	excision bif. and end-to-end repair	-	free of tumour	4	alive
7	right lobectomy (palliative)	-	-	tumour positive	0	cholangitis sepsis
8	left hemohepatectomy	-	-	free of tumour	9	alive

4.5. Discussion

The bifurcation of the portal vein is in direct contact with the hepatic duct confluence (268). In consequence early invasion of the bifurcation by a confluence tumour can be expected. The angiographically screened and operatively verified incidence of portal vein involvement in this series was 45%. Presumably this incidence is representative for all cancers of the hepatic duct confluence since the tumour characteristics in this specific series did not differ from those reported in Chapters 3 and 4, and since angiography was only routinely performed in patients not primarily unsuitable for resection. Corresponding figures were found for initial invasion of the adjacent tissue of the caudate and quadrate lobe.

As reported (616) the angiographic data were in agreement with the findings at operation. Real vein wall invasion by the tumour was demonstrated only once and penetration into the lumen in none of the patients. Apparently the vascular encasements were due to obtrusion of the sclerotic tissue on the vessels. It seems that the abundant peritumoral fibrous reaction, so typical of confluence tumours, strangles the vessel before the actual tumour tissue can reach the vessel wall. This means that vascular involvement in itself is no contra-indication to resection of a proximal bile duct cancer if the involved vascular segment can be removed together with the tumour.

There was no relation between the size of tumour and vascular involvement. Apparently both small and large confluence tumours can affect the portal vein. Likewise a large tumour may as well spare the portal vein (72). All tumours consisted of the common nodular sclerosing adenocarcinoma with the usual preponderance of the well differentiated type. Concomitant arterial involvement was found in 42% of the patients (10/24). Again no actual invasion or penetration of the arterial wall by the tumour was demonstrated (617). The tumours were situated slightly more on the left side of the confluence and the origin of the left portal vein branch was more often affected than the right branch.

The incidence of hepatic metastases in this specific series corresponds well with other series and showed no relation with the side of portal vein or hepatic artery involvement. Also lymphatic and distant metastases occurred as often in these patients as in the average population of patients with bile duct tumours. This supports the concept that impairment of the hilar vessels by the tumour mostly consists in an obtrusion by the sclerotic reaction that surrounds the actual tumour.

Portal hypertension only developed in case of serious obstruction of the portal flow. One normal branch of the portal vein with an unobstructed main stem and bifurcation warrants an adequate outflow tract for the splanchnic blood. This could be expected, as even after right lobectomy the portal pressure does not rise (90), although elevation in pressure may develop postoperatively because of compression of the portal system in the retained liver segments by hyperplasia of the parenchyma (68). Complete obstruction of a portal vein branch or partial obstruction of the ipsilateral main branches of the portal

vein and of the hepatic artery can cause atrophy of the hemi-liver concerned.

As the characteristics and dissemination patterns of confluence tumours with vascular involvement do not differ from tumours without vascular involvement, the treatment of both types of tumours is essentially the same. Although technical problems may arise, and liver resection will be necessary in most cases, theoretically resection of the tumour still is the best treatment, however high the operative mortality may be at present. Although the actual tumour does not usually invade the portal vein wall, the vein is so closely approached that tumour clearance requires at least a resection of the adjacent vein segment.

The right branch of the portal vein in general measures only 2 cm before dividing in the right lateral and right paramedian branch and runs there already intrahepatically. The left branch has a transverse extrahepatic stretch of about 4 cm, from which only minor branches are given off to the quadrate and caudate lobes. At 4 cm from the main bifurcation the left portal branch sharply bends anteriorly, launches the branch to segment II and then widens in the umbilical part (recessus of Rex). For that reason resection of the portal vein bifurcation leaves especially at the left side an extrahepatic vein remnant suitable to re-anastomose.

Just like the bifurcation of the portal vein the confluence of the hepatic ducts is situated at the right side of the liver hilus, and like the right branch of the portal vein the right hepatic duct has an extrahepatic course of only 1 cm before it divides into its lateral and paramedian branch. The left hepatic duct has a length of about 5 cm before entering the left liver lobe. Because of the submucosal spread and the possibility of multifocal or diffuse occurrence of the cancer the extrahepatic biliary system has to be removed as extensive as possible. Consequently a wide excision of a confluence tumour has to include on the right side the first parts of the secondary hepatic ducts. On the left side, a remnant of the left hepatic duct can be retained for later reconstruction.

Therefore resection of a confluence tumour together with portal vein bifurcation is best combined with: 1 diversion of the portal blood flow to the left lobe; 2 right lobectomy, and 3 hepaticojejunostomy with the left hepatic duct remnant.

Right lobectomy is a well established procedure (362, 525, 528), as is left hepaticojejunostomy by means of a Roux-Y loop (330). Resection and reconstruction of the portal vein in combination with a liver resection is less known.

Eight of the 24 tumours of this series were resected (33%). Five times this required a concomitant right lobectomy and three times a left hemihepatectomy, twice a resection of the complete portal vein bifurcation and three times a resection of a lateral segment of the bifurcation, once a resection of the common hepatic artery and once a ligation of the arterial branch to the liver remnant.Still the resection margins of the hepatic duct remnants contained tumour tissue in four of

the eight cases, although the impossibility to obtain tumour clearance was expected in two pre-operatively. Twice a remaining right hemi-liver was supplied with portal blood only. One of these patients showed a good liver regeneration in spite of a stormy postoperative course that required two re-operations. The second patient died from liver necrosis.Therefore reconstruction of both types of afferent blood supply should be pursued.

After partial excision, the portal vein can be reconstructed by 1 end-to-end anastomosis, 2 by interposition of autologous graft, and 3 by transposition of the splenic vein.

1 end-to-end anastomosis of the portal vein remnants. After mobilisation of the head of the pancreas, right colon and mesenteric root the portal trunk can be brought up 6 cm (185, 192, 193, 357, 369);

2 interposition of autologous graft, e.g. internal jugular vein (98, 198, 225, 444, 510, 541). homologous vein graft (185, 198, 541) or vascular prosthesis (356, 424, 522);

3 transposition of the splenic vein. The splenic vein can be mobilised from the dorsal side of the pancreas, distally transected and re-anastomosed with a portal vein remnant in the liver hilus, as proposed by Longmire (357). Use of the splenic vein in a comparable way was successful in bridging defects of the superior mesenteric vein (97, 114, 154) and in the treatment of Budd-Chiari syndrome by interposition of a vein graft or prosthesis between the splenic vein and the pulmonary artery (189), pulmonary vein (346) or right atrium (242, 327, 364).

Three cases of right lobectomy with concomitant resection of the portal vein bifurcation and subsequent end-to-end reconstruction of the portal vein remnants could be traced in the literature (Table XXX). Two patients treated by Fortner (193) soon died after the operation, one from persistent bleeding and the other from septicaemia and portal vein thrombosis. The patient operated on by Longmire (357) recovered after a stormy postoperative course but died one year later from recurrent tumour growth (361). Four other patients who underwent a major liver resection with resection and repair of the portal vein and the extrahepatic biliary tract have also been listed in Table XXX, as well as three patients in whom the portal blood was diverted from the liver remnant by a porto-caval shunt.

The four patients in Table XXX treated by Fortner died shortly after the operation, in all the already abandoned cold perfusion technique was used (191, 194, 197). The two patients reported by Tsuzuki (574) also had their arterial supply to the retained right hemi-liver resected and reconstructed, one by interposition of a saphenous graft and one by an anastomosis with the right gastro-epiploic artery. At least three of the four patients with a carcinoma of the hepatic duct confluence, who survived resection of the tumour in conjunction with a hepatectomy and resection of the portal vein bifurcation, later on died from recurrent tumour growth.

Table XXX. Results of major liver resection in conjunction with resection of the hepatic duct confluence and portal vein bifurcation.

Authors	indication	type of hepatectomy	type of portal vein reconstr.	postoperative survival (months)/cause of death
Fortner et al (193)	carcinoma of hepatic duct confluence	right lobectomy	porto-caval shunt	0 liver insufficiency
Fortner et al. (193)	carcinoma of hepatic duct confluence	right lobectomy	end-to-end repair	0 haemorrhage
Longmire et al. (357)	carcinoma of hepatic duct confluence	right lobectomy	end-to-end repair	12 recurrent tumour growth
Fortner et al. (193)	hepatocellular carcinoma	right lobectomy	end-to-end repair	0 septicaemia,portal vein thrombosis
Schamaun (499)	Echinococcus alveolaris cysts	right lobectomy	porto-caval shunt	5 incarcerated diaphragmatic hernia
Maillard et al.(369)	Echinococcus alveolaris cysts	right hemi-hepatectomy	end-to-end repair	0 haemorrhage from suprahepatic veins
Kajitani (300)	carcinoma of hepatic duct confluence	partial right hepatectomy	porto-caval shunt	16 ?
Fortner et al. (193)	carcinoma of hepatic duct confluence	left hemi-hepatectomy	end-to-end repair	0 haemorrhage
Tsuzuki et al.(574)	carcinoma of hepatic duct confluence	left hemi-hepatectomy	end-to-end repair	15 recurrent tumour growth
Tsuzuki et al. (574)	carcinoma of hepatic duct confluence	left hemi-hepatectomy	end-to-end repair	18 recurrent tumour growth

The three major postoperative complications are 1 thrombosis of the reconstructed portal vein, 2 insufficiency of the liver remnant, and 3 cholangitis with abscesses and septicaemia. Portal vein thrombosis should be dealt with by immediate re-operation and thrombectomy (296). A proper way of liver support to cover a period of insufficiency is not yet known. The presence of a well functioning portal and arterial blood supply to a bile-drained liver remnant of not less than 20 to 25% of the original liver tissue is the best guarantee against liver insufficiency. The danger of infection is clear.

Part B Investigation into the possibility of a curative resection

5. Analysis of the poor results

Of all treatments resection gives the best result in every aspect. Additional liver resection to a resection of the tumour does not increase the operative mortality as long as the resection does not exceed a left hemihepatectomy. Irrespective of the portal vein management, right hemihepatectomy and right lobectomy have a combined operative mortality of 35% and a mean postoperative survival or 17 months (1 to 48 months) when performed for hepatic confluence tumours (combined series of 31 patients from Chapter 2.4.1.; 4.3. and 4.5.).

Cure is virtually never achieved as the patient still dies from recurrence of the tumour after 1½ years. Extension of resection seems not to provide better results as tumour recurrence still occurs in due time, however extensive the excision may have been. However, a wide resection which includes a partial hepatectomy or even the hilar vessels, is performed only if local resection is impossible because of tumour extension. Tumour spread, extent of resection and scantiness of tumour clearance match each other, and so do the results.

Periductal and intraductal spread of the tumour are in the main responsible for the appalling frequency of recurrence.

Periductal spread of the tumour

Criteria to define or to determine the stage of tumour growth at the time of treatment are unknown (75). Tumour growth in the thin walled bile duct soon reaches the periductal tissues. When the tumour has progressed so far that it blocks the bile duct lumen, it already has invaded the periductal tissues. Consequently the same holds true for the moment of treatment.

Metastatic tumour growth in regional lymph nodes is reported in one third of the patients. Lymphatic metastases can easily be overlooked since the lymphatic system is situated on the posterior side of the liver hilus and hepatoduodenal ligament. Incipient invasion of the abundant lymphatic network around the bile ducts is likely to be present in almost all patients. If a lymphatic vessel, however small it may be, is obstructed by the tumour a retrograde lymph flow may occur and consequently retrograde carrying of tumour cells. It is unknown in how far the biliolymphatic permeability in bile duct obstruction results in passage of tumour cells through the wall of grossly dilated bile ducts into the lymphatic system. The highest lymph nodes in the porta hepatis are situated between the left side of the portal vein bifurcation and the anterior surface of the caudate lobe. When there is at least an initial

invasion of the lymphatic system in the liver hilus, then the complete regional area of abundant lymphatic structures in the hilus and the hepatoduodenal ligament must be considered to be affected by tumour.

The curious venous network which drains into the quadrate lobe, described by Petrén and Karlmark (445), is invaded in up to 82% of the patients (433). Therefore initial invasion of the quadrate lobe by this route is plausible in most patients. The parenchyma of the quadrate and caudate lobes is so near to the hepatic duct confluence that incipient invasion by the malignant tumour should be common rather than exceptional. Besides, the problems in histopathologic appreciation discussed on page 13, also apply to the judgement of all resection margins.

Intraductal spread of the tumour

Submucosal spread in the duct wall occurs over long distances and it is wise to consider a primary bile duct carcinoma as a regional rather than as a local affection of the bile duct (433). Attention is usually directed to the proximal and distal extensions of the tumour along the right, left and common hepatic ducts. Probably intraductal extension of the tumour also occurs along small branches from the confluence area into the adjacent liver tissue of the segments IV and I. Such extensions may develop before a major duct becomes blocked as they will produce no symptoms. Finally, multifocal bile duct carcinoma occurs in 9% of the patients and all these may have the indicated spread (page 12).

Conclusion

As it is plausible that intraductal and periductal tumour spread has already occurred at the time of treatment, the complete hepatoduodenal ligament, the liver tissue adjacent to the tumour (segments I and IV) and the extrahepatic biliary tract including the complete right hepatic duct, have to be regarded as a pool of potential tumour invasion. Resection of all lymphatic tissue of the hepatoduodenal ligament, leaving only the bare portal vein and hepatic artery will - similar to retropancreatic dissections in the treatment of pancreas carcinoma (196, 272) - microscopically be insufficient.

Generally, resection of the confluence, part of the biliary tract and the periductal liver tissue gets proper attention, but the retroductal tissues as portal vein bifurcation, hilar lymphatic tissues and the caudate lobe are never resected en bloc, except in case of liver transplantation. Even after liver transplantation the tumour appears to recur and such a recurrence can only arise from tumour-invaded structures in the retained rest of the hepatoduodenal ligament.

6. The possibility of a radical block resection

6.1. Requirements

The poor results of the current types of resections are to be expected as these resections do not follow correct oncologic principles. A radical block resection is the generally accepted procedure of choice for the surgical treatment of a malignant tumour. This treatment consists of removal in one piece of the undisturbed tumour, a considerable amount of apparently normal tissue around the tumour and the area of regional lymph drainage. Hampered by the concentration of vital structures at the hilus of the liver, resection seems impossible for a cancer of the hepatic duct confluence (5, 374, 406, 599). According to oncologic principles, a potentially curative resection of such a malignant tumour should consist in a block resection of

1. the complete extrahepatic biliary tract (which includes the left hepatic duct);
2. the complete right hepatic duct and the central parts of the right lateral and right paramedian ducts;
3. the liver segments IV, V and I;
4. the complete hepatoduodenal ligament with the portal vein, hepatic artery, bile duct and lymphatic tissues up to and including the pancreatoduodenal nodes and the nodes around the base of the common hepatic artery, leaving the bare inferior caval vein as posterior dissection margin.

After such a resection, restoration of the outflow tract of the splanchnic portal blood as well as the reconstruction of the biliary drainage and of the double afferent vascularisation of the remaining liver segments II and III on the left side and VI, VII and VIII on the right side is practically impossible. Moreover such a resection will be an all-or-nothing operation and even by the end of the resection it will be impossible to assess the chances of success of a subsequent reconstruction.

Nevertheless the described block resection is the only rationale for a potentially curative operation and may provide the only way out of the cul-de-sac in which the treatment of proximal bile duct cancers has ended up (168). The feasibility of such a resection should still be considered because of the very properties of the liver, namely the huge functional reserve capacity and regeneration power (6, 436, 494), the segmentally framed vascular and biliary system (142, 206, 247, 267) and the capacity to tolerate a normothermic ischaemia of 45 minutes (281, 282, 283).

Theoretically the resection required can be performed by resecting the hepatoduodenal ligament and the entire liver, followed by autotransplantation of the left lobe and the right lateral sector. This type of surgery is successfully applied in renal operations (103, 340, 425, 549). Simple gravity per-

fusion with 4°C Ringer's lactate offers four hours to perform a similar autotransplantation of liver parts (103, 497). However the precarious outflow tracts may rather be left alone and from oncologic point of view the main suprahepatic veins do not need to be sacrificed either. Therefore resection in situ is preferable to the formidable and hazardous procedure of total resection and autotransplantation.

To preserve as much as possible liver tissue it would be ideal the reconstruct the afferent vascularisation and the biliary drainage of both the left lobe and the right lateral liver sector. Such a procedure is much more difficult and uncertain than the reconstruction of only one liver part. Each part consists of 20 to 30% of the total liver parenchyma. The smallest amount of liver tissue required for a good survival of the patient is 20 to 25% of the normal liver (49, 525, 527, 528). Therefore preservation of only the right lateral sector (segments VI and VII) or the left lobe (segments II and III) may be sufficient.

Extended left hepatectomy, which leaves only the right lateral sector, is an exceptional and difficult operation (530). Right lobectomy, leaving only the left lobe, is a well established procedure (49, 525, 565). In experienced hands the operative mortality is nil to 10% (5, 279, 527, 528), in collective studies this percentage varies from 17 to 25% (200, 201, 406). This operation is the only standard hepatectomy that provides sufficient opportunity to perform a wide resection of the hepatic duct confluence (277). Usually the operation is well tolerated by the patients (5, 49, 279, 383, 436, 525, 528, 565, 566).

After resection of 75% of the liver in dogs, the portal pressure rises slightly (367). In men no rise in portal pressure is seen after right lobectomy (90, 284, 379), but the perfusion of the retained left lobe may rise 200 to 300% (379). Apparently the capacities of the left portal vein branch and the intrahepatic portal system of the left lobe are sufficient to cope with all the portal blood, while the suprahepatic venous system provides an adequate outflow tract for the combined portal and arterial flow.

For these reasons the required radical block resection is possible if 20 to 25% of normal, well vascularised and bile-drained liver tissue can be preserved. Anatomically the left liver lobe can meet this requisite best.

6.2. Possibilities of afferent vascular reconstruction

The arterial stump of the retained liver part can be reanastomosed with the dissected splenic or gastro-epiploic artery or connected to a major artery by interposition of a venous graft or vascular prosthesis. Intraparenchymatous implantation of the splenic artery into the retained liver segments, comparable to implantation of the arteria mammaria interna into the myocardium (584), has been described in animals (151, 203, 220, 221, 222, 293, 461). The implantation has to be performed in a preceding operation as connections with the intra-

hepatic vascular system need time to develop. Besides the resulting blood supply is considerably smaller than the original supply by the hepatic artery (151, 293).

The portal vein remnant of the retained liver part can be re-anastomosed with the splanchnic portal system as outlined on page 71. Since the complete portal vein has to be removed, the gap to bridge between the root of the portal vein and the portal remnant of the retained liver part is too wide for an end-to-end anastomosis. Autologous vein provides the best material for an interposition. Therefore a transposition of the splenic vein or an interposition of an autologous vein graft are the most suitable known ways to replace a completely removed portal vein. When the splenic vein is dissected and peripherally transected, it can be transposed behind the stomach to the retained liver part and anastomosed with the cut edge of the portal branch remnant. As there are no valves in the portal system (157, 479, 615) the portal blood can flow unimpeded in the reverse direction. The use of the central splenic vein as an outflow tract for the portal blood is applied in the central splenorenal shunt for portal hypertension (47, 134), in splenothoracic shunts for Budd-Chiari syndrome (364) and to bridge defects in the superior mesenteric vein (97).

Theoretically a peripheral entrance of the portal blood into the liver must be possible, just like a peripheral outlet of the bile. By a peripheral hepatotomy a large portal vein can be sought. The construction of a connection between such a vein and the extrahepatic system by means of the transposed splenic vein or the interposition of a vein graft should be possible. In this way the portal vein is bypassed even before it has to be transected. A splenic vein implanted into the liver parenchyma was tested in dogs but stayed patent only as long as portal hypertension was present (126, 220, 293). An anastomosis between the re-opened umbilical vein and the portal vein root or splenic vein seems a feasible possibility for a paraportal bypass to the left lobe (Chapter 6.3.).

The entire procedure is simplified if reconstruction of only one afferent blood supply would be enough. A liver part can regenerate when supplied with portal (21, 604) or arterial (85, 264, 499) blood only. After hemihepatectomy the regeneration of a liver remnant only supplied with portal blood, is virtually as good as it is when perfused with both portal and arterial blood, but regeneration is deficient in case only arterial blood is provided (604). The role of the functional competition between the transplant and the liver of the host has been underestimated (265, 496) in the interpretation of the many confusing results of experimental auxiliary liver transplantation (341, 363, 398, 400, 456). Still, reconstruction of the portal blood supply probably prevails over arterial reconstruction. Besides, diversion of the portal blood by a portocaval shunt also requires an anastomosis and causes encephalopathy in more than half of the patients with a previously normal portal circulation (98, 185, 193, 369, 499).

The survival of a patient entirely depends on the function and regeneration of the small liver remnant. A further reduction of the reserve and regenerative capacities is not admissable to meet technical problems. All alternatives considered result in a smaller, ill-balanced or uncertain blood supply. Therefore both portal and arterial vascularisation of the retained liver part have to be reconstructed completely.

The various types of direct vascular reconstruction have been separately employed with good results. The combination of a right lobectomy and resection of the portal vein bifurcation followed by biliodigestive and portal vein reconstruction of the liver remnant is also feasible. Still in the widest resections nowadays performed, the left hepatic duct confluence and the left portal vein branch are transected only halfway along their course - very close to the tumour - to preserve stumps of sufficient length to re-anastomose. Moreover in all known resections most of the hepatoduodenal ligament is left alone as is usually done with the caudate lobe. Lastly all common methods of portal vein reconstruction have an unavoidable all-or-nothing character as a successful outcome of these reconstructions cannot be predicted once a wide resection of the portal vein is performed.

6.3. Outline of a radical block resection

A study was initiated to investigate the practibility of a resection that would meet the oncologic requisites (page 73). For this purpose 40 normal, adult cadaver livers were anatomically explored. It soon became clear that the reconstructive possibilities of the left lobe were such that the tumour area together with the right lobe, caudate lobe and complete hepatoduodenal ligament could be removed at a distance of at least 4 cm from the hepatic duct confluence.

The confluence of the hepatic ducts lies in the right corner of the porta hepatis. Therefore the plane of a radical resection can virtually follow the umbilical fissure on a good 4 cm distance from the confluence with preservation of the 'subhilus' of the left lobe. The left hepatic duct can be cut at this level. The collapsed umbilical vein in the ligamentum teres enters the end of the umbilical part of the left portal vein (recessus of Rex) and can be reopened to a width virtually equal to that of the left portal vein branch. The reopened umbilical vein forms a good access to the hepatic portal system at the outermost left corner of the porta hepatis just outside the plane of resection. The hepatic artery branch to the left lobe runs in the last 2 cm of its extrahepatic course just beside the edge of the left lobe. This part of the artery can be preserved and provides an arterial remnant sufficient for re-anastomosis. In the 22% of patients in whom the main arterial branch to the left lobe arises from the left gastric artery (397) no reconstruction may be necessary at all.

The portal, arterial and biliary reconstructions can all be performed in different areas without mutual interference.

The space available for reconstruction of the hepaticojejunostomy at the umbilical fissure is not impeded by a reconstructed accompanying portal vein branch just beside it. The bile duct of segment III can also be used for biliary drainage. In that case the accompanying portal vein branch of segment III has to be preserved when dissecting the bile duct.

Irreversibility of the procedure can be avoided by the reconstruction of a separate blood supply to the left lobe prior to the actual radical block resection which is proposed as follows:

The reopened umbilical vein is anastomosed end-to side with the central part of the splenic vein (Figs. 10 and 11). Then the portal blood reaches the complete liver by 1 the portal vein itself and 2 by the paraportal bypass formed by the conduit of the splenic vein - umbilical vein - recessus of Rex. The function of the bypass is judged by clamping of the portal vein, as at that time all the portal blood is diverted through the umbilical bypass to the liver. The procedure has to be abandoned when the bypass does not function, in that case all other modalities of treatment still remain available.

Prior to the transection of the portal vein arterial reconstruction of the left lobe should preferably be performed. The last part of the extrahepatic tract of the left hepatic artery is dissected and cut close to its entrance into the left lobe. If the common hepatic artery and the first centimetres of the gastroduodenal artery can be saved without seriously violating the radical intention of resection, such a gastroduodenal artery remnant provides the ideal large vessel for reconstruction of the left lobe. This depends on the individual anatomy but as a rule the artery together with the para-arterial lymph nodes will have to be included in the resection, in which case the splenic artery is the only direct alternative. As the size of the splenic artery resembles that of the hepatic artery, the most adequate and simple reconstruction probably consists in peripheral transection followed by an anastomosis of the transposed splenic artery with the left hepatic artery branch (Fig. 12). Interposition of a saphenous vein graft between the base of the common hepatic artery and the left hepatic artery or use of a transposed gastro-epiploic artery can be good alternatives and can moreover save the arterial supply of the spleen.

If both the umbilical bypass and the separate arterial circulation to the left lobe function properly, the left portal vein branch is cut and closed at the base of the umbilical fissure just before the transition in the recessus of Rex. When this is done both the right and left hepatic lobes each have their own separate afferent blood supply (Fig. 13).

Still radical resection can be abandoned and other methods of treatment can be applied. The haemodynamic situation to be expected after resection is imitated by clamping of the bases of the portal vein and the hepatic artery. In the same way afferent vascular isolation is achieved of the area to be re-

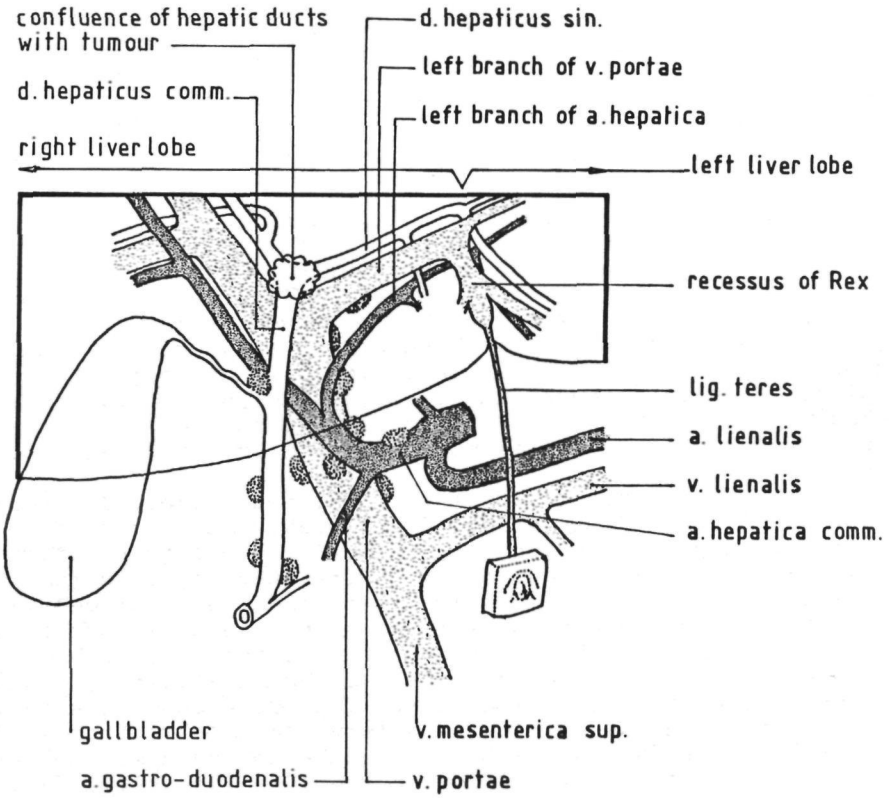

Fig. 10. The anatomy of the hepatoduodenal ligament and the porta hepatis with cancer of the hepatic duct confluence.

sected. Up to that moment the portal blood supply has not been interrupted to any part of the liver and the arterial ischaemia of the left lobe is restricted to the period needed for the reconstruction of the arterial diversion.

After creation of an independent afferent blood supply to the left lobe, the bases of the portal vein and the hepatic artery are cut. Then resection of the complete hepatoduodenal ligament and the right lobe virtually flush with the umbilical scissure are performed in a partially bloodless field (Fig. 14). This vascular isolation does not cover the portal branches to the left side of the quadrate lobe, which sprout from the

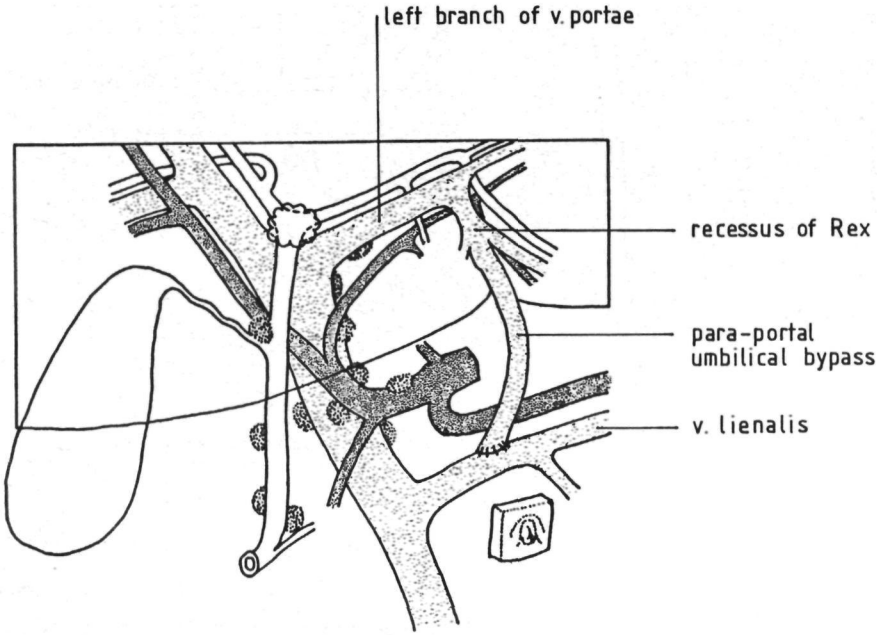

Fig. 11. The collapsed umbilical vein in the ligamentum teres has been reopened and anastomosed end-to-side with the central part of the splenic vein. The portal blood has a double inflow tract to the liver.

recessus of Rex, and the retro- and suprahepatic venous circulation. Bile drainage can be reconstructed in the usual way with a hepaticojejunostomy between the remnant of the left hepatic duct beside the base of the recessus of Rex and a Roux-Y jejunal limb. The final result is comparable with the situation after standard right lobectomy and bilio-enteric reconstruction.

Apart from treatment of a tumour of the hepatic duct confluence, the described procedure may also be used for primary or secondary liver tumours in the hilus, gallbladder and for Echinococcus alveolaris cysts that involve the hepatoduodenal ligament. The paraportal umbilical bypass without a concomitant liver resection may be useful in the treatment of lacerations of the portal vein (382), arteriovenous fistulae (389, 580), isolated portal vein thrombosis or cancers of the common bile ducts.

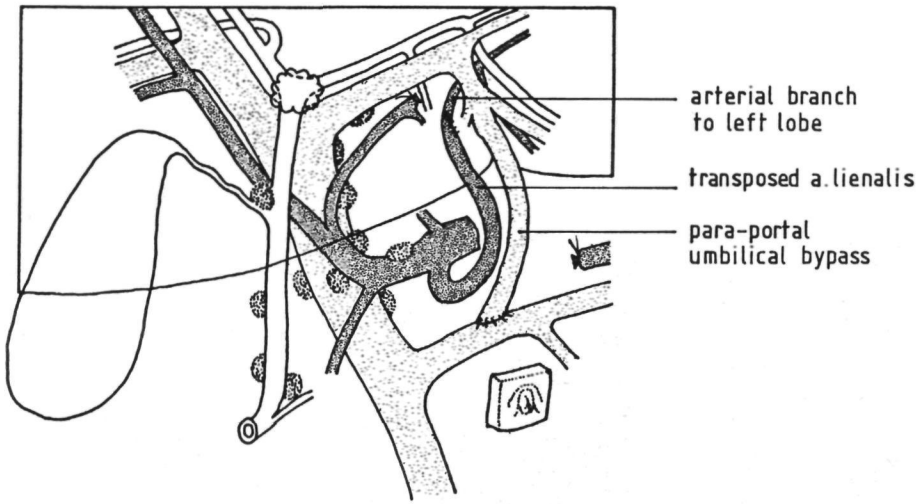

Fig. 12. Double portal inflow to the liver and transposed arterial supply to the left lobe.

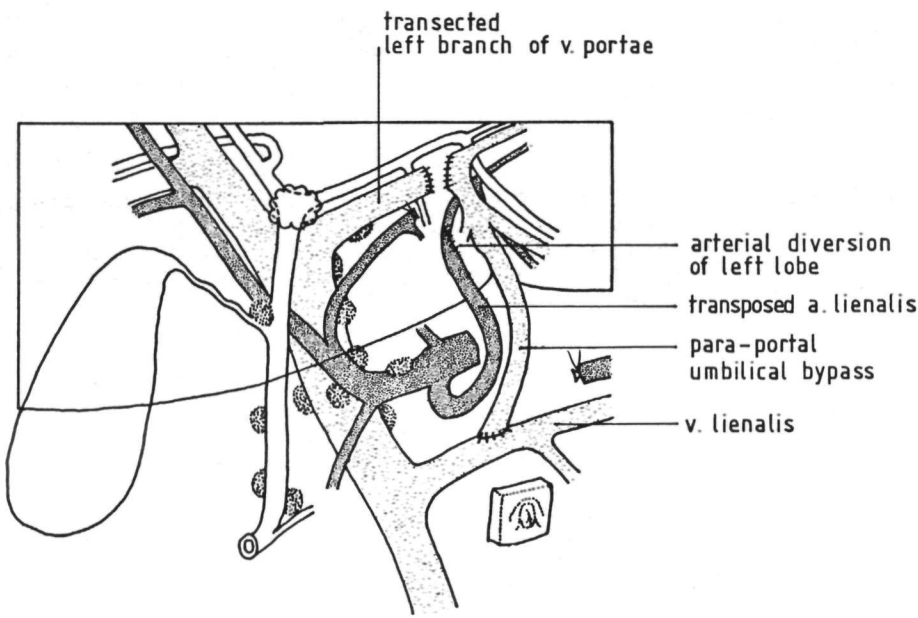

Fig. 13. Right and left liver lobes each have their own separate afferent blood supply.

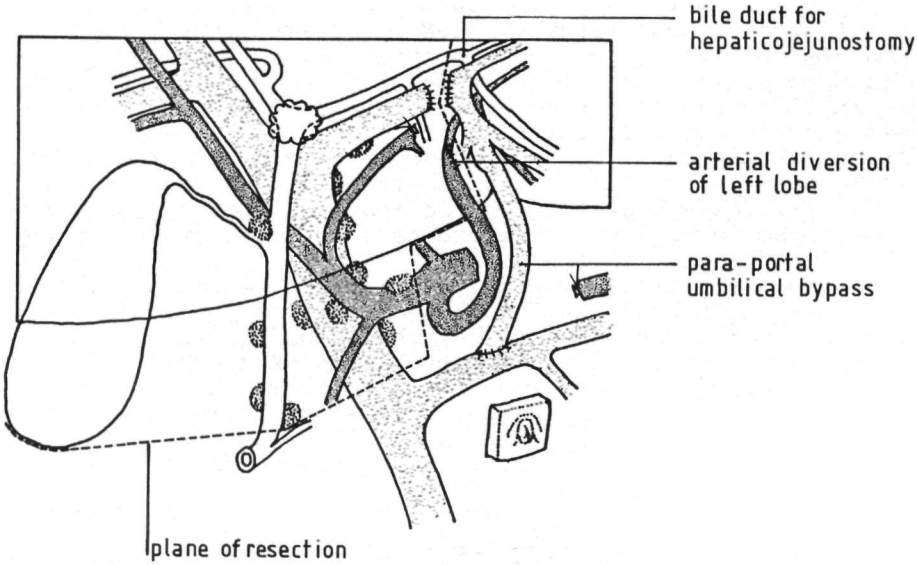

Fig. 14. Proposed radical block resection of a cancer of the hepatic duct confluence after previous construction of a complete and independent afferent vascularisation of the left lobe.

6.4. The umbilical vein

In the foetus the placental blood is conducted by the umbilical vein and ductus venosus Arantii through the liver to the inferior caval vein and the heart (326, 502). Nearly always the umbilical vein enters the portal system at the end of the left portal vein branch, rarely a transition is seen into the portal vein bifurcation or the right branch (305). During the first postnatal months the lumen of the umbilical vein reduces by proliferation of subintimal connective tissue and finally the vessel is closed (99), but the endothelium and surrounding musculo-elastic zone remain intact throughout life. Therefore the lumen of the umbilical vein can be reopened and dilated at any age for access to the portal system (26, 70, 81, 82, 99, 607, 608).

The vein runs from the umbilicus in cranial direction just dorsal to the right side of the linea alba. Halfway between the umbilicus and xiphoid process the collapsed vessel bends and continues in the ligamentum teres to the blind end of the recessus of Rex in the umbilical fissure of the liver. The intraperitoneal stretch of the umbilical vein measures 12 cm (range 8.5 to 20 cm) and has no side branches or valves.

Its microscopic structure is similar to that of other veins (81). At the umbilico-portal junction the tunicae mediae and adventitiae of both vessels merge into each other but the lumen is obstructed by a well defined, 1 cm wide fibrocollagenous plug, which may be a remnant of an umbilico-portal valve. In 95 to 97% of postmortem specimens, the lumen of the vein and the umbilico-portal junction can be reopened and dilated instrumentally to a diameter of 5 to 8 mm without false passage (26, 40, 81). A pressure over 60 mm Hg opens the lumen as well, except at the umbilico-portal junction which remains closed even after exposure to a pressure of 300 mm Hg (81).

In vivo the umbilico-portal junction can become as wide as the portal vein under influence of a chronic portal hypertension, as is the case in liver cirrhosis and Cruveilhier-Baumgarten syndrome (346). In general an open umbilical vein carries the portal blood away from the liver but in 9% of the cases the open umbilical vein serves as an inflow tract (92).

The technique of external dilatation of the umbilical vein to gain access to the portal vein for diagnostic or therapeutic purposes is described in detail (26, 70, 82, 94, 132, 336, 337, 598, 610, 611). Usually the vein is entered at the upper end of its extraperitoneal course and is dilated with Bakes' dilators. Although at that level the lumen is smaller than in the intraperitoneal part of the vein the cannulation is successful in 90 to 95% of the patients (306, 338, 611). This procedure is applied for direct hepatoportography (232, 237, 305, 337, 598, 611), for investigations on selective pressure, flow and metabolism of the portal system (610), for the administration of chemotherapeutics (305, 536, 543, 598) or antibiotics (447) and for temporary portal decompression (607). For portal decompression an open umbilical vein can be anastomosed with a systemic vein (345, 347, 447, 598), although such a shunt is only incidentally reported to be successful on the long term (576). The umbilical vein is anastomosed with various arteries to arterialise the intrahepatic portal system (1, 2, 96, 202), while for liver transplantation the umbilical vein has been proposed as inflow tract for the portal blood (155).

Conclusion

According to literature in about 95% of cases, the umbilical vein can probably be opened and anastomosed with another vein of the extrahepatic portal system. Even if construction of such an alternative connection between the intra- and extrahepatic portal systems is technically feasible, it remains uncertain whether it can properly take over the function of the portal vein.

6.5. Access to the base of the portal vein and the central parts of the splenic vein

The base of the portal vein and the posterior pancreatoduodenal nodes are reached by mobilisation of the right colon

flexure and dissection of the avascular plane between the right kidney, right adrenal gland, inferior caval vein and aorta at the dorsal side and duodenum, head of the pancreas, mesenteric root and superior mesenteric vessels at the ventral side, the so-called 'extended Kocher manoeuvre' (98, 130, 327, 415, 444). The central part of the splenic vein is exposed at its cranial side by incision of the peritoneum at the base of the lesser sac along the cranial margin of the pancreas. Apart from the splenic vein and the portal confluence, the plane between pancreas and posterior abdominal wall is almost avascular. The splenic artery and the splenic vein are brought into view by pulling the pancreas in a caudal direction.

Many small, short veins enter the splenic vein from the posterior side of the pancreas, so dissection of the splenic vein should be confined to an area at its cranial side necessary for the lateral anastomosis with the umbilical vein. Full dissection of the splenic vein from the left side of the pancreas is required if transposition of this vein to the liver is wanted in case the umbilical vein is too short or not available. To that end mobilisation of tail and body of the pancreas is the best approach after distal transection of the splenic vein (47, 134).

6.6. Vascular isolation

Radical resection as outlined in paragraph 3 of this Chapter is already facilitated by selective afferent isolation of the area to be resected. The risk of bleeding and air embolism during the retro- and suprahepatic phases of resection can be reduced by a total vascular isolation of the liver. To that end occlusion of the hepatic veins or the retrohepatic caval segment must be added to the afferent vascular isolation.

Suprarenal and suprahepatic clamping of the inferior caval vein have serious haemodynamic consequences in patients without a collateral circulation (428) and should not exceed 30 minutes (182). The circulatory load is diminished by simultaneous clamping of the aorta just above the coeliac trunk (21, 204, 248, 250, 278, 428, 620) or by an endocaval shunt. Only subhepatic insertion of an endocaval shunt, either suprarenally (89, 125, 136, 182, 568, 620) or via the femoral vein (163, 249, 551, 552) has to be considered, as the operation can probably be carried out by laparotomy only.

Suprahepatic control of the inferior caval vein can be performed just above or just below the diaphragm. In the rather simple supradiaphragmatic approach the inferior caval vein is intrapericardially encircled via a small incision in diaphragm and pericardium (248, 250, 391, 620). Subdiaphragmatic control is more difficult but has the advantage to exclude the phrenic veins. Almost 2 cm of the inferior caval vein can be dissected by careful dissection of the loose fibrous tissue around the inferior caval vein between the liver and the diaphragmatic orifice (276, 329).

Livers of dogs and pigs can stand a normothermic ischaemia of at least 45 minutes without serious consequences, provided

adequate splanchnic decompression is taken care of (324, 367, 368, 375, 422, 540, 585). Normothermic ischaemic periods of one hour are tolerated by the human liver without danger and do not interfere with the regenerative capacity (136, 160, 281, 283, 578, 593). Therefore the safe period of normothermic ischaemia of the human liver can now be fixed on 45 minutes (183, 282), ten years ago this was still believed to be 30 minutes (49, 86). Irrigation of the peritoneal cavity with a cold solution of normal saline prolongs this period (382) but no liver resection needs to last any longer (197,204,248, 358, 551).

The described methods of vascular isolation may be useful during extensive liver resections and vascular reconstructions, but for prevention of thrombosis in the reopened umbilical vein a strong and continuing perfusion through the umbilical bypass is essential. Resection of the caudate lobe will be alleviated when the portal vein is cut and deviated upwards. One of the advantages of a previously constructed portal vein diversion is the very possibility to cut and dissect the portal vein quietly. Consequently, during resection of the caudate lobe the umbilical bypass should be functioning which requires an uninterrupted venous outflow of the left lobe. Therefore, other methods should be applied for retro- and suptrahepatic venous control, if necessary.

The following procedures can be considered:
- simple, brief clamping of the suprarenal caval vein, in conjunction with positive pressure ventilation;
- use of an endocaval balloon shunt, placed in such a position that the inflated balloon selectively blocks the outlets of the hepatic veins to be cut (163, 380, 448);
- subtotal isolation of the retrohepatic caval vein as described by Raia and Huguet (276). In this method the outlets of the suprahepatic veins into the inferior caval vein are dissected until the right and middle suprahepatic veins and the inferior caval vein can be encircled together just below the outlet of the left suprahepatic vein.By pulling the encircling loop the suprahepatic caval vein and the main venous outflow tracts of the right liver lobe are blocked, while the venous outflow of the left lobe remains unhampered.
Clamping of the subhepatic caval vein completes the required subtotal isolation of the retrohepatic caval segment. The method may be hazardous but has been successfully employed (276). Its main disadvantage is the bad tolerance of suprarenal clamping of the inferior caval vein by patients without a paracaval collateral circulation. This drawback can be met by combining the method with the insertion of an endocaval shunt.

The application of the endocaval balloon shunt may be the most suitable method, as it stretches and presents the caval segments to be spared.

7. Anatomical investigation into the feasibility of a radical block resection

An anatomical study was carried out to investigate the possibilities of a radical block resection. At autopsy, of 40 adult patients who had died from other causes than liver or bile duct diseases, the liver was taken out together with the ligamenta teres and falciforme, part of the diaphragm, retrohepatic caval vein, complete hepatoduodenal ligament with gallbladder, arterial coeliac trunk, stomach antrum, pylorus, duodenum and head of the pancreas with the adjacent central splenic vein and superior mesenteric vein (F.B.Bronkhorst, pathologist). The specimens were dissected.

Soon, during dissection of the fifth specimen, the possibility became clear to use the umbilical vein, so that in the next 35 dissections the umbilical vein was reopened and the described radical block resection performed, leaving the left lobe with the inferior caval vein, umbilical vein and left hepatic artery stump. In five cases the umbilical vein was anastomosed end-to-side with the central splenic vein.

The hepatoduodenal ligament and the porta hepatis were partially or completely dissected to assess the mutual relation between vessels and bile ducts and to find the place where the structures to the segments of the left lobe took off. In this context the words'left' and 'right' indicate the left and right side of the patient with the liver in situ. The diameter of the vessels was approximately determined by stretching the lumen over the same inserted forceps. The distance between the forceps blades was measured and the area and diameter of the cross section of the lumen calculated. In some cases portograms, arteriograms, venograms or cholangiograms were made. Initially GRF-glue was applicated to prevent the leaking of contrast medium through the cut edge of the liver remnant (71, 278) but later on this was abandoned as the glue did not stick properly to the cold postmortem specimen, and no leakage was seen through the avascular umbilical scissure.

7.1. The paraportal umbilical bypass

Reopening of the umbilical vein was preceded by cutting the parenchymatous bridge which often connects segments III and IV. In this way the recessus of Rex was exposed, covered only with the left offshoot of the hilar plate and with the peritoneum. The ligamentum teres was freed from the rest of the falciform ligament and the anterior edge of the liver. By opening the upturning fold of loose fibrous tissue between the ligamentum teres and the umbilical fissure, another 1 to 2 cm could be gained.

The umbilical end of the now completely mobilised ligament was freshly transected. The collapsed umbilical vein was visible as a little white strand. By pulling the sides of this

strand with two vascular forcepses an intima-lined lumen was found, in which a closed, moistened pair of slender, blunt scissors could be inserted. Spreading of the scissors opened the lumen. While withdrawing the scissors were closed and after moistening they were re-inserted and spread on a somewhat deeper level perpendicularly to the former dilatation, and so on. The delicate vein walls easily gave way. The procedure had to be carried out cautiously and with careful identification of the lumen to prevent intimal damage or perforation.

At the transition into the recessus of Rex there was more resistance to the dilating scissors. By carefully proceeding step-by-step the umbilico-portal junction could be penetrated easily which was indicated by the appearance of blood in the umbilical vein and by a sudden lack of resistance to the intrusion of the scissors. Opening of the scissors in all directions somewhat more vigorously than before resulted in an equal dilatation of the junction. To prevent a false passage at this level, it was important to keep the ligamentum teres stretched in a direct line with the recessus of Rex, which in vivo runs almost anteroposteriorly. The spreading of the scissors in the depth of the vein could be facilitated by wrinkling the distal portion of the ligamentum teres somewhat down over the inserted scissors.

The procedure could easily be performed in about ten minutes. A careful and atraumatic technique is essential since the feasibility of the proposed resection virtually depends on the outcome of this dilatation.

In all 40 livers the umbilical vein joined the left portal vein branch at the end of the recessus of Rex. In 32 of the 35 specimens (91%) the umbilical vein could be reopened and the left portal vein easily be reached (Fig. 15). Three times a false passage was made at the umbilico-portal junction. During the dilatation of these junctions, the ligamentum teres was held angled to the recessus of Rex. A reopened umbilical vein with a mean length of 10 cm could be obtained, in 16 of the 17 specimens with a ligamentum teres that had been cut close to the abdominal wall during autopsy. The distance between the umbilico-portal junction and the splenic vein was 7 to 8 cm if measured at the central side and decreased to about 6 cm if measured at levels more upstream the splenic vein. The available length of the umbilical vein was ample sufficient to bridge this distance. Once a usable lumen could be found only in the last 4 cm before the umbilico-portal junction. The mean diameter of the umbilical vein was 8.9 mm but decreased at the umbilico-portal junction to 7.7 mm. As the mean diameter of the left portal vein branch, just before the beginning of the recessus of Rex, was 8.2 mm this means that the umbilical vein is virtually as wide as the left portal vein branch (Table XXXI).

After longitudinal cutting of the umbilico-portal junction two or three saccular dilatations were vaguely seen, resembling valve rests. In the junction the intima lining was interrupted circularly for a distance of 2 to 3 mm, but both vessels clearly shaded off each other macro- as well as microscopically (Fig. 16).

Table XXXI. Lengths (in cm) and mean diameter (in mm) of a number of portal vessels measured in normal postmortem specimens.

Portal vessels	length/diameter	no. of specimens
length		
portal vein	mean 6.8 (range 6 to 9)	9
umbilical vein	mean 10.3 (range >6 to >13)	16
diameter		
portal vein	mean 10.2 (range 8.4 to 11.0)	14
left portal vein branch	mean 8.2 (range 7.3 to 9.0)	14
central splenic vein	mean 7.9 (range 6.7 to 9.4)	10
umbilical vein	mean 8.9 (range 7.3 to 9.9)	13
umbilico-portal junction	mean 7.7 (range 6.7 to 8.7)	21

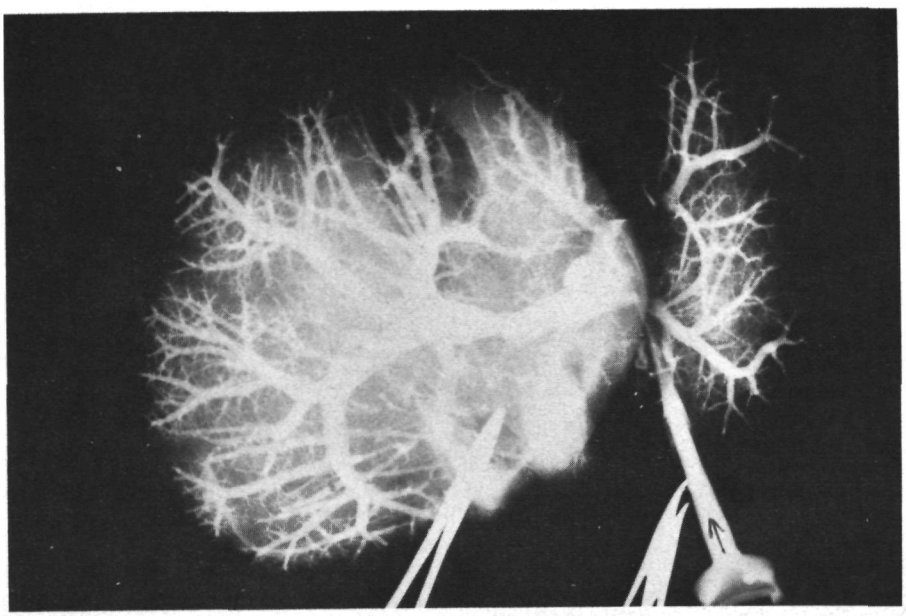

Fig. 15. Portogram of a postmortem liver made via the reopened umbilical vein, showing equal distribution of the contrast material over the complete hepatic portal system. The portal vein has been clamped just upstream of its bifurcation, indicated by forceps.

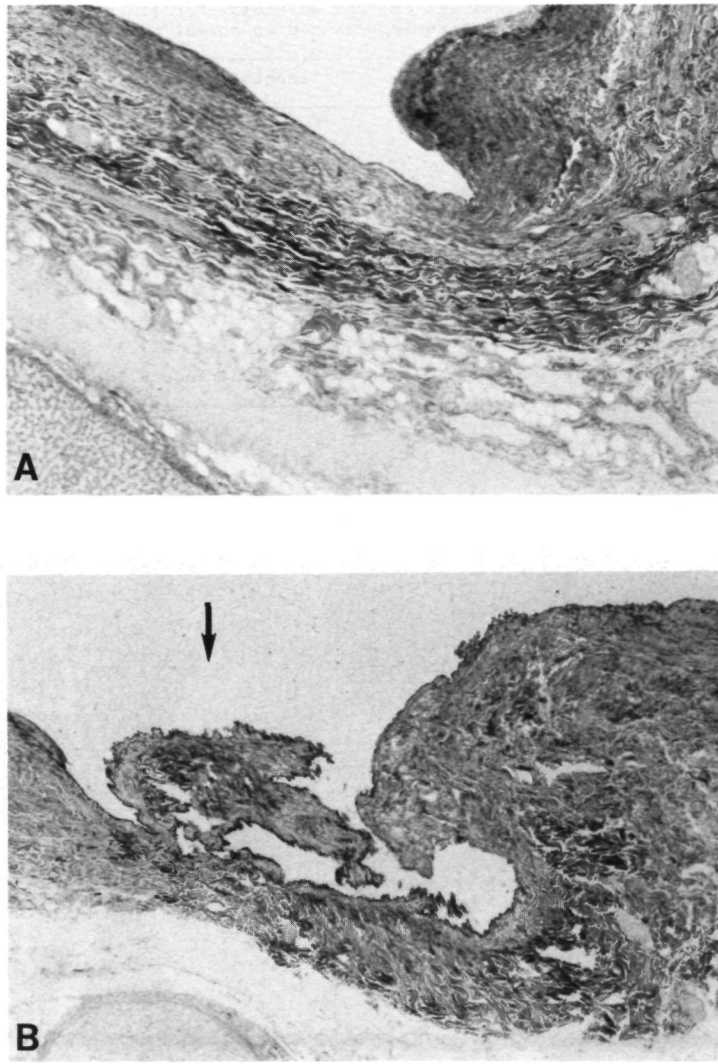

Fig. 16. Transitional zone between a branch of the portal vein or the recessus of Rex (left) and the umbilical vein (right)· A in the vicinity of the portal vein hepatic tissue can be recognized (lower left portion of the field), continuity can be seen between the different coats of both veins; B Reduplication of the lining membrane or valve-like structure protrudes within the lumen (⟶), part of the liver is visible in the lower left portion of the field (El.van Gieson, x36).

After assessment of the required length of the bypass, the ligamentum teres was transected obliquely and the last centimetre of the umbilical vein was carefully freed of surrounding fat. A Satinsky clamp was placed laterally on the splenic vein and an end-to-side anastomosis was made between the umbilical and the splenic vein with a running Prolene(R) 5x0 suture for the posterior side and with interrupted stitches for the anterior side (Figs. 11 and 17).

7.2. The left liver lobe and its subhilus

Portal system

When the left corner of the porta hepatis was dissected, the transition of the left portal vein branch into the recessus of Rex was the first structure encountered after opening of the peritoneum and the hilar plate. The artery to the left lobe was situated at some distance to the left of the left portal vein branch. At the level of the umbilical vein the left hepatic duct or its main branches were in cranial position to the portal vein branch.

As the loose perivascular tissue provided a well dissectable plane, cautious encirclement of the vein at this level could be performed with a dissecting forceps. This dissection should be done carefully as a tear in the vein on this poorly accessible place will present major problems. Two slender vascular clamps were placed in anteroposterior direction and the vein was transected, leaving outside the clamps cuffs of vessel wall just long enough to warrant a closure by vascular sutures. The level of this transection was at a mean distance of 4 cm from the portal vein bifurcation (Table XXXII). Portograms made after completion of the resection via the reopened umbilical vein, showed complete filling of the portal system of the left lobe (Fig. 18).

Arterial system

The proper hepatic artery usually divided about halfway the hepatoduodenal ligament. The last part of the arterial branch to the left lobe had a diameter of about 2.5 mm and ran at some distance caudally to the left portal vein branch and rather superficially in the hepatoduodenal ligament or lesser omentum. It entered the left lobe dorsally to the base of the recessus of Rex. In three of the 13 examined specimens the artery divided before entering the left lobe (Table XXXII). Arteriograms made via the arterial stump showed complete filling of the arterial tree of the left lobe (Fig. 18).

Table XXXII. Data on the anatomy of the vascular and biliary structures of the subhilus of the left liver lobe in the umbilical fissure. (number of examined specimens in brackets).

mean distance between portal vein bifurcation and transection margin of left portal vein branch	(14)	4.1 cm (range 3.5 to 5 cm)
mean distance between confluence of the hepatic ducts and transection margin of left hepatic duct(s)	(13)	4.3 cm (range 3 to 5 cm)
minimal mean length of left hepatic duct	(8)	3.4 cm (range 1 to >5 cm)
position of confluence of segmental ducts II and III in relation to the umbilical fissure	(15)	6 times to the right 9 times to the left
position of the left hepatic duct(s) in relation to the left portal vein branch in the umbilical fissure	(14)	cranial or duct II dorsal duct III cranial
position of the bifurcation of the segmental arteries II and III in relation to the umbilical fissure	(13)	3 times to the right 10 times to the left
position of the left lobe artery (branches) in relation to the base of the recessus of Rex	(11)	dorsal

Biliary system

When followed upstream, the left hepatic duct shifted gradually from a position anterior to the left portal vein to a cranial position. At the umbilical fissure the duct was cranial to the vein and the base of the recessus of Rex. The confluence of the bile ducts of segments II and III was situated at the right side of the umbilical fissure in six of 15 examined specimens (40%) (Table XXXII). In vivo a patient with such a late secondary confluence will require two separate biliodigestive anastomoses after transection of the liver in the proposed way.

When the left portal vein branch had been divided the hepatic duct(s) to the left lobe was (were) encountered. No proper dissectable plane existed between the duct and the strongly adherent liver capsule posterior to the duct. However, it was simple to encircle the duct by penetrating the capsule or to transect directly the duct and the adjacent capsule with a knife. The mean distance between the transection level of the duct and the main confluence was over 4 cm (Tabel XXXII). Cholangiograms made via the remnant of the left hepatic duct showed complete filling of the biliary tree of the left lobe (Fig. 18).

Parenchymatous transection and suprahepatic venous control

After transection of the left branches of the portal vein, hepatic artery and hepatic duct, the parenchymatous part of right lobectomy was carried out just to the right side of the umbilical scissure. The portal branches from the recessus of Rex to segment IV were carefully dealt with since in practice

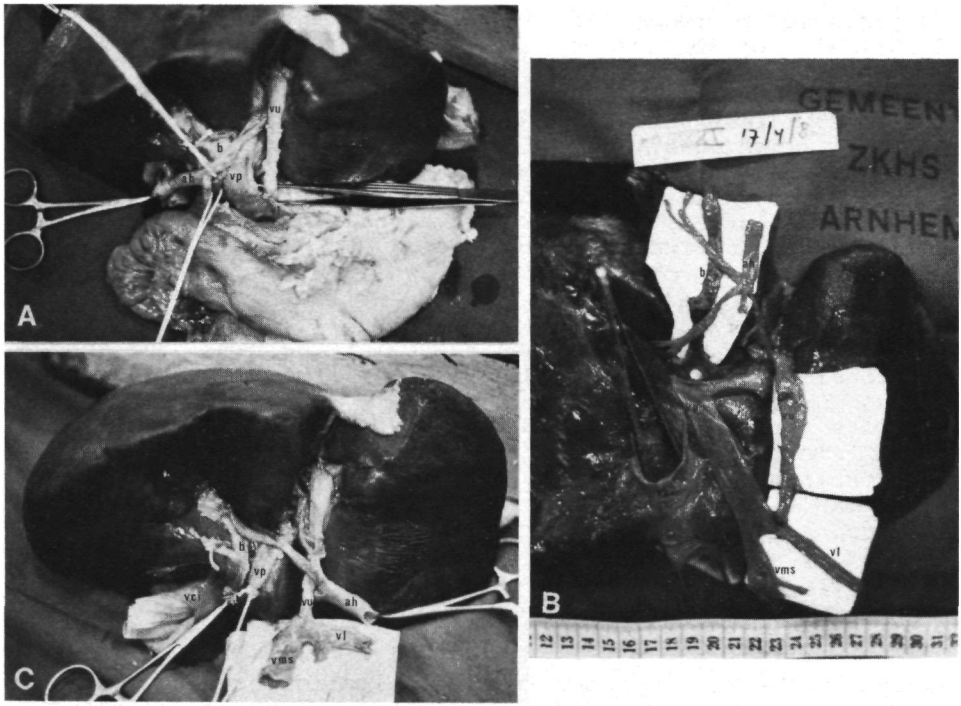

Fig. 17. Postmortem liver: A. reopened umbilical vein, anastomosed end-to-side with the central splenic vein; B. bile duct and hepatic artery have been turned upwards to show the portal vein and the paraportal umbilical bypass (see also Fig. 11); C. portal vein transected.

ah = a.hepatica
vci= v.cava inf.
vms= v.mesent.sup.
vu = v.umbilicalis
b= bile duct
vl= v.lienalis
vp= v.portae

these will be the only perfused afferent vessels to the area to be resected. The largest of these branches had a mean diameter of 6 mm (range 5 to 8 mm) (6 specimens) and could be transected at 1 cm distance from the recessus.

Both the middle suprahepatic vein, entering the inferior caval vein or the left hepatic vein, and the huge right suprahepatic vein were divided at intrahepatic level, which in practice is safer than extrahepatic control (66, 197, 284, 528) and moreover guarantees an undamaged left suprahepatic vein (4). Venograms made via the orifice of the left suprahepatic vein showed complete filling of the venous suprahepatic tree of the left lobe (Fig. 18).

In the specimens, resection of the caudate lobe could not be performed properly. The lobe was situated at the craniodorsal side of the rest of the liver, anterior to and partially around the left side of the inferior caval vein. In some specimens the lobe even lay near to the caudodorsal side of the left suprahepatic vein. As the lobe was so closely related to the dead, flaccid inferior caval vein an intraparenchymatous resection was mostly performed, leaving a cuff of liver tissue on the vein. Proper assistance is indispensable for this resection, and an endocaval balloon that stretches and presents the inferior caval vein will be helpful.

The mean weight of the left lobe was 330 gram (range 145 to 730 gram) which was 22% (range 11 to 33%) of the complete liver (mean weight 1500 gram; range 1000 to 2850), 28 spec.

By the end of dissection and resection the remaining specimen consisted of the left hepatic lobe with its suprahepatic venous outflow tract to the retrohepatic caval vein undisturbed, its portal inflow tract displaced to the reopened umbilical vein, its hepatic duct ending in the umbilical fissure and its artery cut 2 cm from there (Fig. 19). The plain of resection remained at least 4 cm from the hepatic duct confluence. Angiograms showed that the left lobe was supplied completely via the remaining structures and that no other afferent or efferent channels existed.

7.3. Conclusion

From the results of postmortem investigations can be concluded that after construction of a complete and separate afferent blood supply of the left lobe, radical block resection of the area of the hepatic duct confluence, the right liver lobe and the complete hepatoduodenal ligament is possible both anatomically and technically. No indications could be found to deny the supposition that the result of such a procedure is functionally equal to a standard right lobectomy with bilio-enteric reconstruction.

Nevertheless the capacity of the paraportal umbilical bypass in action is as yet unknown. An animal experiment to investigate the efficacy of a paraportal bypass and its application together with arterial revascularisation and liver resection was carried out and will be described in Chapter 8.

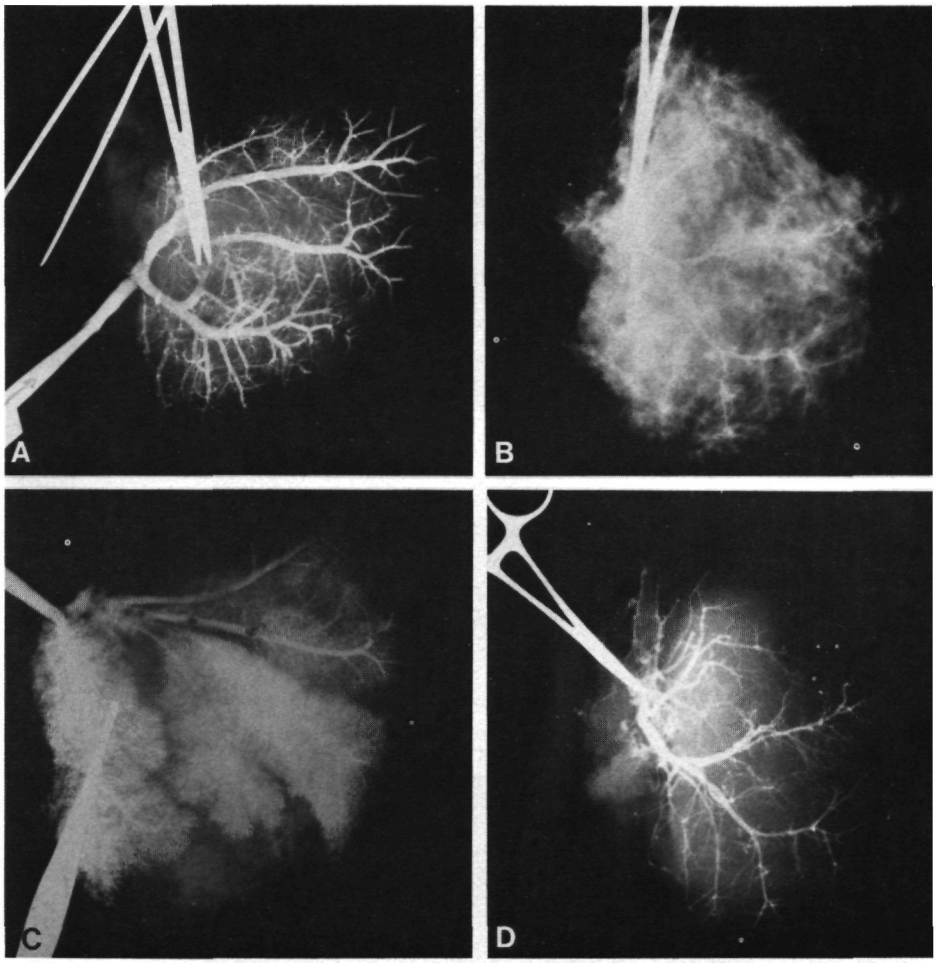

Fig. 18. Postmortem left liver lobe: A. portogram (injection of contrast fluid into the re-opened umbilical vein); B.arteriogram (injection of contrast fluid into the arterial remnant to the left lobe); C. venogram (injection of contrast fluid into the caval orifice of the left supra-hepatic vein); D. cholangiogram (injection of contrast fluid into the left hepatic remnant at the umbilical fissure).

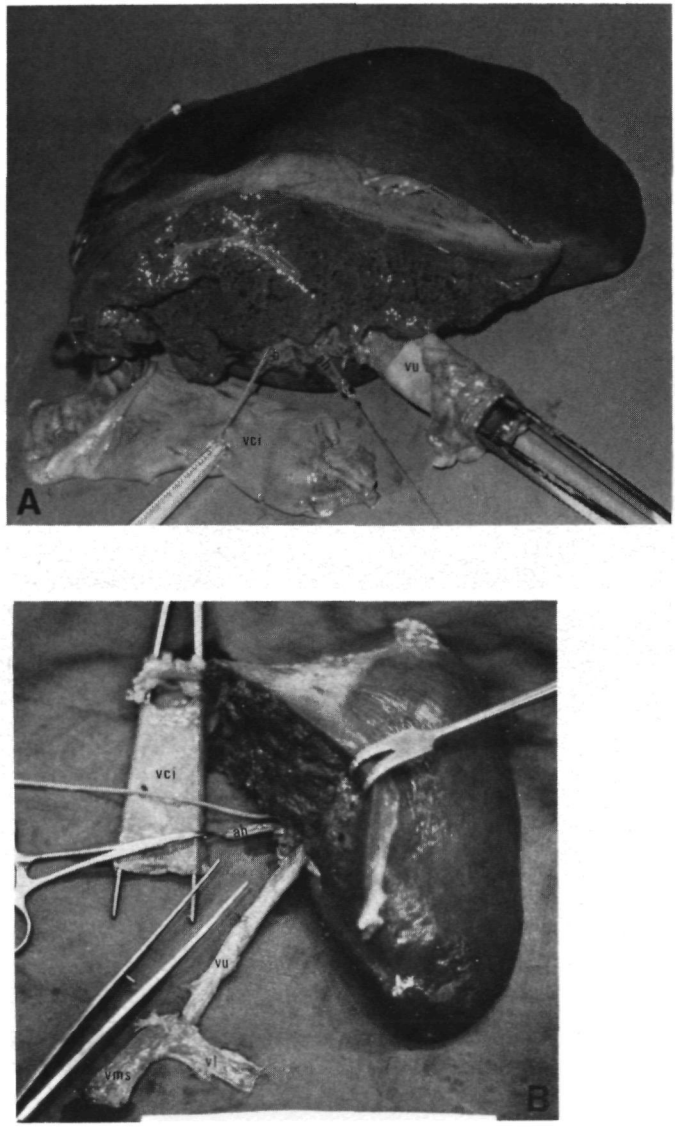

Fig. 19. A. Left liver lobe with access to portal venous system via reopened umbilical vein (forceps inserted), and with left arterial remnant. The left hepatic duct has been cut flush with the parenchyma; B. result after reconstruction of a paraportal umbilical bypass and radical resection of right liver lobe, caudate lobe and the ligamentum hepatoduodenale in a postmortem specimen (see also Fig. 14).

8. Transfer of the afferent hepatic blood supply and hemihepatectomy in the dog

Although the operation proposed is theoretically, anatomically and technically feasible, the practical value has to be proven in vivo. Right lobectomy with bilio-enteric reconstruction is an established procedure and transplantation surgery has demonstrated that all structures which enter and leave the liver can be transected and reconstructed both in animals and in man. The only new element is the reconstruction of a second portal inflow tract to the liver which allows subsequent transection or resection of the portal vein. Therefore an investigation was necessary especially into the action of a paraportal bypass and into the results of partial hepatectomy with transposition of the portal and arterial blood supply to the liver remnant.

The best results of such an investigation would be obtained by performing the same operations in animals as proposed in men, but unfortunately no proper umbilical vein could be found in the common laboratory animals. The only remnant of the umbilical vein in cadavers of pigs, dogs, goats, sheep, rabbits and monkeys consisted of a small, fibrotic strand without a lumen between the left and right liver lobes. Two alternative options remained to bypass the portal vein, namely 1 interposition of an autologous vein graft between the splenic vein and a portal branch beyond the portal bifurcation, and 2 transposition of the splenic vein to such a portal branch. The latter procedure was chosen for being the simplest of the two.

After a pilot study and postmortem investigations in various animals, a protocol was set up. For lack of the human umbilical access to the hepatic portal system, a different but comparable approach had to be found in the animal. Still this approach had to provide the possibility that the paraportal bypass could be constructed prior to transection of the portal vein and without interruption of the portal blood supply to the liver part that would remain after partial hepatectomy. The dog was found to be the most suitable animal as the liver lobes are divided by deep fissures and the portal vessels are large enough to enable macroscopic surgery. Apart from the caudate lobe, all six lobes each have a subhilus, situated at the bottom of these fissures. The dissection of the splenic vein is facilitated by the intraperitoneal position of pancreas and duodenum. Lastly the dog is very sensitive to portal hypertension (10, 129, 209, 459), and so a portal procedure successful in the dog could be expected to be successful in man as well.

8.1. Outline of the operation

In dog cadavers, access to the hepatic portal system could be obtained by dissection of the portal branch of a liver lobe up

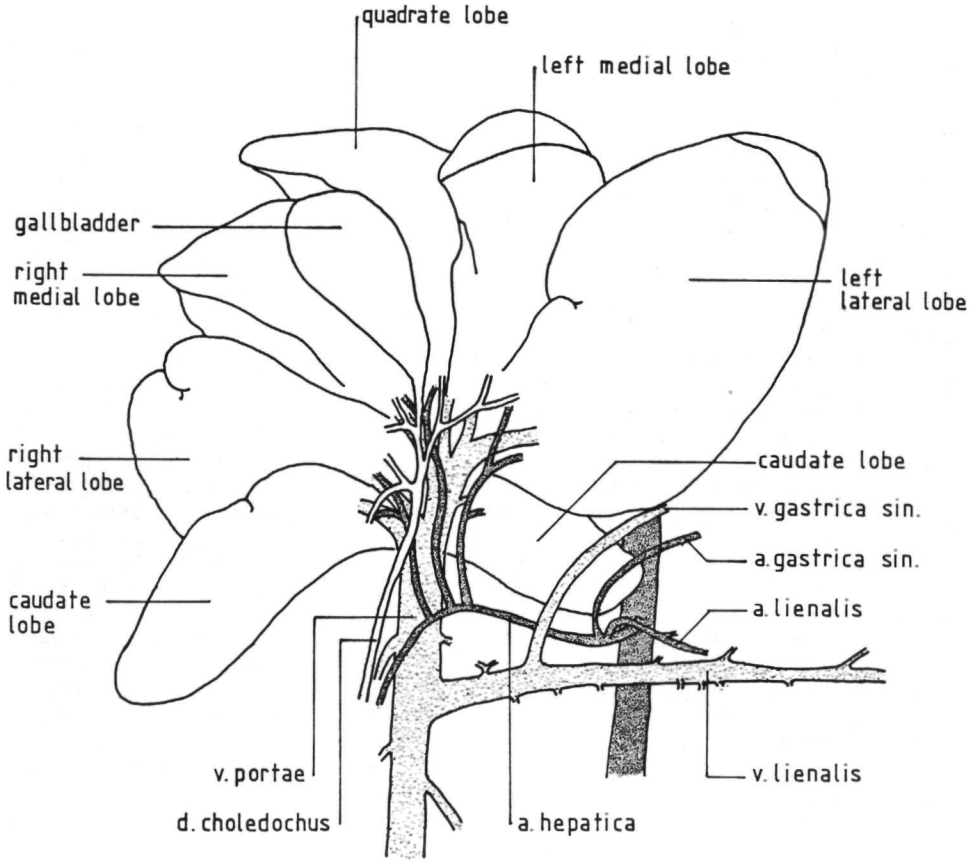

Fig. 20. Anatomy of the liver and its afferent supply in the dog.

into the liver tissue. After transection of this vessel at an intrahepatic level, the part at the central side could be anastomosed end-to-end with the distal end of the mobilised splenic vein. After clamping the portal vein the portal blood was diversed through the transposed splenic vein and the anastomosis into the dissected portal vein branch and from there on to the liver hilus and the rest of the liver (Figs. 20-23).

Since extended liver resections are already recognised operations in man a 50% hemihepatectomy was opted for, to de-

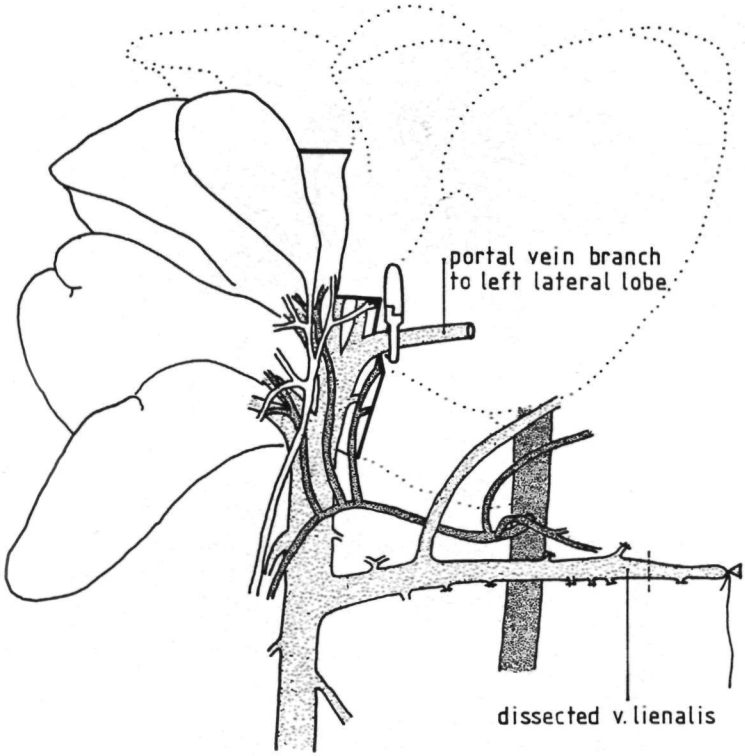

Fig. 21. Situation after dissection of the portal vein branch to the left lateral lobe, left hemihepatectomy and dissection of the splenic vein.

crease the risks of animals dying from the mere resection. The portal vein branch to the left lateral lobe was used for the paraportal bypass being the largest portal branch available in dogs. Consequently a left hemihepatectomy was performed in this animal experiment (instead of right lobectomy as proposed in Chapter 6), leaving the right hemi-liver in situ. After transection of the portal vein the right liver remnant was supplied with portal blood by the splenoportal bypass alone. The required procedure of partial hepatectomy

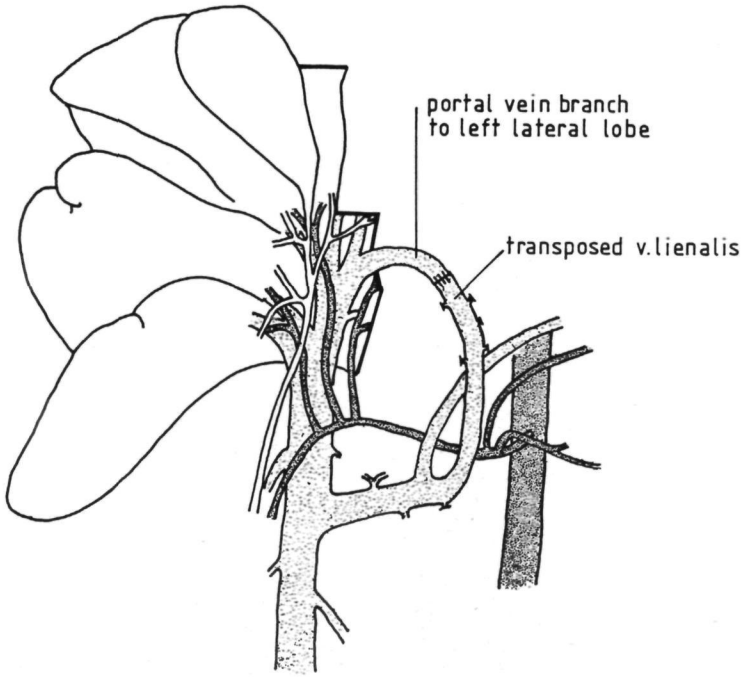

Fig. 22. Left hemihepatectomy and paraportal bypass, constructed by end-to-end anastomosis between the mobilised splenic vein and the dissected portal vein branch to the left lateral liver lobe. The retained right hemi-liver has a double portal inflow tract.

with diversion of the entire afferent blood supply was completed by transposition of the distal part of the hepatic artery to the central part of the splenic artery (Fig. 24).

8.2. Operations performed

In 23 dogs (Beagle, weight 8 to 15 kg), 46 operations were carried out. Anaesthesia was induced by sodium pentothal i.v. and maintained by a mixture of N_2O/O_2/ethrane, administered by a closed ventilation system after endotracheal intubation. During the procedure arterial and portal pressure, ECG and rectal temperature were monitored, angiography of the portal system performed and a liver biopsy taken. The dogs were placed supine on a heated mat and received up to 1000 ml sodium-glucose solution during operation. Autopsies were performed in all animals and the portal and arterial anastomoses were microscopically examined as were the liver specimens. No blood transfusion or other treatment was administered.

In 15 dogs a left hemihepatectomy, construction of a para-

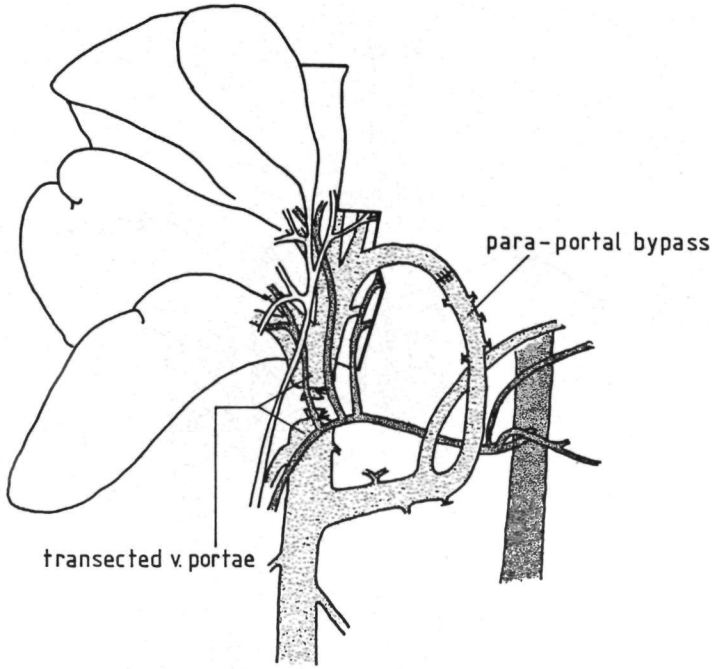

Fig. 23. Left hemihepatectomy, construction of a paraportal bypass and transection of the portal vein. The portal vein is transected if the paraportal bypass appears to function properly.

portal bypass and subsequent transection of the portal vein were performed four died shortly after the operation. After four to six weeks the remaining 11 dogs underwent reoperation, in eight a hepatic artery diversion was performed. After another four to six weeks these animals were operated and subsequently sacrificed, except for one dog that underwent arterial transfer at that time.

In six dogs left hemihepatectomy, portal and arterial diversion were done in one session. All died. In two dogs left hemihepatectomy was combined with arterial transfer only, in one in a single session and in the second arterial diversion was carried out four weeks after liver resection. These two animals underwent reoperation and were subsequently sacrificed four weeks after arterial transfer.

Four to six weeks after reconstruction, the paraportal bypass was examined in 10 dogs and in nine of this series again after another six weeks. The arterial diversion could be examined four to six weeks after being laid out in nine dogs.

In six dogs, a biliary diversion by way of an anastomosis between the gallbladder and a Roux-Y jejunal limb was added

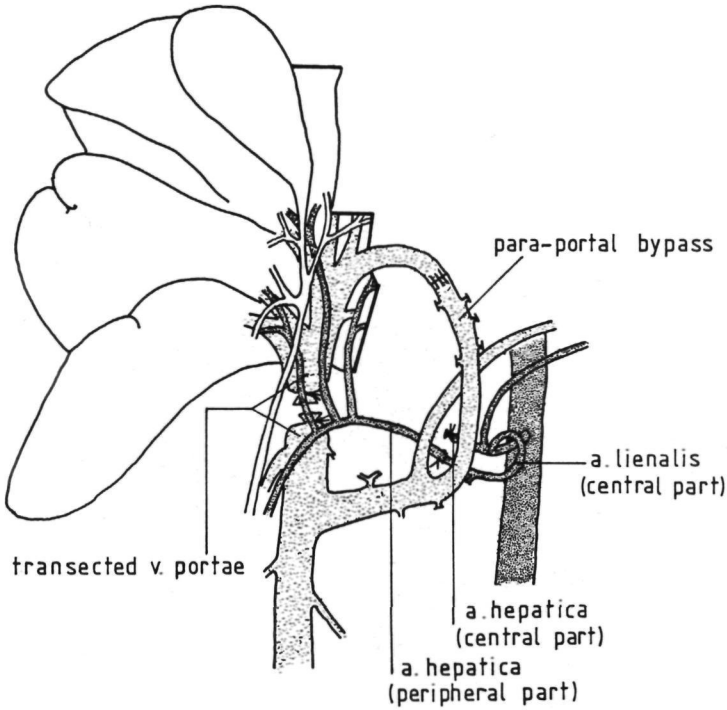

Fig. 24. Left hemihepatectomy with diversion of the afferent blood supply to the retained right hemiliver in the dog. The portal vein has been replaced by a peripheral splenoportal bypass and the hepatic artery has been transected and reanastomosed with the splenic artery.

to the hepatectomy and vascular diversion. Later on this was abandoned as being irrelevant for this study (Table XXXIII).

8.3. Hemihepatectomy and paraportal bypass (21 dogs)

Procedure

A left hemihepatectomy and construction of a paraportal bypass were performed in 21 dogs. After median laparotomy a catheter (internal diameter 1.3 mm) was inserted in a mesenteric vein and guided into the portal vein. The portal pressure was recorded and an arteriogram was made of the portal vein and its intrahepatic branches (Figs. 20 and 25). The portal vein was encircled and the left hepatic lobes mobilised. Then the portal vein branch to the left lateral lobe was scrupulously dissected from the surrounding liver parenchyma, carefully clamped at its base and transected as far distally as possible. This provided a 1 to 2 cm long remnant of a prior intrahepatic portal vein branch with a diameter of 5 mm, suitable for an anastomosis. A standard 50% left hemihepatectomy (509)

Table XXXIII. Operations performed in dogs: left hemihepatectomy (h), construction of paraportal bypass and transection of portal vein (p), construction of arterial diversion (a), construction of biliary diversion (c).

Dog no.	first operation	second operation 4-6 weeks after 1st	third operation 4-6 weeks after 2nd
1	h + p	c	sacrifice after laparotomy
2	h + p	c	-
3	h + p	c	a
4	h + p	a + c	sacrifice after laparotomy
5	h	a	sacrifice after laparotomy
6	h + p + a	-	-
7	h + p + a + c	-	-
8	h + p + a + c	-	-
9	h + p	-	-
10	h + p	a	sacrifice after laparotomy
11	h + p	-	-
12	h + p	a	sacrifice after laparotomy
13	h + p	a	-
14	h + p	-	-
15	h + a + p	-	-
16	h + a + p	-	-
17	h + p	-	-
18	h + p	a	sacrifice after laparotomy
19	h + p	a	sacrifice after laparotomy
20	h + p	a	sacrifice after laparotomy
21	h + p	a	sacrifice after laparotomy
22	h + p + a	-	-
23	h + a	sacrifice after laparotomy	

was then carried out by removal of the left lateral and left medial lobes and half of the quadrate and caudate lobes (Figs. 21 and 26).

Fig. 25. (page 104) A. Normal portogram of the dog showing the splenic vein coming in from the left side of the dog and the portal vein with its hepatic branches; B. portogram just after left hemihepatectomy, construction of a peripheral splenoportal bypass and transection of the portal vein; C. portogram one month after left hemihepatectomy and diversion of the portal blood by a peripheral splenoportal bypass; D. portogram two months after C.

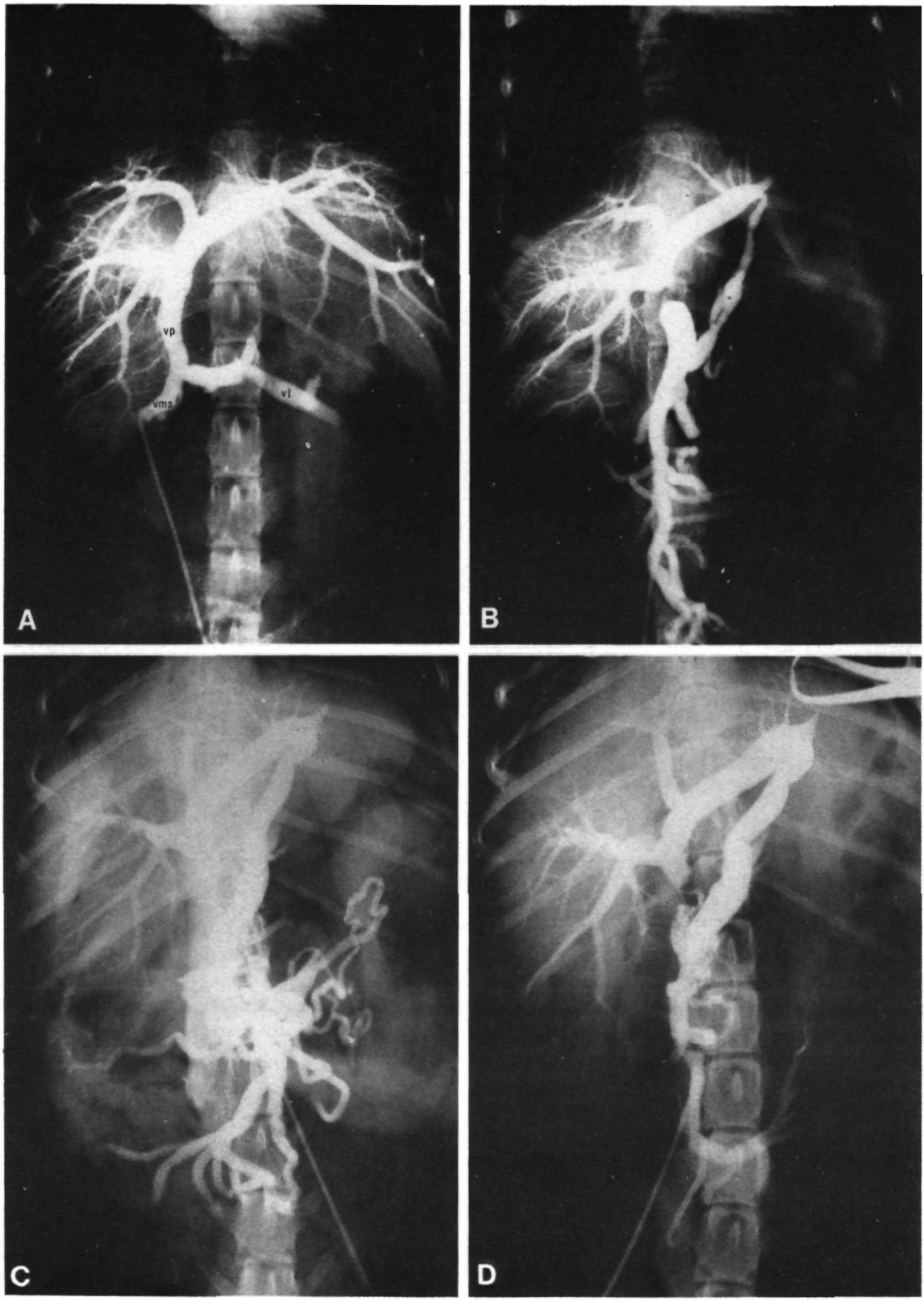

Fig. 25.

The splenic vein (5 mm wide at that level) was carefully dissected and distally cut. After injection with a heparin solution the distal side was ligated (Fig. 26) and moved cranially, behind the stomach, up to the liver and the prepared portal stump. Stabilised by a vascular clamp, the very thin cut edge of the splenic vein was adapted to the portal branch by three stay sutures at 120° distance from each other. Subsequently both vessels were end-to-end anastomosed with interrupted Prolene (R) 6x0 stitches and with microvascular instruments. The clamps were removed and simultaneously the portal vein was closed by pulling the surrounding loop. In this way the portal blood was forced to follow the new hepatic inflow tract. The portal pressure was recorded and the bowel congestion judged. Now the retained liver lobes had a double portal inflow tract (Fig. 22). Only when the bypass was considered to function properly the portal vein was transected, a portogram performed and the abdomen closed (Figs. 23, 25 and 26).

Results

The mean normal portal blood pressure was 6 mm Hg (range 4 to 9 mm Hg) and the mean outside diameter of the portal vein 11 mm (range 10 to 12 mm). Hemihepatectomy caused no change in portal or arterial blood pressures. After opening of the paraportal bypass and closure of the portal vein, the portal pressure rose in all dogs. If the pressure exceeded 30 mm Hg a marked bowel congestion developed and the arterial pressure dropped as described in total occlusion of the portal vein (29).

In all dogs the construction of the bypass was successful. The lumen of the splenic vein was, however, rather unequally narrowed and in all veins stretches not wider than 3 mm remained. Nevertheless, removal of adventitial strings and especially careful grazing of the wall with a pair of vascular pincers resulted in a lumen wide enough to produce a fair decompression in all but one dog. In this dog, the second in the series, the pressure remained 31 mm Hg because of a poor anastomoses. Apparently this bypass provided a decompression

Fig. 26. (page 106) A. Portal vein branch to left lateral lobe dissected between left medial, left lateral and caudate lobes; B. situation after left hemihepatectomy with bulldog on preserved hilar stump of left lateral portal vein branch; C. mobilised and distally transected splenic vein; D. splenic vein transposed to liver hilus and anastomosed end-to-end with hilar part of portal vein branch to resected left liver lobe, forming a peripheral splenoportal bypass.

LML = left medial lobe
LLL = left lateral lobe
CL = caudate lobe
pvb = portal vein branch
vl = v. lienalis
→ = dissected portal vein branch

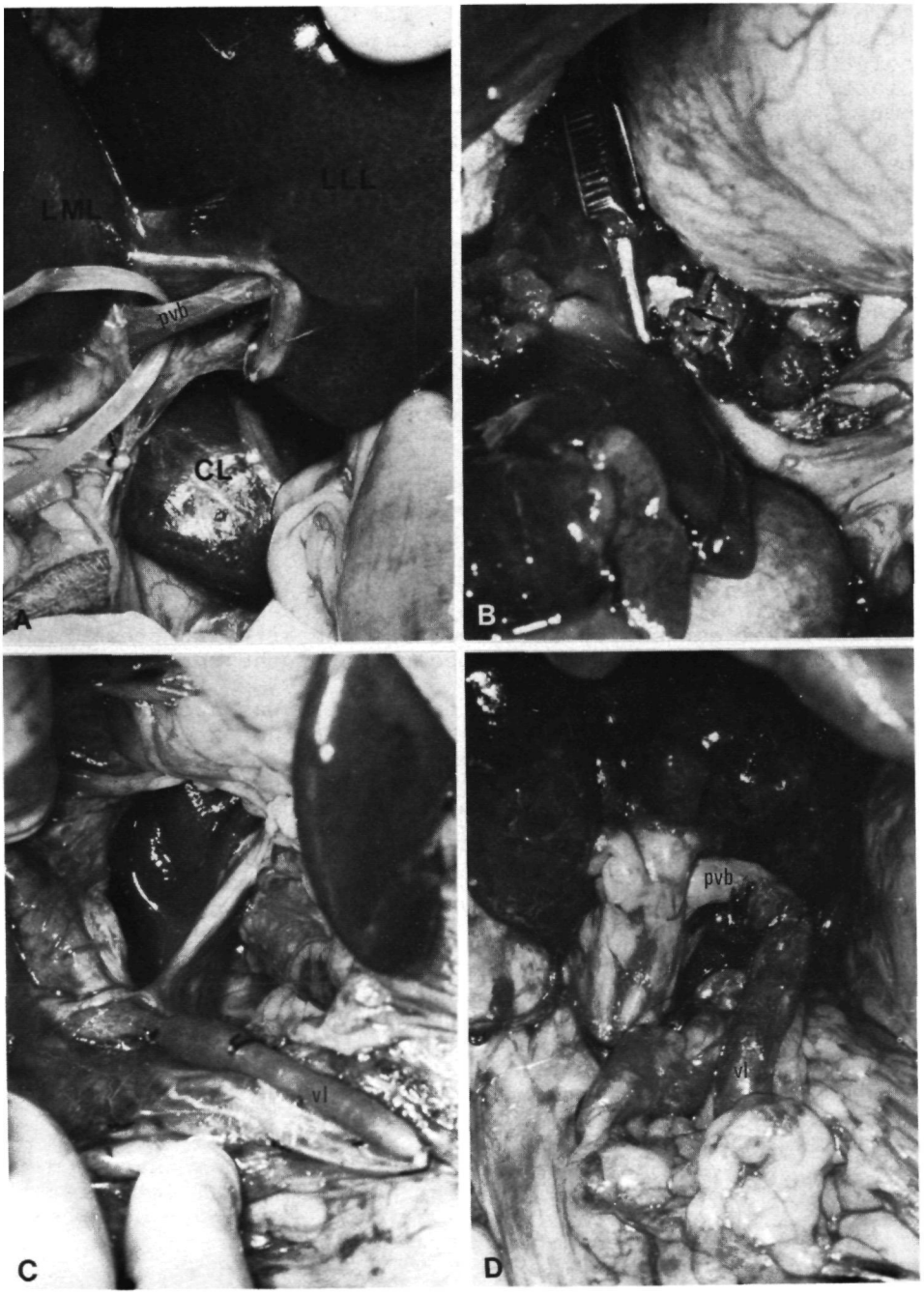

Fig. 26.

Table XXXIV. Development of portal pressure (mm Hg) in dogs after hemihepatectomy and diversion of the portal blood to the retained hemi-liver via a paraportal bypass, consisting of the splenic vein and the left lateral portal vein branch.

Dog no.	original	just after bypass reconstruction	after bypass construction	
			after 4-6 weeks	after 2-3 months
1	4	18	10	6
3	9	15	3	6
4	5	13	4	6
10	7	16	6	5
12	5	15	7	6
13	5	12	7	-
18	6	7	5	4
19	6	12	7	7
20	5	8	6	5
21	4	6	4	6
mean (n = 10)	6	12	6	6

just enough immediately after reconstruction, but this bypass was closed and replaced by a bunch of collateral vessels after six weeks. In the other 20 dogs the mean portal pressure rose to 13 mm Hg (range 6 to 22 mm Hg) with a slight bowel congestion, there were no changes in arterial pressure.

In 15 dogs the first operation consisted in a hemihepatectomy and portal diversion only. Two of these dogs died soon after the operation from postoperative bleed due to technical failures. Two others died within 24 hours after the operation because of bypasses kinked or squeezed by postoperative oedema of a small remnant of hepatic tissue beside the stump of the left lateral portal vein branch. In one dog (page 105) the anastomosis closed postoperatively.

In 10 dogs these complications could be avoided and these animals had an uncomplicated postoperative course. At reoperation four to six weeks later, the bypasses appeared to have widened to large vessels with a concomitant return of the portal pressure to the mean normal value of 6 mm Hg (Table

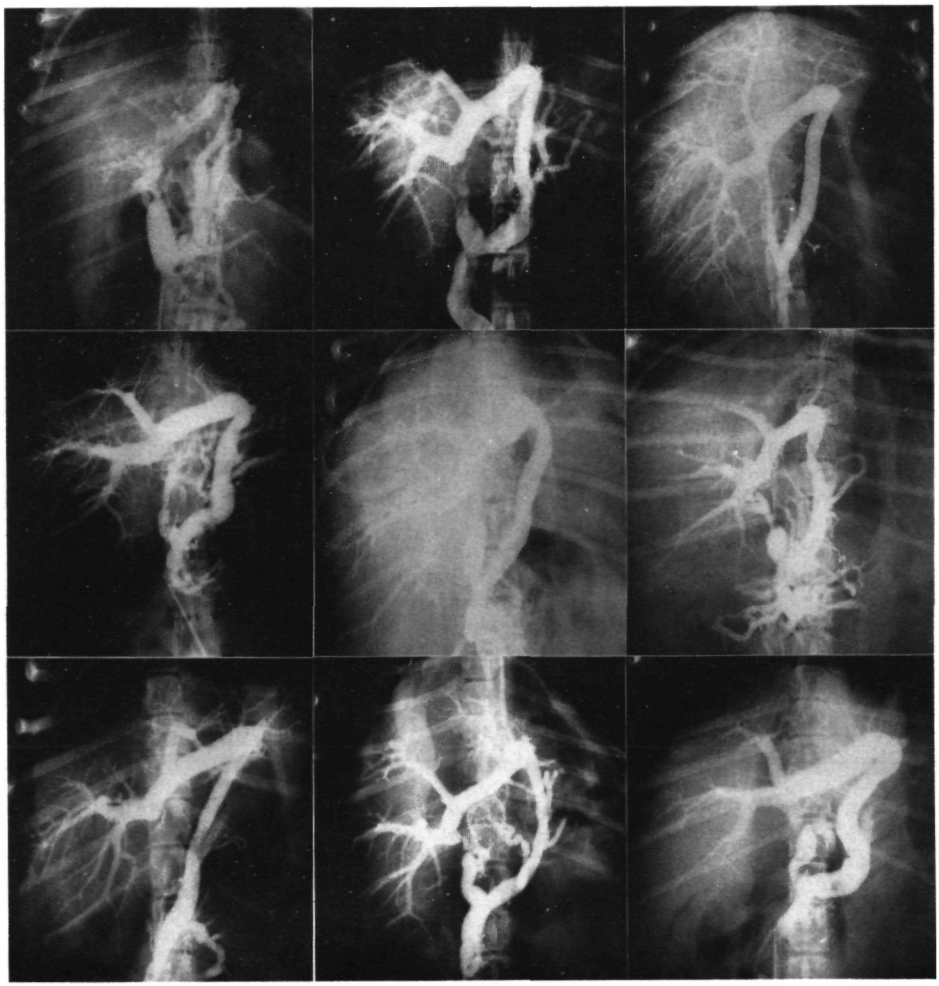

Fig. 27. Peripheral splenorenal bypasses two to three months after construction.

XXXIV). The livers in these dogs showed the bulging picture characteristic of regeneration. At the third operation and subsequent sacrifice two to three months after hemihepatectomy and portal diversion, the bypasses had developed into vessels almost as wide as the original portal vein (Figs. 25 and 27). The mean weight of the regenerated livers was twice that of the hemi-livers removed during the first operation. The splenoportal anastomoses were well healed and as wide as the rest of the bypasses.

Discussion and conclusions

Construction of a paraportal bypass provided the liver with a double inflow tract of portal blood. The diameter of the vessels forming the bypass was only half the diameter of the portal vein and the splenic vein was even further narrowed by spasm immediately after displacement. Nevertheless the bypass provided an outflow tract for the extrahepatic portal system and an inflow tract for the intrahepatic system sufficient to replace the function of the portal vein. This result is consistent with reports that the lumen of the portal vein can be narrowed considerably before the portal flow is influenced (452).

The portal vein was transected only if it was clear that its function would be taken over by the bypass. Hemihepatectomy and transection of the portal vein preceded by the reconstruction of a paraportal bypass, was a feasible and safe procedure, well tolerated by the dog which is more susceptible to an acute rise in portal pressure than man. The rise in portal pressure which reflected the raised resistance in the transferred splenic vein, subsided as the bypass gradually widened. Narrow as it was in the beginning, replacement of the portal vein by the bypass did not interfere with a normal regeneration of the liver remnant. The anastomosis with fine interrupted stitches expanded as much as the rest of the bypass (166, 298). The turned blood flow in the hepatic branch of the portal vein did not raise any problems.

Unfortunately a proper umbilical vein did not exist in the investigated animals. By means of the mobilised splenic vein the situation to be expected after the construction of a paraportal umbilical bypass could be imitated. The relation of the diameters between the splenic and portal vein in the dog (5:11 - 45%) was even less favourable than that between the umbilical and portal vein in man (7.7:10.2 - 75%). Since the peripheral splenoportal bypass functioned properly in the dog, it can be assumed that the umbilical bypass in man will function equally well.

Prospects

Although far from being easy, the construction of the anastomosis between the splenic vein and hepatic portal vein branch was feasible in all dogs. A similar paraportal access to the intrahepatic portal venous system should be possible to any

place in the human liver where a vein of sufficient size can be reached and dissected. Comparable to intrahepatic lateral cholangiojejunostomy with a bile duct of segment III (521), it may be possible to construct an end-to-end anastomosis between the mobilised splenic vein and an intrahepatic portal branch or to connect both vessels with a vein graft. In fact the umbilical paraportal bypass proposed in Chapter 6 is similar and is just a utilisation of a human anatomical advantage.

The mobilised splenic vein may as well be anastomosed end-to-end with the remnant of any main portal branch in the liver hilus with a cuff long enough to allow a vascular suture. This method was suggested by Longmire (357) for reconstruction of the portal blood supply to the left lobe, but is never performed (361). An analogue reconstruction may be considered for the right lateral liver sector as in about 2/3 of men the portal vein branch can be found in the notch which extends from the right side of the porta hepatis (206, 464).

An end-to-end anastomosis between the mobilised splenic vein or a venous graft and the hepatic side of a transected main portal vein branch in the liver hilus will restore the antegrade flow of portal blood to the liver part involved. If such a reconstruction is performed before the portal vein is cut, the extrahepatic portal system remains decompressed in the meantime by the intact contralateral branches. After reconstruction of the portal circulation the situation is similar to that after construction of an umbilical bypass to the left lobe, as outlined in Chapter 6. Transfer of the arterial supply to a near-by large artery completes the construction of an entire and separate afferent blood supply to the relevant liver part. During portal vein reconstruction the arterial supply is undisturbed and during the arterial shift there is a continuing flow through the restored portal branch. By then resection of the rest of the liver and the complete hepatoduodenal ligament can be safely performed.

8.4. Arterial splenohepatic diversion (17 dogs)

Procedure

The trifurcation of the coeliac axis was approached in the lesser sac behind the level of the paraportal bypass or from the left side at the rear of the abdominal cavity. The first 3 to 4 cm of the hepatic and splenic arteries were dissected and both vessels were cut at 2 to 3 cm from their origin. The central part of the splenic artery was then inserted end-in-end into the distal part of the hepatic artery and fixed in this position by three to six interrupted Prolene(R) 6x0 stitches (Fig. 24). In microvascular surgery such a sleeve anastomosis is reported to provide a fast and good anastomosis without stitches entering the perfused lumen (334, 335, 614).

Results

The construction of sleeve anastomoses were not so favourable as expected. The splenic artery which is already smaller than the hepatic artery had to be cut at 2 cm from its origin before becoming too small. The central part had to be bended

sharply to be inserted into the hepatic artery. This and the simultaneous construction of the anastomosis was impaired by the narrow operative field at the bottom of the lesser sac of the dog. Moreover the cut edges of both vessels did not always fit accurately because of the thick walls. Some narrowing was suspected at the anastomotic site though this could not properly be judged during the construction.

Hepatic arterial diversion was performed in 10 dogs that had fully recovered from hemihepatectomy and portal blood transfer (9 dogs) or from hemihepatectomy only (one dog), in one dog the diversion was performed with a concomitant hemihepatectomy only. Two of these 11 dogs died in the second postoperative week;both had a blocked arterial anastomosis. Microscopic examination of the liver of one dog revealed signs of venous liver congestion as seen in the outflow block syndrome. The nine surviving dogs were sacrificed after four to six weeks. In two the arterial anastomosis was blocked by an organised thrombus. One of these two dogs had been nearly moribund in the first postoperative week. The premortal arteriogram of the other showed a collateral circulation between the left gastric artery and the liver.

Seven of the 11 arterial anastomoses were patent and well healed four to six weeks after reconstruction, but all seven showed a slight stenosis. The small gap between the two vessel walls was filled by an organised thrombus and covered with an intimal lining. The liver of the dog that had undergone just a hemihepatectomy and simultaneous arterial diversion had regenerated normally.

Discussion

Splenohepatic arterial transfer both as an isolated operation and combined with a hemihepatectomy was well tolerated by the dog and could safely be performed in dogs recovered from hemihepatectomy with transfer of the portal inflow tract to the retained hemi-liver. In this context the use of a sleeve anastomosis was not beneficial. The procedure was not easier and no less time consuming than a standard or spatulated end-to-end anastomosis. Disadvantages were a slight constriction of the vascular lumen, no expansion and the risk of a thrombosis spreading from the space between the sleeves, as was demonstrated in four of 11 dogs.

Immediately after transection of the splenic vein the spleen became congested and very vulnerable. Four times a hemisplenectomy was performed which consisted in removal of the lower pole. At reoperation four to six weeks after portal diversion the size of the spleen had not changed. However, the spleen and in particular the lower pole, had shrunk considerably if both splenic vein and splenic artery had been used for hepatic blood diversion one to two months before.

8.5. Hemihepatectomy and simultaneous transfer of the portal and arterial blood supply (6 dogs)

Results

All six dogs died in shock within 24 hours. Four had been quite well in the first hours after operation. Postmortem examinations revealed patent anastomoses and no other abnormalities than ascites and slightly congested livers. Microscopic examination of the liver showed acute central and pericentral congestion and necrosis as seen in outflow block with shock (Fig. 28).

Discussion

Hemihepatectomy combined with diversion of the portal and arterial blood supply to the retained liver parts in one session was lethal in the dogs because of an hepatic outflow block and shock. Unlike man, the dog has hepatic venous sphincters reacting upon ischaemia by constriction which results in a blockade of the hepatic venous outflow (414, 524, 592). Diversion of the portal blood from the portal vein to the initially narrow bypass results in a temporarily decreased flow into the liver. Obstruction of the portal blood flow in the dog with an intact hepatic artery shows a reactive increase of the blood flow in that artery (227, 311, 378). The resistance in the splenohepatic arterial diversion was considerably higher than in the original hepatic artery, because of the relatively narrow lumen, the sharp curve of the splenic artery and the still further narrowing at the stenosing anastomosis. This resulted in a substantial decline instead of a reactive increase of the arterial hepatic blood flow.

Hemihepatectomy with a decreased portal or arterial flow was well tolerated. However, a considerable sudden decline of both portal and arterial blood flow apparently caused liver ischaemia serious enough to give a hepatic outflow block in the dogs. If arterial and portal diversions are to be carried out simultaneously, it is advisable to use the largest artery and vein available and also the widest anastomoses possible for vascular reconstructions.

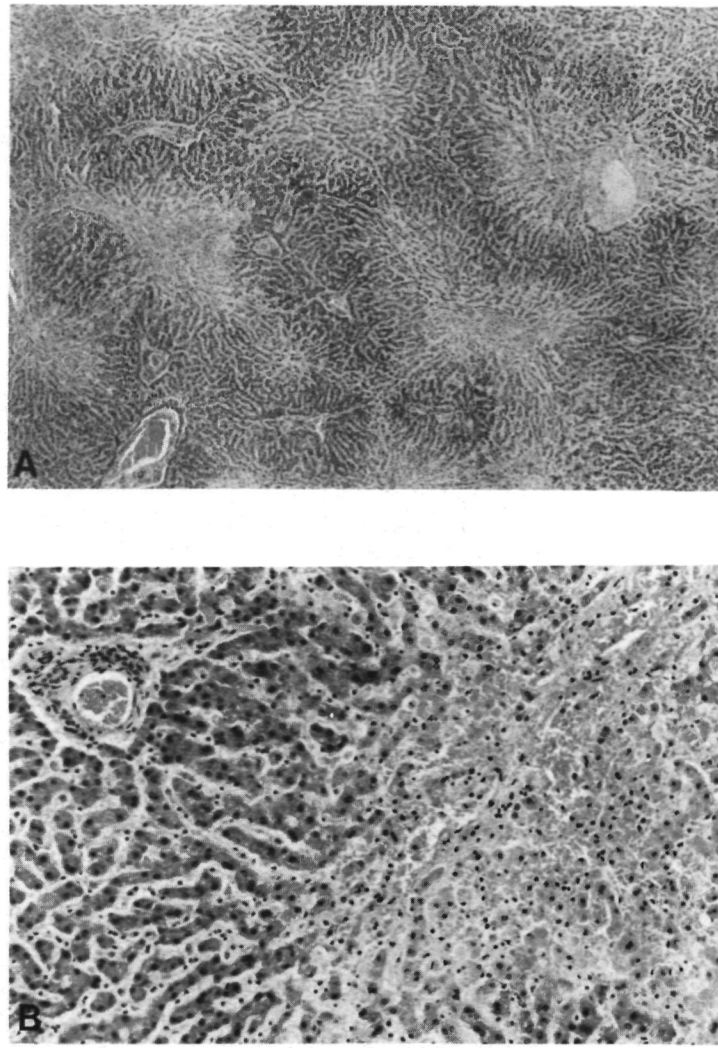

Fig. 28. Section of the liver of a dog that died after progressive shock: A centrilobular pale areas which often merge together, immediately around the portal tracts plates of normal liver cells can still be recognised (HE x36); B Detail of hepatic lobule with normal liver cells around the portal tract (left) and necrosis of centrilobular cells with haemorrhage and congestion and almost no inflammatory response (HE x140).

9. The proposed radical block resection

1 Bilateral subcostal incision, extended Kocher manoeuvre, assessment of tumour dissemination, transection of the tissue bridge between segments III and IV.

2 Dissection and distal transection of the ligamentum teres.

3 Incision of the lesser omentum along the right side of the left lobe (sparing the left arterial branch), dissection of the left corner of the porta hepatis, encirclement of the left hepatic artery branch and of the left portal vein just before the recessus of Rex, palpation of the left hepatic duct.

4 Transection of the common hepatic duct as close as possible to pancreas and duodenum, dissection and encirclement of the bases of the portal vein and hepatic artery, dissection of the central splenic vein.

5 Construction of a Roux-Y loop, mobilisation of the right liver lobe and preparations for right lobectomy.

6 Opening of the umbilical vein. The portal vein base or the encircled left portal vein branch has to be closed at the moment of opening the umbilico-portal junction. Filling of the umbilical vein with heparin solution, distal ligation of the umbilical vein, release of the portal vein clamping.

7 Careful clamping of the umbilical vein, distal transection at the level required, lateral clamping of the central splenic vein and end-to-side anastomosis between the umbilical vein and splenic vein (Fig. 11, page 81). Partial occlusion of the portal vein base may be required to bring about a sufficient flow through the umbilical bypass.

At this stage nothing has been divided yet and the liver has a double portal vascularisation.
If no suitable umbilical vein can be obtained:

7a Mobilisation, distal transection and retrogastric transposition of the splenic vein, filled with heparin solution, towards the left portal vein branch.

8 Transection of the hepatic artery branch to the left lobe 2 cm before its entrance into the parenchyma and re-anastomosis of the hepatic side of the artery with another large artery available, either directly (splenic artery or if possible gastroduodenal artery) or by interposition of a saphenous graft using the hepatic artery stump (Fig.12, page 82).

9 Transection of the left portal vein branch just before the recessus of Rex (Fig. 13, page 82).

If no suitable umbilical vein can be obtained:

9a Transection of the left portal vein branch 1 cm before the

recessus of Rex, end-to-end anastomosis between the hepatic part of the transected vein with the transposed splenic vein.

Now the left and the right liver lobes each have a separate afferent vascularisation.

10 Total clamping of the bases of the portal vein and the hepatic artery, assessment of the haemodynamic situation to expect after radical resection.

Still the procedure can be abandoned. The vascular reconstruction of the area to be retained is finished and tested; there is afferent bloodlessness of the area to be resected.

11 Resection of the complete hepatoduodenal ligament and the right liver lobe with the caudate lobe (Fig. 14, page 83).

12 Hepaticojejunostomy and fixation of the left lobe to prevent postoperative vascular kinking by changement of the patient's posture. Partial or total splenectomy may be necessary during or at the end of the procedure.

Summary

Carcinoma of the hepatic duct confluence causes a non-specific obstructive jaundice only when the bile drainage of the liver is blocked almost completely. Therefore an earlier diagnosis is impossible and the poor results of the current treatment modalities have induced a therapeutic fatalism, especially with respect to the curative prospects. Still the patients die from the consequences of the localised bile duct obstruction and only seldom from a disseminated malignancy. There is ample but fragmentary and non-systematised literature on confluence tumours. This thesis has a twofold aim:

A to inventory the data on the properties of these tumours and on the various surgical treatments (Chapters 1-4), and

B to find better therapeutic possibilities (Chapters 5-9).

In Chapter 1 the anatomy of the liver, liverhilus and ligamentum hepatoduodenale is reviewed. Emphasis is placed on the structures of the left liver lobe and on the lymphatic drainage of the liverhilus in view of the oncological implications and therapeutic possibilities described in Part B.

Chapter 2 presents a literature survey of the pathology and therapy of confluence tumours. At the time of diagnosis the tumour mostly consists of a nodular, firm mass of some centimetres, composed of a well-differentiated adenocarcinoma with strong peritumoral fibrosis. Papillary types are present in 6% of the patients and in 9% of the cases the tumours are multifocal or diffuse. Because of submucosal spread the tumour has to be regarded as a regional affection of the bile duct. Penetration of the thin bile duct wall exists in 70% of the patients and results in periductal tumour spread, in the perineural and other lymph vessels, the peribiliary vascular network, portal vein, hepatic artery or adjacent liver parenchyma.

Metastases in the regional lymph nodes are seen in 33% of the patients, direct spread or metastases in the liver in 50% and distant metastases in 15% of the cases. Some type of regional dissemination exist at the time of diagnosis in 1/2 to 2/3 of the patients but in the others no metastases can be found, even after death.

The stage of growth at the time the tumour is discovered is unknown. A total obstruction or local tumour spread may already exist unilaterally, as obstructive jaundice is the first and only symptom and appears only when the bile drainage of the liver is obstructed nearly completely. The aim of treatment is to raise the bile duct obstruction and to remove the malignant tumour. Resection provides the best possibilities and paratumoral bypass or transtumoral drainage are considered only if resection is impossible.

The pre-operative investigation is directed to the assessment of tumour spread and the biliary and vascular anatomy.

Final assessment of resectability can only occur by laparotomy and exploration. The precarious bilio-enteric anastomosis is made preferably by a careful mucosal suture but hepato-enterostomy may be good as well.

One third of the tumours is resectable. The mean operative mortality of 201 patients from series of at least three patients is 13% and the mean postoperative survival is 19 months. The operative mortality is not increased by a concomitant resection of the quadrate lobe or left hemi-liver, but increases to 35% when performed together with a right hemihepatectomy or right lobectomy. The usual cause of death is bile duct obstruction by recurrent tumour growth, irrespective of the extent of resection.

The various types of paratumoral bypasses and transtumoral drainages are discussed. The mean operative mortality of 349 and 190 patients respectively from series of at least five patients is 25 and 16% and the postoperative survival of both is 11 months. There is no clear difference between the results of the various bypass procedures. Radiotherapy and chemotherapy have no convincing effects. Even after liver transplantation the patients die from recurrent tumour growth. External drainage may be indicated only as terminal palliation. The comparison of various methods of treatment is impaired by the relative incomparability of the patients.

In Chapter 3 the various tumour characteristics and treatment modalities are discussed in a retrospective investigation of 51 Dutch patients. Of these patients 84% had nodular sclerosing tumours and 16% papillary tumours. In 71% the tumour diameter was smaller than 5 cm and in 33% it even did not exceed 2 cm. There were no metastases at the time of operation in half of the patients with a tumour diameter of 5 cm at most. No relation could be found between type or differentiation of the tumour and local spread or dissemination. The cause of death was in general due to cholangitis or liver insufficiency, caused by recurrent biliary obstruction.

The tumour was resected in four patients, resulting in a mean postoperative survival of 22 months although in at least two cases the resection was incomplete. There were no clear differences between the results of paratumoral (9 patients) and transtumoral (20 patients) drainage, the operative mortality was 20 to 22% and the mean postoperative survival was six to nine months. No obvious effect of radiotherapy could be demonstrated except perhaps in one patient.
Nine patients survived the first operative year and no relation could be found with age, duration of pre-operative jaundice, type of tumour or operation, administration of radiotherapy and cause of death. Only one patient is alive, eight years after paratumoral bypass and radiotherapy. It is concluded that resection, if need be incomplete, is the best treatment.

Chapter 4 contains the results of a prospective investigation in a specialised clinic in London into the properties and resection possibilities of confluence tumours with portal

vein involvement in 24 patients. Involvement of the bifurcation area of the portal vein was demonstrated by angiography in 45% of patients with a nodular, sclerosing confluence tumour and nearly half of these 45% also had involvement of the heaptic artery, caudate lobe or quadrate lobe. The vascular affection was not related to the size or differentiation degree of the tumour or to the occurrence of metastases. The involvement of the vessels did not consist in intraluminal invasion by the tumour but in compression or strangulation by peritumoral sclerotic tissue. Therefore vascular involvement is no contra-indication for resection.

A paratumoral bypass or transtumoral drainage was constructed in 12 patients and resulted in an operative mortality of 17% and a mean postoperative survival of seven months. Resection of the tumour together with right lobectomy or left hemihepatectomy was performed in eight patients. In three of these cases a lateral segment of the portal vein bifurcation was removed and twice the complete bifurcation was resected, followed by end-to-end repair.

The case histories and operations of six patients are discussed in detail, three of the eight patients died in the postoperative period, the mean postoperative survival of the other patients was 11 months. Finally the methods of portal vein reconstruction are reviewed and the literature on hepatectomy with simultaneous portal vein resection is discussed.

Chapter 5 presents an analysis of the poor results. In half of the patients no metastases are found but because of its characteristic submucosal and periductal spread the tumour must already have invaded the tissues around the confluence at the time of treatment. In all current types of resection these tissues are removed only partially and especially the retro-hilar structures are left alone. Therefore the usual tumour recurrence is not surprising.

In Chapter 6 the requirements and possibilities of a radical resection are discussed. Block resection of the right liver lobe, hilus and hepatoduodenal ligament, leaving the left lobe with complete portal and arterial vascularisation, is the best possiblity. Revascularisation methods of the remaining left lobe are discussed and an operative procedure is proposed in which the reconstruction of the vascularisation is performed prior to the actual resection.

In man the umbilical vein remains after birth in collapsed but intact form in the ligamentum teres. By opening the umbilical vein the left hepatic portal system can be entered and by anastomosing the re-opened umbilical vein with the splenic vein a communication is acquired between the splanchnic and the hepatic portal system, which can replace the portal vein. After construction of such a paraportal bypass the portal vein can be resected. The hepatic artery branch to the left lobe can be transected and re-anastomosed with another large upper abdominal artery, which results in a separate arterial supply of the left lobe. If both portal and arterial reconstructions appear to function well, the desired block resection of the right liver lobe and hepatoduodenal ligament can be carried

out safely.

The literature on the use of the umbilical vein is reviewed and the conclusion is that the proposed application of the vein will probably be effective. Finally the access to the portal vein confluence and the desirability of retrohepatic vascular isolation are discussed.

Chapter 7 presents an anatomical postmortem investigation of 40 livers into the feasibility of the proposed block resection. In 91% of the dissections the umbilical vein could be re-opened to a lumen almost as wide as the left portal vein branch. After encirclement and transection of the left portal vein branch in the umbilical fissure of the liver, the portal venous system of the left liver lobe can be reached via the re-opened umbilical vein. The artery branch to the left lobe enters the parenchyma dorsally to the portal vein branch, the left hepatic duct is on this level cranial to the portal vein branch.

The plane of transection along the umbilical scissure stays a good 4 cm from the hepatic duct confluence. After parenchymatous transection the left lobe remains with the umbilical vein, arterial remnant and hepatic duct stump. The described portal and arterial revascularisation of the left lobe prior to transection of the liver was a well feasible procedure in the autopsy specimen. The final result after radical block resection seems haemodynamically comparable to standard right lobectomy and biliodigestive reconstruction.

In Chapter 8 an experimental investigation is presented into the functional effectivity of paraportal bypasses, arterial revascularisation and liver resection in 23 dogs. It appeared that no usable umbilical vein existed in laboratory animals and therefore the splenic vein was mobilised, distally transected and end-to-end anastomosed with the stump of a hepatic portal vein branch, which was preserved during left hemihepatectomy. The retained right hemi-liver then had a double portal inflow tract. The portal vein could be cut safely when it was clear that the paraportal bypass functioned properly. The hepatic and splenic arteries were cut during the same operation or six weeks later, followed by an end-in-end sleeve anastomosis of the central side of the splenic artery into the distal side of the hepatic artery. Forty-six operations were performed.

Left hemihepatectomy with replacement of the portal vein by the paraportal bypass was well tolerated by the dogs. The portal pressure rose from 6 to 13 mm Hg because of the initially narrow lumen of the bypass, but at re-operation one to three months later the bypass had widened to a vessel almost as wide as the original portal vein and the portal pressure had returned to normal.

Arterial transfer as a separate procedure or together with hemihepatectomy resulted in a patent but narrow anastomosis in seven of the 11 dogs. Hemihepatectomy with simultaneous portal and arterial transfer in six dogs was lethal within 24 hours. The animals died in shock, probably caused by a hepatic outflow block induced by liver ischaemia.

The conclusion is made that a splenic paraportal bypass, comparable with a human umbilical bypass, can function properly. If in a patient no usable umbilical vein is available, a splenic paraportal bypass may serve the same function. For a simultaneous portal and arterial transfer the larges available artery or venous graft interposition should be used with the widest anastomosis possible.

Finally in Chapter 9, the proposed radical block resection is outlined step by step.

Het zeldzame carcinoom van de ductus hepaticus confluentie manifesteert zich pas in een aspecifieke obstructie icterus als de galafvloed van de lever vrijwel volledig is afgesloten. Een vroegere diagnose is daarom onmogelijk en de povere resultaten van de huidige vormen van behandeling hebben geleid tot een therapeutisch fatalisme, vooral voor wat betreft de curatieve mogelijkheden. Toch sterven de patiënten aan de gevolgen van de gelokaliseerde galwegobstructie en slechts zelden aan een gedissemineerde maligniteit. Er bestaat een uitgebreide literatuur over confluentietumoren doch deze is fragmentarisch en niet gestructureerd. Deze dissertatie heeft een tweeledig doel:

A het inventariseren van hetgeen bekend is over de eigenschappen van deze tumoren en over de verschillende chirurgische behandelingen (Hoofstuk 1 t/m 4);

B het zoeken naar betere therapeutische mogelijkheden (Hoofdstuk 5 t/m 9).

Hoofdstuk 1 bevat een anatomisch rappel van lever, leverhilus en ligamentum hepatoduodenale. In verband met de oncologische implicaties en therapeutische mogelijkheden die beschreven worden in Deel B, wordt enige nadruk gelegd op de structuren van de linker leverkwab en op de lymphatische drainage van de leverhilus.

Hoofdstuk 2 geeft een literatuuroverzicht van pathologie en therapie van confluentietumoren. Op het tijdstip van diagnose bestaat de tumor meestal uit een nodulaire, vaste massa van enkele centimeters grootte, opgebouwd uit een goed gedifferentieerd adenocarcinoma met een sterke peritumorale fibrose. Beoordeling van de aanwezigheid van tumorcellen kan erg moeilijk zijn door uitgebreide fibrose. Papillaire vormen worden bij 6% van de patiënten aangetroffen, en in 9% van de gevallen betreft het multifocale of diffuse tumoren. Door submucosale verspreiding moet de tumor worden beschouwd als een regionale aantasting van de galwegwand. Penetratie van de dunne galwegwand bestaat bij 70% van de patiënten en resulteert in periductale doorgroei van tumorweefsel in perineurale lymphspleten, grotere lymphe banen, peribiliaire vaat plexus, v. portae, a. hepatica of aangrenzend leverparenchym.

Metastasering in de regionale klieren wordt bij 33% van de patiënten aangetroffen, doorgroei of metastasering in de lever bij 50% en metastasering op afstand bij 15% van de gevallen. Een vorm van regionale metastasering bestaat bij 1/2 tot 2/3 van de patiënten, doch bij de overigen ontbreekt elke vorm van aantoonbare disseminatie, zelfs na het overlijden van de patiënt.

Het groeistadium waarin de tumor zich bevindt op het moment van ontdekking is onbekend. Aangezien icterus eerst ontstaat

als de galafvloed van de lever bijna volledig is afgesloten, kan unilateraal al een totale obstructie en lokale doorgroei zijn opgetreden met alle gevolgen van dien.

Het doel van de behandeling is opheffing van de galwegobstructie en verwijdering van de maligne tumor. Resectie biedt daartoe de beste mogelijkheden en eerst als resectie onmogelijk is, komen paratumorale bypass of transtumorale drainage in aanmerking.

Het pre-operatieve onderzoek is gericht op het bepalen van de uitbreiding van de tumor en op het bepalen van de biliaire en vasculaire anatomie. Definitieve vaststelling van de resectabiliteit kan alleen geschieden tijdens laparotomie. De precaire bilio-enterische anastomose kan het beste worden gemaakt met een zorgvuldige mucosanaad, doch hepato-enterostomie biedt ook goede mogelijkheden.

Een derde van de tumoren is resectabel. De gemiddelde operatieve mortaliteit van 201 patiënten uit series van tenminste drie patiënten is 13% en de gemiddelde postoperatieve overlevingsduur is 19 maanden. De operatiemortaliteit wordt niet vergroot door een gelijktijdige resectie van de lobus quadratus of linker leverhelft, maar stijgt tot 35% wanneer een rechter hemihepatectomie of lobectomie wordt uitgevoerd. De gebruikelijke doodsoorzaak is galwegobstructie door recidiverende tumorgroei, onafhankelijk van de omvang van de resectie.

De verschillende typen paratumorale bypasses en transtumorale drainages worden vervolgens besproken. De gemiddelde operatie mortaliteit van respectievelijk 349 en 190 patiënten afkomstig uit series met tenminste 5 patiënten is 25 en 16% en de postoperatieve overleving van beide is 11 maanden. Een duidelijk verschil in resultaat tussen de verschillende bypass procedures is niet aantoonbaar. Een overtuigend effect van radio- of chemotherapie is niet te vinden. Ook na levertransplantatie sterven de patiënten aan tumorrecidief. Externe drainage kan alleen geïndiceerd zijn als terminale palliatie. De vergelijking van de verschillende vormen van therapie wordt echter belemmerd door de betrekkelijke onvergelijkbaarheid van de patiënten.

In Hoofdstuk 3 worden de verschillende tumoreigenschappen en vormen van therapie vergeleken in een retrospectief onderzoek van 51 Nederlandse patiënten. 84% Van de tumoren was nodulair skleroserend, 16% papillair. Bij 71% van de patiënten was de tumor diameter kleiner dan 5 cm en bij 33% zelfs niet groter dan 2 cm. Bij de helft van de patiënten met een tumor doorsnede van hoogstens 5 cm waren geen metastasen aanwezig ten tijde van de operatie. Er bestond geen relatie tussen type of differentiatiegraad van de tumor enerzijds en het optreden van metastasen of lokale uitbreiding anderzijds. De doodsoorzaak was meestal het gevolg van recidiverende biliaire obstructie, namelijk cholangitis en liverinsufficientie.

Bij vier patiënten werd de tumor gereseceerd en ondanks het feit dat tenminste bij twee patiënten de resectie incompleet was, werd een gemiddelde postoperatieve overlevingsduur van 22 maanden bereikt. Er bestonden geen duidelijke verschillen

tussen de resultaten van paratumorale (9 patiënten) en transtumorale (20 patiënten) drainage, de operatiemortaliteit was 20 tot 22% en de gemiddelde overlevingsduur 6 tot 9 maanden. Van radiotherapie was geen duidelijk effect aantoonbaar. Negen patiënten leefden langer dan één jaar, zonder aantoonbare relatie met leeftijd, duur van de voorafgaande icterus, tumortype, aard van de operatie, toediening van radiotherapie en doodsoorzaak. Acht jaar na paratumorale bypass en radiotherapie is één patiënt in leven. Geconcludeerd kan worden dat resectie, desnoods incompleet, de beste behandeling vormt.

Hoofdstuk 4 bevat de resultaten van een prospectief onderzoek in een gespecialiseerde kliniek in Engeland naar de eigenschappen en resectiemogelijkheden van 24 patiënten met confluentietumoren die de v. portae hadden aangetast. Angiografisch aantoonbare aantasting van het bifurcatiegebied van de v.portae bestond bij 45% van de patiënten met een nodulaire sklerоserende tumor en bij bijna de helft van deze 45% bestond eveneens aantasting van de a. hepatica, lobus caudatus of lobus quadratus. De vasculaire aantasting was niet gerelateerd aan grootte of differentiatiegraad van de tumor of aan het vóórkomen van metastasen. Deze aantasting bestond niet uit intraluminale tumorinvasie maar uit verdrukking van de vaten door peritumoraal sklerotisch weefsel. Vaataantasting vormt daarom geen contra-indicatie voor resectie.

Een paratumorale bypass of transtumorale drainage werd geconstrueerd bij 12 patiënten en gaf een operatiemortaliteit van 17% en een gemiddelde postoperatieve overlevingsduur van zeven maanden. Resectie van de tumor, samen met rechter lobectomie of linker hemihepatectomie, werd verricht bij acht patiënten; in drie gevallen werd daarbij een lateraal segment van de v. portae bifurcatie verwijderd en tweemaal werd de volledige bifurcatie gereseceerd, gevolgd door end-to-end reconstructie.

De ziektegeschiedenis en operatie van zes patiënten worden nader besproken. Drie van de acht patiënten overleden in de postoperatieve periode, de gemiddelde overlevingsduur van de overigen is 11 maanden. Tenslotte worden de methoden voor v. portae reconstructie aangegeven en de literatuur over hepatectomie en gelijktijdige v. portae resectie besproken.

Hoofdstuk 5 geeft een analyse van de slechte resultaten. Hoewel bij de helft van de patiënten geen metastasering wordt aangetroffen, moet op grond van de submuceuze en periductale groeikenmerken van de tumoren toch worden aangenomen dat ten tijde van de behandeling reeds tumorweefsel in de weefsels om de hepaticus confluentie aanwezig is. Bij alle bestaande vormen van resectie worden deze weefsels slechts partieel verwijderd. Het gebruikelijke optreden van een lokaal tumorrecidief is daarom dan ook niet verwonderlijk.

In Hoofdstuk 6 worden de vereisten en mogelijkheden van een radicale resectie besproken. Blokresectie van de rechter leverkwab, hilus en ligamentum hepatoduodenale, waarna de linker-

kwab met een arteriële en volledige portale vascularisatie overblijft, vormt de beste mogelijkheid. Revascularisatiemethoden van de resterende linkerkwab worden besproken en een operatieve procedure wordt voorgesteld waarbij de reconstructie van de vascularisatie van de linkerkwab wordt uitgevoerd vóór de eigenlijke blokresectie.

Door de v.umbilicalis, die bij de mens in gecollabeerde vorm aanwezig blijft, te openen wordt toegang tot het linker hepatische v. portae stelsel verkregen. Door de heropende v. umbilicalis te anastomoseren met de v. lienalis ontstaat een verbinding tussen het splanchnische en het hepatische portale systeem, die de v. portae kan vervangen. Na constructie van een dergelijke umbilicale paraportale bypass kan de v.portae worden gereseceerd. Door de arterietak die de linkerkwab verzorgt te klieven vlak voor intrede in het parenchym en te re-anastomoseren met een andere bovenbuiksarterie, krijgt de linkerkwab een afzonderlijke arteriele circulatie. Wanneer deze portale en arteriele reconstructies goed blijken te functioneren, kan de gewenste blokresectie van de rechter leverkwab en het ligamentum hepatoduodenale veilig worden uitgevoerd.

De ervaringen die in het verleden met het gebruik van de v.umbilicalis zijn opgedaan worden besproken. Op grond van deze ervaringen is de effectiviteit van de voorgestelde toepassing waarschijnlijk. Tenslotte wordt de wenselijkheid van retrohepatische vasculaire isolatie belicht.

Hoofdstuk 7 bevat het verslag van een anatomisch postmortem onderzoek van 40 levers naar de uitvoerbaarheid van de voorgesteld blokresectie. Bij 91% van de dissecties kon de v. umbilicalis worden heropend.tot een lumen dat vrijwel even wijd was als dat van de linker v. portae tak.Na omsingeling en klieving van de linker v. portae tak in de fissura umbilicalis kan het v. portae stelsel van de linker leverkwab worden bereikt via de heropende v. umbilicalis. De arterietak van de linker kwab gaat het parenchym binnen dorsaal van de v.portae tak, de linker ductus hepaticus ligt op dat niveau craniaal van de v. portae tak.

Het transsectievlak langs de scissura umbilicalis blijft ruim 4 cm van de hepaticus confluentie verwijderd. Na parenchym transsectie blijft aan de linkerzijde de linkerkwab over met v. umbilicalis, arteriestomp en galwegstomp. Portale en arteriele revascularisatie van de linker kwab op deze wijze, voorafgaande aan transsectie van de lever, bleken in het obductiepreparaat goed uitvoerbaar. Het eindresultaat na radicale blokresectie zal haemodynamisch vergelijkbaar zijn met een standaard rechter lobectomie en biliodigestieve reconstructie.

In Hoofdstuk 8 wordt een experimenteel onderzoek gepresenteerd naar de functionele effectiviteit van paraportale bypasses, arteriele revascularisatie en leverresectie in 23 honden. Omdat er bij proefdieren geen bruikbare v. umbilicalis bleek te bestaan, werd de v. lienalis gemobiliseerd, distaal doorge-

knipt en end-to-end geanastomoseerd met een stomp van een v. portae tak die gespaard was tijdens een linker hemihepatectomie. De overblijvende rechter leverhelft had op die wijze een dubbel instroomkanaal voor het portale bloed. De v. portae werd veilig doorgeknipt wanneer duidelijk was dat de paraportale bypass goed functioneerde. De a. hepatica en de a.lienalis werden in dezelfde zitting of zes weken later doorgeknipt, gevolgd door een end-in-end sleeve-anastomosis van het centrale deel van de a. lienalis in het perifere deel van de a.hepatica. Er werden 46 operaties uitgevoerd.

Linker hemihepatectomie met vervanging van de v. portae door de paraportale bypass werd goed door de honden doorstaan. Door het aanvankelijke nauwe lumen van de bypass steeg de portale druk van 6 tot 13 mm Hg maar bij re-operaties één tot drie maanden later bleek dat de bypasses zich hadden verwijd tot vaten die bijna even wijd waren als de oorspronkelijke v. portae, terwijl de portale druk was genormaliseerd.

Arteriele verplaatsing als een afzonderlijke procedure of samen met linker hemihepatectomie leverde een open doch nauwe anastomose op in zeven van de 11 honden. Hemihepatectomie met gelijktijdige portale èn arteriele verplaatsing bij zes honden was dodelijk binnen 24 uur ten gevolge van shock, waarschijnlijk door een hepatische afvloedobstructie op basis van leverischaemie.

Geconcludeerd wordt dat de splenische paraportale bypass, vergelijkbaar met de menselijke umbilicale bypass, goed kan functioneren. Wanneer bij de mens geen bruikbare v.umbilicalis voor handen is, kan een splenische paraportale bypass dezelfde functie vervullen. Voor een gelijktijdige arteriële verplaatsing moet de grootst beschikbare arterie of veneus interpositie transplantaat worden gebruikt met de wijdste anastomose.

In Hoofdstuk 9 tenslotte wordt de voorgestelde radicale blokresectie stap voor stap besproken.

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APPENDIX I

Results of resection of primary tumours of the hepatic duct confluence

Authors	patients	operative mortality	mean survival (months)	alive after (yrs)					
				½	1	1½	2	3	5
Akwari and Kelly (7)	4	0	33	4	3	3	3	3	
Alexandre et al.(9)	3	0	14	3	1	1	1	0	
Altemeier (15)	1	0	42	1	1	1	1	1	
Andersson et al. (17)	3	1	36	2	2	2	2	1	
Bérard et al. (39)	4	0	13	4	2	1	0	0	
Bertrand et al.(43)	1	0	16	1	1	0	0	0	
Bird et al. (46)	1	0	12	1	1	0	0	0	
Bismuth et al.(56)	1	0	78	1	1	1	1	1	1
Blumgart (68,70)	17	3	15						
Braasch et al.(77, 80)	2	0	60	2	2	2	2	2	2
Brown and Myers (87)	2	1	8	1	0	0	0	0	
Cameron et al. (109)	10	0	21						1
Camprodon et al.(110)	1	0	16	1	1	0	0	0	
Chitwood et al. (131)	5	0	17						
Crismer et al.(145)	1	1							
Crozetti (146)	1	0	60	1	1	1	1	1	1
Engler et al.(109)	1	0	10	1	0	0	0	0	
Evander et al.(178)	16	2	?						
Finney (184)	1	0	16	1	1	0	0	0	
Fortner et al.(195)	6	3	20	3	3	1	1	0	
Foster et al. (199)	1	1							
George et al. (208)	3	0	12	2	2	1	0	0	
Hart and White (243)	3	0	11	3	1	0	0	0	
Haynes et al. (246)	1	0	9	1	0	0	0	0	
Heully et al. (263)	1	0	?						
Hoevels and Ihse (270)	3	1	28	2	1	1	1	1	
Hollender et al.(273)	2	0	8	1	0	0	0	0	
Ingis and Farmer (290)	1	0	24	1	1	1	1	0	
Innocenti et al.(291)	1	0	?						
Iwasaki et al.(294) [a]	(9)	(1)	(12)	6	(4	2	1	0)	
Kajitani (300)	2	0	12	2	1	0	0	0	0
Karakousis (303)	2	0	15	2	1	1	0	0	0
Kelly (304)	1	0	19	1	1	1	0	0	
Kozaka et al.(315)	2	0	20	2	2	2	0	0	
Launois (330,332,333)	11	2	28	9	8	6	5	3	1
Longmire et al.(357)	2	1	12	1	1	0	0	0	
Mann (373)	1	0	13	1	1	0	0	0	
McDermott and Peinert(384)	7	0	10	4	1	0	0	0	
McDermott (385,386)	3	0	?					1	
Mistilis and Schiff (402)	1	0	30	1	1	1	1	0	
Nicoli et al.(418)	2	0	9	1	1	0	0	0	
Nishimura et al.(420)	2	0	17	2	2	1	0	0	
Ong and Lee (432)	2	2	?						
den Otter (435)	3	1	13	2	1	1	0	0	
Philippakis et al.(446)	1	0	13	1	1	0	0	0	
Quattlebaum (458)	3	1	±12	2	2	0	0	0	
Rodgers et al.(471)	1	0	¯28	1	1	1	1	0	
Rohner (476)	4	2	36	2	2	1	1	1	0
Roux (481)	1	0	15	1	1	0	0	0	
Salembier (487,489)	5	1	16	3	1	1	1	1	
Saubier et al. (491)	3	0	?						
Skoog and Thorén (512,558)	8	2	23	5	4	7	3	1	1
Todoroki et al. (561) [a]	23	1	±16	?	14	9	9	7	2
Tompkins et al. (564)	22	5	¯ ?						
Tsuchiya (570,571)	9	2	17						
Tsuzuki et al.(572,574)	11	0	15	9	7	4	2	0	
Van Vroonhoven (589)	1	1							
Warren et al.(596)	9	0	26	9	8	5	4	3	
Whelton et al.(606)	2	1	20	1	1	1	0	0	
White (609)	1	0	23	1	1	1	0	0	

(a) the series of Iwasaki has been worked into the series of Todoroki except for the ½ year survival

APPENDIX II

Results of paratumoral bilio-enteric bypass for primary tumour of the hepatic duct confluence

Authors	patients	operative mortality	mean survival (months)	alive after (yrs)				
				½	1	1½	2	3
Alvarez (16)	2	1	18	1	1	1	0	0
Andersson et al.(17)	8	1	8					
Arianoff et al.(19)	10	4	11	4	1	1	1	0
Bertrand et al.(43)	11	6	20	5	5	3	2	0
Bismuth (48)	45	2	13					
Bodner (69)	4	1	8	2	0	0	0	0
Cahow (101)	9	3	15	?	3	1	1	0
Chapuis (124)	32	10	?					
Chigot et al. (128)	20	7	9	6	4	3	0	0
Corlette (137)	1	0	18	1	1	1	0	
Couinaud (143)	8	4	5	2	0	0	0	0
Crismer et al. (145)	2	1	12	1	1	0	0	0
Cukier et al. (147)	4	0	10	2	2	0	0	0
Dalmas et al. (148)	2	0	12	2	1	1	0	0
Gautier et al. (207)	1	0	5	0	0	0	0	0
Hepp et al. (253)[a]	3	0	9	3	0	0	0	0
Hepp et al. (258)[a]	38	7	15					
Hollender et al.(273)	3	0	3	0	0	0	0	0
Huguet (277) [b]	24	2	?					
Huguet et al.(286)[b]	13	4	17					
Launois et al.(330)	14	5	6					
LeNeel et al. (348)	3	2	10	1	0	0	0	0
Levasseur et al.(349)	1	0	15	1	1	0	0	0
Longmire(355,357,359)[c]	11	2	7	4	3	2	2	1
Malt et al.(371)	4	0	12	2	2	1	0	0
Mercadier (394)	16	4	8	2	2	2	0	0
Moreno Gonzalez (407)	12	1	?	11	?			
Mourgue Molines (409)	3	2	16	1	1	0	0	0
Osborne et al.(434)	1	0	15	1	1	0	0	0
Patel et al. (438)	32	11	?					
Pelissier (441)	2	0	3	0	0	0	0	0
Perissat et al.(443)	12	5	5	2	1	0	0	0
Prioton et al. (457)	3	0	19	2	2	1	0	0
Ragins et al. (460)	5	1	14	3	3	1	1	0
Saegesser et al.(484)	6	2	10					
Salembier (487,489)	16	5	4	3	1	0	0	0
Schamaun (498)	1	0	5	0	0	0	0	0
Schutt (500)	1	0	26	1	1	1	1	0
Seigert et al.(503)	1	0	20	1	1	1	0	0
Seiro (504)	7	2	?					
Sullivan (539)	1	0	29	1	1	1	1	0
Tsuzuki (572)	3	0	6	2	0	0	0	0
Wagenaar (591)	1	0	?					
White (609)	3	0	16					
Wong (618)	1	0	12	1	1	0	0	0

[a] and [b] there may be an overlap in these series

[c] the survival of one patient (more than 4½ years) is not included in the total mean survival

APPENDIX III

Results of operative transtumoral drainage for primary tumour of the hepatic duct confluence (numbers in brackets indicate the number of patients who survived the operation. In these series no total number of patients is reported)

Authors	patients	operative mortality	mean survival (months)	alive after (yrs)				
				½	1	1½	2	3
Akwari and Kelly (7)	?(26)	?	10					
Altemeier et al. (13)	?(18)	?	?	?	?	?	5	2
Black et al. (59)	2	0	13	2	1	1	0	0
Blumgart (63)	5	0	±12	?	?	2	1	0
Cameron et al.(107)	14	2	12	10	4	3	2	0
Eriksson (176(	5	1	8	3	1	0	0	0
Everson (180)	1	0	18	1	1	1	0	0
Green et al. (226)	2	0	10	2	1	0	0	0
Grill (228)	30	2	?	19	5	4	3	0
Huguet et al.(285,286)	21	2	13					
Launois et al.(330)	2	0	7	1	0	0	0	0
Lees et al. (343)	31	1	10½	19	14	?	3	1
Longmire (359)	2	0	8	1	1	0	0	0
Marais (374)	2	0	5	1	0	0	0	0
Mercadier (394)	1	0	5	0	0	0	0	0
Reynolds (465)	1	0	19	1	1	1	0	0
Ritchie and McLean(469)	3	0	12	2	2	1	1	0
Sako et al. (485)	1	0	1	0	0	0	0	0
Salembier(487,489)	6	1	7	4	1	0	0	0
Schamaun (498)	1	0	5	0	0	0	0	0
Shidler (508)	1	0	15	1	1	0	0	0
Smoron (518)	?(12)		10					
Stoppa et al (535)	4	3	4	0	0	0	0	0
Terblanche (545, 548)	9	1	15	5	4	2	2	1
Thorbjarnarson (555)	3	0	20	3	2	2	2	0
Tompkins et al. (564)	24	4	?					
Tsuzuki (572)	12	6	17	6	3	1	1	1
VanderVelde (581)	6	3	8	1	1	0	0	0
Whelton et al.(606)	20	7	16					
White (609)	7	0	7					

Acknowledgements

This study was performed in various locations where I enjoyed the invaluable assistance of many people. To mention all of them is an impossible task and yet I must mention a number of individual names.

To Professor Dr H.H.M. de Boer I am much indebted for his attentive and clear assistance.

Dr. M.N. van der Heyde I thank for the stimulation and the opportunity he gave me to extend the original patient study into a thesis.

Professor C. Huguet and Professor B. Launois kindly introduced me to the amazing quality of French liver surgery. My stay in their hospitals was made possible through financial support of the Boerema Fund.

Dr F.B. Bronkhorst I wish to thank for the fine hepatic specimens.

Mr P.M. Huggers arranged the anatomical investigations and Ms Marjo Willemse prepared the hepatic angiograms together with her fellow röntgen assistants. I am grateful for their willingness to do this, often in the middle of the night.

To the staff and co-workers of the Centraal Dieren Laboratorium of the Catholic University of Nijmegen, I am indebted for the dedicated and skilful co-operation.

My special thanks to Professor Dr U.J.G.M. van Haelst for guiding me in the field of histology in the animal experiments.

The practice of resection of hilar bile duct cancers was demonstrated to me by Professor L.H. Blumgart and I feel much obliged to him for his hospitality and for allowing me to use some of the fruits of his vast experience.

Dr D.J. Allison kindly granted permission for the reproduction of the angiograms in Chapter 4.

Professor Dr W.H. Brummelkamp offered me the opportunity to spend half a year in Professor Blumgart's Hepatobiliary Surgical Unit, for which I wish to thank him.

Dr. M. Flannigan kindly corrected my English.

Ms Marijke Windhorst skilfully arranged the huge original list of references and I enjoyed the professional assistance of Ms L. Kok-Noorman in the final completion of the manuscript. My thanks are due to them.

I am grateful to the Jan Dekkerstichting for their financial contribution to the production of this thesis.

My wife Saskia Boerma-Binkhorst I wish to thank for the very clear drawings.

Finally I am quite grateful to her and our children for not killing me.

Curriculum Vitae

Egge Jan Boerma was born July 16th, 1943 in Renkum, The Netherlands. He completed Gymnasium beta at the Stedelijk Gymnasium, Arnhem and studied medicine at the State University of Groningen and the University of Amsterdam, where he graduated in 1971. After two years of practical training including 1½ year surgy in the St Joannes de Deo Hospital in Haarlem, he went with his wife and three children to Zambia and served 2½ years as a district medical officer in Kashikishi Hospital, Nchelenge.

In January 1976 he started his official surgical training in the St Joannes de Deo Hospital (head of the Department of Surgery Dr J.P.E. Bouwes Bavinck), Haarlem and continued his training in September 1976 in the Municipal Hospital (head of the Department of Surgery Dr M.N. van der Heyde), Arnhem. In 1979 he spent five weeks in the Gastro-intestinal Surgical Departments of the Hôpital St Antoine (head Professor J. Loygue and Professor C. Huguet), Paris and the Centre Hospitalo-Universitaire (head Professor B. Launois), Rennes.

In 1981 he worked for half a year in the Department of Surgery of the St Radboud Hospital (head Professor H.H.M. de Boer), Catholic University of Nijmegen and during that period the experimental work described in this thesis was performed in the Central Animal Laboratory of the Catholic University.

May 1st 1982 he was registered as a surgeon.

In June 1982 he joined the Department of Surgery of the Academic Medical Centre (head Professor Dr W.H. Brummelkamp), University of Amsterdam. He spent half a year in the Hepatobiliary Unit of the Department of Surgery of the Royal Postgraduate Medical School and Hammersmith Hospital (head and director Professor L.H. Blumgart, BDS, MD, FRCS), London.

STELLINGEN

1

Een hilair galwegcarcinoom is reseceerbaar tot het tegendeel is bewezen.

2

Wanneer bij een grote leverresectie ook de afferente vascularisatie van de lever verwijderd moet worden verdient het aanbeveling om voorafgaand aan de resectie de reconstructie van de vaten uit te voeren.

3

Klieven van het ligamentum teres mag alleen op stricte indicatie worden uitgevoerd, aangezien daarmee een autoloog veneus transplantaat verloren gaat.

4

Sparende miltchirurgie is zeker geïndiceerd in gebieden waar malaria endemisch is.

5

De behandeling van een tropisch ulcus met een split skin graft is eerst zinvol als de oppervlakte van het ulcus groter is dan 2x2 cm.

6

Levermetastasen van colorectale carcinomen dienen vaker gereseceerd te worden.

7

Medische miskenning van het obligate karakter waarmee vocht zich in een ontstekingsgebied sequestreert heeft menigeen zijn nieren gekost.

8

Tussen geneeskunst en geneeskunde bestaat geen relatie.

9

Diagnostische wildgroei is gebaseerd op angst.

10

De beste reggae en rum komen nog steeds uit Jamaica, maar de Jamaicaanse bush thee blijkt niet beter dan chemotherapeutica in het veroorzaken van veno-occlusive disease van de lever.

E.J. Boerma NIJMEGEN, 6 mei 1983