Surgical anatomy of the extra- and intrahepatic arteries of the human liver on corrosion casts

PhD thesis

Károly Németh MD.

Semmelweis University
Doctoral School of Pathological Sciences

Tutor:	Prof. László Kóbori MD, Ph.D.
Official reviewers:	Ákos Szűcs MD, Ph.D.
	Zsolt Csapó MD, Ph.D.

Head of the Final Examination Board:	Prof. József Sándor MD, Ph.D.
Members of the Final Examination Board:	Attila Bursics MD, Ph.D.
	Ákos Balázs MD, Ph.D.

Budapest
2016

1. Table of Contents

2. List of abbreviations

Abbreviation	Meaning
a-	accessory
A1	artery of segment I.
A2A3	common trunk of segment II and III arteries
CA	celiac axis
CHA	common hepatic artery
CyA	cystic artery
dist.	distal
DPA	dorsal pancreatic artery
GDA	gastroduodenal artery
HCC	hepatocellular carcinoma
IAPD	inferior anterior pancreaticoduodenal artery
IMA	inferior mesenteric artery
IPPD	inferior posterior pancreaticoduodenal artery
IVC	inferior vena cava
LDLT	living donor liver transplantation
LGA	left gastric artery
LHA	left hepatic artery
MHA	middle hepatic artery
OLT	orthotopic liver transplantation
PHA	proper hepatic artery
prox.	proximal
r-	replaced
RAHA	right anterior hepatic artery
RGA	right gastric artery
RHA	right hepatic artery
RPHA	right posterior hepatic artery
S	segment
SA	splenic artery
SAPD	superior anterior pancreaticoduodenal artery
SLT	split-liver transplantation
SMA	superior mesenteric artery
SPPD	superior posterior pancreaticoduodenal artery
UC	unclassified
2x	twice, two different origins
@	anastomosis (members are between brackets)

3. Introduction

3.1 Segmental anatomy of the liver

An extensive knowledge of hepatic anatomy is required for the transplant and hepato-pancreato-biliary surgeon. For explantation and implantation of the whole liver graft, the main interest is focused on the hilar structures embedded in the hepatoduodenal ligament, with the proper hepatic artery located ventromedially, the common bile duct running ventrolaterally, and the portal vein dorsally. The prevailing pattern of the three hepatic veins is a right hepatic vein and a common trunk formed by the middle and left hepatic veins, which drain into the inferior vena cava (IVC). Regarding segmental liver transplantation and liver resections, a closer analysis of the functional and intrahepatic anatomy of the liver is recommended.

From a functional point of view the liver is divided into right and left lobes along the Rex–Cantlie line (H. Rex, 1888; J. Cantlie, 1898), that corresponds an extrapolated line from the posthepatic IVC across the diaphragmatic surface of the liver to the site where the fundus of the gallbladder typically contacts the inferior margin of the liver; approximates the anterior aspect of the plane of the middle hepatic vein and demarcates anteriorly the right and left lobes of the liver. The Rex–Cantlie line forms the boundary between the two portal distribution areas, i.e. the boundary between liver parts supplied by the right and the left branches of the portal vein. Regarding the contribution to the liver volume, the right hemiliver generally constitutes two thirds and the left lobe one third of the total liver volume, which typically represents at least 2% to 2.7% of the body weight [1].

The right lobe can be divided into the anterior section (segments V and VIII) and the posterior section (segments VI and VII), whereas the left lobe consists of a medial section (segment IV) and a lateral section (segments II and III). The borders between these sections concur with the course of the right, middle and left hepatic veins within the corresponding parenchymal scissures – which are not visible on the diaphragmatic surface (Figure 1).

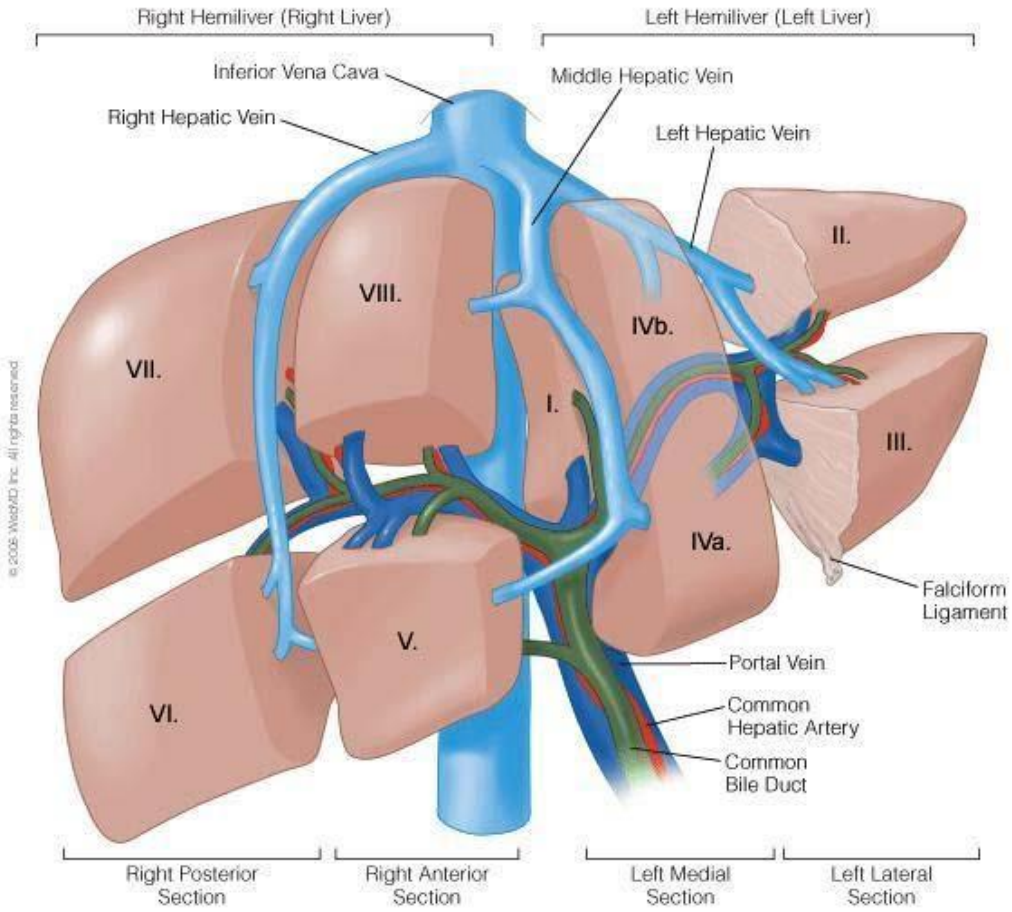


Figure 1: Sectors and segments of the liver.

(Source: https://www.socialtext.net/acs-demo-wiki/couinard_s_segments)

This commonly applied concept of hepatic segmentation, which divides the organ into eight segments, delimited by three vertical and one transverse plane, is credited to the French surgeons Couinaud [2] and Bismuth [3]. The caudate lobe is called segment I, the remaining segments are located clockwise, starting from the upper left side of the liver.

The left medial section includes S I dorsally and S IV ventrally. The latter can be further divided into the cranial IVb and caudal IVa subsegments. Such organization of the subsegments is similar to S V and S VIII within the right anterior section, where S V is located caudally and S VIII cranially.

This segmental anatomy serves as basis for the anatomical and non-anatomical surgical resections, split liver transplantation and living donor liver transplantation [4].

3.1.1 The left lobe of the liver

The left liver lobe displays less variant vascular and biliary anatomy than the right lobe. The fissure of ligamentum venosum (containing the remnant of the ligamentum venosum Arantii) and the fissure of ligamentum teres (containing the remnant of the left umbilical vein) divide the left lobe into a posterior sector (S I and S II) and an anterior sector. The anterior sector is further divided into S III and S IV by the sinus of Rex placed in the umbilical fissure and defined as physiological dilatation of the main left portal branch and congenital mesenterico-portal shunt. The left portal vein travels a relatively long way between the IVb subsegment and the left lateral lobe (S II and S III), covered by a short parenchyma bridge.

Transection of this bridge grants easy access to important structures. From the sinus of Rex, the portal branches to segments II and III run to the left and from the right part, the branches to segment IV arise. Additional segment IV branches may arise from the portal vein bifurcation or the right portal vein. The portal branches to segment I are not constant and originate from the left portal vein, the bifurcation, the right portal pedicle, the main portal vein or a combination of 2 or all of these [5].

Parallel to the A4 the segment IV bile duct comes up. Dissection of all portal segment IV branches gives access to the left lateral bile duct within the umbilical plate (Figure 2). The confluence of the segment II and segment III bile ducts into the left lateral duct can be found in the umbilical plate in 55% of the cases [6]. The biliary anatomy of the umbilical plate underlies numerous variations and is of high interest for the splitting surgeon to limit the risk of biliary complications in the recipient. It is also important to secure the arterial supply of the bile ducts to avoid ischemic cholangiopathy.

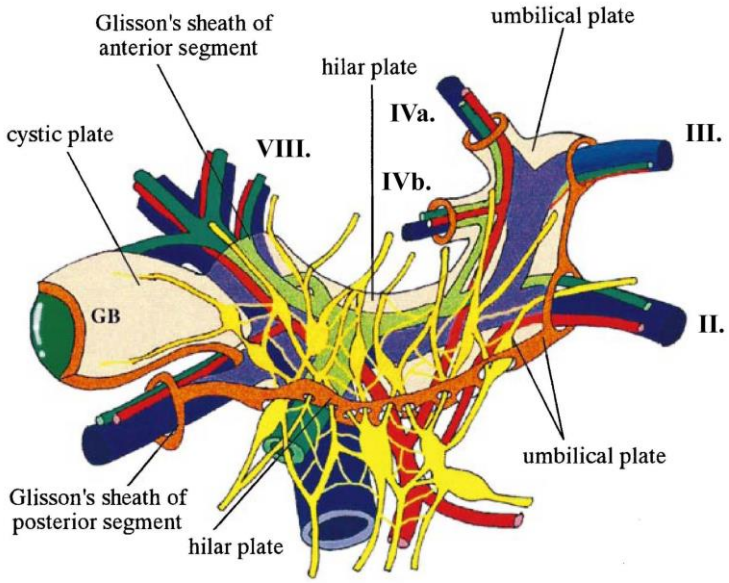


Figure 2: View of the liver plate system. (GB: gallbladder)

Source: Kawarada Y, Das BC, Taoka H. (2000) Anatomy of the hepatic hilar area: the plate system. J Hepatobiliary Pancreat Surg, 7(6): 580-586.

The vascular plexus surrounding the extrahepatic bile ducts derives from the gastroduodenal (GDA), cystic (CyA), and right hepatic artery (RHA) [7], while the intrahepatic bile ducts are supplied by the LHA, RHA and segmental artery branches, and indirectly from the GDA via the arteries supplying the common bile duct [8]. The venous drainage of the left liver lobe contains the left and middle hepatic veins. The left hepatic vein drains segments II, III, and small parts of segment IV while the middle hepatic vein drains the majority of segment IV, but also portions of segments V and VIII. Drainage of segment I is constituted by 1 or 2 separate veins draining directly into the vena cava.

3.1.2 The right lobe of the liver

The right hemiliver is divided by the right parenchymal scissure into the anterior sector, representing segments V and VIII and the posterior sector with segments VI and VII. The right branch of the portal vein arises from the bifurcation in the liver hilum and in 15% an extrahepatic trifurcation with a right anterior and right posterior branch can

be found [2]. The extrahepatic course of the RHA is relatively long and crosses the common bile duct usually posterior to the right.

The anatomy of the right hemiliver bile ducts shows plenty of variations. In the case of split liver transplantation and right-sided liver resections, variations are relevant in which right bile duct branches drain directly into the left hepatic duct. During full-left full-right splitting, complex bile duct variations can result in contraindication for this procedure. In most cases of left lateral split liver transplantation, the variations of the right liver bile ducts are not relevant if the transection plane remains far enough to the left behind the left branch of the portal vein [9].

The venous drainage of the right liver lobe is given mainly by the right hepatic vein, draining segments VI, VII, and parts of segment VIII and V. The middle hepatic vein drains the major portion of segments V and VIII, and a large portion of segment IV. The major drainage of segment VIII into the middle hepatic vein has been described in up to 67%, while the drainage of segment V mainly passed into the right hepatic vein in 70% [10]. In addition to these three major hepatic veins, there can be numerous venous tributaries of different size which directly enter the retrohepatic IVC. Segment VI and VII veins draining directly into the IVC were first described by Makuuchi and colleagues [11]. There can also be found a segment VIII vein draining directly into the IVC close to the right hepatic vein [12].

3.2 Development of the liver vasculature

The primitive fetal blood supply is initially a dual one, with double aortas and both a dorsal and a ventral arterial supply to the abdominal viscera. During subsequent differentiation, much of this dual vascular supply regresses. Varying degrees of persistence of certain portions of the dual blood supply account for the many variations that are seen in the mesenteric circulation (Figure 3).

A series of vitelline arteries arise from the paired, fused dorsal aortas in the fetus, and are initially connected through a ventral anastomotic channel. During fetal development, three of these vitelline segments (10^{th}, 13^{th}, and 21^{st}) persist to individually form the celiac axis (CA), superior mesenteric (SMA), and inferior mesenteric (IMA) arteries; the remaining segments regress before birth. If a portion of

the ventral anastomosis fails to regress, or there is abnormal persistence of vitelline segments during development, anatomic variants result (e.g. arch of Buhler, hepatic artery replaced to the SMA) [13, 14, 15, 16].

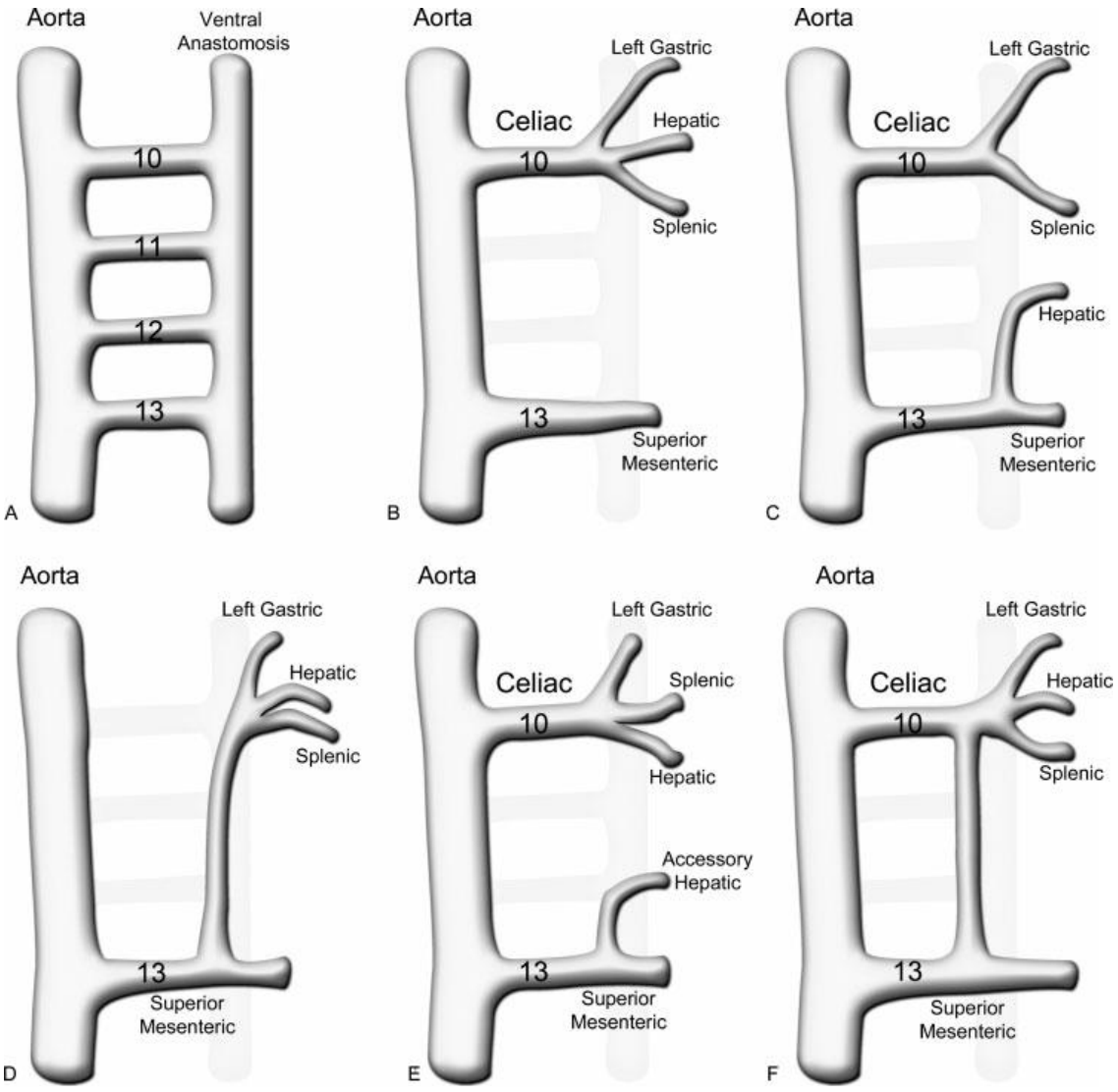


Figure 3: Embryology of normal and variant celiaco-mesenteric vascular anatomy. (A) In the primitive vasculature, the 10th to 13th vitelline arteries communicate between the aorta and a primitive ventral anastomotic artery. (B) Normally the ventral anastomosis and the 11th and 12th vitelline arteries regress, leaving the 10th root as the celiac trunk and the 13th as the superior mesenteric artery (SMA). (C) With replacement of the hepatic artery to the SMA, there is incomplete regression of the ventral anastomosis, forming a hepatomesenteric trunk. (D) A celiacomesenteric trunk occurs when the 10th to 12th vitelline arteries regress and a large portion of the ventral anastomosis persists to connect the celiac artery and branches to the SMA. (E) A partially replaced or accessory hepatic artery occurs in similar fashion to a completely replaced hepatic artery, through failure of a portion of the ventral anastomosis to regress. (F) The arc of Buhler results from persistence of the ventral anastomosis, connecting the celiac and SMA, despite regression of the 11th and 12th vitelline arteries.
Source: Walker TG. (2009) Mesenteric vasculature and collateral pathways. Semin Intervent Radiol, 26: 167-174.

Embryologically, there are 3 lobes in the early stage of hepatic formation, each supplied by an embryonic artery of its own: the lateral sector (S II) by the embryonic LHA, the medial and anterior sectors (S III, IV, V, VIII) by the embryonic middle hepatic artery, and the posterior sector (S VI, VII) by the embryonic RHA [5] (Figure 4). During the early period of human fetal life, the liver is bulky, the gut is very small, and the hepatic artery is predominant. Later, the size of the liver proportionally decreases while the gut reciprocally increases; the 3 principal hepatic arteries are anastomosed in the hilum of the liver, and some of them regress while the enteric branches expand. If the right or left embryonic artery does not totally regress, it becomes the right or left aberrant hepatic artery. Miyaki [17] reported that 1 or 2 aberrant hepatic arteries were found in 30% of human fetuses (over 5 months of pregnancy).

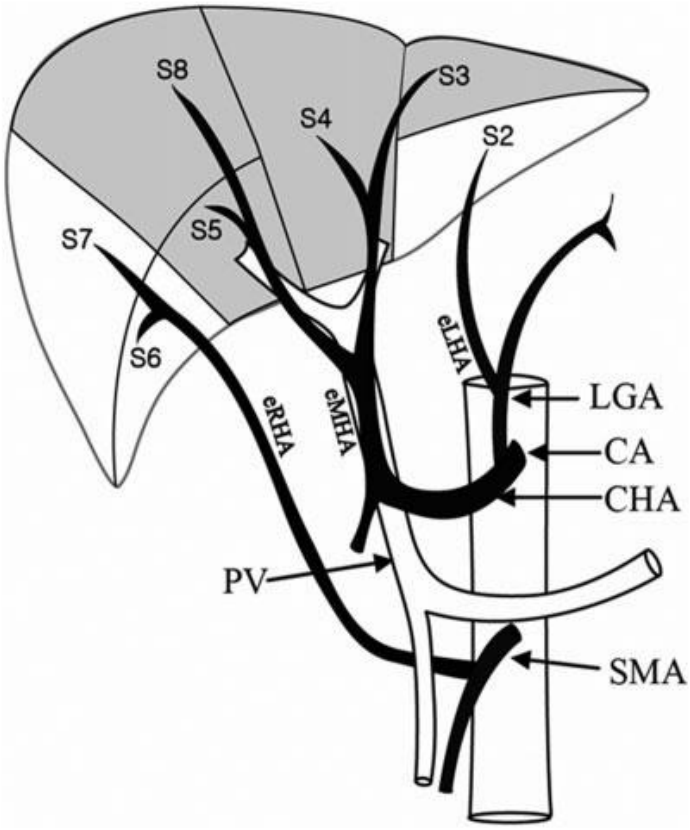


Figure 4: A schematic presentation of 3 embryonic hepatic arteries and their branches representing the number of supplying subsegments of Couinaud. Abbreviations: eRHA, embryonic right hepatic artery; eLHA, embryonic left hepatic artery; eMHA, embryonic middle hepatic artery. Source: Jin GY, Yu HC, Lim HS, Moon JI, Lee JH, Chung JW, Cho BH. (2008) Anatomical variations of the origin of the segment 4 hepatic artery and their clinical implications. Liver Transpl, 14: 1180-1184.

In the adult liver, S IV is a part of the medial sector, and it has been called by various other names such as medial segment, left medial segment, and quadrate lobe. Likewise, the artery for S IV has many names, such as middle hepatic artery (MHA) [18, 19, 20], medial segment artery [21], left medial artery [22], and segment IV artery (A4) [18]. This inconsistency in nomenclature, stemming from various origins, leads only to more confusion. Michels [18] defined this artery as a middle hepatic artery that courses in the umbilical fossa to supply the quadrate lobe and thought that the branches originate in equal proportions from the RHA or LHA. Most of the anatomical studies have used the term middle hepatic artery synonymously for A4.

The hepatic, splenic, and portal venous systems originate through the development of a liver bud that forms between the pericardial cavity and the primitive yolk sac stalk. Hepatic sinusoids are formed when liver cords insinuate between vitelline and umbilical vein tributaries. Branches of the right vitelline vein around the duodenum ultimately become the central portal vein, while the right umbilical vein involutes and the left umbilical vein becomes the primary venous inflow vessel to the liver. This left umbilical vein inflow (portal venous inflow) is connected via the ductus venosus to the hepatic venous outflow. The hepatic venous outflow consists of the hepatic veins and the intrahepatic portion of the IVC formed by the right vitelline vein. Shortly after birth, the ductus venosus and the left umbilical vein close and form the ligamentum venosum and the ligamentum teres, respectively [13, 14].

3.3 Classification systems of the arterial supply of the liver

3.3.1 Classification according to Michels and other authors

The first description of the CA and the aberrant hepatic arteries was published in 1756 by Haller [23]. However, later studies of the frequency of those variations required large series of anatomic autopsies, such as first performed in 1928 by Adachi [24]. In the radiologic literature, publications on selective angiographies dealing with accessory hepatic arteries date from 1958 [25]; extensive studies were performed by Lunderquist in 1967 [26]. Michels proposed an internationally recognized classification of these hepatic abnormalities in 1966 (Table 1) [18]. In his classic autopsy series of 200

dissections, the basic anatomical variations in hepatic arterial supply were defined and this classification has served as the benchmark for all subsequent contributions in this area. Michels' motivation was to maximize the database of the surgeon performing procedures in and around the porta hepatis, to avoid injury to vascular and ductal structures.

The dominant scheme, in which the liver receives its total inflow from the hepatic branch of the CA, is often modified by aberrant hepatic arteries. According to Michels' definition, these vessels may be accessory, occurring in addition to the normal arterial supply, or replaced, representing the primary arterial supply to the lobe.

Table 1: Michels' classification.

Michels' classification of hepatic arterial anomalies	
Type	Description
I.	normal anatomy
II.	r-LHA from LGA
III.	r-RHA from SMA
IV.	r-LHA from LGA + r-RHA from SMA
V.	a-LHA from LGA
VI.	a-RHA from SMA
VII.	a-LHA from LGA + a-RHA from SMA
VIII.	a-LHA from LGA + r-RHA from SMA
IX.	CHA from SMA
X.	CHA from LGA

This classification was modified (and actually simplified) by Hiatt in 1994 (Figure 5) [27] and Varotti in 2004 (Figure 6) [28]. These two systems share the same logic, the main difference is whether they distinguish between accessory and replaced arteries. In Hiatt's classification an accessory and a replaced artery from the same origin (e.g. a-/r-LHA from LGA) belongs to one type, whereas Varotti set up subtypes for these two kinds of aberrant vessels.

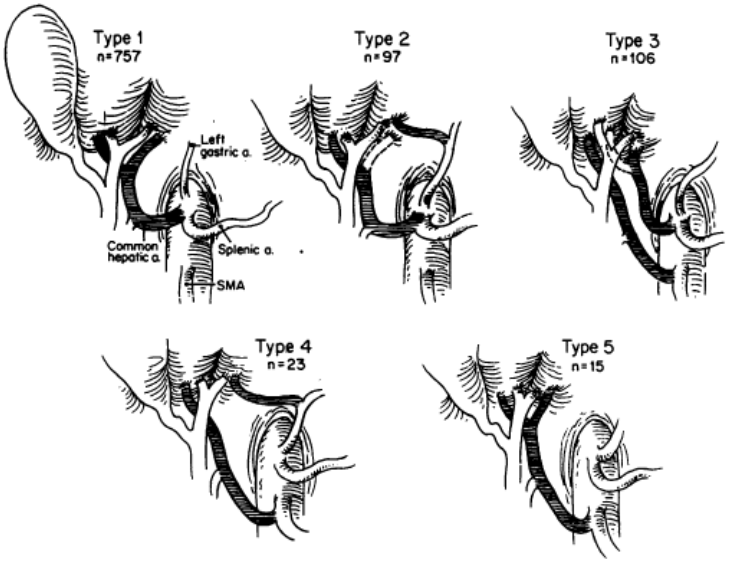


Figure 5: Hiatt's classification. Type 1: normal anatomy. Type 2: Accessory or replaced LHA from LGA. Type 3: Accessory or replaced RHA from SMA. Type 4: Double replaced system. Type 5: Replaced CHA from SMA. Type 6 (not shown): CHA takes direct origin from the aorta.

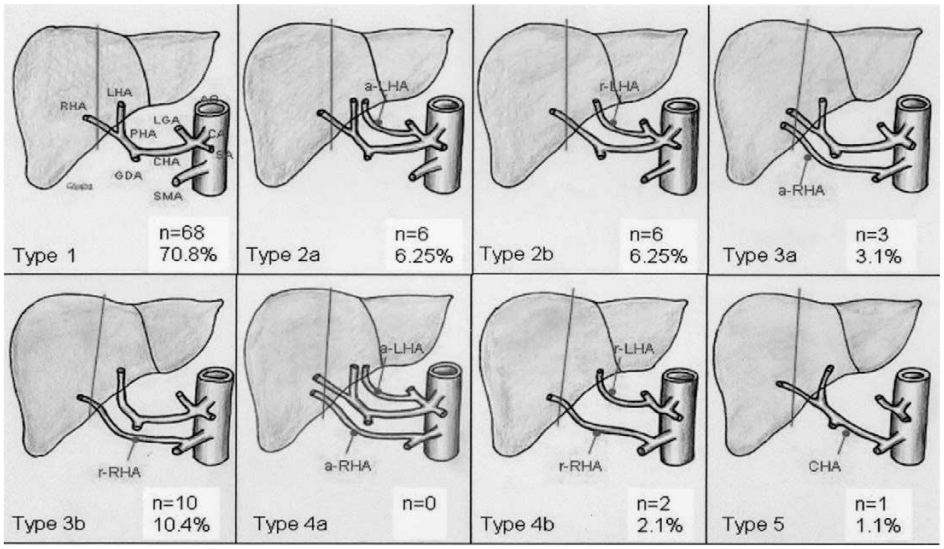


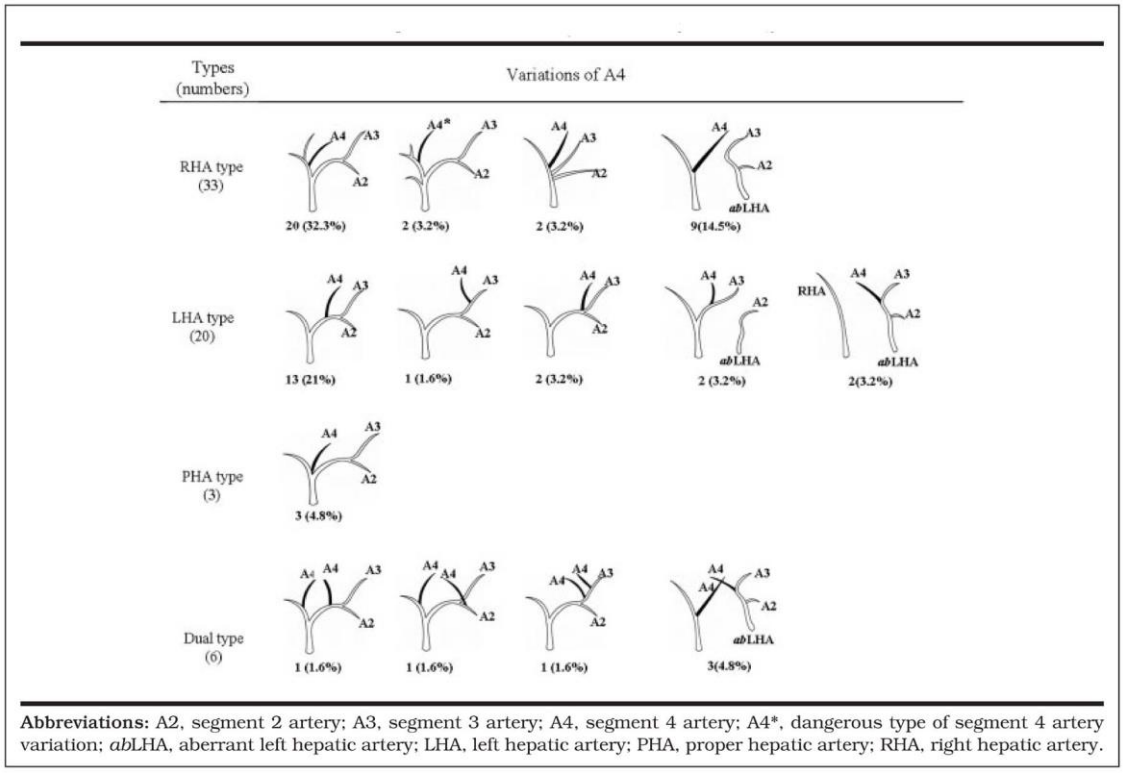
Figure 6: Classification of Varotti. Type 1: normal anatomy. Type 2: Accessory or replaced LHA from LGA. Type 3: Accessory or replaced RHA from SMA. Type 4: Double replaced or double accessory system. Type 5: Replaced CHA from SMA.

3.3.2 Classification of the branching and coursing patterns of the intrahepatic arteries

Compared to the numerous studies on the extrahepatic arterial supply, the intrahepatic arteries have been less frequently investigated, mostly regarding the arterial branches to the segment IV and the caudate lobe. Few data are available on the segmental branching patterns of the arterial tree in the right hemiliver.

Most commonly the A4 derives from the LHA, RHA, from both LHA and RHA, from PHA (Table 2) [19, 29, 30, 31, 32, 33]. In very rare cases the A4 can originate from CHA [29], the CA [31], both LHA and PHA, both RHA and PHA, from RHA and LHA and PHA [32].

Table 2: Four different types of A4 in livers. Source: Jin GY, Yu HC, Lim HS, Moon JI, Lee JH, Chung JW, Cho BH. (2008) Anatomical variations of the origin of the segment 4 hepatic artery and their clinical implications. Liver Transpl, 14: 1180-1184.



Abbreviations: A2, segment 2 artery; A3, segment 3 artery; A4, segment 4 artery; A4*, dangerous type of segment 4 artery variation; *ab*LHA, aberrant left hepatic artery; LHA, left hepatic artery; PHA, proper hepatic artery; RHA, right hepatic artery.

Concerning the arterial structures of the right lobe we stick to the definitions given by Michels [18]: the RHA divides into RAHA and RPHA, which then irrigate the right anterior sector (S V, S VIII) and the right posterior sector (S VI, S VII) of the

liver, respectively. The most important contributions to the description of the coursing and branching patterns were made by Couinaud [34], Yoshioka [35] and Radtke [36].

3.4 Technical aspects of liver transplantation

3.4.1 The short history of liver transplantation

Orthotopic liver transplantation (OLT) has thrived extensively since it was first described by Starzl in 1963 [37]. As the procedure and the outcomes proved to be a success with the innovative surgical techniques, advanced clinical care and improved immunosuppression, the popularity of transplantation increased rapidly. Unfortunately, available donor supply could not keep up with the growing number of recipients; causing longer waiting times and increased waiting list mortality [38]. To overcome this growing imbalance, transplant centers developed strategies to expand the organ donor pool. These strategies have included live donor liver transplantation, split-liver transplantation (SLT), extended criteria donor livers such as elderly livers, and donation after cardiac death donors.

Early experience using the reduction of an adult graft for pediatric liver transplantation was first described by Bismuth et al. [39] In reduced-size OLT, the remaining part of the graft was discarded. Reduced-size OLT became the gold standard for pediatric transplantation, with excellent graft and patient outcomes and decreased waiting list mortality among children. The concept of splitting a liver allograft between 2 recipients was reported almost simultaneously by Pichlmayr et al. [40] and Bismuth et al. [41] Early experiences with SLT were poor, especially in adult recipients and this technique was almost abandoned in the early 1990s [42]. Later, however, with a better understanding of intrahepatic anatomy, better established donor and recipient selection criteria for SLT and the introduction of the in situ split technique [43], splitting livers between child and adult recipients has gained more popularity. Starting in the mid-1990s, many centers worldwide started SLT programs, performing mostly cases serving 1 child who received the left-lateral lobe and 1 adult, who received the extended right lobe [44, 45].

3.4.2 Whole-liver transplantation

Since the pioneering times of OLT in the early 1960s, innumerable improvements have been made, not only in immunosuppression [46], but also in surgical technique.

The technique of OLT as first developed at the University of Colorado in Denver [37], and afterwards at the University of Pittsburgh [47, 48] and at Cambridge [49, 50], has been improved by some technical procedures mainly focused on achieving haemodynamic stability, adequate vascular and biliary anastomoses, and perfect haemostasis.

Abdominal incision and exposure

Adequate exposure is crucial to allow the appropriate dissection needed for native liver hepatectomy. The most commonly used incisions are the bilateral subcostal incision with midline extension, named by Sir Roy Calne as the ”Mercedes” incision (Figure 7), and the inverted J incision, named the Makuuchi incision. With any of these incisions exposure is excellent. The use of Makuuchi's incision is advocated, except in cases of previous surgery or huge splenomegaly that can necessitate the part of the left-sided incision.

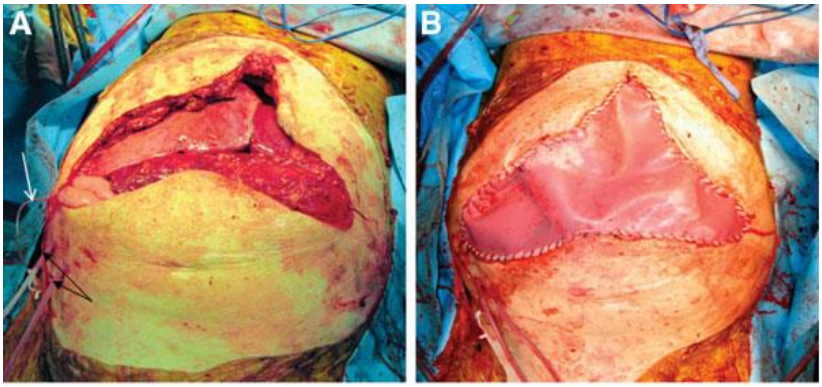


Figure 7: Mercedes incision used during temporary abdomen closure. (A) Open abdomen after liver transplantation with 2 Jackson-Pratt (JP) drains (black arrows) placedinto abdomen and 8-Fr pediatric feeding tube (white arrow) temporarily tied to the distal common bile duct. (B) After silastic mesh is sutured to the skin.
Source: Jafri MA, Tevar AD, Lucia M, Thambi-Pillai T, Karachristos A, Trumbull L, Buell JF, Thomas MJ, Hanaway MJ, Woodle ES, Rudich SM. (2007) Temporary silastic mesh closure for adult liver transplantation: a safe alternative for the difficult abdomen. Liver Transpl, 13: 258-265.

Native liver removal

The native liver removal begins with dissection of the hepatic hilum. The dissection is carried down to the hepatic artery, which is divided above its bifurcation. The most frequent site chosen for the subsequent anastomoses is the region of the CHA and GDA. Then the cystic duct is cannulated and division of the common duct above the cystic duct is carried out. It is important to take care to preserve the longitudinal vessels supplying the common bile duct.

The next step is the portal vein transection. Management of the portal vein will depend on the chosen OLT technique (IVC cross-clamping with veno-venous bypass or preservation of the IVC).

It had already been demonstrated by the initial experimental OLT in dogs that some type of bypass would be needed to tolerate the anhepatic phase [51]. Even though cirrhotic patients could better tolerate portal clamping, it was shown by Pittsburgh studies in the early 1980s that OLT with veno-venous bypass, without anticoagulation [52], was performed with better haemodynamic stability and lower blood needs (Figure 8).

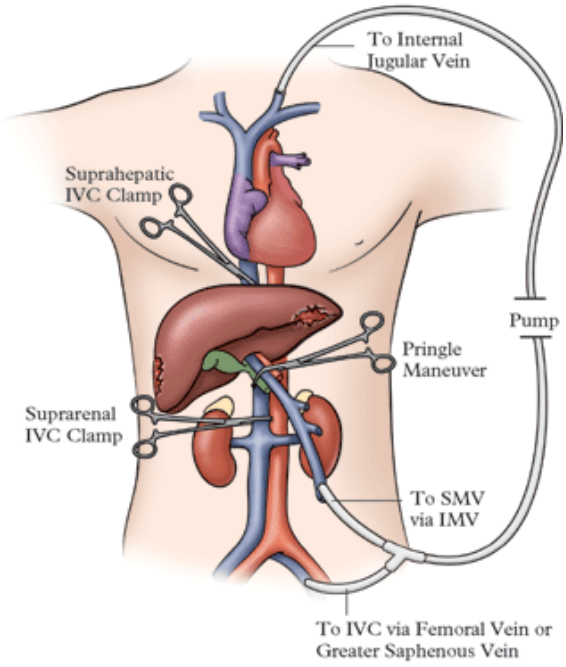


Figure 8: Portofemorojugular venous bypass.
(Source: http://www.snipview.com/q/Veno-venous_bypass)

Nevertheless, some complications have been described when bypass is used [53], and some haemodynamic alterations cannot be avoided [54]. For this reason, in 1989 Tzakis et al. described the technique of OLT with preservation of the IVC, called the ”piggy-back” technique [55]. It has been shown that with the use of this technique OLT can be performed with better haemodynamic stability, lower blood transfusion requirements and shorter operative time [56, 57, 58].

Although the piggy-back technique improves the haemodynamic stability during the anhepatic phase, the portal vein clamping induces portal hypertension and splanchnic congestion, which can induce renal dysfunction and make dissection more difficult. In 1993, Tzakis et al. [59], and afterwards Belghiti et al. in 1995 [60, 61], described the use of temporary portocaval shunt in OLT. It has already been demonstrated that the use of temporary portocaval shunt associated with the preservation of the IVC can be performed safely in most patients, without the need for veno-venous bypass, and with haemodynamic and renal improvements [62, 63]. This fact is confirmed with a prospective randomized study that demonstrated that the association of a temporary portocaval shunt with the preservation of the IVC technique achieved better haemodynamic stability during the anhepatic phase, and this was associated with lower blood transfusion requirements and better renal function, mainly in those patients with higher portal blood flow or higher portocaval gradient [64]. It should be noted that temporary portocaval shunt is particularly useful in patients with fulminant hepatitis. These patients do not have hepatofugal circulation and tolerate poorly the fluid overload necessary to maintain haemodynamic status during the anhepatic phase [61].

After arterial and hepatic duct section, the portal vein is dissected widely. Afterwards, the anterior face of IVC is exposed. After clamping and section of the portal vein, the vena cava is laterally clamped, and an end-to-side anastomosis with a running suture with 5/0 polypropylene (Prolene 5/0; Ethicon, Somerville, NJ, USA) is performed. The hepatectomy can then be performed safely, with the preservation of the caval and portal flow. The early hepatic devascularization, and the absence of splanchnic congestion due to the portocaval shunt, facilitate the division of hepatic ligaments, ligation of small hepatic veins and further mobilization of the liver.

Subsequently, the complete dissection of the anterior face of the lower caval vein is done by ligation of the small vessels that join the caudate lobe to the caval vein. To complete the exposure of the right hepatic vein, the hepatocaval or Makuuchi's ligament must be divided. Depending on the anatomic distribution and the size of the hepatic veins, we will decide to ligate and section the right hepatic vein, or if possible join it creating a common hole with the middle and left hepatic veins. In some cases the anatomic distribution of the recipient hepatic veins makes it difficult to join them. In such cases, it is preferable to create an orifice from the middle and left hepatic veins and extended caudally to the lower caval vein, thus performing a ”face-à-face” venacavaplasty [65, 66].

It is essential to achieve enough long venous cuffs for the subsequent anastomoses. Once all hepatic veins have been exposed, they are clamped, while avoiding clamping the IVC.

Vascular anastomoses

After the removal of the native liver, the liver graft can be implanted. Before initiating the vascular reconstruction, the surgical field must be completely ready, with perfect haemostasis, and adequate vascular cuffs prepared.

The liver allograft implantation begins with the suture of the donor upper vena cava to the cuff created with the three recipient hepatic veins (or the hepatic venous orifice created with the middle and left hepatic veins with an extension inferiorly onto the inferior caval vein), by a running 3/0 polypropylene suture (Prolene 3/0; Ethicon).

Afterwards, we can perform the portal or arterial anastomoses. If we begin with the portal one, then the portocaval anastomosis has to be taken down. If we begin with the arterial anastomoses, the portocaval shunt can be maintained until the arterial anastomoses are finished. The ”piggy-back” technique with temporary portocaval shunt allows one to choose the order of graft revascularization.

To take down the portocaval shunt, the portal side is clamped, and a vascular stapling device closes the caval defect. Before performing the portal anastomoses, the graft is washed with 1 liter of lactate Ringer at 38 °C (washing with blood or cold perfusion has also been described), with the patient positioned in the Trendelenburg

position. The inferior opening of the donor vena cava is left open to allow the drainage of the effluent from the flush of the graft. After washing the graft, the caudal opening of the donor IVC is closed with a vascular stapler.

Then the portal anastomosis is performed, with a running 5/0 polypropylene suture (Prolene 5/0; Ethicon).

The arterial anastomosis is performed by a running or interrupted 6/0 polypropylene suture (Prolene 6/0; Ethicon) generally at the level of the CHA bifurcation, or at the cuff created with the GDA take-off. If the graft has some kind of arterial anomaly it should be solved at the bench surgery to create an appropriate vascular cuff. On the other hand, if the recipient hepatic artery is not adequate to perform a safe anastomosis, several alternatives have been evolved to solve this problem. In most cases, the arterialization of the graft will be achieved using the aorta, through either direct anastomoses or the placement of an arterial conduit between the aorta and the donor hepatic artery [67]. Although different grafts can be used, the preferable one is a graft of donor iliac artery. The location of choice for the graft is an end-to-side anastomosis to the supracoeliac aorta. Sometimes it is not possible to anastomose the graft at the supracoeliac aorta; in such cases, the origination of the arterial graft could be the infrarenal aorta [68]. The graft then has to be passed through the transverse mesocolon, anterior to the pancreas, to reach the hepatic hilum.

One alternative to the graft, particularly suitable when splenomegaly is present, is to use the recipient's splenic artery (SA) for arterial reconstruction [69].

Biliary tract reconstruction

After performing the cholecystectomy, the biliary tract reconstruction is done. The standard technique is the end-to-end choledochostomy with an interrupted (or running) suture using PDS 5/0 (Violet polydioxanone; Ethicon), without a T-tube stent.

In some cases of inadequacy of the biliary duct size, or depending on the underlying disease (primary sclerosing cholangitis), a Roux-en-Y hepatojejunostomy will be needed.

Some centers use a choledochostomy stented with a T-tube. Nevertheless, since the report from Rouch et al. in 1990 demonstrating that most of the early biliary

complications were T-tube-related [70] most centers decided to use a choledochostomy without a T-tube, whenever possible.

3.4.3 Split liver transplantation

The development of split liver transplantation started with the left lateral ex situ split procedure, creating a left lateral graft for a child and an extended right graft for an adult recipient by Pichlmayr in 1989 [40]. The feasibility of ex situ technique and its safe application was also shown [44]. Rogiers introduced the technical modification by performing the procedure in situ in 1995 [43, 45]. Nowadays, the decision as to whether to perform the split procedure in situ or ex situ is often a logistical question. Performing the full split procedure by dividing a deceased donor liver along the line of Cantlie into hemilivers for transplantation of 2 adults marked a further progression of the art [71].

In situ splitting

The in situ split procedure is performed in the heart-beating deceased donor and was developed on the basis of the experiences with living donation [45]. The in situ technique shows some advantages compared with the ex situ procedure; the cold ischemic time remains short, avoiding any additional graft injury. Second, due to the chance of complete hemostasis and an easier detection of bile leaks of the perfused cut surface, bleeding and biliary complications can be reduced and the surgeon who implants the graft is faced with a more controlled situation. Due to the shorter ischemic time and a minimizing of back-table preparation, the sharing of grafts between different centers is simplified and the respective implanting surgeon does not necessarily need the skill for splitting but the competence to handle a segmental graft. The potential disadvantage of in situ splitting is a higher amount of logistical needs. The operating time in the deceased donor is lengthened and that might mean a higher burden for the respective hospital and waiting time for other teams to harvest (e.g., the thoracic organs). The deceased donor must be correctly managed to assure a hemodynamic stable situation while splitting.

In Situ Left Lateral Split. The first step during the left lateral split procedure is the exploration and assessment of graft quality and anatomic evaluation. Therefore, the

abdomen and thorax of the heart-beating deceased donor is opened by midline laparotomy and sternotomy. Before any further steps, the standard technique to prepare the organ harvesting must be performed. The split procedure starts with the mobilization of the left lateral liver lobe by transection of the left triangular ligament and the attachments to the diaphragm. The lesser omentum is dissected and thereby examined for an accessory or replaced hepatic artery arising from the left gastric artery, which has to be saved.

The hilar dissection starts at the lower margin of segment IV with the preparation of the left hepatic artery throughout its course to the left liver. Attention should be paid to a probable A4 arising from the LHA or PHA, which should also be dissected. Depending on the significance of inflow to segment IV, this artery should be preserved for the extended right graft. Dissection of the RHA should be avoided to prevent arterial and biliary complications to the extended right graft. During the next step, the parenchymal bridge between the left lateral lobe and segment IV is divided, exposing the left portal vein and the umbilical recessus. To prepare and ligate the portal branches to segment IV, the right bordered peritoneal sheath of the umbilical recessus is opened. Portal branches to segment I arising from the left portal vein are also divided. The LHA and left portal vein are marked with vessel loops. The complete mobilization of the left portal vein gains access to the umbilical plate. To dissect the left hepatic vein, the obliterated ductus venosum within the sulcus of Arantius should be dissected down to the IVC. The left hepatic vein is also marked with a vessel-loop to allow the vessel-loop-guided parenchymal dissection [72]. In case of an intrahepatic common middle and left hepatic vein, the left vein must be separated by parenchymal division. In rare variations, segments II and III drain separately into the IVC, which must be recognized to assure a reconstruction on the back-table for optimal venous outflow [8]. If available, intraoperative ultrasound can be used to detect larger hepatic veins crossing the transection line.

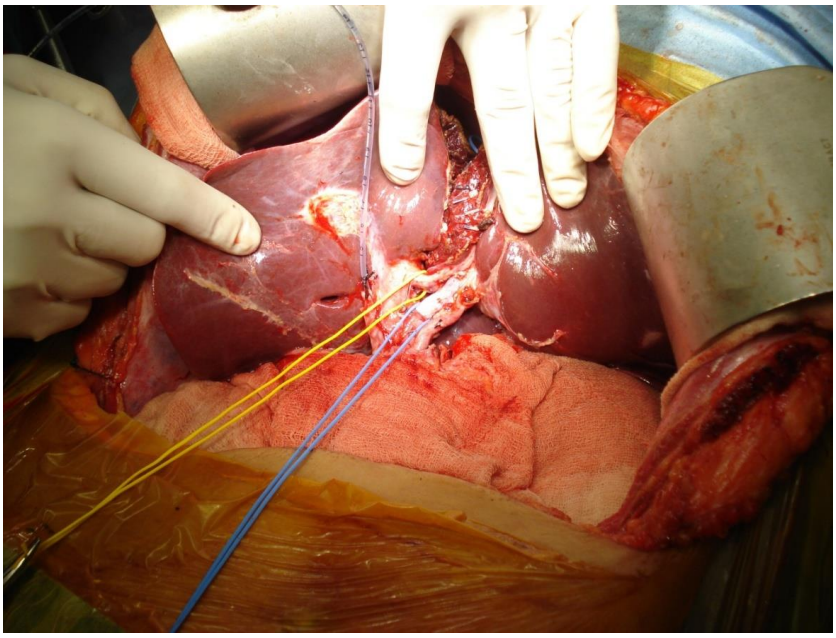


Figure 9: Partial liver transplantation. The exposed vascular structures are marked with vessel loops. (Source: www.vkumaran.net/domino_liver_transplant)

The hypovascular plane for the parenchymal transection runs along the right side of the falciform ligament. The liver parenchyma is divided using either the Harmonic Scalpel (Ethicon Endo-Surgery, Miami, FL) or an ultrasound dissector for optimal control and minimal parenchymal loss or damage (Figures 9 and 10). After reaching the umbilical plate, this region is controlled by using a dissection clamp to allow sharp transection of the umbilical plate containing the left lateral bile duct. No electrocautery should be used in this region to avoid any devascularization of the bile duct. The challenge is to end up with only 1 bile duct orifice to anastomose in the recipient. Toward the left, one might probably meet 2 bile duct openings-segment II and III separately. The more one moves to the right, one might raise the risk of narrowing or at least injuring bile ducts of segment IV and segment I.

After cutting the hilar plate, the right side of the bile duct orifice is sewn over as well as the rest of the hilar plate in a running matter to avoid bile leakage from the smallest additional bile ducts. Then, the parenchymal division is finished by transection between middle and left hepatic veins, guided by the vessel-loop placed behind the left lateral lobe and anterior to the left portal vein and artery.

The explantation of both grafts can now be finished after standard cold perfusion in the deceased donor or the left lateral lobe can be harvested first and perfused on the

back-table. Therefore, the left hepatic artery is clamped and cut at its origin from the proper hepatic artery, followed by the left portal and the left hepatic vein. The stumps of the vessels are sutured and the extended right lobe is harvested after perfusion in the standard fashion and both grafts are stored in ice-cold preservation solution.

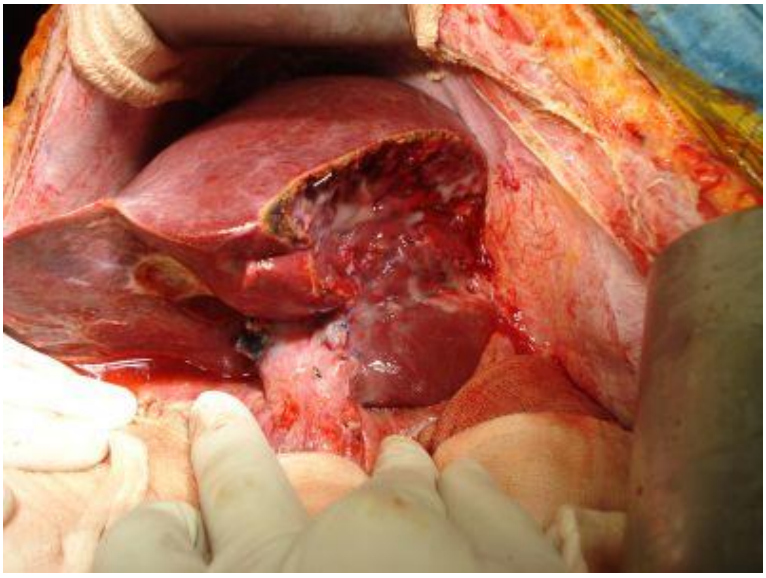


Figure 10: The remaining liver segments (IV – VIII) within the donor, following left lateral splitting. (Source: http://www.sgrh.com/supercat.aspx?id=35)

In Situ Full-Left Full-Right Split. To obtain a full left lobe (segments I-IV) and a full right lobe (segments V-VIII) by in situ split procedure in the deceased donor, the first steps are similar to those described for left lateral splitting. Respecting the frequently occurring variations of the right liver bile duct anatomy, which can possibly contraindicate the full split procedure, cholangiography via the cystic duct should be performed if available. Thereafter, hilar dissection and preparation of the hepatic artery starts with special attention to and preservation of the A4. The portal vein is dissected up to its bifurcation. The hepatic veins are identified and the right hepatic vein is marked by a vessel-loop to perform the vessel-loop-guided transection [72]. The retrohepatic veins are preserved to allow further implantation in the recipient if needed. Intraoperative ultrasound can help to identify crossing major segment V or VIII veins to prevent bleeding. For demarcation of the division line of Cantlie, a short clamping of the right hepatic artery and right portal vein can be performed. The parenchymal transection is performed by using the ultrasonic dissector. Crossing segment V and VIII veins are isolated and marked with removable large clips to enable back-table

reconstruction. The middle hepatic vein remains with the left liver lobe (Figure 11). The left bile duct is sharply divided a few millimeters left to the main hepatic bifurcation and cold perfusion starts after complete parenchymal dissection. The right hemiliver is harvested by cutting the RHA and right portal vein at their origins and the right hepatic vein is divided with a caval patch. The back-table procedure includes the bile duct flushing and reconstruction of the venous outflow of segments V and VIII on the cut surface. The left hemiliver is removed as well and on the back-table, the orifices of the RHA, right portal vein, and the right hepatic vein are oversewn. The IVC remains with the right lobe if splitting of the IVC was not possible.

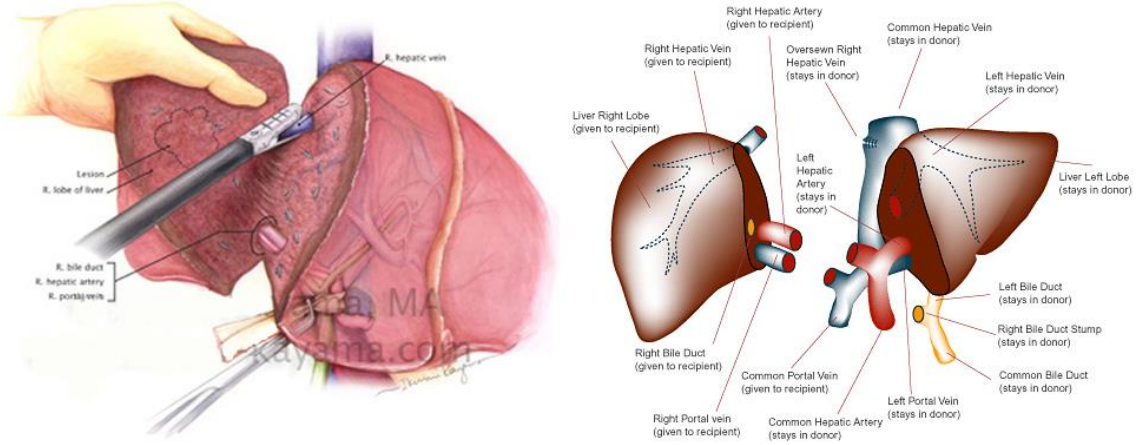


Figure 11: Full-left full-right split.
(Source: http://intensivecarehotline.com/clinical-pictures/liver-transplant/)

Ex situ splitting

To divide a liver graft ex situ offers a more flexible and practical application of liver splitting in situations where the logistical conditions for an in situ split are not available and opens up other possibilities for making use of the anatomy of the deceased donor liver. The main obstacle remains the prolonged ischemic time and the risk of premature rewarming of the graft during the split procedure.

Ex Situ Left Lateral Split. The hepatectomy in the deceased donor can be performed standardized en bloc [73] after perfusion, stored in ice-cold preservation solution, and transported to the respective transplant center. The optimal temperature of 0°C to 4°C will be reached after 1 to 2 hours. The total cold ischemic time including the

split procedure of 1 to 2 hours has to be minimized as far as possible. To prevent further rewarming during the back-table preparation, the graft has to maintain in cold preservation solution with crushed ice. The preparation starts with the evaluation of the graft's anatomy and cholecystectomy. The further steps are similar to the in situ technique; the left hepatic artery is prepared and the presence of an accessory or replaced left artery branch is checked. The portal vein is dissected toward the main bifurcation to exclude absence of the left portal vein. Dissection of the bile ducts is obsolete to avoid devascularization.

The splitting procedure starts contrary to the in situ technique with the division of the vessels. The question of where to leave the vessels remains not clearly defined yet. But it is favorable to leave the main trunk with the graft for the primarily allocated recipient. In the majority of cases this will be the child, so the CA stays with the left lateral lobe, cutting the RHA at its orifice. Due to a larger diameter of the RHA, facilitating the arterial anastomosis of the extended lobe, this approach is favorable. The left portal vein is divided flush from the portal trunk and ensures a comfortable length for further anastomosis. The openings of the cut-down vessels of arterial and portal trunks are sutured in a transverse fashion to avoid stenosis.

The left hepatic vein is cut with a small venous cuff for reconstruction of an optimal venous outflow in the recipient. The IVC and right and middle hepatic veins remain with the right extended graft and the defect is also closed by transverse suture or by a plastic patch if necessary.

The parenchymal transection is then performed using the sharp knife technique. First, the parenchymal bridge between segment IV and the left lateral lobe is dissected for the preparation of the left portal vein branch within the sinus of Rex, and after cutting all portal branches to segment IV, full access to the umbilical plate is gained. The transection line follows the right border of the falciform ligament, corresponding to the in situ technique. The splitting surgeon must control the correct dissection plane and keep the position of the middle and left hepatic veins in mind to avoid losing the way. The goal is to cut the liver in a single even plane thereby allowing precise hemostasis by suture every single vessel opening, which can be performed similarly by 2 surgeons, 1 for each graft side [44].

Ex Situ Full-Left Full-Right Split. The full split procedure deals with some special issues. Besides the resulting graft sizes, the main technical problem and most discussed question is how to achieve an optimal venous outflow of both grafts and the sharing of the middle hepatic vein. By leaving the middle hepatic vein with the left lobe like it is described for the in situ technique, the right median sector of the right hemiliver is predisposed to develop venous congestion [74] because these segments V and VIII are partly drained by the middle hepatic vein. To perform an ex situ split has in this case the advantage of undisturbed access to the complete anatomy to create the optimal venous outflow in both grafts. This can be realized by splitting the IVC [75] and the middle hepatic vein [76, 77] (Figure 12).

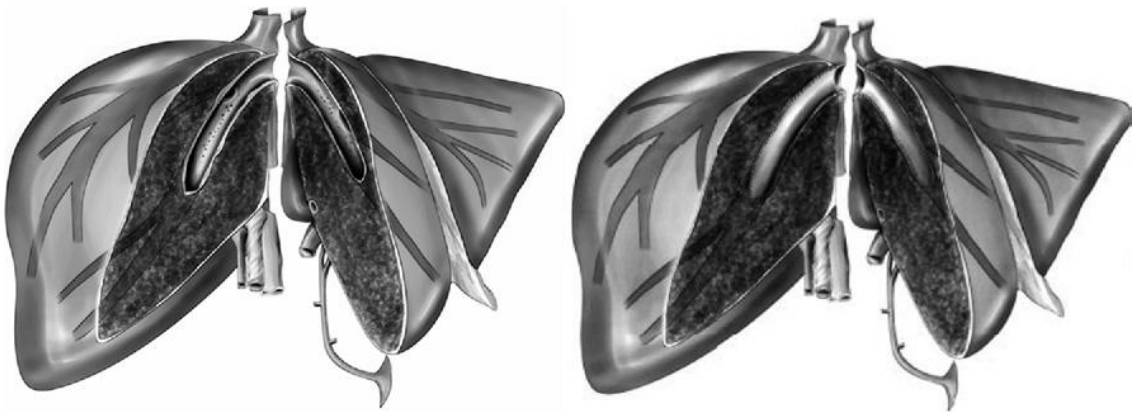


Figure 12: Technique of full-right, full-left splitting with splitting the IVC and the middle hepatic vein. Reconstruction of the middle hepatic vein is made with an iliac vein patch. Source: Broering DC, Bok P, Mueller L, Wilms C, Rogiers X. (2005) Splitting of the middle hepatic vein in full-right full-left splitting of the liver. Liver Transpl, 11: 350-352.

Hilar dissection starts by identification and preparation of the hepatic artery bifurcation and A4. The artery transection will depend on the origin of the A4. The portal vein is dissected down to the main bifurcation and the main portal vein stays with the left hemiliver to preserve the segment I branches. The division of the bile duct results in leaving the main bile duct with the right liver lobe due to frequently more biliary variants of the right hemiliver. Before starting the parenchymal transection, the dorsal and ventral wall of the IVC is cut in the midline, acquiring 2 hemicaval patches [75]. The parenchyma dissection is performed using the sharp knife technique described

by Azoulay along the line of Cantlie to achieve a plane cut surface. Ignoring splitting of the middle hepatic vein, the cutting line is right to the middle hepatic vein. During the first applications of splitting the middle hepatic vein, it was cut in the middle from inside the IVC, preserving one half of the middle hepatic vein in each of the hemilivers [77]. Those were then reconstructed with donor iliac vein patches, but the resulting diameters of reconstructed veins were not always sufficient and this led to improvement of this technique. Now, the division of the middle hepatic vein starts within the joining of the segment VIII vein leaving the proximal 1 to 2 cm of the middle hepatic vein intact with the left lobe [76] (Figure 13).

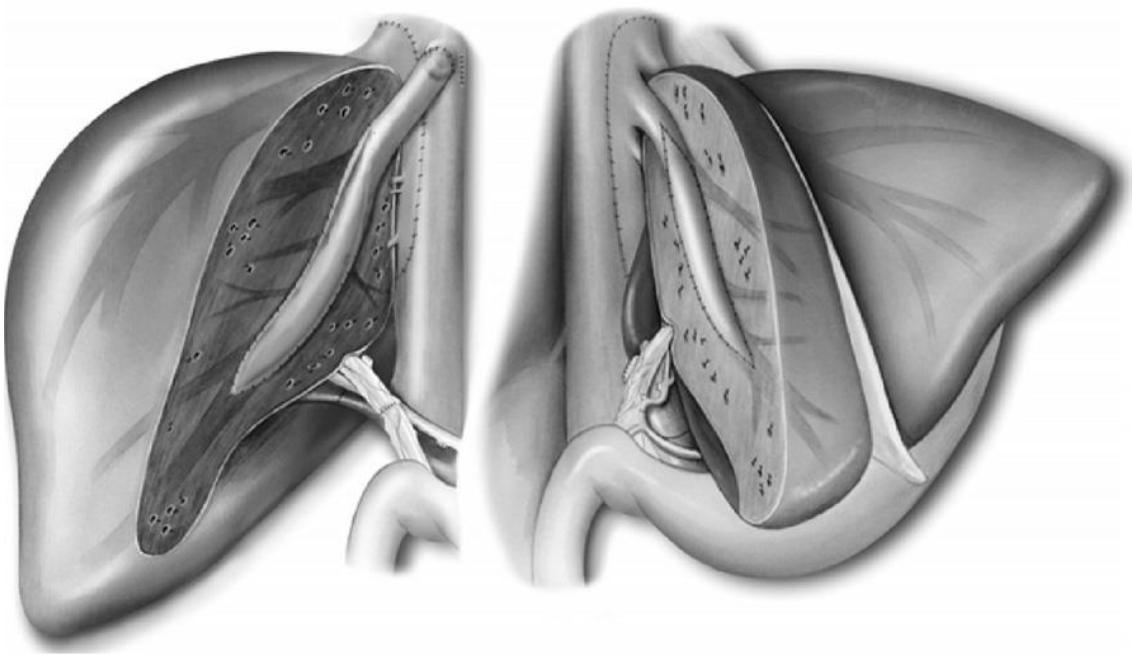


Figure 13. Modified technique of ex situ full-left full-right splitting including split of the IVC and middle hepatic vein. The proximal part of the middle hepatic vein stays with the left graft.
Source: Broering DC, Wilms C, Lenk C, Schulte am Esch J 2nd, Schonherr S, Mueller L, Kim JS, Helmke K, Burdelski M, Rogiers X. (2005) Technical refinements and results in full-right full-left splitting of the deceased donor liver. Ann Surg, 242: 802-812, discussion 812-3.

Then, the split portion of the middle hepatic vein of the left hemiliver is reconstructed with half an iliac artery and for the right portion an entire iliac vein graft is used. The benching procedure is finished by oversewing the vessel openings at the cut surface as described for ex situ left lateral split.

Implantation of the segmental liver graft

In general there will be no major differences to the implantation of a whole organ graft, but the implanting surgeon must keep the special features of the segmental grafts in mind to provide optimal venous outflow, arterial and portal perfusion, and biliary drainage. The recipient operation starts with the hepatectomy and regardless of the type of segmental graft, it is favorable to keep the recipient's hepatic artery, portal vein, and bile duct as long as possible. Particularly those grafts without the main vessel trunks can have relatively short vessels and the use of interposition grafts should be avoided by using the recipient's hilar structure as much as possible.

Left lateral liver lobe. For implantation of the left lateral lobe, the orifice of the recipient's right hepatic vein can be oversewn and the small retrohepatic veins can be ligated and sutured. The confluence of the left and middle hepatic veins is preserved and the opening is enlarged by a longitudinal incision of the ventral wall of the IVC. The corresponding orifice of the graft's left hepatic vein can also be widened dorsally to provide optimal venous outflow. The graft's left hepatic vein is then anastomosed in an end-to-side fashion to the common trunk IVC opening [78]. The anastomosis of the portal vein is performed end-to-end between the graft's left portal vein and the recipient's portal vein bifurcation in a running fashion. To prevent stenosis, we use a growth factor of at least one third of the portal vein diameter. Before closure of the portal vein anastomosis, the graft is perfused with human albumin via the portal vein to wash out the potassium-rich preservation solution. Caval and portal vein anastomoses are then finished. The arterial reconstruction is a crucial step in the transplantation of small children due to a reported risk of hepatic artery thrombosis of 7% to 8% [79]. The arterial anastomosis can be performed in several ways, depending on the presence of a graft's CA and the size of the recipient's artery. Preferably it can be performed end-to-end between the graft's LHA and the recipient's PHA. If the recipient's artery is not of appropriate size, the graft's CA, if present, can be anastomosed end-to-side to the infrarenal aorta or an interposition graft must be used. Microsurgical techniques such as the use of magnification (4x) loupes or a surgical microscope are strongly encouraged. The biliary reconstruction in left lateral split liver transplantation is performed by Roux-en-Y hepaticojejunostomy (Figure 13). Each significant bile duct must be anastomosed.

To prevent bile leaks from the smallest accessory bile ducts, the hilar plate should be sutured up to the anastomosis.

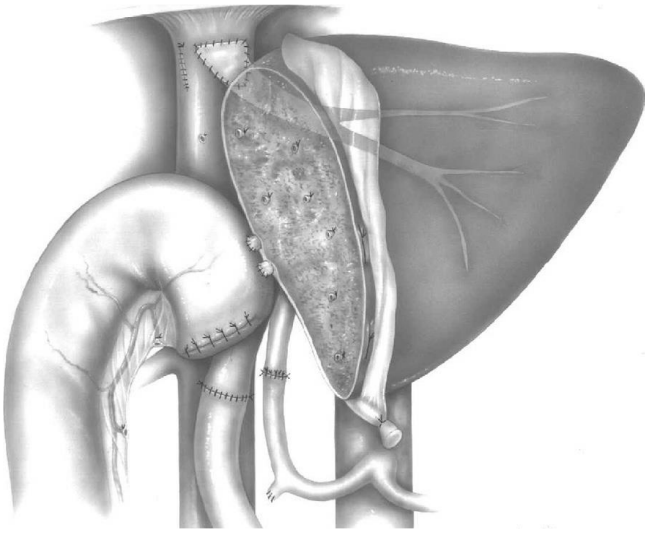


Figure 13: Implantation of the left lateral liver lobe.
Source: Broering DC, Walter J, Braun F, Rogiers X. (2008) Current status of hepatic transplantation. Anatomical basis for liver transplantation. Curr Probl Surg, 45: 587-661.

After reperfusion, immediate Doppler ultrasound of the graft is performed with measurement of the different perfusion modalities. Measurement must be repeated before and after closing the abdominal wall to avoid any perfusion deficits due to increased intra-abdominal pressure. To avoid necrosis of the graft, the abdomen should be secured by a mesh to allow shrinking of the graft.

Extended right liver lobe. After left lateral splitting of a liver, the extended right lobe is left with the IVC, the portal trunk, the RHA only, and the common bile duct. Then the implantation hardly differs from the implantation of a whole graft.

Before starting the recipient's operation, care must be taken that the graft's phrenic veins are ligated, the orifices of the left hepatic vein, left portal vein, LHA (in presence of the CA), and the left bile duct are sutured and the cut surface should be inspected. In the recipient, the hepatectomy follows the piggyback technique [55]. The IVC is partially clamped at the level of the common trunk. To ensure a large outflow, the openings of the left and middle hepatic veins are reunited and widened ventral to the IVC. After closing the distal IVC of the graft, the dorsal wall of the IVC is

longitudinally incised according to the incision length of the recipient's caval opening. Anastomosis is then performed side-to-side with a running suture (Figure 14).

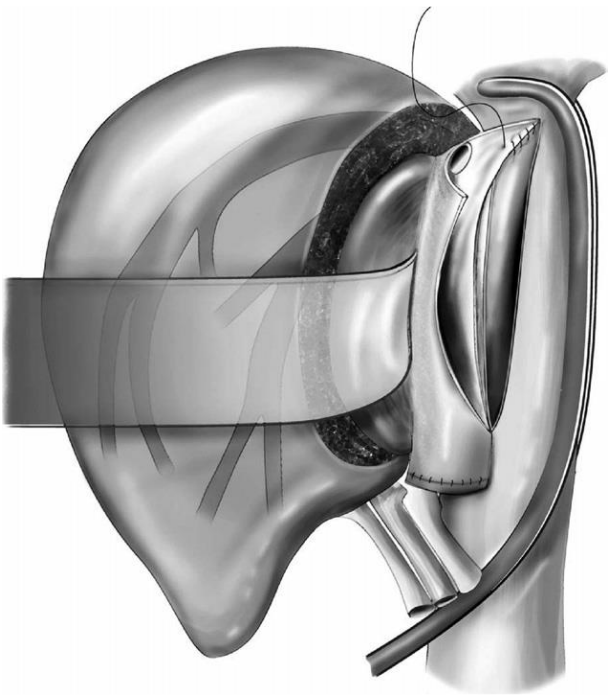


Figure 14: Implantation of the extended right liver lobe using the piggyback technique. Source: Broering DC, Walter J, Braun F, Rogiers X. (2008) Current status of hepatic transplantation. Anatomical basis for liver transplantation. Curr Probl Surg, 45: 587-661.

The portal vein anastomosis is performed in an end-to-end fashion. In terms of a recipient's portal vein thrombosis or anomaly, the portal venous reconstruction should be performed by using an iliac vein of the same donor. The arterial reconstruction depends on the anatomical conditions of the graft. In case of a separate A4 or additional accessory right arteries arising from the SMA, the implanting surgeon must decide whether to perform a reanastomosis. In optimal situations with a single RHA, it will be anastomosed favorably in a branch patch technique [80] end-to-end to the recipient's hepatic artery in the area of the GDA passage. Microsurgical technique is again of paramount importance. The biliary anastomosis can usually be performed in an end-to-end fashion, except in cases of sclerosing cholangitis or special situation in which a Roux-en-Y hepaticojejunostomy seems to be useful. While performing end-to-end anastomosis of the bile duct, the surgeon must ensure that both bile ducts are well vascularized to avoid ischemic necrosis. Intraoperative ultrasound should be used to

prove regular perfusion of the graft. The cut surface must be checked once more before closing the abdomen to prevent bile leakage.

Full left liver lobe. Using the refined full split technique as described above, the implanting surgeon is faced with a full left graft, which is retained with a hemicaval patch, the main arterial trunk, main portal vein, and the left bile duct. The proximal part of the middle hepatic vein is left untouched; the remnant is reconstructed with an iliac venous patch, enabling only 1 venous anastomosis. In the recipient, the right hepatic vein can be dissected and sutured while the common orifice of middle and left hepatic vein is enlarged by a more leftward placed ventral longitudinal incision as described for the implantation of the extended right lobe. The left hemicava of the graft is then anastomosed in a side-to-side fashion. The following steps of the implantation do not differ from that described for whole organ or extended right lobe implantation. Special attention should be paid to the large resection plane and the stump of the hilar plate. Before biliary reconstruction integrity of the segment I bile duct must be proven by probing of the left main bile duct and inspection of the left umbilical plate.

Full right liver lobe. The right hemiliver resulting from the full split procedure is generally presented with the right hemicaval patch, the right portal vein, the RHA, and the common bile duct. Using the technique of splitting the middle hepatic vein, the longitudinal opening of the middle hepatic vein has been reconstructed with an entire iliac vein of the deceased donor. The implantation of the full right graft slightly differs from that of the full left liver graft regarding the venous reconstruction. The opening of the right hepatic vein in the recipient is longitudinally incised ventral to the IVC to allow a wide side-to-side cavocavostomy [81]. The right hemicaval patch includes most of the retrohepatic veins and ensures their venous drainage. The described reconstruction of the middle hepatic vein enables an end-to-end anastomosis between the opening of the iliac vein and the recipient’s stump of the common trunk (Figure 15).

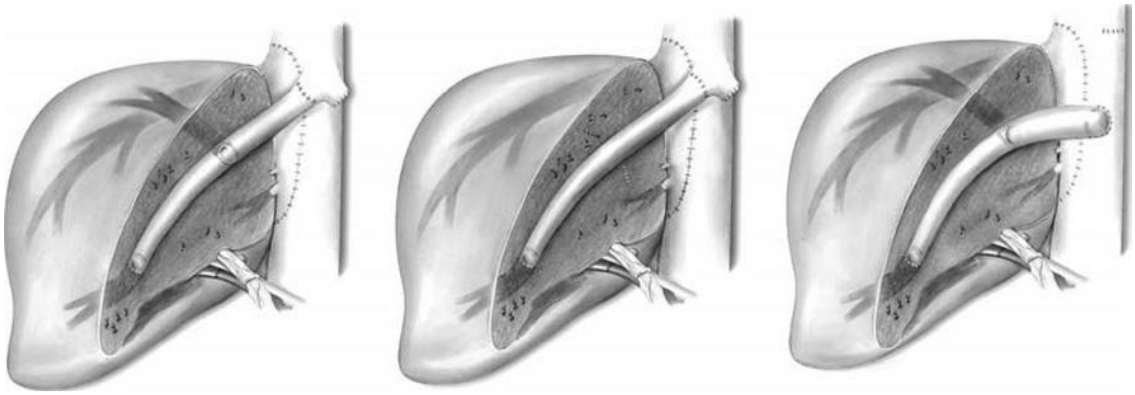


Figure 15: Implantation of the right liver lobe. Different variations of reconstruction of the middle hepatic vein using iliac vein patches to provide optimal venous drainage.
Source: Rogiers X, Bismuth H, Busuttil RW, Broering DC, Azoulay D. Split Liver Transplantation. Springer, Darmstadt, 2002.

The remaining procedure of implantation is in accordance with that of the extended right lobe and the full left lobe as described above. The portal vein branch may be shorter than that of the full left lobe, but the length will be sufficient in the majority of cases. Regarding arterial reconstruction, the individual anatomy is of interest and end-to-end anastomosis to the recipient's hepatic artery without interposition graft is the aim. Using magnification loupes and performing intraoperative ultrasound is mandatory.

3.4.4 Living donor liver transplantation (LDLT)

Since the demand for liver transplantation has continuously expanded through the last few decades due to growing utilization, and marked by improvement of the surgical techniques and the postoperative management, the discrepancy between available deceased donor organs and patients awaiting liver transplantation accelerated as well. The resulting increased mortality on the waiting lists [82] prompted the transplant community to extend the possibility of harvesting segmental liver grafts from healthy volunteer living donors to transplant them into beloved recipients. The first living donor liver transplantations (LDLT) were performed in pediatric recipients using left lateral liver grafts from 1 parent. Rapid advancement in technique and care enabled the transplant surgeons to nearly eliminate the mortality of children on the waiting lists

[83, 84]. The adoption of LDLT for adult recipients was primarily hindered by the higher demand of liver volume for the adult patient but could be realized and further established by using right liver lobes. The living donor procedure faces the transplant community (besides the ethical discussion and justification of putting an otherwise healthy person in a not negligible risk) with the burden of responsibility for this "additional" patient and his or her outcome.

Selection of the living donor. Since donor safety is of paramount interest during all steps of living donor liver transplantation, the first and pivotal step is the careful selection and evaluation of the donor. The living donor should be a healthy volunteer with a comprehensible close relationship with the recipient, not limited to blood-born relatives in most countries. The donor age should be between 18 and 60 years. The blood group is preferably compatible with the recipient, although there are reports about successful ABO-incompatible donations particularly in children, but also in adults using different approaches and immunosuppressive regimens leading to survival rates of 60% to 80% [85, 86, 87, 88].

The donor should present normal liver function, no medical comorbidities, and no history of major abdominal surgery. The body mass index must be below 30 kg/m2 to be aware of thromboembolic events [89]. Any other procoagulatory disorders and risk factors for thrombosis must be excluded to prevent perioperative development of pulmonary embolism as one of the most feared complications in living donation [90].

The evaluation of the donor follows a stepwise program with assessments of the medical risk, assessment of the remnant liver, suitability of the potential graft for the recipient, and the psychological evaluation of the donor [91]. Due to the fact that only approximately one third of the potential donors will be suitable [92], the invasive and cost-effective steps are performed in late stages of evaluation.

The examination of the liver quality and especially the prospective remnant liver volume is of high importance to the donor's outcome. Particularly in adult-to-adult LDLT, where right liver lobe donation is planned, the donor must be safeguarded from postoperative liver failure due to insufficient remnant volume. The critical threshold of remnant liver volume has been assumed to be 30% of the standard liver volume to provide a safety margin, since the lowest limit was reported to be 27% [93]. Presence of relevant steatosis must be checked as well, as it would lower the functional liver mass.

To exclude fatty degeneration of the liver, liver biopsy in cases of right lobe donation is demanded during evaluation.

Examination of the vascular anatomy is also crucial to identify potential contraindications. Those might be the absence of the main portal vein bifurcation or significant biliary or arterial malformations, explaining the need for MRI or CT angiography and cholangiography. Again, right lobe donation will require the closest investigations, particularly regarding the arterial blood supply of segment IV, which can arise from the right side as well as from the left side and also regarding the various biliary anatomy of the right hemiliver.

Selection of the graft. Generally, the decision regarding which kind of graft should be used depends on the demand of the recipient matched with the individual situation in the donor. To fulfill the recipient's metabolic demands, a liver volume of at least 0.8% to 1% of the body weight (graft-to-recipient weight ratio, GRWR) should be targeted. Living donation for children up to 25 kg will require resection of the left lateral liver lobe (segments II-III) in the donor. Children and small adults between 30 and 60 kg need the implantation of a full left lobe (segments I-IV), representing approximately 40% of liver volume of the donor. Donation for adult recipients weighing more than 60 kg mainly necessitates harvesting a right liver lobe (segments V-VIII). In rare cases, the transplantation of the right lateral sector including segments VI and VII, the extended right lobe, the extended left lobe, or monosegmental transplantations have been reported, but these procedures are still exceptional cases [94, 95, 96]. Another unique solution in situations in which the prospective remnant liver volume is too small for the donor or the graft size is not sufficient for the recipient or both (small-for-size syndrome), is transplantation of 2 smaller grafts from 2 donors, which can result in a sufficient combined graft volume. This dual graft LDLT is an immense technical and logistical challenge, but is feasible and remains available to individual cases and specialized centers [97, 98].

Selection of the recipient. In principle, evaluation of the recipient does not differ from the evaluation for deceased donor liver transplantation. The LDLT recipient must meet all the criteria and must at least be formally listed on the waiting list for deceased donor transplantation. If available, a deceased donor organ will be preferred before LDLT, always in respect of the donor risk. The pivotal question remains, which patient

will benefit the most from LDLT. In countries where there is a choice and the resource of deceased donors, LDLT should be reserved for elective patients, whereas highly urgent recipients might show an inferior outcome after LDLT [99, 100, 101] (e.g., with 1-year survival rates of 57% in urgent cases compared with 82% after deceased donor transplantation) [100]. One reason might be a higher demand of liver mass in critical situations, with an aimed graft weight of more than 1% of the recipient's body weight, which can rarely be realized by living donation. Potential higher complication rates (e.g., biliary morbidity after segmental transplantation) will also hinder the urgent recipient's outcome.

Living donor liver transplantation for hepatocellular carcinoma (HCC) respecting or beyond the current criteria is still under debate. Because of the individual constellations and willingness to donate for a beloved person, the criteria to perform LDLT might be eased, even if the patient would not have a chance to receive a donor organ and is therefore faced with death. LDLT beyond the Milan criteria shows significantly lower survival rates, demonstrated in a Japanese collective with 138 recipients within the Milan criteria and 171 beyond. Three-year disease-free survival rates were 79% and 53%, respectively [102]. Those results might be in contrast to the principle of LDLT, that the risk of the donor should be justified by the expectation of an acceptable outcome in the recipient.

Further attention should be paid to portal hypertension in the recipient. As described in split liver transplantation, a high portal inflow might lead to graft dysfunction in small grafts.

Altogether, donor, graft, and recipient must be carefully matched to provide a safe procedure and acceptable outcome for both donor and recipient.

Left lateral LDLT

Donor Operation. Harvesting of the donor's left lateral segment for a pediatric recipient is nowadays a standardized safe procedure in experienced centers. Regarding the timing of both donor and recipient operation, the procedures can be performed in an overlapping manner if 2 teams with experienced transplant surgeons are available, to

reduce the cold ischemic time. The operative procedure itself is similar to the in situ split described above, since this was adopted from LDLT. After opening the abdomen through a midline incision, the lesser omentum is opened after looking for an aberrant left artery. The left portal vein is prepared by cutting the parenchymal bridge between segment IV and the left lateral segment and then the portal segment IV branches are cut. The LHA gets isolated and marked and the left hepatic vein is then prepared and a vessel loop is placed to allow the vessel-loop-guided transection. The parenchymal division along the falciform ligament is accomplished by using an ultrasound dissector for controlled and parenchyma-saving transection. The left bile duct within the umbilical plate is sharply transected. The vessels are then clamped and cut and the graft is taken back-table and perfused with preservation solution by the portal vein and the artery. Meanwhile, the vessel stumps in the donor are sutured; the hilar plate is sutured in a running manner and the cut surface is checked for bile leakage. After meticulous hemostasis and placing of drainage, the abdomen is closed. Soubrane and colleagues published their experience in laparoscopic left lateral sectionectomy for LDLT with 16 procedures including 1 conversion due to left portal vein injury [103]. The remaining 15 cases could be performed successfully and safely, with significantly lower blood loss but longer operating time compared with a control group of 14 open resections. Hospital stay and amount of analgesics did not differ among the groups, querying an evident advantage at this stage.

Recipient Operation. The implantation of the living donor left lateral graft starts after caval preserving hepatectomy without veno-venous bypass. The recipient's RHA and LHA and the portal vein should have been left as long as possible. To achieve a wide open caval anastomosis, a longitudinal incision of the recipient's IVC after partial clamping is performed and the opening of the left hepatic vein of the graft is also widened. Small children might even tolerate a complete clamping of the IVC during the anastomoses. All anastomoses are performed like described for split grafts above. Measurement of the flow patterns by Doppler ultrasound is mandatory as well.

Hemiliver LDLT

Donor Operation. Harvesting a full right or left graft begins by opening the abdomen through a right subcostal incision with median extension toward the xyphoid.

After close exploration of the abdomen and particularly the liver quality and volume of the respective hemilivers, cholecystectomy and cholangiography via the cystic duct are performed. Based on the findings, a decision of which graft will be harvested is made and the procedure is performed in a fashion similar to the in situ full split technique as described above. Care must be taken about the blood supply of the main bile duct; depending on the type of graft, the main bile duct must be left as untouched as possible to the remnant side. Regarding the hepatic veins, significant retrohepatic veins must be marked to allow anastomoses in the recipient. The question where to leave the middle hepatic vein is still under debate. Some transplant surgeons harvest the graft including the middle hepatic vein to provide an optimal venous outflow for the recipient and showed that this can be accomplished safely and successfully [104, 105, 106]. Notwithstanding, this proceeding might harm the donor by leaving a remnant liver with disturbed venous outflow and the risk of venous congestion. Respecting the donor's safety as highest priority, some centers always leave the middle hepatic vein with the donor and isolate significant veins crossing the line of Cantlie, which will be segment V and VIII veins. All of those veins can be back-table reconstructed by using cryopreserved iliac veins, if possible, to allow further anastomoses in the recipient. This additional procedure is similar to the in situ full split and the left lateral LDLT.

Recipient Operation. The operation of the recipient is in accordance with the implantation of a full split graft and with the approach described for left lateral LDLT. There might be differences in caval anastomoses due to the absence of any cava patch of the graft, and eventually shorter vessels. The venous anastomosis is performed between the longitudinal incisured recipient's IVC and the widened graft's hepatic vein. In case of reconstructed veins on the cut surface or presence of large retrohepatic veins (larger than 5 mm), those must be anastomosed to the IVC as well (Figure 16).

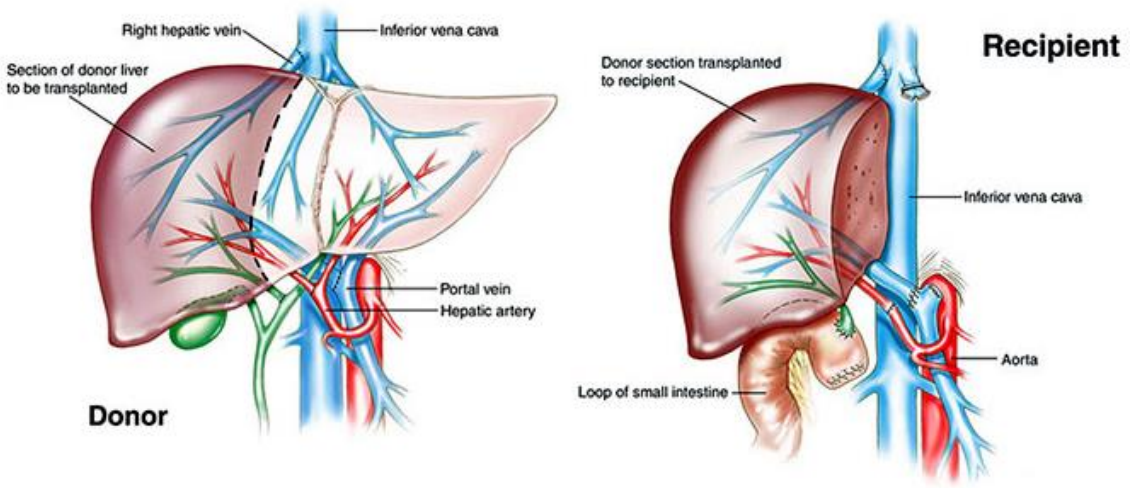


Figure 16: Full-left full-right LDLT. The middle hepatic vein is left with the donor. (Source: http://www.indiahospitaltour.com/liver-transplant-hospital-india.htm)

The biliary anastomosis remains a problem in LDLT as a major cause of morbidity due to smaller and often multiple bile ducts present with the graft. End-to-end bile duct anastomosis is preferred if possible without placement of T-tubes or stents.

4 Objectives

Different vascular anatomic variations in the upper abdomen are part of the daily experience of surgeons and radiologists. The various forms of the hepatic arterial structure are of great importance in terms of liver resections, living donor hepatectomies and split liver transplantation. The anatomic variations of both the extra- and intrahepatic arteries are associated with increased perioperative morbidity and mortality due to the ischemic complications implied, such as insufficient remnant liver volume in the donor, reduction of the functional liver mass in the recipient, intrahepatic abscess formation, ischemic cholangiopathy and hepatic artery thrombosis [107, 108, 109, 110, 111]. Moreover, aberrant hepatic arteries can be of major surgical significance during operations of the upper intestinal tract, the gallbladder and pancreas [112, 113]. They can also present technical challenges for infusion therapy and transarterial chemoembolization of neoplasms in the liver [114, 115, 116]. For this reason, the comprehensive understanding and the precise preoperative identification of the hepatic vasculature is essential to avoid injuries that might compromise the arterial blood supply of the graft.

Concerning the arterial blood supply, in case of deceased donor whole-organ liver transplantation mostly the extrahepatic arterial variations are in the focus of the transplant surgeon. However, during the evaluation of living donors, special attention must be paid on both the extra- and intrahepatic arteries, which greatly influence the planning and performance of the entire transplantation process with special regards to the viability of segment IV [30, 117]. For more than 20 years the left lateral segmentectomy is considered to be the primary approach for obtaining an allograft from a living donor. This procedure allows the left lateral segment of a non-heart-beating donor to be implanted into a pediatric recipient, while the remaining extended right lobe is suitable for an adult patient. In a technically more challenging version of the splitting procedure the donor liver is divided into the left and right lobes (full-left full-right split), the left side can be used for a small adult or teenager, and the right side for a medium-size adult patient. Due to the technical difficulties and the potential increase in postoperative morbidity and mortality, there are only a few reports of true full-left full-right splitting [118, 119, 120, 121, 122, 123].

The first description of the aberrant hepatic arteries was published in 1756 by Haller [23]. Since then the anatomic variations of the extrahepatic arteries have been examined by several authors worldwide. In 1966, Michels' classic series of 200 autopsies [18] defined the basic anatomic variations in hepatic arterial supply and has served as the benchmark for all subsequent contributions in this area, such as the simplified classifications of Hiatt (1994) [27] and Varotti (2004) [28]. According to the international data available, the incidence of the so called "normal" anatomical variation varies between 50.7% [124] and 80.9% [125]. Some authors report about sporadic unusual, surprising anatomical variations, which cannot always fit into (or cannot be explained by) the currently accepted developmental theory of the liver vasculature [126, 127].

Compared to the numerous studies on the extrahepatic arterial supply, the intrahepatic arteries have been less frequently investigated, mostly regarding the arterial branches to the segment IV and the caudate lobe [30, 117, 128, 129]. Few data are available on the segmental branching pattern of the arterial tree in the right lobe [36].

The aim of our present study was to investigate the segmental arterial supply of the human liver by using vascular corrosion casting technique, paying particular attention to rare variations not reported in previous studies. To our knowledge, this is the first study in which corrosion procedure is used to demonstrate the extra- and the intrahepatic arteries together by injecting resin into human cadaveric abdominal organ complexes in relatively large numbers. Our results may contribute to the better understanding of the arterial supply of the liver and may reduce the risk of perioperative complications of surgical and radiological interventions in the upper abdomen.

5 Methods

5.1 Preparation of 3-dimensional vascular corrosion casts

Vascular corrosion casts were prepared from a total of 50 abdominal organ complexes obtained from fresh human cadavers of Caucasoid race, which were delivered to the Department of Forensic and Insurance Medicine, Semmelweis University, Budapest. Written permission was obtained beforehand from the Ethical Commission of Semmelweis University (TUKEB number: 185/2004). The corpses neither had any history of liver disease, nor presented any signs of abdominal trauma or macroscopic alteration (Figure 17).

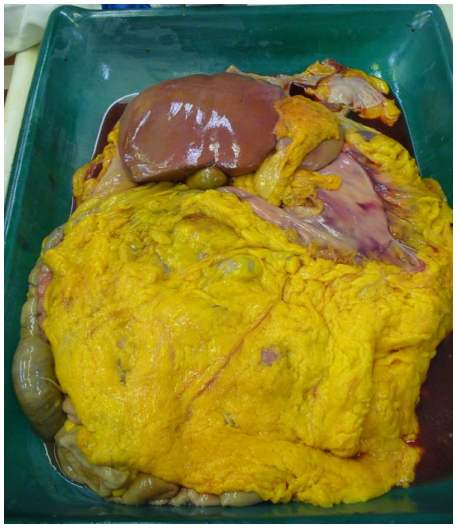


Figure 17: Human abdominal organ complex before preparation.

Following the preparation of the abdominal aorta (Figure 18) a polyethylene cannula was inserted into its proximal end. Lumbar branches, renal arteries and the aorta above the origin of the IMA were ligated (Figure 19).

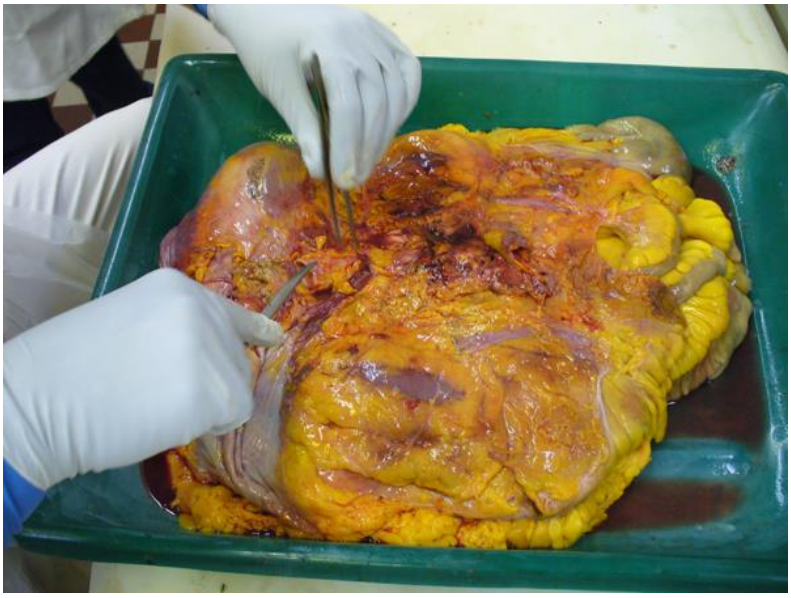


Figure 18: Preparation of the abdominal aorta.

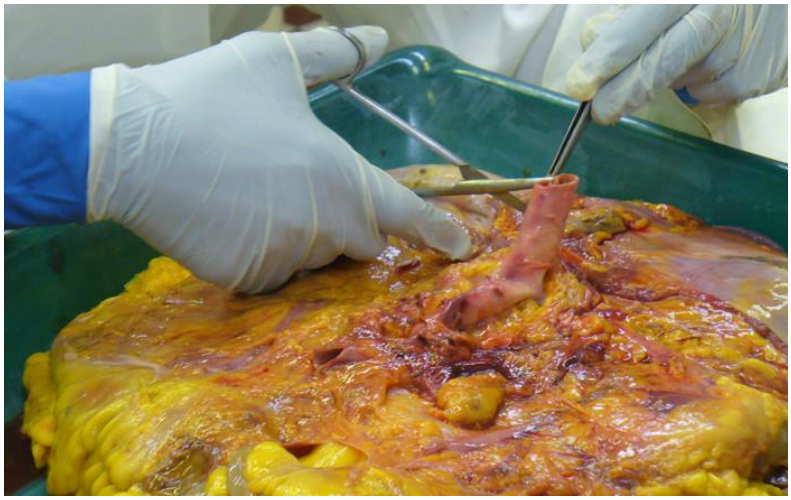


Figure 19: The dorsal side of the aorta is prepared. Lumbar branches are to be ligated.

In order to begin the investigation of the spatial relationship between the main portal vein and the hilar arteries, the portal vein was also injected in 16 cases. For leak control, the specimens were flushed with warm tap water through the abdominal aorta (and the portal vein, if cannulated) to detect and eliminate resin outflow further on. (Figure 20).

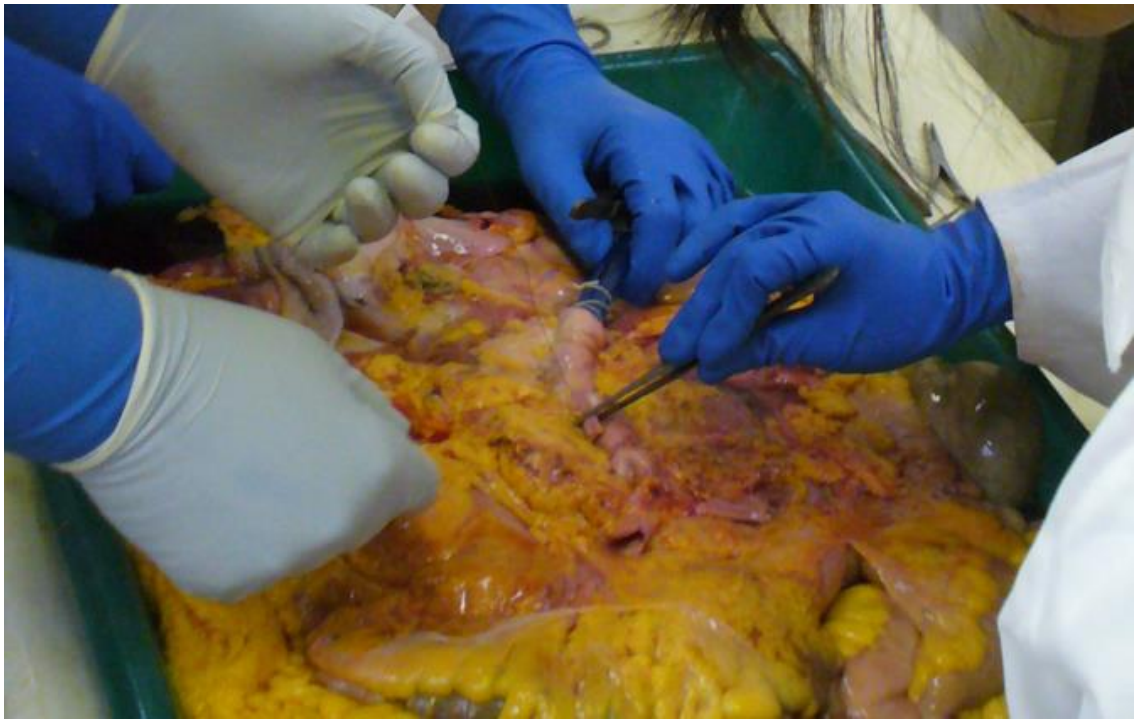


Figure 20: Suture of leakages. The aorta is already cannulated.

In order to ensure the correct anatomical orientation of the organs, the abdominal complex was submerged into tap water at room temperature.

Our research group developed several types of resin mixtures to improve the visualization technique of the arterial system on macro- and microvascular scale in humans and other species as well [130]. A special Vinyl Ester resin mixture developed by M. Kiss was prepared for the purpose of injecting the arteries of the human organ complexes. The components of the mixture were: 1. Resin: Novolac-based Epoxy Vinyl Ester Resin (Derekane 470-300 by Ashland); 2. Pigments (5%): FP3000 red and FP1021 yellow (by Cytec Surface Specialties Austria GmbH); 3. Accelerator (2%): Cobalt 2-ethylhexanoate, N,N-Dimethyl aniline (Accelerator NL-23 by AkzoNobel); 4. Catalyst (2%): Methyl Ethyl Ketone Peroxid (Butanox M-50 by AkzoNobel). The pigment guaranteed not only different colors of the different vessels, but also provided suitable CT density (approx. 250 HU). During the injection the liver was afloat in tap water at room temperature. The viscosity of the resin was set to enter the arteries with a diameter of 0.3-1 mm. The amount of the injected resin was considered enough when it appeared in the subserosal arteries of the large intestines. After filling the arteries with

resin, the proximal end of the aorta was clamped. The resin polymerized in 3-5 minutes, after which, concentrated KOH was added to the surrounding water to commence digestion of the soft tissues. The corrosion process lasted one week at 60-65°C temperature. Residual fat and liver parenchyma were removed by rinsing in warm water, leaving only the cast behind (Figures 21 and 22).

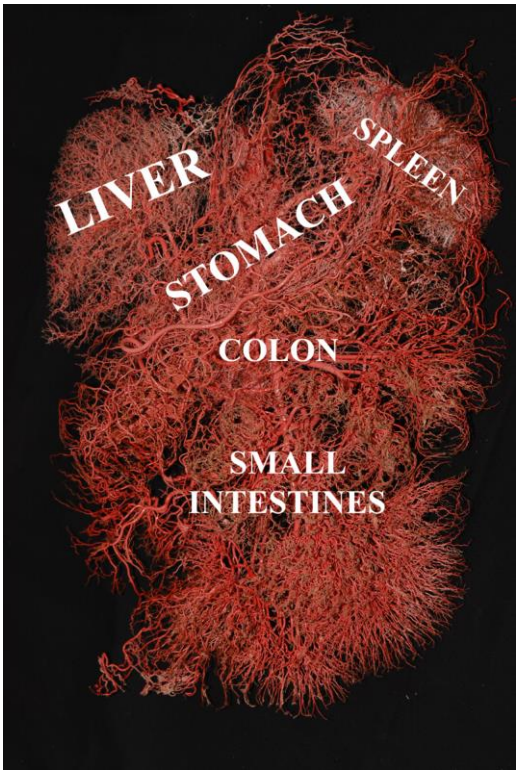


Figure 21: Corrosion cast of organ complex No. 1.

As a result, we could examine the branching system of the CA, the SMA and in some cases, the inferior phrenic artery as well. In 16 cases the portal vein system was also visible with a different colored resin.

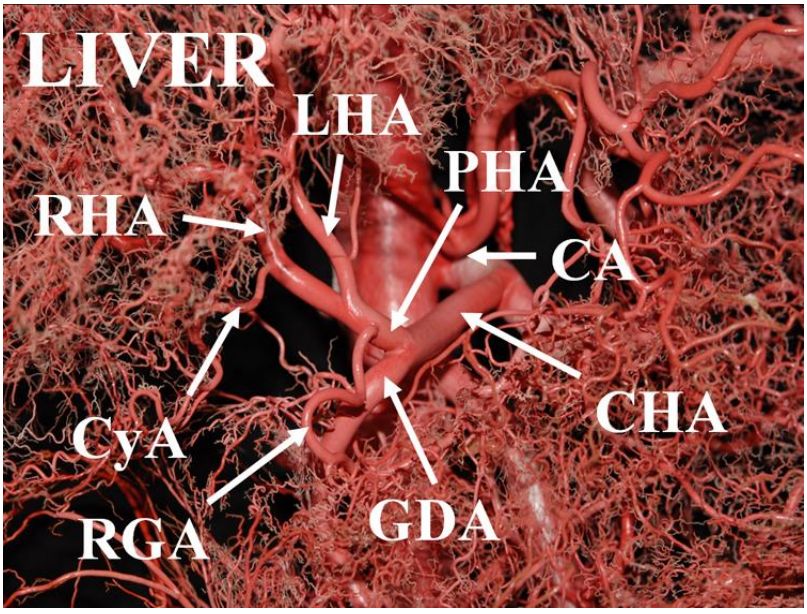


Figure 22: Close-up photography of complex No. 1. Michels I: normal anatomy.

In our present research we examined the patterns of both the extrahepatic and the intrahepatic arterial supply of the human liver. The branching order of the hepatic arteries is demonstrated in Table 3.

Table 3: Branching order of the hepatic arteries.

1st order	RHA, LHA
2nd order	RAHA, RPHA, A2A3, A4
3rd order	segmental arteries: A2, A3, A5, A6, A7, A8
4th order	subsegmental arteries

The point of reference is the PHA, first-order branches are the RHA and LHA; second-order branches are the right anterior and right posterior hepatic arteries (RAHA and RPHA), on the left side one segmental artery and the common trunk of the remaining two segment arteries (most frequently A4 and A2A3); third-order branches are the segmental arteries (except one segmental artery in the left lobe); fourth-order branches are the subsegmental arteries and so on. With our corrosion method we were able to visualize arteries up to 8th-order branching, but according to our experience a liver cast can be precisely analyzed with no more than sixth-order arteries, otherwise the corrosion cast is too dense and the coursing and arborization pattern of even the higher-caliber arteries are difficult to evaluate.

The classification of the extrahepatic arteries was based on Michels' classic results of 200 autopsies [18], where the normal extrahepatic arterial structure is defined as *(1)* the proper hepatic artery dividing into the right and left hepatic arteries, *(2)* the right hepatic artery further dividing into the right anterior and posterior hepatic arteries, *(3)* the left hepatic artery dividing into branches supplying segment II and segment III, and *(4)* one or more branches supplying segment IV arising from the right, left, or proper hepatic artery. Hepatic arterial anatomy that differed from the typical anatomy was considered to represent an anatomic variation. Aberrant hepatic arteries can be accessory, occurring in addition to the normal arterial pattern and supplying only partially the left or the right lobe; or replaced, representing the primary arterial supply to the lobe. In addition, since our organ complex casts were suitable for investigation of the complete upper abdominal vascular structure, the right gastric artery (RGA), the GDA and the interrelationship between the main portal vein and the hilar hepatic arteries were also taken into account due to their high surgical and radiological significance.

During the inspection of the corrosion casts the liver could be divided into the left and right lobes by the "functional plane", which on the visceral surface of the liver, corresponds with the line extending from the gall bladder inferiorly to the fossa of the IVC superiorly, according to Cantlie's description. The right lobe was divided into the anterior sector (segments V and VIII) and the posterior sector (segments VI and VII) by a sectoral fissure, which was best seen while looking at the casts sideways. A sectoral fissure was distinguishable within the left lobe as well, dividing it into a medial sector (segment IV) and a lateral sector (segments II and III). The right sectoral fissure, the functional plane and the left sectoral fissure concur with the course of the right, middle and left hepatic veins. After macroscopical examination, digital photos and high-resolution CT-scans were taken of the casts for better digital documentation.

5.2 3D volumetric CT reconstruction of the corrosion casts

CT images of each cadaveric specimen were acquired, anonymized and interpreted in random order by an experienced radiologist. For CT examinations we used a Philips Brilliance 16 unit (parameters: 140 kV, 300mAs, collimation: 16x0.75mm, overlap 50%) with Philips Brilliance 16 Workspace 2007 software Specimens were placed in anatomical position. Images with a pixel spacing 0.08x0.08mm and with 0.4mm axial resolution were obtained, and multiplanar reconstructions were used for image evaluation.

After segmentation of the hepatic arteries, based on different densities and manual cut and fill tools, volume rendering and surface shaded display was used with color encoding at the different main branches for reconstruction (Figure 23).

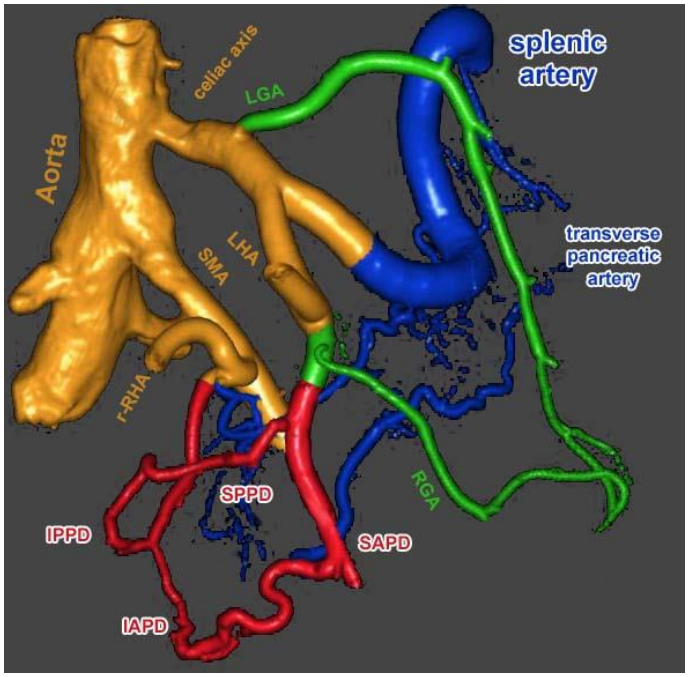


Figure 23: 3D CT reconstruction of organ complex No. 25. Michels III: r-RHA from SMA.

6 Results

6.1 Anatomical variations of the extrahepatic arterial supply of the human liver

6.1.1 Michels types

In our series of 50 corrosion casts, 41 casts (82%) could be classified according to Michels. Twenty-one cases (42%) showed normal arterial pattern (Michels I), while 29 casts (58%) presented different types of extrahepatic arterial variations. However, 9 casts (18%) displayed variations not described in the Michels' classification.

Replaced hepatic arteries

Replaced left hepatic artery (r-LHA) arising from the LGA – Michels II – was observed in 3 (6%), while replaced right hepatic artery (r-RHA) originating from the SMA – Michels III – was present in 7 (14%) cases. Double replaced system (Michels IV – Figure 24) was present in 2 casts (4%). One cast showed a replaced common hepatic artery (r-CHA) arising from the SMA (Michels IX – Figure 25).

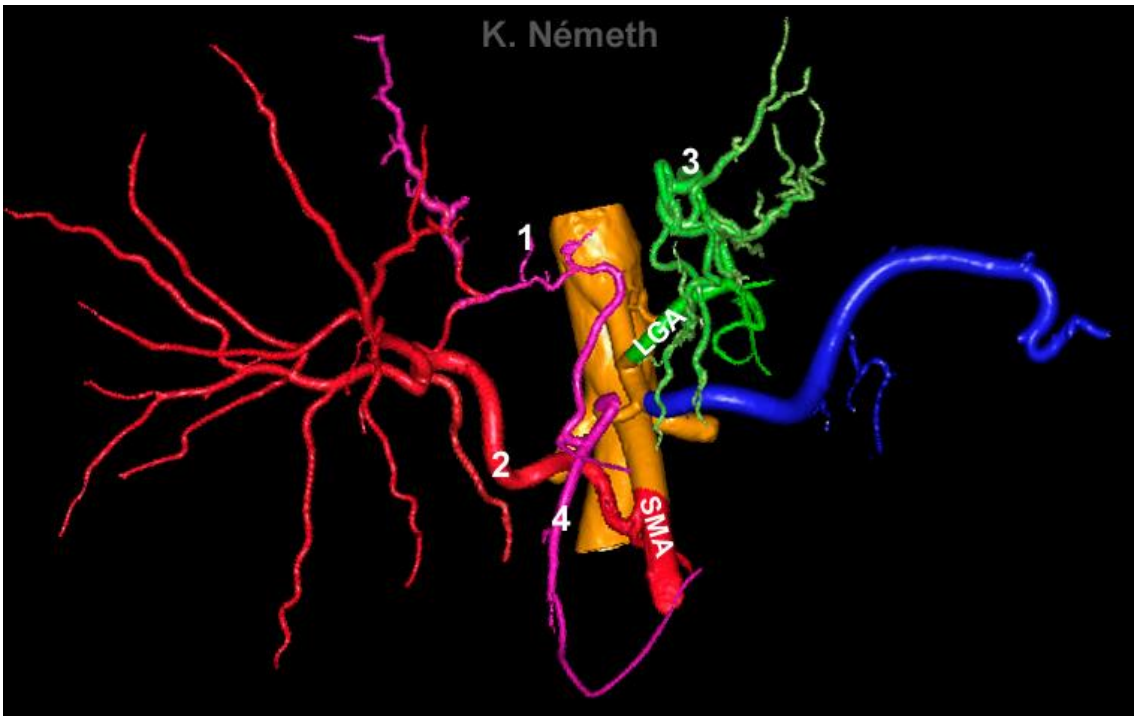


Figure 24: Double replaced system. Green: left lobe; red: right lobe; purple: caudate lobe and GDA; blue: SA; 1: artery of segment I; 2: r-RHA; 3: r-LHA; 4: GDA.

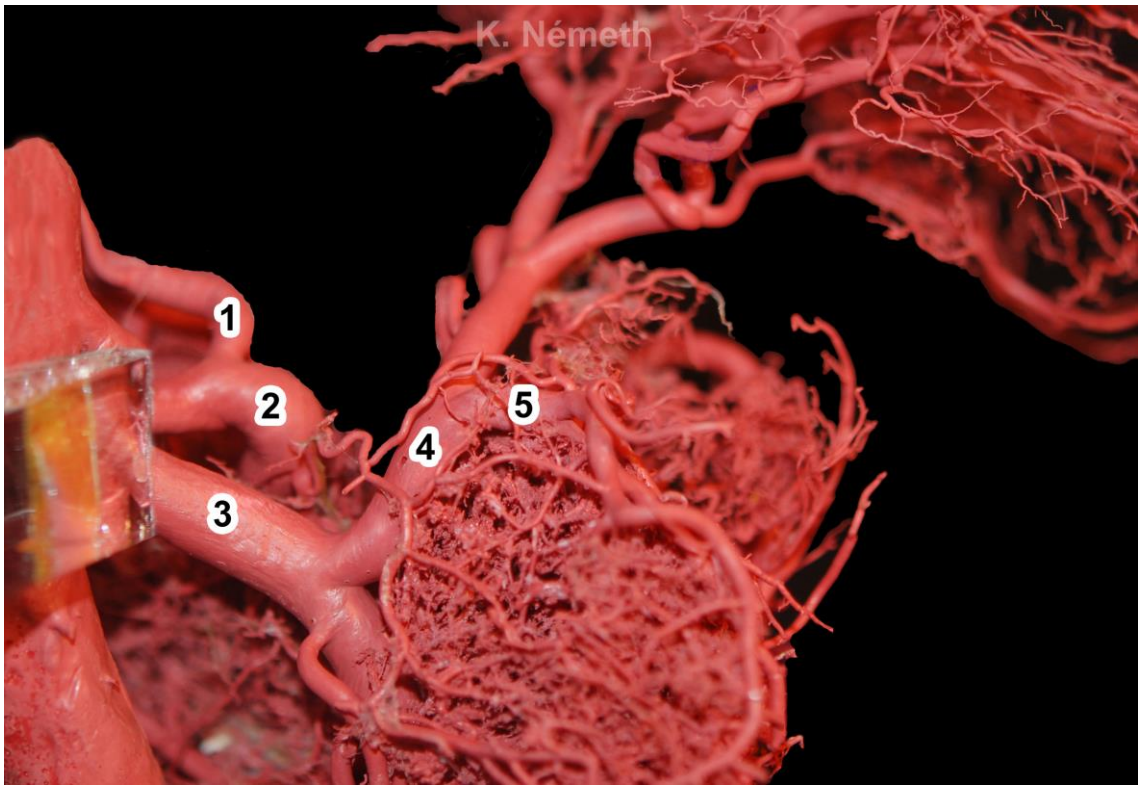


Figure 25: Replaced CHA originating from SMA. 1: LGA; 2: SA; 3: SMA; 4: CHA; 5: GDA.

Accessory hepatic arteries

Accessory left hepatic artery (a-LHA) - Michels V – was present in 4 (8%) cases, accessory left and right hepatic arteries together (a-LHA and a-RHA) – Michels VII – in 1 (2%) case, while combined a-LHA and r-RHA – Michels VIII – were found in 2 (4%) cases.

It is notable that in 4 cases we observed well-defined Michels types showing additional arteries with extrahepatic destination. In the Michels I group one cast presented RGA and a-LGA originating from LHA, another cast showed an a-LGA arising from the artery of segment II (A2) and segment III (A3). In Michels type V, we found one cast having an a-LGA from the a-LHA. The only case of Michels type VII presented two a-LGA arteries branching from LHA, which represents a triple accessory system, a structure which can be considered as a subtype of Michels VII (Figure 38).

No casts presented a single a-RHA from SMA (Michels VI) or r-CHA originating from the left gastric artery (Michels X).

6.1.2 Arterial variants not mentioned in Michels' classification (Unclassified variations – UC)

Out of 50 cases, 9 corrosion casts (18%) showed unusual arterial patterns that could not be classified according to Michels.

The UC variations of our series could be divided into 2 groups. The first group consisted of 5 cases presenting arborization abnormalities of the CHA. Trifurcation of CHA was observed in 4 cases overall, with the CHA giving off the RHA, LHA and the GDA in 3 casts. One cast showed an early, proximal origin of the RHA from the CHA, which results in the CHA trifurcating into LHA, GDA and RGA (Figures 26A-C). In this newly described variation the RHA ran behind the portal vein.

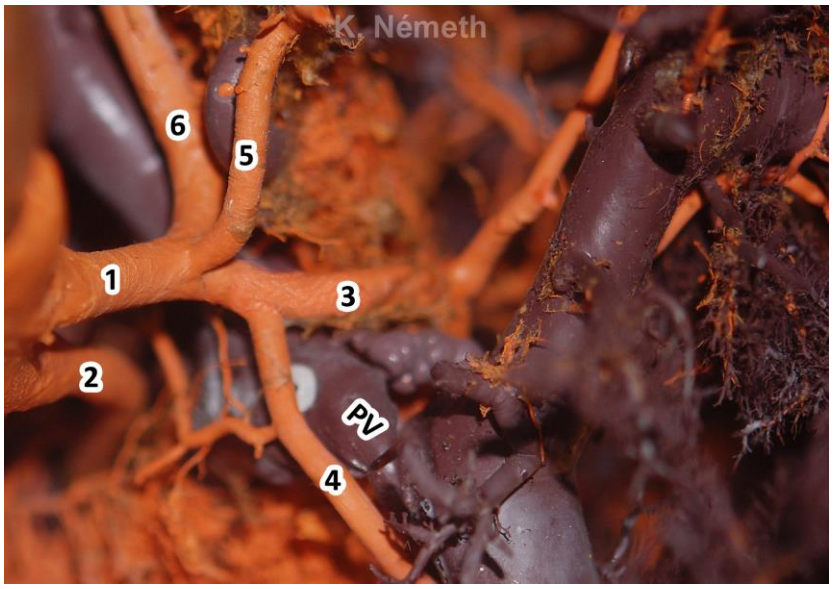


Figure 26A: Proximal branching of RHA with retroportal course. CHA trifurcation into LHA, GDA, RGA. New UC variation. 1: CA; 2: SMA; 3: CHA; 4: RHA; 5: LGA; 6: SA; P: pancreas; PV: portal vein. Right lateral, dorsocranial aspect.

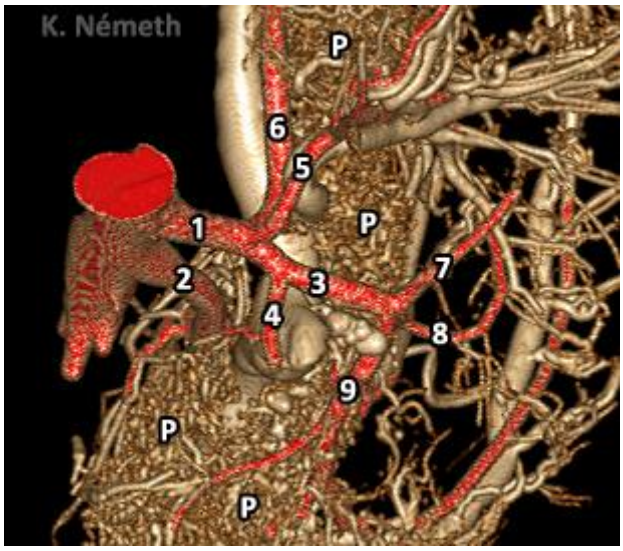


Figure 26B: CT image of proximal, retroportal RHA. Right lateral, dorsocranial aspect.

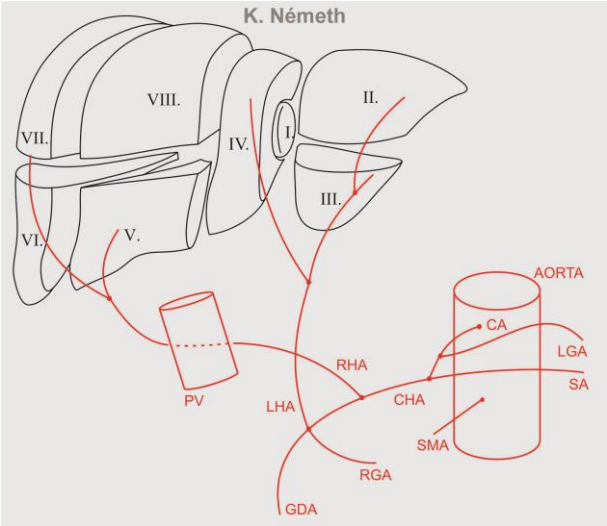


Figure 26C: Schematic drawing of proximal, retroportal RHA.

The fifth case within this group is also a new variant, the CHA branching into five arteries: the LHA, RHA, A4, GDA and RGA (Figures 27A-C). It is also remarkable that the caudate artery (artery of segment I - A1) originated dorsally from the RHA only 4 mm away from the point of pentafurcation.

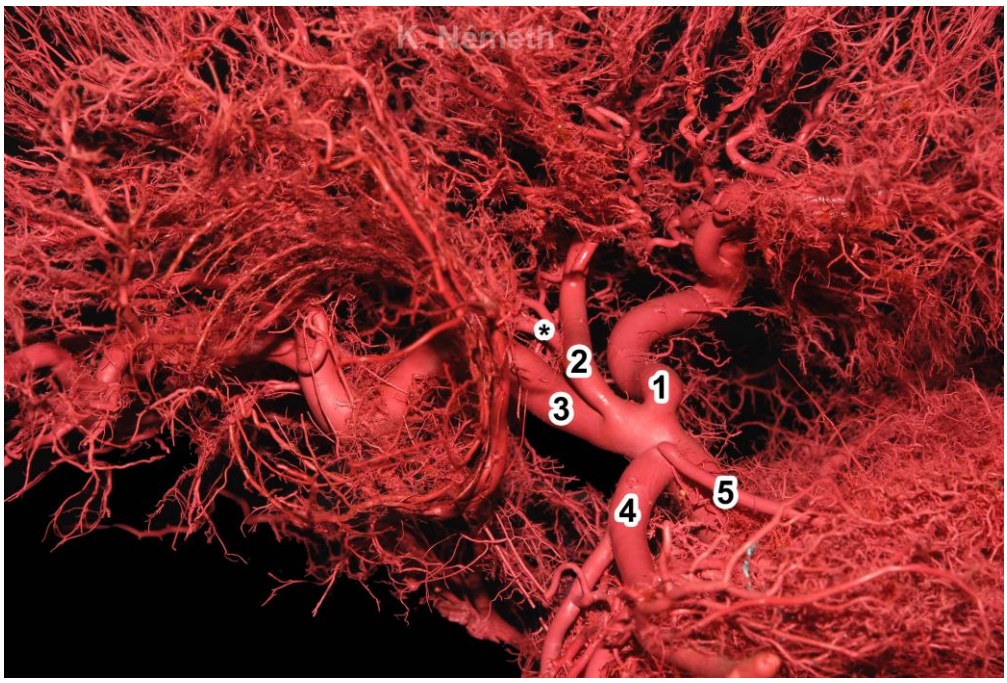


Figure 27A: CHA pentafurcation. New UC variant.
1: LHA; 2: A4; 3: RHA; 4: GDA; 5: RGA; 6: CA; 7: SMA; *: A1. Anterior aspect.

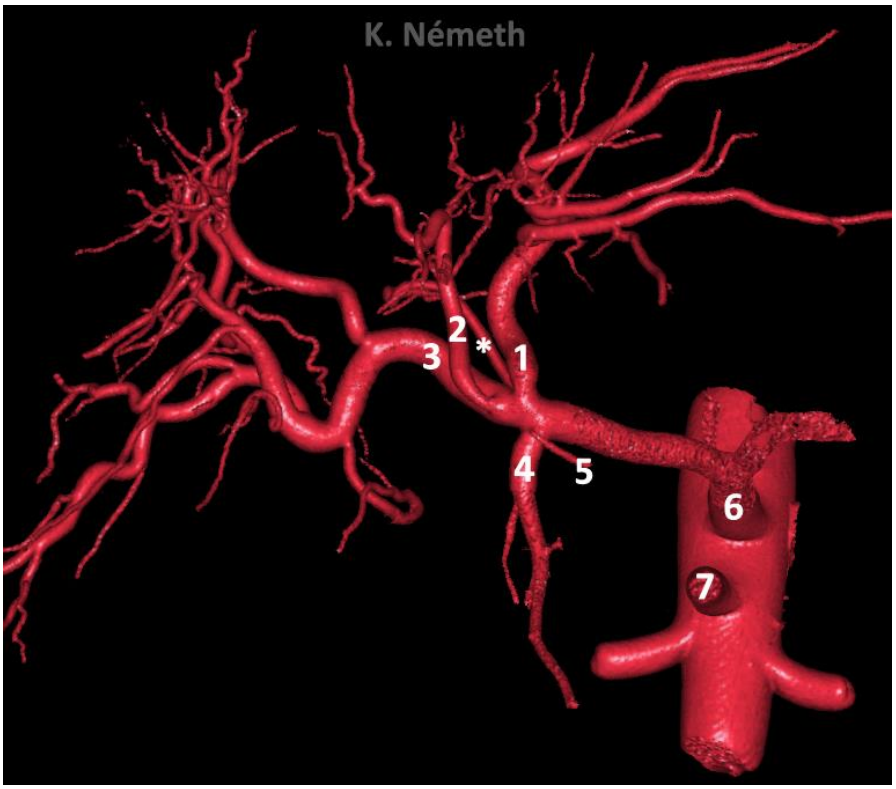


Figure 27B.

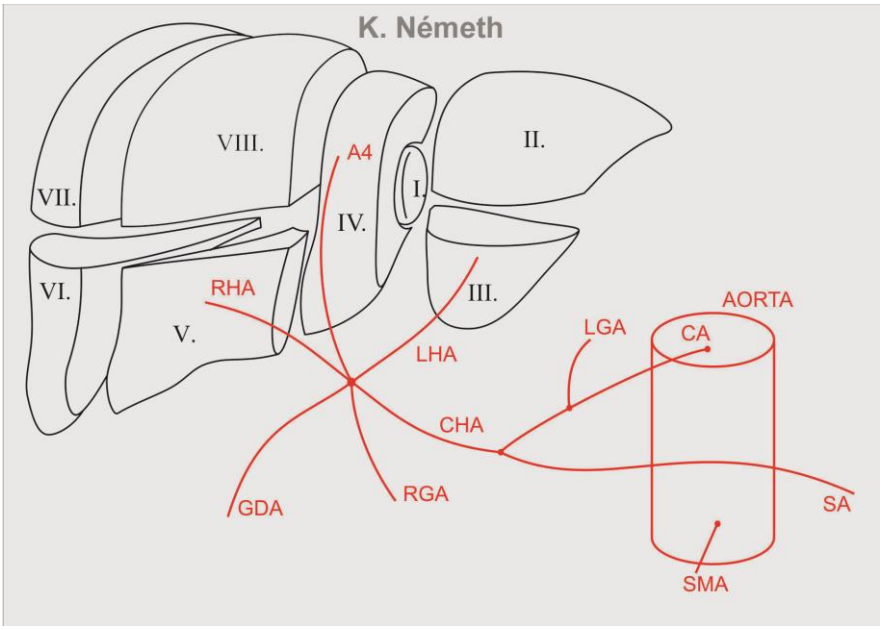


Figure 27C.

The second group of the UC variations is formed by the 4 cases displaying anomalous origins and courses of the lobar and sectorial arteries, the RHA, LHA and RPHA. In one case the RHA arose from the CA and coursed behind the portal vein. In another cast, displaying a new variant, the RHA originated from the proximal part of the CHA and then passed in front of the portal vein. Thus CHA did not bifurcate into PHA and GDA, but into RHA and LHA-GDA trunk (Figures 28A-C).

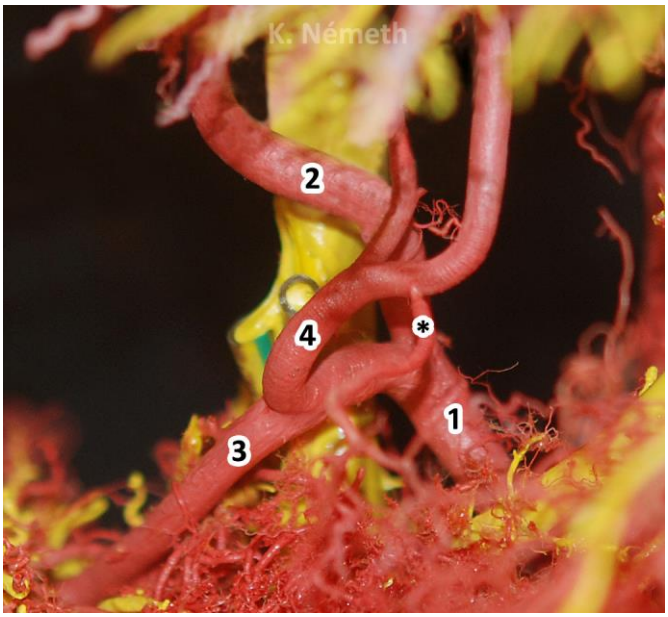


Figure 28A: Proximal branching of RHA, passing in front of the portal vein. New UC variant. 1: CHA; 2: RHA; 3: GDA; 4: LHA; 5: CA; 6: LGA; 7: SA; 8: SMA; *: RGA; PV: portal vein. Anterior aspect.

Figure 28B: Left lateral aspect.

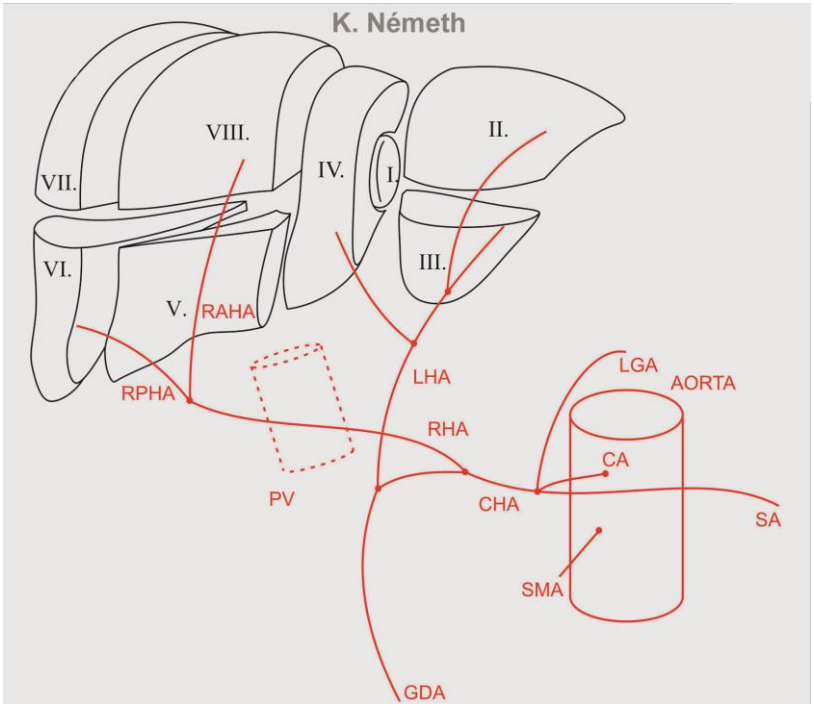


Figure 28C.

In one cast, exhibiting the fourth new variant, the RPHA arose directly from the CHA, in close proximity to the CA-bifurcation. It then passed around the portal vein to reach the right posterior sector of the liver. The CA bifurcated into SA and CHA; and the LGA arose independently from the aorta (Figures 29A-C).

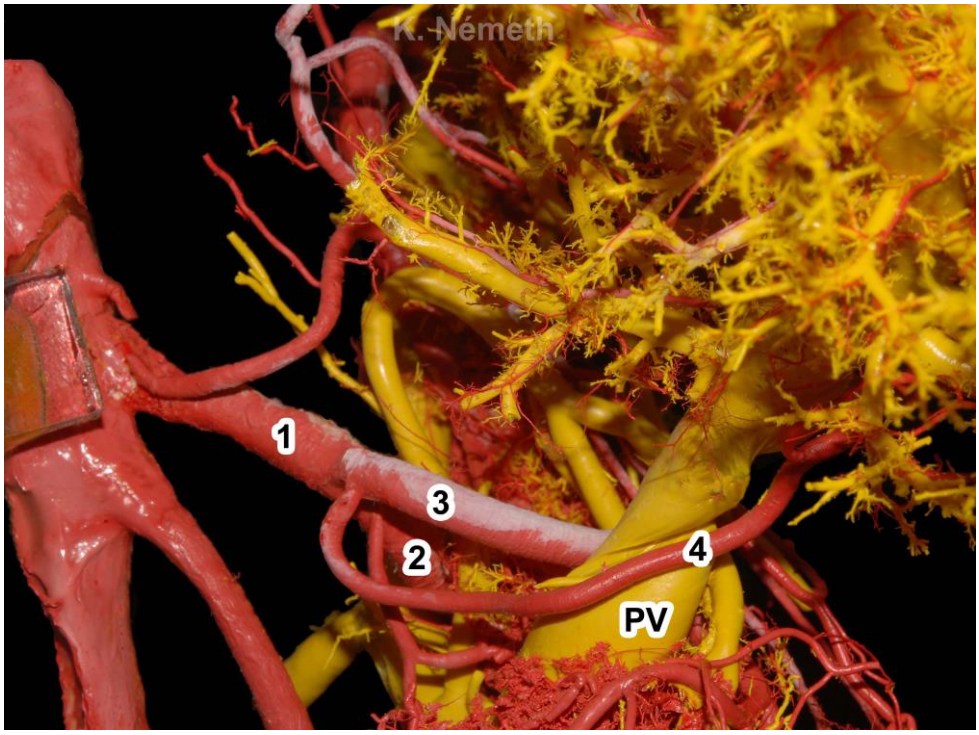


Figure 29A: Proximal branching of RPHA with retroportal course. New UC variant.
1: CA; 2: SA; 3: CHA; 4: RPHA; 5: LHA-RAHA common trunk; 6: GDA; PV: portal vein.
Right lateral aspect.

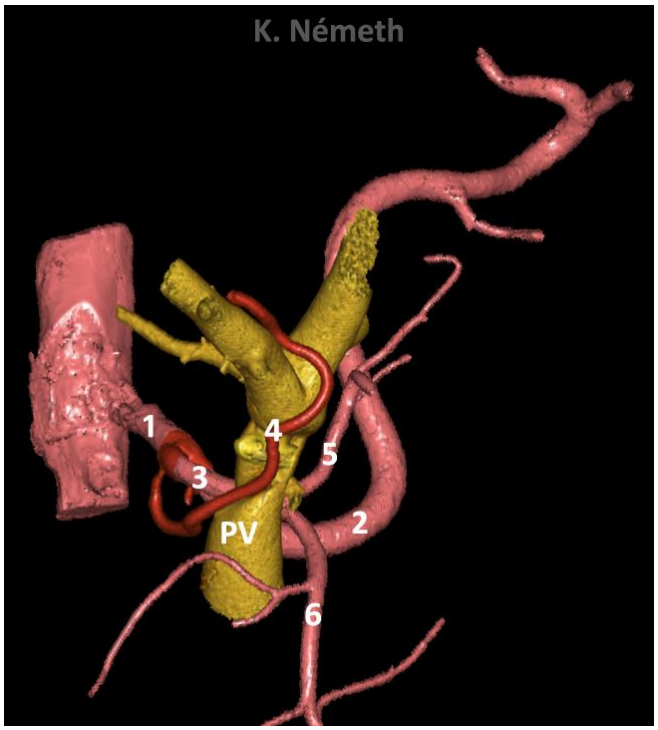


Figure 29B: Right lateral aspect.

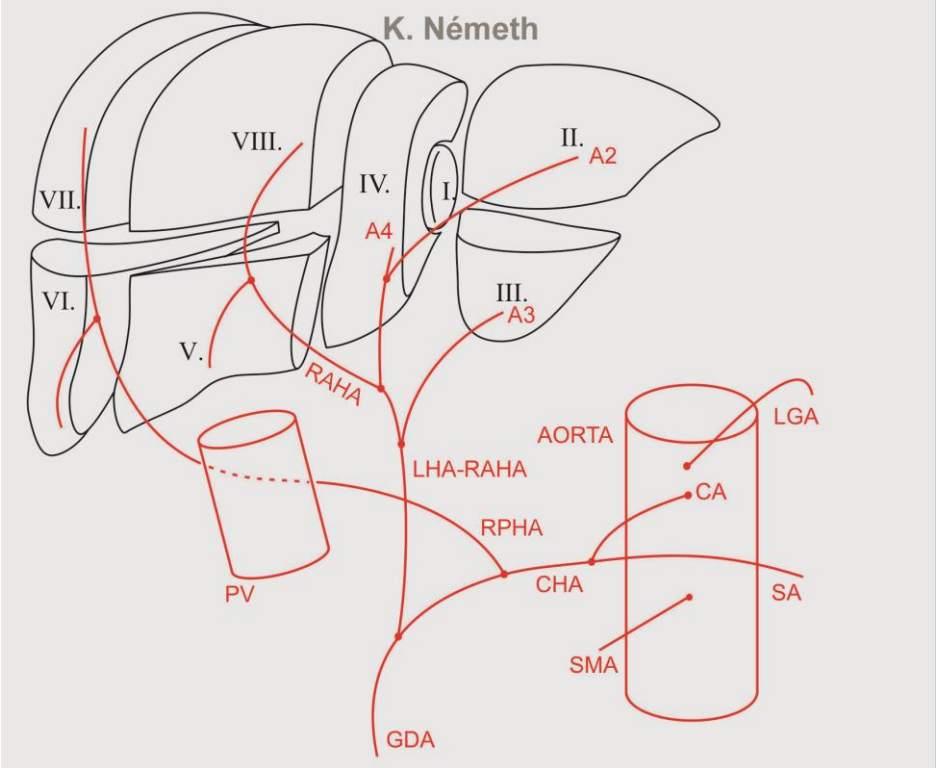


Figure 29C.

The last, already known UC variation of this group is a proximal branching of the LHA with a considerable distance between the origins of the LHA and RHA. In this case RHA took off of GDA (Figure 30).

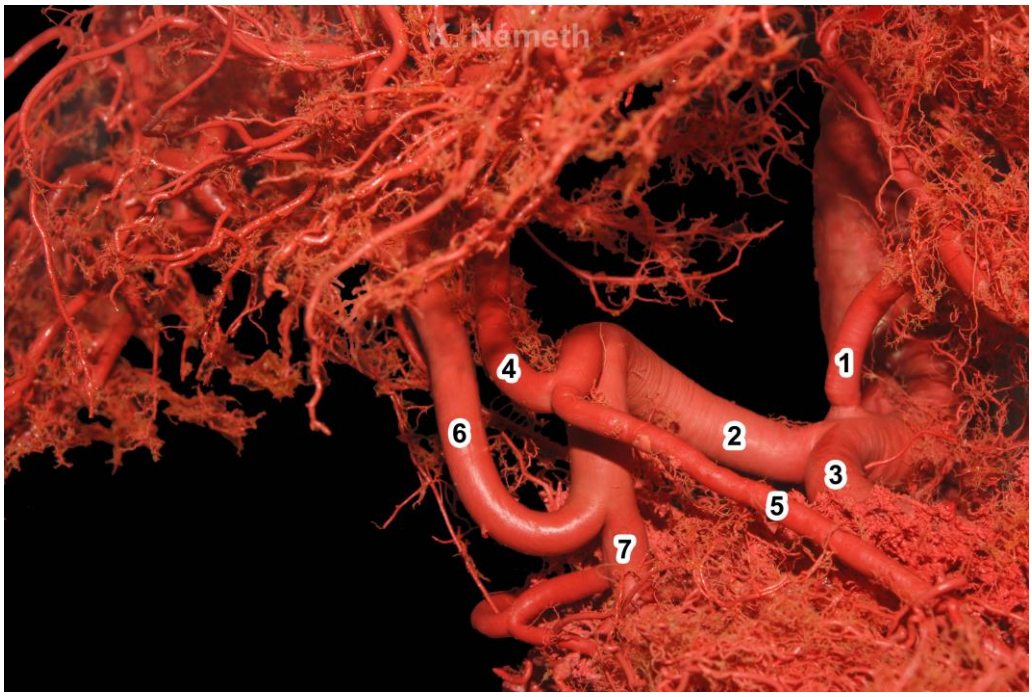


Figure 30: Proximal branching of LHA and RHA originating from GDA.
1: LGA; 2: CHA; 3: SA; 4: LHA; 5: RGA; 6: RHA; 7: GDA. Anterior aspect.

Thus, among these 9 UC variations we encountered 4 cases which, to the best of our knowledge, have not been reported before: CHA pentafurcation; Proximal origin of RHA from CHA with retroportal course, CHA trifurcates into LHA, GDA and RGA; Proximal origin of RPHA from CHA with retroportal course, CHA gives the LHA-right anterior hepatic artery (RAHA) common trunk and GDA (LGA originates separately from aorta); Proximal origin of RHA from CHA with a course in front of the portal vein, CHA later divides into LHA and GDA.

Vessels of approximately 1 mm diameter were visualized during 3D CT evaluation of the specimens and all variations were recognized. Therefore, radiological and anatomical results were identical.

6.2 Branching and coursing patterns of the lobar arteries

We observed numerous abnormalities within almost every Michels type (I, II, III, V, VII, IX) based on the branching and coursing variations of the segmental/subsegmental branches of the RHA and LHA. In Michels types IV and VIII the arborization of the RHA always showed the conventional pattern.

6.2.1 Right hepatic artery

- ***RPHA absence***. Proximal deriving of segmental arteries (A6 or A7) were found in 7 cases. Normal branching of the RHA into RAHA and RPHA was not observable due to an early branching of a right-sided segmental artery. Whenever a segmental artery displays a proximal origin, the corresponding sectoral artery (in these cases the RPHA) does not actually exist. This fact is underlined when the complementary sectoral artery originates between the two separate segmental arteries, e.g. proximal origin of the A7 and RAHA origin between the separated A7 and A6. In this particular case the segmental branches of the RPHA are independent of one another, definite RPHA is non-existent (Figures 31, 32).

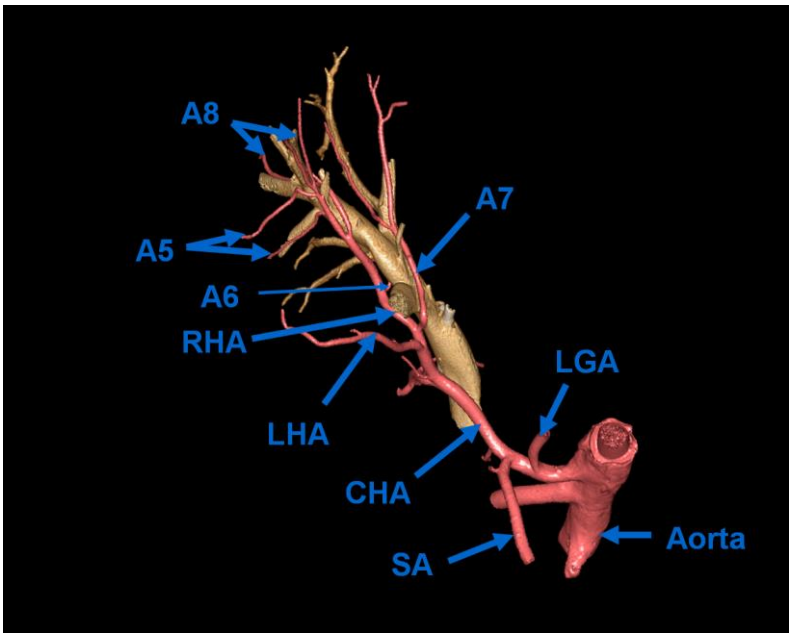


Figure 31: RPHA absence: Proximal origin and retroportal course of A7. (Yellow: portal vein).

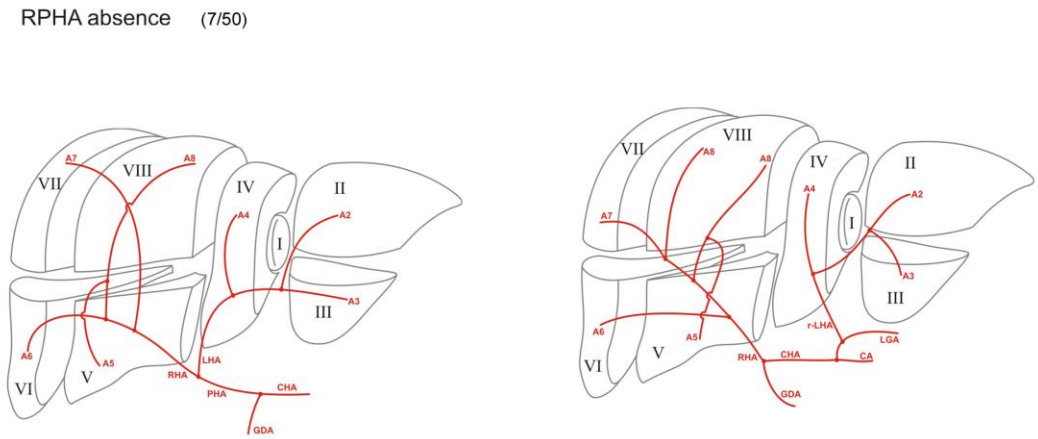


Figure 32: RPHA absence. Proximally originating segmental artery (A7 or A6) from RHA. RAHA originates between A7 and A6.

- ***Accessory segmental arteries*** (Figures 33, 34) deriving from RHA or LHA, proximally to the normal RAHA-RPHA division (i.e. double or triple segmental supply) were seen in 8 cases (6 cases in the Michels group + 2 cases in the UC group). In 1 remarkable case the RHA gave 8 separate branches: RAHA, RPHA, 2xA6, 2xCyA, A4, A1 – the S VI was supplied by 3 arteries (two a-A6 from RHA + A6 from RPHA).

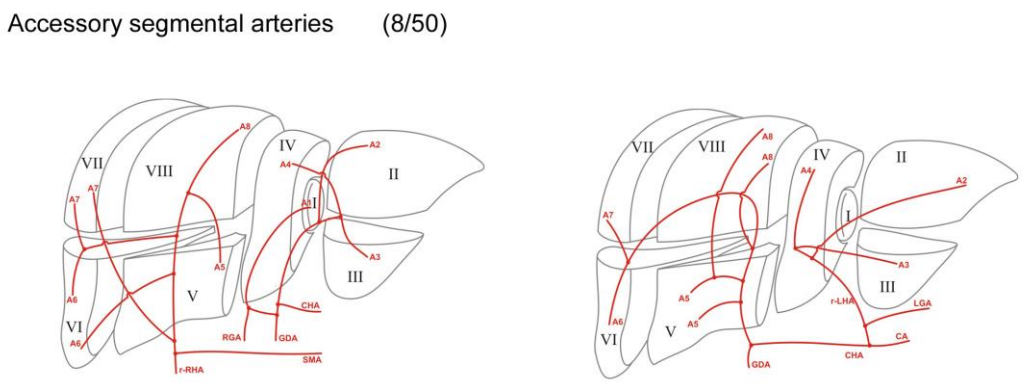


Figure 33: Accessory segmental arteries (a-A6 + a-A7; a-A5 + a-A8) arising from RHA. The related segments have double supply.
(On the left picture additionally: RGA-A1 trunk arises from GDA.)

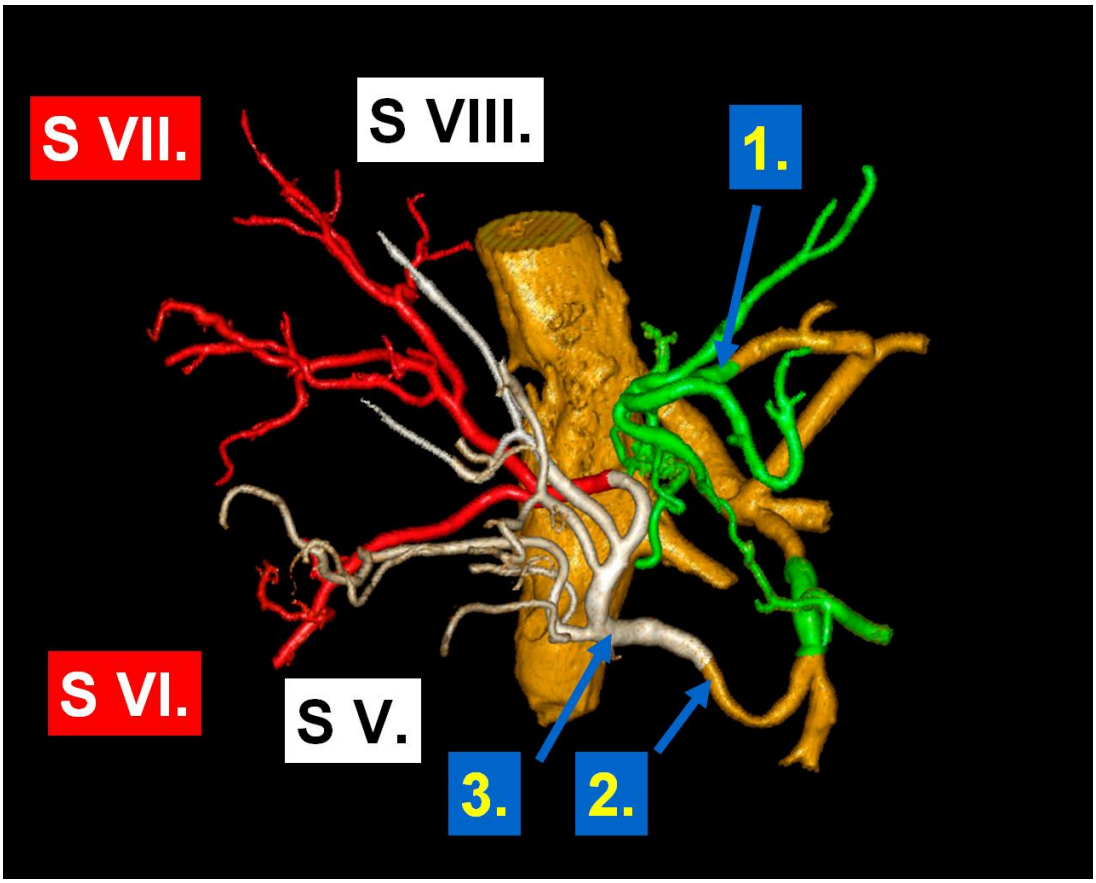


Figure 34: CT image of a-A5 + a-A8 accessory segmental arteries.
The right anterior sector has double supply from RHA.
(1: r-LHA. 2: RHA. 3: Three branches of RHA: a-A5, RAHA, a-A8)

- ***RAHA absence:*** A4A8 trunk from RHA (2 cases, Figure 35).

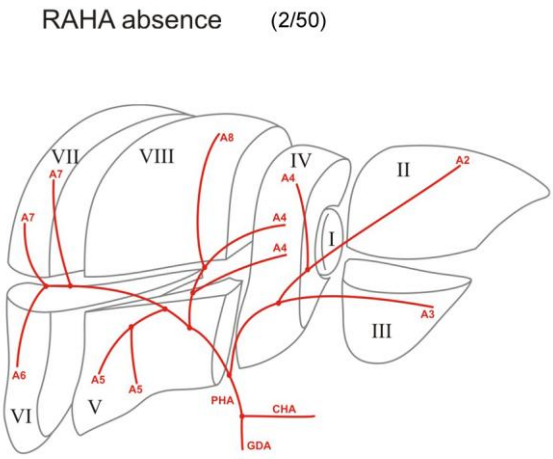


Figure 35: Schematic structure of RAHA absence.
A4A8 trunk originates from RHA, A5 takes off distally. RPHA is intact.

- ***Neither RAHA, nor RPHA are present.*** (3 cases – Figures 36, 37)
  - RHA branches: A5A6 + A7A8.
  - RHA branches: A5A6 + A5A8 + A6A7A8.
  - RHA branches: A4A5, A6A7A8, A6.

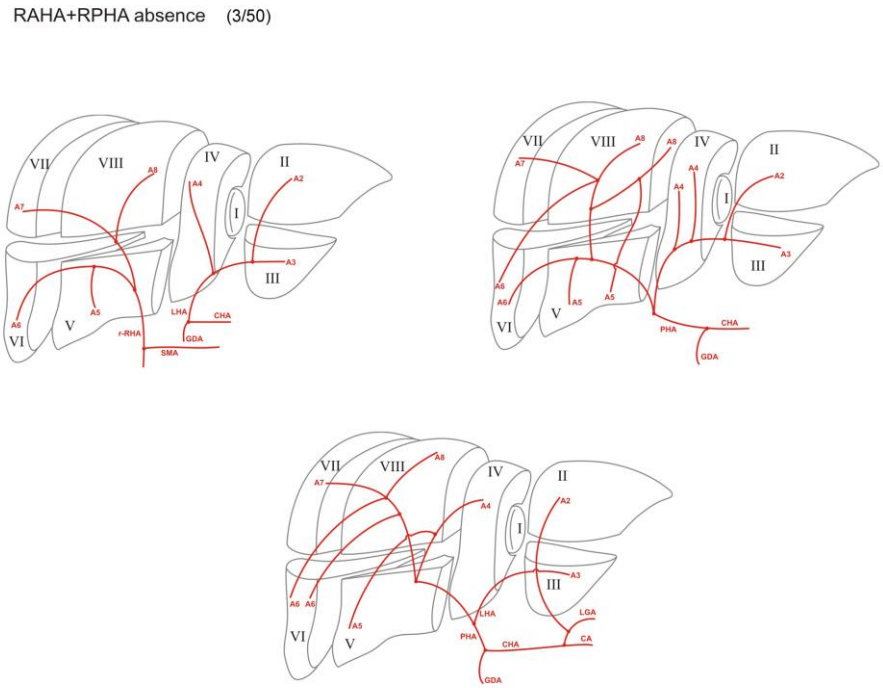


Figure 36: RAHA+RPHA absence. RHA branching is completely abnormal.

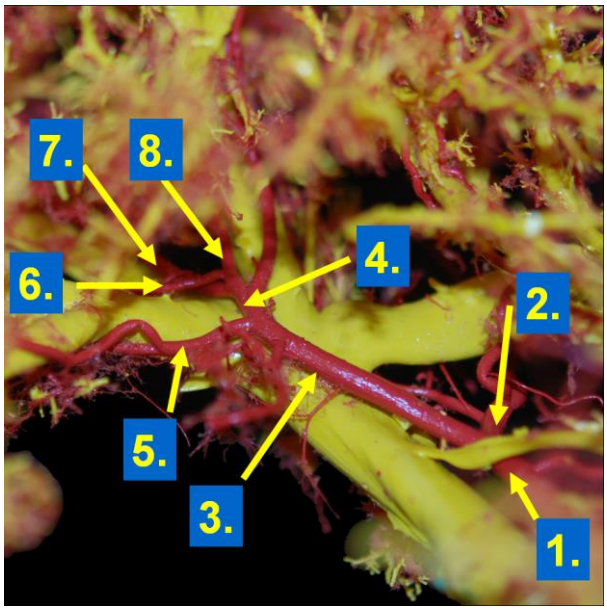


Figure 37: Liver No. 18. RAHA and RPHA are absent. RHA branches: A5A6, A5A8, A6A7A8. (1: CHA; 2: LHA; 3: RHA; 4: A6A7A8 trunk; 5: A5A6 trunk; 6: A6; 7: A7; 8: A5A8)

- ***Double RAHA.*** (2 cases – including 1 case of RAHA trifurcation into A5 and two A5A8 trunks)
- ***RHA trifurcation.*** (2 cases, including 1 case of double RPHA)

6.2.2 Left hepatic artery

- Accessory LGA (a-LGA) originating from A2 and A3 (1 case)
- Accessory LGA (a-LGA) and RGA take off of LHA (1 case)
- Accessory LGA (a-LGA) arises from accessory LHA (a-LHA). (1 case)
- Triple accessory system: a-LHA + a-RHA + a-LGA (Figure 38). (1 case)

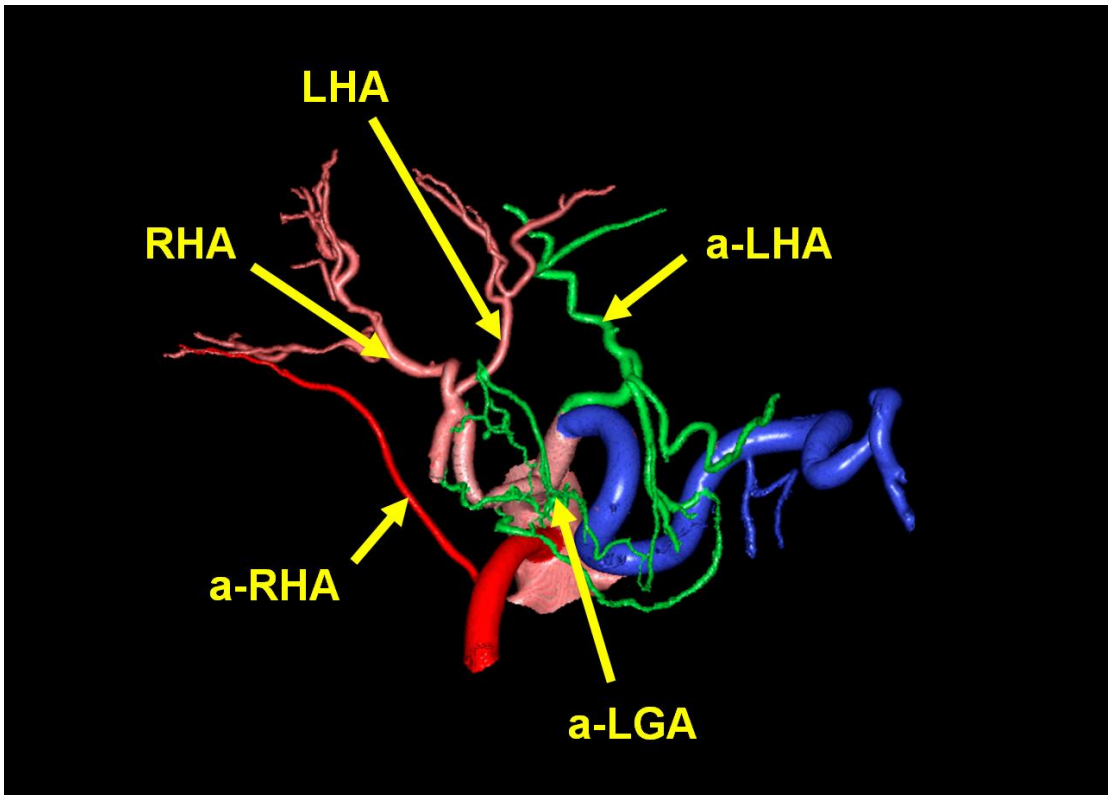


Figure 38: Triple accessory system.

In 5 cases we found a combination of extrahepatic and intrahepatic variations as follows:

- Proximal branching of LHA + a-A8 from RHA (Figure 39)
- CHA trifurcation + a-A5 from RHA
- CHA trifurcation + retroportal RHA
- CHA trifurcation + RGA-A1-CyA trunk from LHA (Figure 40)
- CHA pentafurcation + double RAHA

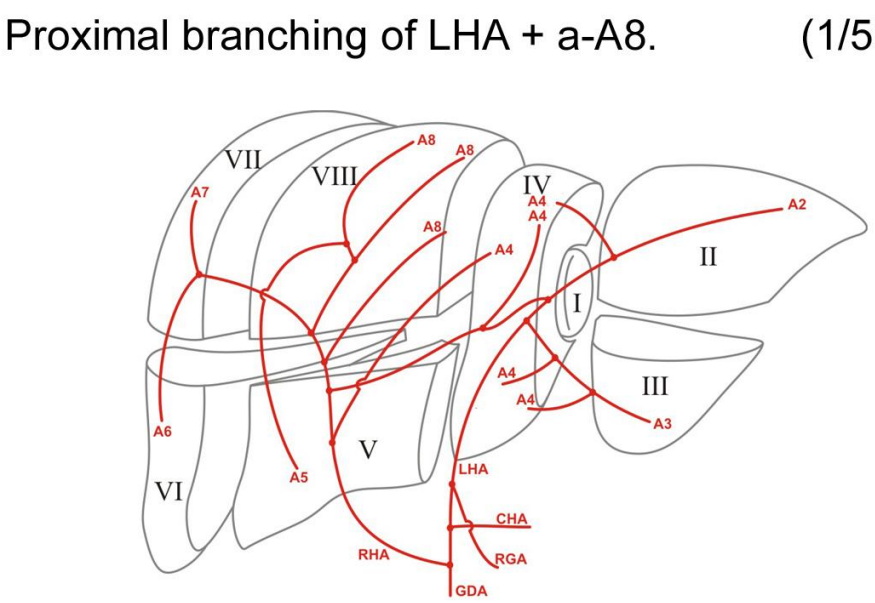


Figure 39: Liver No. 45. One case of a combined extra- and intrahepatic variation. S IV is fed by 5 arteries.

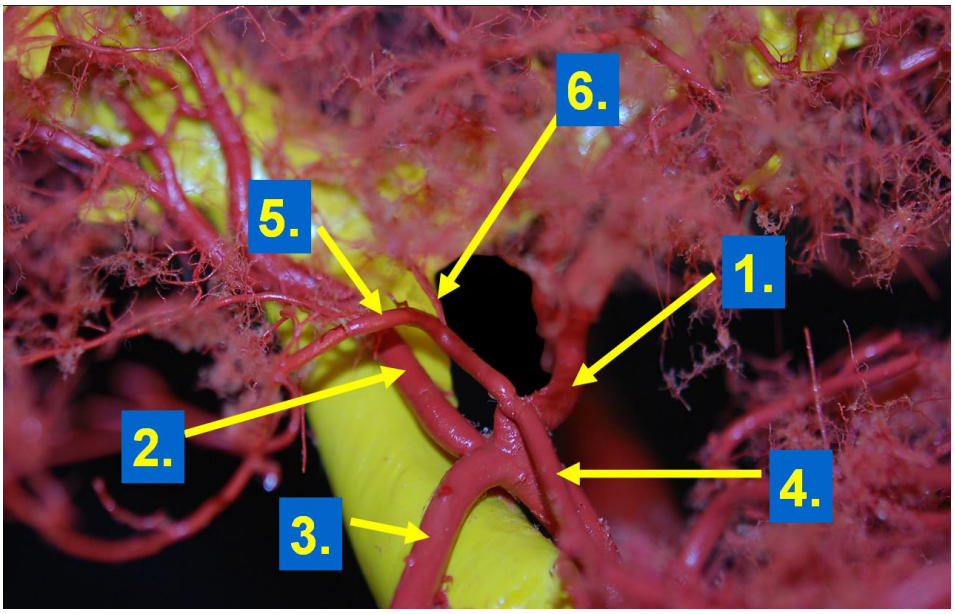


Figure 40: Liver No. 49. RGA-A1-CyA trunk from LHA combined with CHA trifurcation. 1: LHA; 2: RHA; 3: GDA; 4: RGA; 5: CyA; 6: A1. Yellow: portal vein.

6.3 Segmental arterial supply of the human liver

6.3.1 Caudate lobe (Segment I. – S I.)

Table 4: Arterial supply of the caudate lobe in different types of extrahepatic vascular anatomy.

Liver No.	Michels	S I.
1	I.	(RHA @ RPHA @ IPPD) + A2
2	I.	(RHA @ LHA) + A4
3	I.	(RAHA @ A4) + A8
4	I.	(A2A3 @ A4)
5	I.	(RAHA @ RPHA @ RHA @ A6 @ A4 @ LHA) + A2
6	I.	(RAHA @ GDA @ A4) + A2 + A8
7	I.	(RAHA @ A4) + A4
8	I.	(RPHA @ A4)
9	I.	(RPHA @ 2xRAHA @ 2xLHA)
10	I.	(RPHA @ 3xA1A8 @ LHA @ A4 @ A2A3)
11	I.	(RHA @ GDA @ 2xA8 @ A4 @ A4 @ [A4 @ A4])
12	I.	(RPHA @ 2xA8 @ A4 @ LHA)
13	I.	(RHA @ LHA)
14	I.	RAHA + PHA
15	I.	(A8 @ A2A4)
16	I.	(RPHA @ RHA @ LHA)
17	I.	(A7 @ LHA)
18	I.	(A7 @ LHA @ A4)
19	I.	(RAHA @ RPHA @ LHA @ A2A4)
20	I.	(RAHA @ A4 @ A2) + A2
21	I.	(A7 @ A2A3)
22	II.	(2xRPHA @ 2xRAHA @ RHA @ CyA @ SAPD @ A3A4)
23	II.	(RHA @ A8 @ A4)
24	II.	(RAHA @ RPHA @ RGA @ LHA)
25	III.	(RHA @ RAHA @ RPHA @ 2xSAPD @ RGA @ LHA @ A2)
26	III.	RAHA + A3
27	III.	(2xRHA @ GDA @ LHA)
28	III.	(RHA @ GDA @ CyA @ A2A4)
29	III.	(2xA4 @ 2xA2)
30	III.	(RHA @ A2 @ A4)
31	III.	(RPHA @ RGA @ A8 @ GDA @ 2xLHA)
32	IV.	(RAHA @ 2xA8 @ GDA @ A4)
33	IV.	(RAHA @ GDA)
34	V.	(RHA @ GDA @ A4)
35	V.	(RHA @ A8 @ A4)
36	V.	(RPHA @ GDA @ 2xA4)
37	V.	(A4 @ 2xA3)
38	VII.	(2xRHA @ A7 @ 2xLHA)
39	VIII.	(RAHA @ GDA @ A4)
40	VIII.	(RPHA @ GDA @ 2xA2)
41	IX.	(A5A8 @ A6 @ A8 @ CyA @ 2xLHA @ A4 @ A2)
42	NC	(dist. RAHA @ 2x prox.RAHA @ RHA @ A2)
43	NC	(RHA @ GDA @ A4)
44	NC	(RAHA @ LHA)
45	NC	(a-A8 @ A4) + 2x a-A8
46	NC	(RPHA @ A2A3 @ CyA @ 2xA4)
47	NC	(RPHA @ LHA @ A2)
48	NC	(RPHA @ CyA)
49	NC	(RAHA @ LHA @ A7 @ A4 @ A2)
50	NC	(RAHA @ RPHA) + A3

In the present study we described 49 different types of arterial supply of the caudate lobe (49/50) (Table 4). The (RHA @ GDA @ A4) pattern was observed on 2 casts. It is notable that the (RHA @ A8 @ A4) pattern was described on 2 casts as well,

however, the A4 originated from the LHA on one cast and from the RHA on the other one – consequently the arterial structure is different.

In 45 out of 50 cases (90%) communicating arcades with multiple anastomoses were observed between different arteries of the left and the right lobe (Figures 41, 42), often involving extrahepatic vessels, such as GDA in 12 cases, CyA in 5 cases, RGA in 3 cases, SAPD in 2 cases and IPPD artery in 1 case. These arcades also served as origin for the always fork-like stemming end-arteries of the caudate lobe.

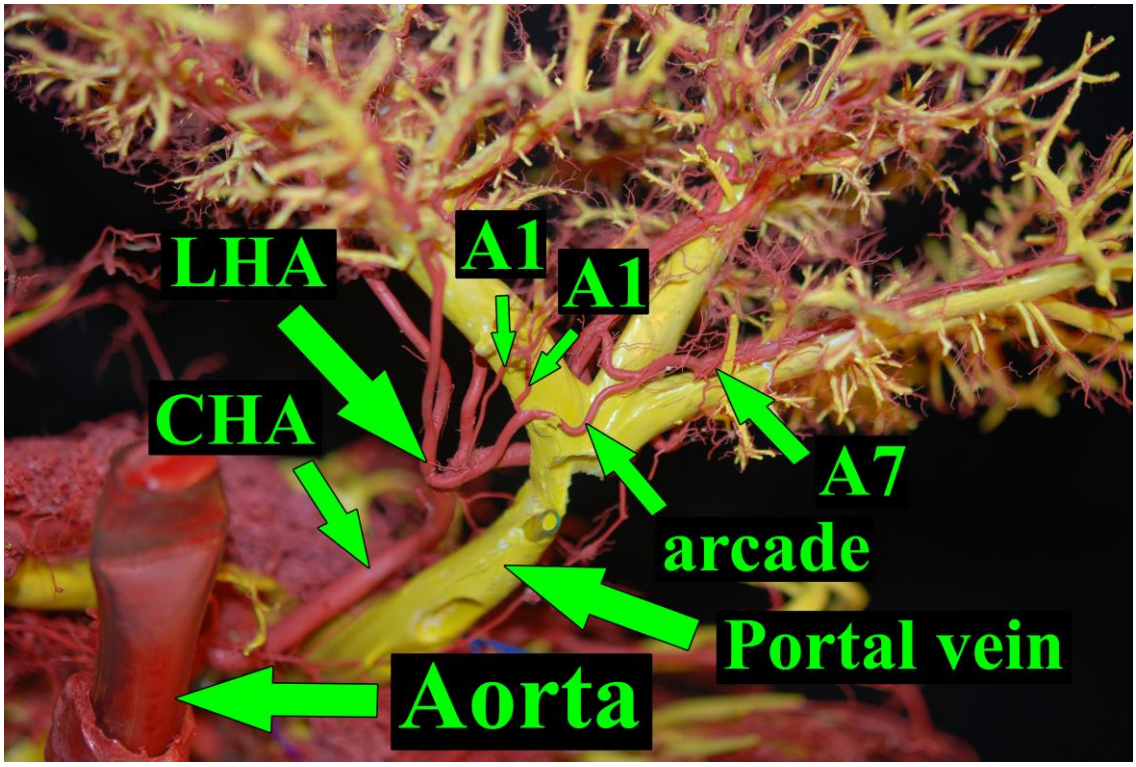


Figure 41: Liver No. 17.
Caudate lobe arteries (A1) originate from the vascular arcade between LHA and A7.

Out of the remaining 5 cases 2 casts showed S I arteries coming from both lobes without forming anastomosis [(RHA @ A6A8 @ IPPD) + A2 and RAHA + A3]; 1 cast showed S I arteries originating from the left lobe only (A2A3 @ A4); 1 cast showed S I arteries stemming from the coeliac axis and the right lobe only (RAHA + PHA); and S I. arteries from the right lobe only were present on 1 cast (RPHA @ CyA).

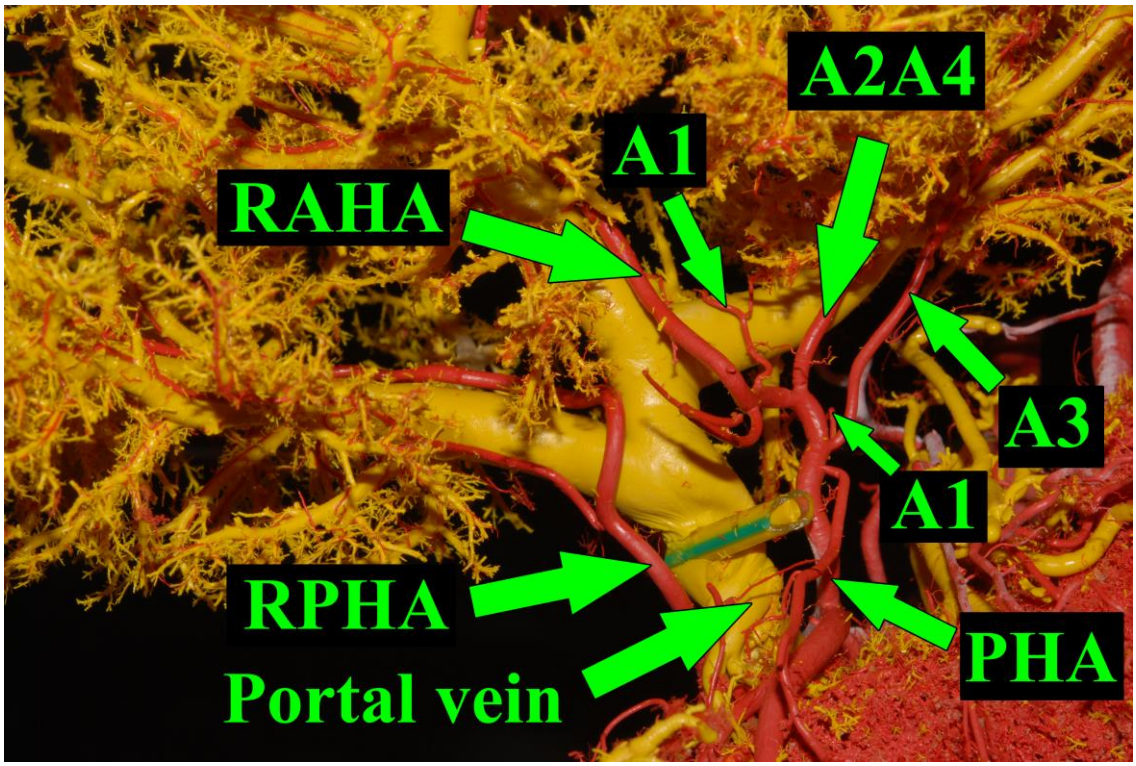


Figure 42: Liver No. 50. One of the feeding arteries of the caudate lobe derives from A3. (The other A1 arising from the anastomosis between RAHA and RPHA is not shown.)

6.3.2 Segment II. (S II.)

In our series we observed 14 different patterns of arterial supply.

The A2 deriving from the A2A3 common trunk was the most frequent variation with 28% (14/50), two segmental arteries from the A2A3 were observed in 1 additional case. In 16% (8/50) a single A2 was the first branch of the LHA, in 2 more cases the LHA gave rise to two S II arteries. The S II received its arterial inflow from the LGA in 14% (7/50). The A2A4 served as origin for the A2 in 10% (5/50), the A2A4a with 6% (3/50) with the A3 being the first branch of the LHA.

Two segmental arteries to S II with two different origins were present in 16% (8/50).

We also observed rare variations of the S II supply. The A2 was the first branch of the RHA in 1 case and a common trunk of the A2A3A4aA4b was the root of the A2 in 1 case.

6.3.3 Segment III. (S III.)

We revealed 14 different arterial patterns of the blood supply of S III.

A single segmental artery was present in 70% of cases (35/50): the A2A3 trunk was the origin in 26% (13/50), the LHA in 24% (12/50) and the A3A4 in 18% (9/50) and in 1 case the LGA was the root of the S III segmental artery. Two segmental arteries were observed in 30% (15/50), where the other segmental arteries of the left lobe (A2, A4, A4a, anastomosis between A3 and A4) gave additional branches to the S III There were no double S III segmental arteries originating from the same root. In 1 case we found 3 arteries feeding S III.

Figure 43 summarizes the branching patterns of the LHA in our series.

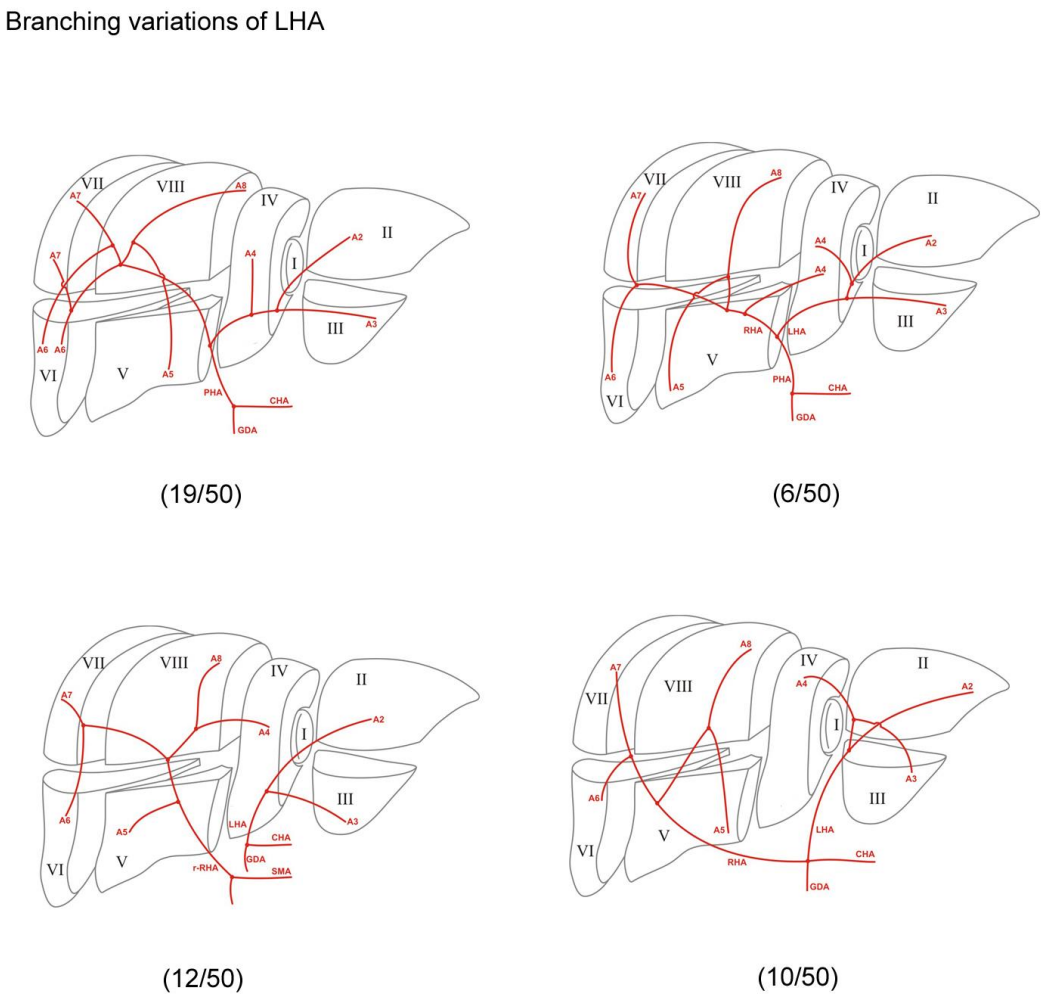


Figure 43: Arteries of segments II and III.
Upper left: The first branch of LHA is A4, segments II and III are fed by A2A3. (Double RPHA.)
Upper right: First LHA branch is A3, therefore A2A4 trunk supplies the remaining left lobe segments.

Bottom left: A4 originates from RHA, LHA consists of A2 and A3 only. (RAHA absence – A4A8 trunk from RHA)
Bottom right: The first branch of the LHA is A2. (CHA trifurcation.)

6.3.4 Segment IV. (S IV.)

We revealed 27 types of arterial supply of S IV, as shown on Table 5.

Table 5: Arterial supply of segment IV in our series.

Origins of A4 in our series							
Left-sided	No. of cases	***Right-sided***	No. of cases	***Both sides***	No. of cases	***Miscellaneous***	No. of cases
LHA	12	RHA	3	RHA + A2A4	2	Pentafurcation	1
A2A4	2	RAHA	1	RHA + A3A4	1		
A3A4	5	2xRHA	1	RAHA + A2A4	1		
2xLHA	2			RAHA + A3A4	1		
2xA3A4	1			RHA + 2xA3A4	1		
LHA + A2	1			RHA + A2 + A3	2		
LHA + A3	3			dist. RAHA + A2	1		
2xLHA + A2	1			RHA + 2xA3 + A2	1		
2xLHA + A3	1			RHA + 2xA2 + 2xA3	1		
LHA + A2A3	1			RHA + A2A4 + (A4 @ GDA)	1		
2xLHA + a-LHA	1			RHA + 2xLHA + A3 + (A4 @ GDA)	1		
A3A4 + A2 + A3	1						
Summary	**31**		**5**		**13**		**1**

In 24% (12/50) the A4 was the first branch of the LHA, representing the most frequent variation. In 10% of cases (5/50) the A4 was the second branch from the LHA, deriving from its A3A4 trunk. Segment IV received its arterial inflow from the left side in 62% (31/50), from right-sided origins in 10% (5/50), from both sides in 26% (13/50). In one case A4 took off of CHA as part of the pentafurcation.

One supplying artery irrigated S IV in 48% of cases (24/50), we found two arteries in 30% (15/50), three feeding arteries in 16% (8/50), four arteries in 1 case and five arteries in 2 cases.

We observed extrahepatic vessels contributing to the blood supply of S IV in 3 cases. Anastomoses between A4 and GDA were found on 2 casts and a-LHA gave S IV branches on one cast.

6.3.5 Segment V. (S V.)

Compared to the left lobe, the right lobe segments have more than one segmental artery in most of the cases.

The arterial structure of the S V exhibited 20 different patterns, nonetheless, S V can have various numbers of segmental arteries from the same origin (e.g. 3 A5 arteries from RAHA).

The S V segmental artery (or arteries) representing only RAHA origin were present in 58% (29/50), branches from A8 joined the RAHA in 10% (5/50), RAHA and RPHA fed S V simultaneously in 8% (4/50), proximal segmental artery from RHA accompanied by branches from RAHA in 6% (3/50) or A5A8 in 4% (2/50), proximally arising S V artery from RHA in 4% (2/50). Arteries from RAHA and A1, double RAHA, A5A6 common trunk, A5A6 trunk combined with A5A8 trunk, and A4A5 common trunk were observed in 1 case each.

6.3.6 Segment VI. (S VI.)

The arterial structure of S VI showed 18 forms in our series, with a single origin from RPHA being the most frequent variant (28/50 = 56%), regardless of how many segmental arteries derived from it. Proximal S VI segmental artery arising directly from the RHA was observed in 14% of cases (7/50), A7 branches provided supply of S VI beside RPHA in 6% (3/50), both RPHA and RAHA fed S VI in 6% (3/50) and A1 branches joined RPHA in 4% (2/50). Accessory A6 arteries from RHA were observed in 6% (3/50). A6A7A8 common trunks were found in 4% (2/50), one of them presented A5A6 trunk as well. We found 1 case of each following variation: RPHA + SMA (as part of the triple accessory system); A5A6 common trunk being the only supply of S VI.

Whenever RPHA was present, it always gave high caliber segmental artery (or arteries) to S VI.

6.3.7 Segment VII. (S VII.)

We observed 8 forms of arterial structures of S VII.

One or two high caliber segmental arteries arose from the RPHA in 70% of cases (35/50) (Figure 44), proximally originating A7 from RHA was found in 12% (6/50), A1 branches joined RPHA in feeding S VII in 6% (3/50), A6A7A8 and A7A8 trunks provided supply in 4-4% (2 cases each), RAHA and RPHA simultaneously fed S VII in 1 case, RPHA and an a-A7 branch from RHA in 1 case.

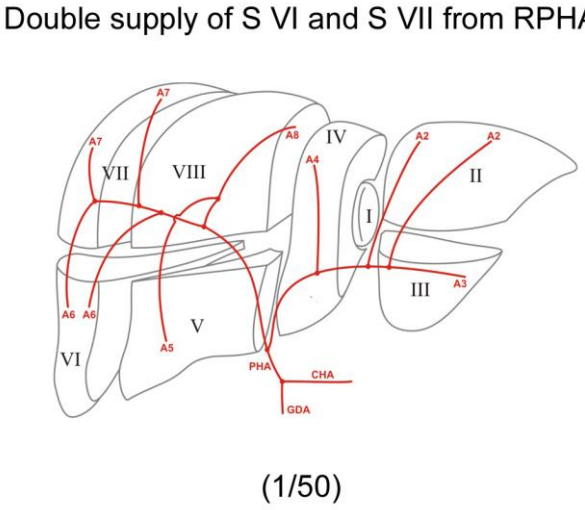


Figure 44: Double supply of S VI and VII. Both segmental arteries arise from RPHA.

6.3.8 Segment VIII. (S VIII.)

Out of 50 corrosion casts in this series we described 17 different arterial structures feeding S VIII.

In 62% (31/50) the RAHA was the single source of the S VIII supplying arteries and it took part in supplying the S VIII in 11 more cases: joined by branches from other segmental arteries (A1, A5, A1A8, A7A8 – 6 cases), the RHA (2 cases), RPHA (1 case), the CyA (1 case) and the RPHA together with CyA (1 case). Double RAHA supplied S VIII in 1 case. In 7 cases, where RAHA was not present, common trunks of different segmental arteries irrigated S VIII, combined with one another or with different segmental arteries. Such cases include: A4A8; A7A8; A6A7A8; A4A8 + A5; A5A8 + 2xA8; A5A8 + A6A7A8; A5A8 + A8.

7 Discussion

7.1 Corrosion casts and other modalities in the investigation of hepatic arteries – an overview

Couinaud in his classical work [34] analyzed arterial vascular casts which were prepared by injection of the arteries at the level of the hepatic pedicle without specifying the source of these arteries except for the left gastric artery that had been checked. Therefore we designed a study to reveal the hepatic arterial vascular system originating from normal and variant sites. For this purpose we investigated the vascular structure of abdominal organ complexes instead of liver casts that provide only limited information about the blood supply from extrahepatic arterial source. Moreover, our casts provide 3D data on the whole upper abdominal vascular system making these data equally important for all interventions in this region. Furthermore the 3D CT reconstructions of these casts simulate the preoperative angiographies. Our series of 50 human liver casts is, to our knowledge, the largest sample of its kind.

We would like to emphasize that vascular corrosion casting, when performed correctly, is an effective and reliable technique for the purpose of clinical anatomical investigation of the hepatic arterial system. The proper setting of the viscosity, the CT density and the color coding of the resin mixture developed by M. Kiss has the advantage of a highly detailed, real 3-dimensional demonstration of the hepatic arteries up to the 8^{th}-order branching from the proper hepatic artery. This allows us to provide information about the complete abdominal vascular anatomy, including detection of new hepatic variations, identifying subvariants of previously reported cases and describing vascular structures that can be overlooked on CT angiographies or conventional angiograms. Kishi et al. [131] stated that the accuracy of preoperative conventional subtraction angiography for the assessment of hepatic arterial anatomy was better than the less invasive MR or CT angiographic examinations, however, the same authors reported the use of 3D CT for preoperative evaluation [132]. Takatsuki et al. [133] relied on findings at hilar dissection rather than conventional angiography. Lee et al. [134] reported that multidetector CT was unable to depict the origin of A4 in 18% of liver donors, small accessory hepatic arteries in 13%, second-order branches of the left hepatic artery in 18%, second-order branches of the right hepatic artery in 6% due to

technical limitations and respiratory motion artifacts. Bogetti et al. [135] found that 3-MDCT reconstruction of hepatic arteries was better than or equivalent to angiography in 5 out of 9 donor patients. Contrary to Bogetti's results, Stemmler et al. [125] concluded that the quality of 2D images were superior in the identification of small diameter accessory left hepatic arteries to those of 3D images, however, the 3D MDCT angiograms are useful in preoperative assess of hepatic arterial anatomy. Similarly, in the studies of Coşkun et al. [136] and Kamel et al. [137], multidetector multiphase CT angiography did not depict one a-RHA from the SMA and an A4 in 1 case, respectively. De Cecco et al. [138] and Koops et al. [139] share the opinion that the wrong positioning of the angiographic catheter, the small caliber and the slow flow in the aberrant hepatic arteries are the main reasons for problematic identification of these vessels. Therefore, high resolution CT imaging and meticulous analysis of the images are strongly advised.

Our results show substantial differences concerning the variations of the extrahepatic arteries, compared to the literary data (Table 6). While, according to several authors, the incidence of the Michels I type ranges between 50.7% and 80.9% [32, 124, 125, 132, 136, 138, 139, 140], we found normal anatomy in only 42% of cases. The incidence of Michels III is the second most frequent type (6% - 18.3%) in the series of the majority of authors [28, 32, 124, 138, 139, 141, 142], but it is the third one with 14% in our study. Surprisingly, the second most frequent variation in our study was the UC type with 18%. Only Coşkun et al. [136] reported high frequency of UC type (16.6%), however, they described similar frequency for the occurrence of a-LHA from LGA (Michels V). Other publications report lower frequency, between 0% and 14.7% [32, 125, 138, 139, 142, 143, 144, 145]. Our series shares the general findings of the low percentage of Michels' types VII, VIII and IX, however, discrepancies of other patterns are obvious. These may be explained with the low number of cases in our series, population differences, misinterpretation of radiological findings in other investigations due to respiratory motion artifacts, wrong catheter positioning, narrow diameter or slow flow in the small aberrant vessels. It is notable that unintentional wrong catheter positioning can be relatively common during radiological interventions and selective angiographies.

The limitation of our study is the relatively low number of cases, however, our results and other angiographic investigations in which the patient numbers are ranged between 40 and 63 [125, 136, 137, 142] are comparable to the larger series as well [32, 139, 145, 146].

Table 6: Incidence of variations (%) compared to other authors

Author (Year)	Michels' types										
	I.	II.	III.	IV.	V.	VI.	VII.	VIII.	IX.	X.	NC
Our series (2015) *(n = 50)* corrosion casts	***42***	6.0	14.0	4.0	8.0	0	2.0	4.0	2.0	0	***18***
Kamel (2001) *(n = 40)* MDCTA	70.0	5.0	7.5	2.5	7.5	2.5	0	5	0	0	0
Coşkun (2005) *(n = 48)* 16-row CTA	54.1	0	6.3	0	16.6	2.1	4.2	0	0	0	16.6
Ferrari (2007) *(n = 60)* 64-row CTA	60.0	10.0	18.3	5.0	1.7	0	0	1.7	0	0	3.3
Stemmler (2004) *(n = 63)* 4/8-row CTA	80.9	0	6.3	0	7.9	0	1.6	1.6	1.6	0	0
Varotti (2004) *(n = 96)* liver graft	70.8	6.25	10.4	2.1	6.25	3.1	0	0	1.1	0	0
Ugurel (2010) *(n = 100)* 16-row MDCT	52.0	11.0	17.0	1.0	10.0	1.0	1.0	1.0	2.0	0	4.0
De Santis (2000) *(n = 150)* angiography	52.0	10.0	15.5	0.6	0.6	2.0	0.6	0	4.0	0	14.7
Michels (1966) *(n = 200)* cadaver dissection	55.0	10.0	11.0	1.0	8.0	7.0	1.0	2.0	2.5	0.5	0
Rygaard (1986) *(n = 216)* arteriographies	75.5	4.6	13.4	0.9	0	0	0.5	0.5	1.4	0	3.2
Kishi (2004) *(n = 223)* angiography	61.0	14.0	4.0	0	12.0	3.0	2.0	0	6.0	0	0
De Cecco (2009) *(n = 250)* 64-row CTA	66.0	5.2	9.2	2.0	5.2	4.0	2.0	0.6	2.0	0	3.3
Kishi (2010) *(n = 361)* angiography + CTA	68.6	10.2	6.9	4.2	4.7	1.4	0.6	0.6	2.5	0	0.3
Winston (2007) *(n = 371)* 4-row CTA	50.7	14.5	8.1	0	3.5	0	0	0	1.6	0	12.5
Covey (2002) *(n = 600)* DSA	61.3	3.8	8.7	0.5	10.7	1.5	1.0	3.0	2.0	0	7.5
Koops (2004) *(n = 604)* DSA	79.1	2.5	8.6	1.0	0.5	3.3	0.2	0.2	2.8	0	1.8
López-Andújar (2007) *(n = 1081)* liver graft	70.0	9.7	7.8	3.1	3.9	0.6	0.6	0.3	2.5	0	1.0
Saba (2011) *(n = 1629)* MDCTA	61.37	7.48	10.56	1.35	6.69	6.99	0.73	1.9	1.59	0.31	1.09

7.2 Surgical relevancies of the anatomical variations of the extrahepatic arteries, with special regard to rare variations

In the 1990s, livers displaying aberrant or accessory right and left arteries and requiring multiple anastomoses were not frequently used [147, 148]. Since the routine use of arterial reconstruction techniques, such exclusions are extremely uncommon. However, the surgeons should take special care in identifying and distinguishing their size and position of these accessory and replaced arteries, based on preoperative high resolution MDCT and/or MR angiographic data [149]. The surgical strategy depends largely on the diagnostic accuracy of the patient's vascular morphology. In contrast to the concepts of novel classifications (Hiatt et al. [27]; Abdullah et al. [146]), we share the opinion of Michels and recent clinical studies [145, 150, 151] making categorical distinction between accessory and replaced arteries. These authors point out that whereas replaced arteries must be always preserved, accessory vessels do not necessarily need to be reconstructed if intrahepatic anastomoses result in adequate back-flow or if intraoperative Doppler ultrasonography confirms sufficient perfusion of every liver segment [146]. The presence of accessory arteries, however, might necessitate reconstruction of multiple vessels which, due to their narrow diameter, leads to an increased risk of hepatic artery thrombosis. Consequently, not only the volume of the supplied liver parenchyma, but the length and caliber of these vessels are important factors in the planning, performance and efficiency of arterial reconstruction.

Identification of a replaced RHA from the SMA, present in 14% in our study, is critical when performing pancreaticoduodenectomy, pancreas procurement and porta hepatis dissection. Involvement of this vessel in pancreas head tumors precludes the patient from surgical resection (consequence is the same when the CHA originates from SMA). Performing back-table arterial reconstruction for such a r-RHA occasionally produces twisting and flow problems, requiring special vascular surgery techniques, such as the Carrel-patch anastomosis with the splenic artery stump [151]. An aberrant RHA, giving off the CyA, also can be a source of complications during laparoscopic cholecystectomy through accidental clipping or bleeding.

Some abnormalities, however, can be beneficial. Types II, III and IV, present in 24% in our series, are favorable variants to obtain reduced-sized liver grafts from either

a deceased or a living donor. A r-LHA may allow a rapid, facilitated dissection of the porta hepatis or may serve as an ideal artery for anastomosis in left lobe liver transplantation. Sakamoto et al. [150] stated that aberrant LHA or CHA provided thicker and longer arterial branches, resulting in safer anastomosis and decreased risk of hepatic arterial occlusion during living donor liver transplantation.

Whereas some authors [137] state that small-diameter arteries included in Michels' classification are of no clinical relevance, others [125, 138, 140, 152] point out that these vessels do affect the surgical planning and the placement of chemotherapy pump or embolization catheter in patients subjected to primary or metastatic liver tumor treatment. The reason is that variant anatomy may be the cause of incomplete embolization of the tumor, incomplete perfusion of the liver or liver remnant and extrahepatic perfusion, which may result in vessel thrombosis, misperfusion of chemotherapeutic or radiotherapeutic agents [125] pancreatitis or gastroduodenal ulcerations [153, 154]. This underlines the necessity of the precise and accurate description of not only the main, but even the smaller hepatic arteries. Regarding the rapid development of imaging modalities, it may be concluded that in the second decade of the 21st century, the routine evaluation of vascular anatomy with high accuracy CT and/or magnetic resonance angiography, replacing the use of DSA, is imperative in hepatobiliary pancreatic surgery, interventional radiological treatments and LDLT [32, 141, 155].

Regarding the UC variants, Abdullah et al. [146] published the highest number, actually 19 types (in 50 cases) which could not fit into Michels' classification, in their series of 932 surgical dissections in liver transplantation. Covey et al. [140] published 17 types (in 45 cases out of 600), followed by Winston [124] with 10 UC types (11 cases out of 50). Kishi et al. [132] report about r-RHA from dorsal pancreatic artery (DPA), r-LHA from CA, accessory S VI arteries (a-RHA) from PHA, CA and superior posterior pancreaticoduodenal artery (SPPD), combination of Michels V and IX – however, they only considered the latter case as UC variation. Table 7 shows a summary of extrahepatic variations not classified by Michels, observed in the current study and those described by other authors. Bold-italic letters show UC variations found also by us [32, 124, 126, 127, 134, 136, 138-143, 145, 146, 147, 155-160].

Table 7: Unclassified variations published by other authors.

AUTHOR	NC variations
New variations in our series	CHA pentafurcation
	proximal RHA from CHA with anteportal course
	proximal RPHA from CHA with retroportal course
	prox., retroportal RHA + CHA trifurcation: LHA, GDA, RGA
Ferrari	CHA from aorta
	r-LHA from IPD + r-RHA from SMA
De Cecco	r-RHA / r-LHA directly from aorta
	a-/r- RHA or LHA from CA, IPD, Bühler-arch
Coskun	a-RHA from CA
	GDA from RHA
	CHA trifurcation
	a-RHA from CHA
	a-RHA from SMA + a-LHA from GDA
Koops	r-LHA from GDA
	a-RHA from CA
	r-RHA from aorta
	r-LHA from GDA + r-RHA from SMA
	RHA + LHA separately from CA + GDA from LHA
	RHA + LHA separately from CA + GDA from RHA
Johnson	celiacomesenteric trunk +
	proximal branching of LHA from CHA + LGA from aorta
Chaib	LHA from aorta or SMA
Covey	CHA from aorta
	CHA trifurcation
	a-LHA from RAHA
	CHA quadrifurcation
	GDA from RHA or LHA
	RHA and/or LHA from CA
	a-RHA + a-LHA from LGA
	RHA and/or LHA from aorta
	r-RHA from right phrenic artery
	r-PHA from SMA + GDA from CA
	a-RHA from right phrenic artery or GDA or CA or LGA
Soin	CHA from aorta
	a-/r-LHA from CA
	a-/r-RHA from CA
	a-LHA + a-RHA from CA
	a-/r-LHA from supracoeliac aorta
	a-LHA from CA + r-RHA from SMA
	a-/r-LHA from LGA arising from aorta
	a-/r-LHA from GDA + a-/r-RHA from SMA
	dual origin of single CHA from SMA and CA
	a-/r-RHA and CHA from SMA + a-/r-LHA from LGA
Rygaard	RHA from aorta
	a-RHA from GDA
	double LHA from CHA
	r-LHA from LGA + LGA from aorta
	RHA from aorta + r-LHA from LGA
	RHA and LHA arise separately from CA
	r-RHA from SMA + a-RHA from GDA + r-LHA from LGA + LGA from aorta

AUTHOR	NC variations
Lee SS	RHA from CA
	CHA trifurcation
	celiacomesenteric trunk
	hepaticomesenteric trunk
	a-RHA from GDA or DPA
	proximal branching of LHA from CHA
	separate origins of S II. and S III. branches
Winston	LHA from CA
	RHA from CA
	LHA from CHA
	RHA from GDA
	GDA from RHA
	RHA from aorta
	CHA from aorta
	GDA from SMA
	CHA trifurcation
	S IV. branch from GDA
Braun	r-RHA from right renal artery
Wadhwa	retroportal course of PHA
Nakamura	a-LGA from LHA
Polguj	a-RHA from GDA
Saba	CHA from aorta
	r-CHA from SMA + a-LHA from LGA
Lopez-Andujar	CHA from aorta
	r-CHA from SMA + a-LHA from LGA
Fasel	***RHA from CA with retroportal course***
Gordon	a-RHA from dorsal pancreatic artery
Ugurel	CHA from aorta
	RHA from aorta
	RHA from middle colic artery
	LHA from CHA
Abdullah	CHA from aorta
	CHA trifurcation
	CHA with variations of GDA
	PHA with more than 2 branches
	a-/r-LHA from CA and/or a-/r-RHA from CA
	r-LHA from CA + r-RHA from SMA
	r-LHA + r-RHA from CHA
	r-RHA from IMA
	CHA but LHA gives PHA and RHA gives GDA
	retroportal CHA
	CHA + r-LHA + r-RHA (LGA from aorta)
	CHA from aorta + r-LHA
	CHA + r-LHA + r-RHA (CA and SMA origins at the same level)
	CHA + r-LHA (LGA from aorta)
	CHA (GDA from RHA) + a-RHA
	CHA (gives LHA + A4) + r-RHA
	CHA trifurcation (RHA, LHA, GDA) + a-RHA
	CHA trifurcation (RHA, LHA, GDA) + a-LHA
	CHA (gives GDA, RGA, LHA, RHA) + a-LHA + a-RHA

To the best of our knowledge, this study is the first to demonstrate four previously undescribed extrahepatic hilar arterial variants, which are to be recognized accurately before surgery in order to avoid graft injury and ensure a safe hepatectomy. These newly presented variants are: a) CHA pentafurcation; b) CHA trifurcation into LHA, RGA and GDA together with a proximally originating, retroportal RHA; c) proximal branching and anteportal course of RHA from CHA and d) RPHA deriving from CHA and travelling a retroportal course.

Pentafurcation of the CHA can be beneficial, if the right lobe, segment IV or segments II and III are involved in tumorous transformation. The sufficient length, large diameter and easy identification of the RHA, LHA and A4 – as seen on our preparation – allows the surgeon to safely perform right hepatectomy, left lateral split or Taj Mahal

resection [161], without compromising the arterialization of the liver remnant. This new arterial variation may not necessarily cause problems during superselective chemoembolizations, however, when whole organ chemo- or radioembolization is needed, this anomaly can potentially lead to significant gastrointestinal side effects by shunting the therapeutic agents to the non-hepatic arteries. While this anatomic variant is manageable during whole-organ recovery from a deceased donor, it may be a problem potentially for a full left lobe donation.

Particular attention must be paid when the RHA or the RPHA displays a proximal origin from the CHA (or CA). After passing behind or in front of the portal vein, the distal part of the vessel reaches the right side of the hepatoduodenal ligament. Therefore, one has to be careful during dissection not to inflict accidental damage to the common bile duct, which runs close to it in the hepatic pedicle. In case of proximal branching of the RHA, the point of origin usually lies deep, next to the CA division, consequently – if liver volumetry allows – the left lobe is more preferable for donation in living donor liver transplantation due to its easier accessibility. However, in this case left lateral splitting may endanger the blood supply of segment IV, causing ischemia.

In case of the proximal branching of the RPHA, the right anterior sector of the liver is supplied by the common trunk of the LHA and RAHA and the first branch of this trunk is A3 on our cast. Subsequently, the intrahepatic distribution of the segmental arteries (A3 from LHA-RAHA trunk; A2 from A4) would result in 2 arterial stumps during left lateral splitting and the arterial inflow of S V and S VIII could also be endangered. Furthermore, a separate RPHA arising from CHA may imply a relative contraindication for right lobe living donation and full left – full right split as well, due to the double source of arterial supply of the right lobe (S V and S VIII from LHA; S VI and S VII from CHA). On the other hand, the proximal origin of the RHA or RPHA may have the advantage of an easier selective catheterization and a reduced risk of chemo- or radiotherapeutic agents reaching the wrong liver lobe.

7.3 Left liver lobe

In reference to the branching of the LHA, our results are different from those of Couinaud's. He found the division of left hepatic artery into A4 + A2A3 to be the most frequent (86%), exhibiting A4 as the first branch of the LHA, while type A3A4 + A2

division was present in 10.8%. According to our data, the LHA gave its first branch to the S IV in only 38% of cases (19/50), the A2 was the first to derive from the LHA in 20% (10/50). We observed 4 new intrahepatic arborization variations of the LHA: a-LGA from A2 and A3 (1/50); a-LGA from a-LHA (1/50); triple accessory system (1/50); RGA-A1-CyA trunk (1/50).

7.4 Right liver lobe

In order to accurately describe the arterial supply of the right lobe segments, the terminology of the sectoral arteries has to be clarified. Based on the definition given by Michels, the PHA divides into RAHA and RPHA, which then irrigate the right anterior sector (S V, S VIII) and the right posterior sector (S VI, S VII) of the liver, respectively. We propose a terminology based on these easily understandable definitions and we determine the right-sided branching structures on the presence or absence of the RAHA and RPHA. This makes the description and comprehension of the anatomical variations less complicated, even if the segmental arteries are taken into account.

Couinaud, in his series of 93 corrosion casts [34], found single RAHA and RPHA in 61.3%, double RPHA in 34.4%, double RAHA in 2.1%, double RAHA + double RPHA in 1.1% and complex branching pattern in 1.1%. In contrast to his observations, we found normal RHA segmental/sectoral arborization in 50% of cases (25/50), while anomalous RHA patterns were seen in a high incidence (50% - 25/50). The most common variation of the RHA branching was the accessory segmental artery (or arteries) in 16% of cases (8/50), followed by the RPHA absence with 14% (7/50) – while double RAHA were seen on 2 casts and double RPHA was seen on 1 cast only.

The importance of RPHA absence is underlined in the study of Yoshioka et al. [35], who found that separate origins of A6 and A7 (referred to as ”combined type RPHA”) can lead to unexpected iatrogenic injury during hepatectomy and this pattern requires arterial reconstruction of both vessels during right lateral sector resection.

Radtke et al. [36] proposed a more complex classification for the right-sided segmental variations (Figure 45) and set up 7 different types of the peripheral branching patterns: normal (A), abnormal linear (B), stellate-shaped (C), early segmental (D) and double sectorial branching (E), crossover segmental transposition (F), early segmental branch combined with crossover segmental origin (G).

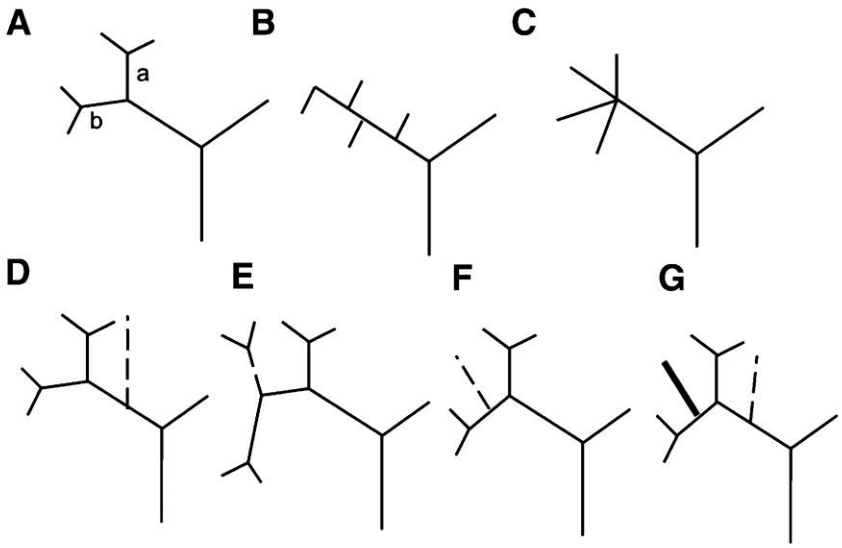


Figure 45: Classification of the right-sided segmental branching according to Radtke.

According to our terminology, type (B) means RAHA-RPHA absence, type (C) is equal to RHA quadrifurcation, type (D) – (F) – (G) are proximally originating accessory segmental arteries, type (E) means double RAHA/RPHA. In our series normal RHA branching was observed in 29 cases out of 50, type (B) was present in 3 cases, type (D) was seen in 4 cases, type (E) in 1 case. We did not observe casts with type (C), (F) and (G) patterns.

On the other hand, we found 7 arterial structures described neither by Radtke et al., nor by other authors. These variations include RAHA trifurcation (1/50), RHA trifurcation (2/50), absence of RAHA (2/50), absence of RPHA (7/50), nonexistent RAHA and RPHA (3/50), RHA giving A2 (1/50), proximally originating RPHA coursing behind the portal vein (1/50).

7.5 Liver segments of high surgical interest

7.5.1 Caudate lobe

The caudate lobe lies concealed between the right and the left lobes. Due to its deep position and the proximity of the hepatic hilum and the IVC, surgical treatment is associated with a considerable technical difficulty and high mortality rate. Resection of this liver segment was rarely performed in the past due to the challenging dissection and

the early invasion of the IVC or the portal vein by the tumor [162]. However, improved surgical techniques, perioperative treatment and accurate descriptions of the caudate lobe HCC arteries led to a more frequent use of caudate lobe segmentectomy worldwide, with different clinical outcomes [163, 164]. Therefore, transarterial chemoembolization (TACE) plays a crucial role in the treatment of HCC, however, its clinical results are limited [165, 166].

According to previous international results, the arterial supply of caudate lobe is highly variable with multiple vessels arising from different origins [167, 168, 169]. Extensive studies have been conducted on the description of the feeding arteries of the HCC in the caudate lobe, however, little attention has been paid to the vascular structure of the healthy segment I and its relationship to the interlobar communicating arcade. This study was designed to precisely describe the arterial anatomy of the caudate lobe arteries with the extrahepatic variations taken into account.

To our knowledge, this is the first study to describe the anatomy of the caudate lobe arteries on corrosion cast preparations without any kind of intervention to the hepatic arteries, such as temporary occlusion or ligation of the LHA or RHA [170, 171]. As mentioned before, the proper setting of the viscosity of the resin mixture allowed us to demonstrate the hepatic arteries up to the 8th-order branching from the proper hepatic artery. This resulted in highly accurate visualization of the caudate arteries.

Among the observed intrahepatic arterial variations, the caudate lobe showed the most variable pattern of arterial supply. We found 49 different types of vascular structure. Anastomoses were detected between the right and left lobes, between intra- and extrahepatic vessels (CyA, RGA, GDA, SAPD, IPPD) and we succeeded in describing the A1 origins on segmental and subsegmental arterial levels (Table 4). In the left lobe the three most frequent origins of the caudate lobe arteries were as follows: A4 in 44%, LHA in 38%, A2 in 24%. On the right side: RAHA in 40%, RPHA and RHA in 34% each, A8 in 24%. Rare origins were for example the CyA (10%), RGA (6%), A3 (6%), A6 (4%), SAPD (4%), IPPD (2%), PHA (2%). These findings also confirm that the interlobar communicating arcade not only plays a remarkable role in the supply of the hilar bile ducts, but it is also the main factor in feeding the caudate lobe as well [10,169].

Our results are similar to the data of Mizumoto's cadaveric dissections [20], who found the caudate arteries arise from the RPHA and the LHA in 32.1%, from the RPHA and the MHA in 26.4% and from three arteries in 20.8%. The current study also found concordant data with Tohma [170] and Gunji [172], who also described the A4 as the most common origin of the communicating arcade on the left side (55% and 62%, respectively), and the RAHA in the right lobe (73% and 46%, respectively).

Regarding the tumor feeding arteries in the caudate lobe, Miyayama et al. showed that the caudate arteries originate from different parts of the RHA in 27.6%, from the LHA in 20.7%, from RAHA in 20.7%, from RPHA 21.6%, from MHA in 6%, from PHA in 0.9% and from extrahepatic origins (right inferior phrenic artery, a-LGA) in 2.6%. The number of tumor feeding arteries was 1, 2 or 3, which depended on the tumor location within the caudate lobe [128]. Other rare origins can be the CHA and the cystic artery, while recurrent tumors are more frequently supplied by extrahepatic arteries, such as right inferior phrenic, right or left gastric, dorsal pancreatic, right adrenal, right renal capsular arteries [169]. Kim et al. [165] describe the origin of these arteries as the RHA (32.7%), the RAHA (26.6%), RPHA (16.3%), LHA (18.3%) and MHA (6.1%), with one tumor feeding artery present in 80%, two arteries in 17,5% and three arteries in 2,5%. It is notable that multiple tumor feeding vessels are significant factors in shorter overall survival, therefore the accurate identification of the A1 origins is of crucial importance.

This research extends our knowledge of the diversity of the S I vascular anatomy. Considering that multiple tumor feeding vessels are significant factors in shorter overall survival [165], therefore accurate identification and selective catheterization of the caudate arteries is of crucial importance [173]. Overall, our results suggest that the high rate of recurrence of caudate lobe HCC [163, 174] might be explained by the number and variability of its supplying arteries.

7.5.2 Segment IV.

The arterial supply of segment IV has been in the focus of surgeons for a long time, because this vessel plays a remarkable role in left lateral splitting and full-left full-right

splitting as well, carrying the risk of iatrogenic injury and subsequent segment IV ischemia, necrosis and abscess formation.

Several authors investigated the arterial supply of S IV, their results are shown in Table 8.

Table 8: The origins of A4 described in the literature.

Origins of segment IV feeding arteries according to available data									
AUTHOR	LHA	RHA	LHA + RHA	PHA	CHA	CA	LHA + PHA	RHA + PHA	LHA + RHA + PHA
Suzuki	54%	34%	-	8%	4%	-	-	-	-
Onishi	61.5%	27.5%	5.5%	5.5%	-	-	-	-	-
Jin	32.2%	53.2%	9.8%	4.8%	-	-	-	-	-
Wang	36.9%	56.3%	-	-	-	6.8%	-	-	-
Saba	55.01%	31.25%	6.63%	3.99%	-	-	1.1%	1.41%	0.61%

It is notable that our study revealed the arterial supply of S IV in much further details. Whereas the hereby mentioned data label only the main hepatic arteries (from CA to lobar arteries) as origins of A4, we were able to identify segmental, even extrahepatic vessels, which contributed to the feeding of S IV. Moreover, our results clearly show that S IV has more than one supplying artery in a remarkable part of cases. In our series two to five A4s were observed on 52% of the casts.

Our results are similar to Jin's and Wang's, who found LHA to be the origin of A4 in a relatively small number of cases. However, pure RHA origin was shown in only 6% in our study, which is significantly lower than any other data.

7.6 Intersegmental arterial anastomoses

Regarding the intersegmental anastomoses, Cho et al. [171] detected collaterals between the medial and the left lateral segments using 12 liver casts after ligating the right hepatic artery; Tohma et al. [170] performed a CT angiographic study with 13 patients applying temporary occlusion of the right and left hepatic arteries and described the left-sided origins of the communicating arcade as being the A4 in 62%, the LHA in 38% - and the RAHA in 46%, the RHA in 15% and both arteries in 38% on the right side. Stapleton et al. [10] and Vellar [175] prepared organ complexes as well and their series of 9 casts and 9 dissected complexes, respectively, showed the anastomosis between the left and right liver lobes, irrigating the caudate lobe. According to these authors the interlobar communicating arcade has a close relationship to the arterial supply of the biliary tract. In our series we found arterial anastomoses between the left

and right lobes in 90% (45/50) of cases, which always gave rise to the caudate lobe arteries. The origins of these anastomoses were highly variable, we observed 49 patterns in 50 cases.

Beside these, we found 44 anastomoses on 24 organ complexes – 16 of these arterial connections were intrasegmental (within segments III, IV, V, VI, VII, VIII), 11 were intralobar/intersegmental (e.g.: S III – S IV; S V – S VIII), 12 were extrahepatic (e.g.: GDA – SMA; SA – SMA; RGA – SPPD; A1 – GDA; A5 – CyA) and 5 were combined anastomoses (e.g.: IPPD – RHA – A1 – A6; RHA – SPPD – RGA – LHA).

8 Conclusions

This method, simulating the MDCT angiography allows us to provide accurate information about the complete upper abdominal vascular anatomy, including detection of new hepatic arterial variations, identifying subvariants of previously described cases. Given the fact that all four new unclassified variations were accurately visualized on our 3D CT reconstructions, these structures should be identifiable during clinical CT examinations as well. Being of great surgical and radiological importance, unusual variations must always be in the focus of surgeons and radiologists during the preoperative evaluations and interventions in the upper abdomen.

In this present study we described the detailed arterial supply of the human liver on 50 corrosion cast preparations and found numerous anatomical variations on both extrahepatic and intrahepatic levels, including newly observed vascular structures which have not been reported before. New extrahepatic variations include combinations of already known variants (CHA trifurcation + accessory A5; proximal branching of LHA from CHA + accessory A8) and entirely new structures (CHA pentafurcation + double RAHA; CHA trifurcation + proximal RHA from CHA with retroportal course; CHA trifurcation + RGA-A1-CyA trunk; proximal RHA from CHA with anteportal course; proximal RPHA from CHA with retroportal course).

The arborization patterns of the RHA and LHA are summarized on Table 9.

Table 9: Levels of RHA/LHA arborization patterns.
The variations can be combined independently of one another.

Different levels of RHA/LHA branching variations in our series			
Extrahepatic	**Lobar**	**Sectoral**	**Segmental**
Michels I.	RHA trifurcation RGA from LHA a-LGA from LHA or A2+A3	double RAHA double RPHA RAHA trifurcation RAHA and/or RPHA absence	a-A5 a-A6 a-A8 A2 from RHA
Michels II-IX.	a-LGA from LHA a-LGA from a-LHA	RAHA and/or RPHA absence	a-A6 a-A7
CHA trifurcation CHA pentafurcation	proximal LHA from CHA RHA from CA with retroportal course proximal RHA with retroportal course proximal RHA with anteportal course	double RAHA proximal RPHA with retroportal course	a-A5 a-A8 RGA-A1-CyA

Concerning the segmental arterial supply, our findings show that the liver segments are often fed by more than one artery. S I receives multiple feeding arteries in 100% of cases; S II in 22%; S III in 30%; S IV in 52%, S V in 74%; S VI in 56%; S VII in 22% and S VIII in 32%.

The different liver segments can be sequenced in the order of arterial variability, which means not as much the number of variations described, but the incidence of the most frequent case of arterial supply (Table 10).

Table 10: Distribution of liver segments according to the number of variants and the incidence of the most frequent arterial supply.

	No. of arterial variations	Incidence of the most frequent variation	Most frequent origin of segmental artery
S I.	49	4%	(RHA # GDA # A4)
S II.	14	28%	A2A3
S III.	14	26%	A2A3
S IV	27	24%	LHA
S V.	20	58%	RAHA
S VI.	18	56%	RPHA
S VII.	8	70%	RPHA
S VIII.	17	62%	RAHA

According to our data, the caudate lobe has the most variable arterial anatomy, while segment VII is the most stable one displaying only 8 types of arterial patterns and with 70% of the most common structure present on the casts. Although an uncommon source of arteries supplying a particular liver segment is rare, surprising anatomic variations may occur, e.g. S VIII arteries arising from the cystic artery.

It is of note, that in our series there is a significant difference between the left and the right lobe in terms of arterial variability, the right lobe has a much more diverse, colorful vascular anatomy. In contrast to our findings, some authors [133] claimed that surgical anatomy and the technique of hepatic arterial reconstruction are simpler when using right lobe liver grafts during LDLT due to the higher incidence of multiple graft arteries irrigating the left lobe. Takatsuki [133], Kishi [131] and Marcos [176] found similar, low incidence of double arteries feeding the right lobe graft (1.3%-11.6%).

The increasing number of living donor and cadaveric liver transplantations, anatomical and non-anatomical hepatic resections, laparoscopic and radiological interventions are the main reason for a renewed interest in the investigation and statistical analysis of arterial variations and reports of new variants. The hepatic arterial anatomy is highly variable and some variations may necessitate the modification of the surgical approach [6, 32, 149, 150]. We believe that our hereby presented data can contribute to the better understanding of the segmental arterial supply of the human liver and therefore lead to the reduction of complications during surgical and radiological interventions in the upper abdomen.

Main findings

1. Description of 4 new extrahepatic and 11 new intrahepatic arterial variations.
2. First complete documentation of the associated extra- and intrahepatic arterial variations, including previously unknown cases.
3. First highly detailed description of the caudate lobe arteries in healthy livers.
4. To the best of our knowledge, we were the first visualize the intrahepatic arteries up to subsegmental levels.

9 Summary

Aims The arterial anatomy of the liver is highly variable with normal anatomy present in 50.7%-80.9% of cases. Recognition of abnormalities is crucial, especially during liver transplantation, because an impaired hepatic arterial blood supply may result in ischemic complications. The purpose of our study was to investigate the anatomical variations of the extra- and intrahepatic arterial structures of the liver and their potential impact on liver surgery.

Methods Human abdominal organ complexes were used to prepare 50 corrosion casts. A multicomponent resin mixture was injected into the abdominal aorta to visualize the arterial structure of the upper abdominal organs. The portal vein was injected with a differently colored resin in 16 cases. Digestion of soft tissues was achieved using cc. KOH solution. The extrahepatic arterial variations were classified according to Michels and the arterial supply of the liver segments was described. All specimens underwent 3D volumetric CT reconstruction.

Results Normal extrahepatic anatomy was seen in 42% of cases and variants were observed in the other 58%. No Michels type VI and X variations were present, and in 18% of cases the extrahepatic arterial anatomy did not fit into Michels' classification. We report 4 extrahepatic arterial variations which have not been described before. Branching and coursing variations of the lobar arteries were observed in 56% of cases. We detected a total of 11 new arborization variants of the left and right hepatic arteries. The arterial supply of the liver segments was described by the origins of their feeding arteries, including the first highly detailed description of the caudate lobe arteries in healthy livers. We provided the first complete documentation of the associated extra- and intrahepatic arterial variations, including previously unknown cases.

Conclusions In contrast to the available data, different frequencies of extrahepatic arterial variations were found in our series compared to other publications. We detected new variations including extrahepatic, segmental and combined patterns. Our data may contribute to the reduction of complications during surgical and radiological interventions in the upper abdomen.

10 Összefoglalás

Célok A máj artériás anatómiája rendkívül változatos. Normál, tankönyvi anatómiával az esetek 50,7%-80,9%-ában találkozunk. A variációk felismerése nagy jelentőséggel bír, különösképpen májtranszplantáció során, hiszen a máj artériás vérellátásának zavarai ischaemiás szövődményekhez vezethetnek. Kutatásunk célja a humán máj extra- és intrahepatikus artériás variációinak feltérképezése, és ezen struktúrák májsebészetre gyakorolt hatásának vizsgálata volt.

Módszerek Összesen 50 db humán hasi szervkomplexet használtunk korróziós öntvények készítéséhez. Egy többkomponensű gyantakeveréket injektáltunk az aortába, hogy láthatóvá tegyük a felhasi zsigerek artériás rendszerét. Tizenhat esetben a vena portae-t is feltöltöttük egy eltérő színű gyantával. A lágyrészek korróziójához tömény KOH-oldatot használtunk. Az extrahepatikus variációkat Michels szerint osztályoztuk és leírtuk a máj artériás vérellátását szegmentális szinten. Az összes preparátumról 3D volumetriás CT rekonstrukció készült.

Eredmények Normál extrahepatikus anatómia az esetek 42%-ában volt megfigyelhető, a maradék 58%-ban variációkat találtunk. Michels VI és X típusú variációt nem észleltünk. Az esetek 18%-ában az extrahepatikus anatómia Michels szerint nem volt klasszifikálható. Négy olyan extrahepatikus variációról számolunk be, amelyek korábban nem kerültek leírásra. A lebenyartériák eredési és lefutási variációit az esetek 56%-ában figyeltük meg, az arteria hepatica dextra és sinistra arborizációjának összesen 11 új változatát írtuk le. A májszegmentek artériás vérellátását a tápláló artériáik eredési helyeinek leírásával jellemeztük, beleértve a lobus caudatus ellátásának első részletes leírását egészséges májban. A társuló extra- és intrahepatikus artériás variációk első komplett dokumentációját szolgáltattuk, beleértve korábban ismeretlen eseteket is.

Következtetések Az extrahepatikus artériás variációk incidenciájával kapcsolatban eltérő eredményeket kaptunk a nemzetközi irodalmi adatokhoz képest. Több új artériás variációt találtunk extrahepatikus, szegmentális szinten és ezek kombinációit tekintve. Eredményeink hozzájárulhatnak a felső hasi régióban végzett sebészi és radiológiai beavatkozások szövődményeinek csökkentéséhez.

11 Bibliography

1. Henderson JM, Heymsfield SB, Horowitz J, Kutner MH. (1981) Measurement of liver and spleen volume by computed tomography. Assessment of reproducibility and changes found following a selective distal splenorenal shunt. Radiology, 141: 525-527.
2. Couinaud C. Le Foie: Études anatomiques et chirurgicales. Masson, Paris, 1957.
3. Bismuth H. (1982) Surgical anatomy and anatomical surgery of the liver. World J Surg, 6: 3-9.
4. Strasberg SM. (2005) Nomenclature of hepatic anatomy and resections: a review of the Brisbane 2000 system. J Hepatobiliary Pancreat Surg, 12(5): 351-355.
5. Couinaud C. Surgical Anatomy of the Liver Revisited. Couinaud, Paris, 1989.
6. Reichert PR, Renz JF, D'Albuquerque LA, Rosenthal P, Lim RC, Roberts JP, Ascher NL, Emond JC. (2000) Surgical anatomy of the left lateral segment as applied to living-donor and split-liver transplantation: a clinicopathologic study. Ann Surg, 232: 658-664.
7. Northover JM, Terblanche J. (1979) A new look at the arterial supply of the bile duct in man and its surgical implications. Br J Surg, 66: 379-384.
8. Stapleton GN, Hickman R, Terblanche J. (1998) Blood supply of the right and left hepatic ducts. Br J Surg, 85: 202-207.
9. Kiss M, Deshpande RR, Nemeskéri A, Nguyen TT, Kürti Z, Kovács S, Pápai Z, Németh K, Szuák A, Dudás I, Kóbori L. (2015) Optimal line of hepatotomy for left lateral living donor liver transplantation according to the anatomical variations of left hepatic duct system. Pediatr Transplant, 19(5): 510-516.
10. Guiney MJ, Kruskal JB, Sosna J, Hanto DW, Goldberg SN, Raptopoulos V. (2003) Multi-detector row CT of relevant vascular anatomy of the surgical plane in split-liver transplantation. Radiology, 229: 401-407.
11. Makuuchi M, Hasegawa H, Yamazaki S, Bandai Y, Watanabe G, Ito T. (1983) The inferior right hepatic vein: ultrasonic demonstration. Radiology, 148: 213-217.
12. Imamura H, Makuuchi M, Sakamoto Y, Sugawara Y, Sano K, Nakayama A, Kawasaki S, Takayama T. (2000) Anatomical keys and pitfalls in living donor liver transplantation. J Hepatobiliary Pancreat Surg, 7: 380-394.

13. Reuter SR, Redman HC, Cho KJ. Gastrointestinal Angiography. Third edition. WB Saunders, Philadelphia, 1986.
14. Kaufman JA, Lee MJ. Vascular and Interventional Radiology: The Requisites. Mosby, St. Louis, 2004.
15. McNulty JG, Hickey N, Khosa F, O'Brien P, O'Callaghan JP. (2001) Surgical and radiological significance of variants of Buhler's anastomotic artery: a report of three cases. Surg Radiol Anat, 23: 277-280.
16. Iezzi R, Cotroneo AR, Giancristofaro D, Santoro M, Storto ML. (2008) Multidetector-row CT angiographic imaging of the celiac trunk: anatomy and normal variants. Surg Radiol Anat, 30: 303-310.
17. Miyaki T. (1989) Patterns of arterial supply of the human fetal liver: the significance of the accessory hepatic artery. Acta Anat, 136: 107-111.
18. Michels NA. (1966) Newer anatomy of the liver and its variant blood supply and collateral circulation. Am J Surg, 112: 337-347.
19. Onishi H, Kawarada Y, Das BC, Nakano K, Gadzijev EM, Ravnik D, Isaji S. (2000) Surgical anatomy of the medial segment (S4) of the liver with special reference to bile ducts and vessels. Hepatogastroenterology, 47: 143-150.
20. Mizumoto R, Suzuki H. (1988) Surgical anatomy of the hepatic hilum with special reference to the caudate lobe. World J Surg, 12: 2-10.
21. Healey JE, Schroy PC. (1953) The intrahepatic distribution of the hepatic artery in man. J Int Coll Surg, 20: 133-148.
22. Yoshimura H, Uchida H, Ohishi H, Honda N, Ohue S, Kinoshita Y, Katsuragi M, Matuso N, Hosogi Y, Hatakeyama M. (1986) Evaluation of "M-point" in hepatic artery to identify left medial segment of liver angiographic study. Eur J Radiol, 6(3): 195-198.
23. Haller A. Icones anatomicae in quibus aliquae partes corporis humani delineatae proponuntur et arteriarum potissimum historia continetur. Vandenhoeck, Göttingen, 1756.
24. Adachi B. Das Arteriensystem der Japaner. Kenkyusha Press, Kyoto, 1928.
25. Ödman P. (1958) Percutaneous selective angiography of the celiac artery. Acta Radiol Suppl, 159: 1-168.

26. Lunderquist A. (1967) Arterial segmental supply of the liver. Acta Radiol Stockh Suppl, 272.
27. Hiatt JR, Gabbay J, Busuttil RW. (1994) Surgical anatomy of the hepatic arteries in 1000 cases. Ann Surg, 220: 50-52.
28. Varotti G, Gondolesi GE, Goldman J, Wayne M, Florman SS, Schwartz ME, Miller CM, Sukru E. (2004) Anatomic variations in right liver living donors. J Am Coll Surg, 198: 577-582.
29. Suzuki H. Correlation and anomalies of the vascular structure in Glisson's area around the hepatic hilum, from the standpoint of hepatobiliary surgery. (1982) Arch Jpn Chir, 51: 713-731.
30. Jin GY, Yu HC, Lim HS, Moon JI, Lee JH, Chung JW, Cho BH. (2008) Anatomical variations of the origin of the segment 4 hepatic artery and their clinical implications. Liver Transpl, 14: 1180-1184.
31. Wang S, He X, Li Z, Peng Z, Tam NL, Sun C, Hu A, Huang J. (2010) Characterization of the middle hepatic artery and its relevance to living donor liver transplantation. Liver Transpl, 16: 736-741.
32. Saba L, Mallarini G. (2011) Anatomic variations of arterial liver vascularization: an analysis by using MDCTA. Surg Radiol Anat, 33: 559-568.
33. Rogiers X, Bismuth H, Busuttil RW, Broering DC, Azoulay D. Split Liver Transplantation. Springer, Darmstadt, 2002.
34. Couinaud C, Houssin D. Controlled partition of the liver for transplantation. Couinaud, Paris, 1991.
35. Yoshioka Y, Ebata T, Yokoyama Y, Igami T, Sugawara G, Nagino M. (2011) "Supraportal" right posterior hepatic artery: an anatomic trap in hepatobiliary and transplant surgery. World J Surg, 35: 1340-1344.
36. Radtke A, Sgourakis G, Sotiropoulos GC, Molmenti EP, Nadalin S, Schroeder T, Saner F, Schenk A, Cincinnati VR, Broelsch CE, Lang H, Malagó M. (2009) Vascular and biliary anatomy of the right hilar window: its impact on recipient morbidity and mortality for right graft live donor liver transplantation. World J Surg, 33: 1941-1951.
37. Starzl TE, Marchioro TL, Vonkaulla KN, Hermann G, Brittain RS, Waddell WR. (1963) Homotransplantation of the liver in humans. Surg Gynecol Obstet, 117: 659-676.

38. SRTR/OPTN Annual Report 2009.
http://www.srtr.org/annual_Reports/archives/2009/2009_Annual_Report/default.htm
39. Bismuth H, Houssin D. (1984) Reduced-sized orthotopic liver graft in hepatic transplantation in children. Surgery, 95: 367-370.
40. Pichlmayr R, Ringe B, Gubernatis G, Hauss J, Bunzendahl H. (1988) Transplantation of a donor liver to 2 recipients (splitting transplantation)--a new method in the further development of segmental liver transplantation. Langenbecks Arch Chir, 373: 127-130.
41. Bismuth H, Morino M, Castaing D, Gillon MC, Descorps Declere A, Saliba F, Samuel D. (1989) Emergency orthotopic liver transplantation in two patients using one donor liver. Br J Surg, 76: 722-724.
42. Broelsch CE, Emond JC, Whitington PF, Thistlethwaite JR, Baker AL, Lichtor JL. (1990) Application of reduced-size liver transplants as split grafts, auxiliary orthotopic grafts, and living related segmental transplants. Ann Surg, 212: 368-377.
43. Rogiers X, Malago M, Habib N, Knoefel WT, Pothmann W, Burdelski M, Meyer-Moldenhauer WH, Broelsch CE. (1995) In situ splitting of the liver in the heart-beating cadaveric organ donor for transplantation in two recipients. Transplantation, 59: 1081-1083.
44. Azoulay D, Astarcioglu I, Bismuth H, Castaing D, Majno P, Adam R, Johann M. (1996) Split-liver transplantation. The Paul Brousse policy. Ann Surg, 224: 737-748.
45. Rogiers X, Malagó M, Gawad K, Jauch KW, Olausson M, Knoefel WT, Gundlach M, Bassas A, Fischer L, Sterneck M, Burdelski M, Broelsch CE. (1996) In situ splitting of cadaveric livers. The ultimate expansion of a limited donor pool. Ann Surg, 224: 331-341.
46. Calne RY, Rolles K, White DJ, Thiru S, Evans DB, McMaster P, Dunn DC, Craddock GN, Henderson RG, Aziz S, Lewis P. (1979) Cyclosporin A initially as the only immunosuppressant in 34 recipients of cadaveric organs: 32 kidneys, 2 pancreas and 2 livers. Lancet, 2: 1033-1036.
47. Starzl TE, Groth CG, Brettschneider L, Penn I, Fulginiti VA, Moon JB, Blanchard H, Martin AJ Jr, Porter KA. (1968) Orthotopic homotransplantations of the human liver. Ann Surg, 168: 392-415.

48. Starzl TE, Iwatsuki S, Esquivel CO, Todo S, Kam I, Lynch S, Gordon RD, Shaw BW Jr. (1985) Refinements in the surgical technique of liver transplantation. Semin Liver Dis, 5: 349-356.

49. Calne RY, Williams R. (1968) Liver transplantation in man. I. Observations on technique and organization in five cases. Br Med J, 4: 535-540.

50. Calne RY. (1969) Surgical aspects of clinical liver transplantation in 14 patients. Br J Surg, 56: 729-736.

51. Starzl TE, Kaupp HA, Brock DR, Lazarus RE, Johnson RV. (1960) Reconstructive problems in canine liver homotransplantation with special reference to the postoperative role of hepatic venous flow. Surg Gynecol Obstet, 111: 733-743.

52. Griffith BP, Shaw BW, Hardesty RL, Iwatsuki S, Bahnson HT, Starzl TE. (1985) Veno-venous bypass without systemic anticoagulation for transplantation of the human liver. Surg Gynecol Obstet, 160: 271-282.

53. Khoury GF, Mann ME, Porot MJ, Abdul-Rasool IH, Busuttil RW. (1987) Air embolism associated with venovenous bypass during orthotopic liver transplantation. Anesthesiology, 67: 848-851.

54. Shaw BW Jr, Martin DJ, Marquez JM, Kang YG, Bugbee AC Jr, Iwatsuki S, Griffith BP, Hardesty RL, Bahnson HT, Starzl TE. (1984) Venous bypass in clinical liver transplantation. Ann Surg, 200: 524-534.

55. Tzakis A, Todo S, Starzl TE. (1989) Orthotopic liver transplantation with preservation of the inferior vena cava. Ann Surg, 210: 649-652.

56. Salizzoni M, Andorno E, Bossuto E, Cerutti E, Livigni S, Lupo F, Maritano M, Massano G, Marchesa PE, Pinna Pintor M. (1994) Piggyback techniques versus classical technique in orthotopic liver transplantation: a review of 75 cases. Transplant Proc, 26: 3552-3553.

57. Figueras J, Sabate A, Fabregat J, Torras J, Drudis R, Rafecas A, Dalmau A, Bartolomé C, Jaurrieta E. (1993) Hemodynamics during the anhepatic phase in orthotopic liver transplantation with vena cava preservation: a comparative study. Transplant Proc, 25: 2588-2589.

58. Jovine E, Mazziotti A, Grazi GL, Ercolani G, Masetti M, Morganti M, Pierangeli F, Begliomini B, Mazzetti PG, Rossi R, Paladini R, Cavallari A. (1997) Piggy-back versus

conventional technique in liver transplantation: report of a randomized trial. Transpl Int, 10: 109-112.

59. Tzakis AG, Reyes J, Nour B, Marino IR, Todo S, Starzl TE. (1993) Temporary end to side portocaval shunt in orthotopic hepatic transplantation in humans. Surg Gynecol Obstet, 176: 181-183.

60. Belghiti J, Noun R, Sauvanet A. (1995) Temporary portocaval anastomoses with preservation of caval flow during orthotopic liver transplantation. Am J Surg, 169: 277-279.

61. Belghiti J, Noun R, Sauvanet A, Durand F, Aschehoug J, Erlinger S, Benhamou JP, Bernuau J. (1995) Transplantation for fulminant and subfulminant hepatic failure with preservation of portal and caval flow. Br J Surg, 82: 986-989.

62. Cherqui D, Lauzet JY, Rotman N, Duvoux C, Dhumeaux D, Julien M, Fagniez PL. (1994) Orthotopic liver transplantation with preservation of the caval and portal flow. Transplantation, 58: 793-796.

63. Steib A, Saada A, Clever B, Lehmann C, Freys G, Levy S, Boudjema K. (1997) Orthotopic liver transplantation with preservation of portocaval flow compared with venovenous bypass. Liver Transpl Surg, 35: 518-525.

64. Figueras J, Llado L, Ramos E, Jaurrieta E, Rafecas A, Fabregat J, Torras J, Sabate A, Dalmau A. (2001) Temporary portocaval shunt during liver transplantation with vena cava preservation. Results of a prospective randomized study. Liver Transpl, 7: 904-911.

65. Belghiti J, Panis Y, Sauvanet A, Gayet B, Fékété F. (1992) A new technique of side to side caval anastomoses during orthotopic hepatic transplantation without caval occlusion. Surg Gynecol Obstet, 75: 270-272.

66. Bismuth H, Castaing D, Shellock DJ. (1992) Liver transplantation by "face-à-face" venacavaplasty. Surgery, 111: 1515.

67. Goldstein RM, Secrest CL, Klintmalm GB, Husberg BS. (1990) Problematic vascular reconstruction in liver transplantation. Part I. Arterial. Surgery, 107: 544-548.

68. Shaked AA, Takiff H, Bussutil RW. (1991) The use of the supraceliac aorta for hepatic arterial revascularization in transplantation of the liver. Surg Gynecol Obstet, 173: 198-202.

69. Figueras J, Parés D, Aranda H, Rafecas A, Fabregat J, Torras J, Ramos E, Lama C, Lladó L, Jaurrieta E. (1997) Results of using the recipient's splenic artery for arterial reconstruction in liver transplantation in 23 patients. Transplantation, 64: 655-658.
70. Rouch DA, Emond JC, Thistlethwaite JR, Mayes JI, Broelsch CE. (1990) Choledochocholedocostomy without a T tube or internal stent in transplantation of the liver. Surg Gynecol Obstet, 170: 239-244.
71. Colledan M, Andorno E, Valente U, Gridelli B. (1999) A new splitting technique for liver grafts. Lancet, 353: 1763.
72. Broering DC, Rogiers X, Malago M, Bassas A, Broelsch CE. (1998) Vessel loop-guided technique for parenchymal transection in living donor or in situ split-liver procurement. Liver Transpl Surg, 4: 241.
73. Starzl TE, Miller C, Broznick B, Makowka L. (1987) An improved technique for multiple organ harvesting. Surg Gynecol Obstet, 165: 343-348.
74. Lee S, Park K, Hwang S, Lee Y, Choi D, Kim K, Koh K, Han S, Choi K, Hwang K, Makuuchi M, Sugawara Y, Min P. (2001) Congestion of right liver graft in living donor liver transplantation. Transplantation, 71: 812-814.
75. Gundlach M, Broering D, Topp S, Sterneck M, Rogiers X. (2000) Split-cava technique: liver splitting for two adult recipients. Liver Transpl, 6: 703-706.
76. Broering DC, Wilms C, Lenk C, Schulte am Esch J 2nd, Schönherr S, Mueller L, Kim JS, Helmke K, Burdelski M, Rogiers X. (2005) Technical refinements and results in full-right full-left splitting of the deceased donor liver. Ann Surg, 242: 802-813.
77. Broering DC, Bok P, Mueller L, Wilms C, Rogiers X. (2005) Splitting of the middle hepatic vein in full-right full-left splitting of the liver. Liver Transpl, 11: 350-352.
78. Broering DC, Kim JS, Mueller T, Fischer L, Ganschow R, Bicak T, Mueller L, Hillert C, Wilms C, Hinrichs B, Helmke K, Pothmann W, Burdelski M, Rogiers X. (2004) One hundred thirty-two consecutive pediatric liver transplants without hospital mortality: lessons learned and outlook for the future. Ann Surg, 240: 1002-1012.
79. Stringer MD, Marshall MM, Muiesan P, Karani JB, Kane PA, Mieli-Vergani G, Rela M, Heaton ND. (2001) Survival and outcome after hepatic artery thrombosis complicating pediatric liver transplantation. J Pediatr Surg, 36: 888-891.
80. Quinones-Baldrich WJ, Memsic L, Ramming K, Hiatt J, Busuttil RW. (1986) Branch patch for arterialization of hepatic grafts. Surg Gynecol Obstet, 162: 488-490.

81. Lerut J, de Ville de Goyet J, Donataccio M, Reding R, Otte JB. (1994) Piggyback transplantation with side-to-side cavocavostomy is an ideal technique for right split liver allograft implantation. J Am Coll Surg, 179: 573-576.

82. Gridelli B, Remuzzi G. (2000) Strategies for making more organs available for transplantation. N Engl J Med, 343: 404-410.

83. de Ville de Goyet J, Hausleithner V, Reding R, Lerut J, Janssen M, Otte JB. (1993) Impact of innovative techniques on the waiting list and results in pediatric liver transplantation. Transplantation, 56: 1130-1136.

84. Broering DC, Mueller L, Ganschow R, Kim JS, Achilles EG, Schäfer H, Gundlach M, Fischer L, Sterneck M, Hillert C, Helmke K, Izbicki JR, Burdelski M, Rogiers X. (2001) Is there still a need for living-related liver transplantation in children? Ann Surg, 234: 713-722.

85. Troisi R, Noens L, Montalti R, Ricciardi S, Philippé J, Praet M, Conoscitore P, Centra M, de Hemptinne B. (2006) ABO-mismatch adult living donor liver transplantation using antigen-specific immunoadsorption and quadruple immunosuppression without splenectomy. Liver Transpl, 12: 1412-1417.

86. Kozaki K, Egawa H, Kasahara M, Oike F, Yoshizawa A, Fukatsu A, Tanaka K. (2005) Therapeutic strategy and the role of apheresis therapy for ABO incompatible living donor liver transplantation. Ther Apher Dial, 9: 285-291.

87. Egawa H, Oike F, Buhler L, Shapiro AM, Minamiguchi S, Haga H, Uryuhara K, Kiuchi T, Kaihara S, Tanaka K. (2004) Impact of recipient age on outcome of ABO-incompatible living-donor liver transplantation. Transplantation, 77: 403-411.

88. Egawa H, Teramukai S, Haga H, Tanabe M, Fukushima M, Shimazu M. (2008) Present status of ABO-incompatible living donor liver transplantation in Japan. Hepatology, 47: 143-152.

89. Kucher N, Tapson VF, Goldhaber SZ. (2005) Risk factors associated with symptomatic pulmonary embolism in a large cohort of deep vein thrombosis patients. Thromb Haemost, 93: 494-498.

90. Durand F, Ettorre GM, Douard R, Denninger MH, Kianmanesh A, Sommacale D, Farges O, Valla D, Belghiti J. (2002) Donor safety in living related liver transplantation: underestimation of the risks for deep vein thrombosis and pulmonary embolism. Liver Transpl, 8: 118-120.

91. Broering DC, Sterneck M, Rogiers X. (2003) Living donor liver transplantation. J Hepatol, 38: S119-135.
92. Middleton PF, Duffield M, Lynch SV, Padbury RT, House T, Stanton P, Verran D, Maddern G. (2006) Living donor liver transplantation-adult donor outcomes: a systematic review. Liver Transplantation, 12: 24-30.
93. Fan ST, Lo CM, Liu CL, Yong BH, Chan JK, Ng IO. (2000) Safety of donors in live donor liver transplantation using right lobe grafts. Arch Surg, 135: 336-340.
94. Lo CM, Fan ST, Liu CL, Chan JK, Lam BK, Lau GK, Wei WI, Wong J. (1999) Minimum graft size for successful living donor liver transplantation. Transplantation, 68: 1112-1116.
95. Enne M, Pacheco-Moreira L, Balbi E, Cerqueira A, Santalucia G, Martinho JM. (2005) Liver transplantation with monosegments. Technical aspects and outcome: a meta-analysis. Liver Transpl, 11: 564-569.
96. Kasahara M, Kaihara S, Oike F, Ito T, Fujimoto Y, Ogura Y, Ogawa K, Ueda M, Rela M, D Heaton N, Tanaka K. (2003) Living-donor liver transplantation with monosegments. Transplantation, 76: 694-696.
97. Lee SG, Hwang S, Park KM, Kim KH, Ahn CS, Lee YJ, Cheon JY, Joo SH, Moon DB, Joo CW, Min PC, Koh KS, Han SH, Choi KT, Hwang KS. (2001) Seventeen adult-to-adult living donor liver transplantations using dual grafts. Transplant Proc, 33: 3461-3463.
98. Broering DC, Walter J, Rogiers X. (2007) The first two cases of living donor liver transplantation using dual grafts in Europe. Liver Transpl, 13: 149-153.
99. Marcos A, Ham JM, Fisher RA, Olzinski AT, Shiffman ML, Sanyal AJ, Luketic VA, Sterling RK, Posner MP. (2000) Emergency adult to adult living donor liver transplantation for fulminant hepatic failure. Transplantation, 69: 2202-2205.
100. Testa G, Malagó M, Nadalin S, Hertl M, Lang H, Frilling A, Broelsch CE. (2002) Right-liver living donor transplantation for decompensated end-stage liver disease. Liver Transpl, 8: 340-346.
101. Kam I. (2002) Adult-adult right hepatic lobe living donor liver transplantation for status 2a patients: too little, too late. Liver Transpl, 8: 347-349.
102. Todo S, Furukawa H. (2004) Living donor liver transplantation for adult patients with hepatocellular carcinoma: experience in Japan. Ann Surg, 240: 451-461.

103. Soubrane O, Cherqui D, Scatton O, Stenard F, Bernard D, Branchereau S, Martelli H, Gauthier F. (2006) Laparoscopic left lateral sectionectomy in living donors: safety and reproducibility of the technique in a single center. Ann Surg, 244: 815-820.
104. Fan ST, Lo CM, Liu CL, Wang WX, Wong J. (2003) Safety and necessity of including the middle hepatic vein in the right lobe graft in adult-to-adult live donor liver transplantation. Ann Surg, 238: 137-148.
105. Shah SA, Grant DR, Greig PD, McGilvray ID, Adcock LD, Girgrah N, Wong P, Kim RD, Smith R, Lilly LB, Levy GA, Cattral MS. (2005) Analysis and outcomes of right lobe hepatectomy in 101 consecutive living donors. Am J Transplant, 5: 2764-2769.
106. Scatton O, Belghiti J, Dondero F, Goere D, Sommacale D, Plasse M, Sauvanet A, Farges O, Vilgrain V, Durand F. (2004) Harvesting the middle hepatic vein with a right hepatectomy does not increase the risk for the donor. Liver Transpl, 10: 71-76.
107. Mäkisalo H, Chaib E, Krokos N, Calne R. (1993) Hepatic arterial variations and liver-related diseases of 100 consecutive donors. Transpl Int, 6: 325-329.
108. Todo S, Makowka L, Tzakis AG, Marsh JW Jr, Karrer FM, Armany M, Miller C, Tallent MB, Esquivel CO, Gordon RD, Iwatsuki S, Starzl TE. (1987) Hepatic artery in liver transplantation. Transplant Proc, 19: 2406-2411.
109. Tzakis AG, Gordon RD, Shaw BW Jr, Iwatsuki S, Starzl TE. (1985) Clinical presentation of hepatic artery thrombosis after liver transplantation in the cyclosporine era. Transplantation, 40: 667-671.
110. Warner P, Fusai G, Glantzounis GK, Sabin CA, Rolando N, Patch D, Sharma D, Davidson BR, Rolles K, Burroughs AK. (2011) Risk factors associated with early hepatic artery thrombosis after orthotopic liver transplantation - univariable and multivariable analysis. Transpl Int, 24: 401-408.
111. Duffy JP, Hong JC, Farmer DG, Ghobrial RM, Yersiz H, Hiatt JR, Busuttil RW. (2009) Vascular complications of orthotopic liver transplantation: experience in more than 4,200 patients. J Am Coll Surg, 208: 896-905.
112. Scott-Conner CE, Hall TJ. (1992) Variant arterial anatomy in laparoscopic cholecystectomy. Am J Surg, 163: 590-592.
113. Weimann A, Meyer HJ, Mauz S, Ringe B, Jähne J, Pichlmayr R. (1991) Anatomic variations in the course of the left hepatic artery. A problem for systematic

lymphadenectomy in gastrectomy or proximal stomach resection before stomach tube formation. Chirurg, 62: 552-556.

114. Chen CY, Lee RC, Tseng HS, Chiang JH, Hwang JI, Teng MM. (1998) Normal and variant anatomy of hepatic arteries: angiographic experience. Chin Med J, 61: 17-23.

115. Daly JM, Kemeny N, Oderman P, Botet J. (1984) Long-term hepatic arterial infusion chemotherapy. Anatomic considerations, operative technique, and treatment morbidity. Arch Surg, 119: 936-941.

116. Kemeny MM, Hogan JM, Goldberg DA, Lieu C, Beatty JD, Kokal WA, Riihimaki DU, Terz JJ. (1986) Continuous hepatic artery infusion with an implantable pump: problems with hepatic artery anomalies. Surgery, 99: 501-504.

117. Xie YZ, Liu J, Chung GH, Kong X, Li XJ, Zhang LT, Ma ZB, Chai OH, Kim HT, Song CH. (2014) Visualization of the segment IV hepatic artery using 128-section MDCT angiography. Clin Radiol, 69: 965-973.

118. Vagefi PA, Parekh J, Ascher NL, Roberts JP, Freise CE. (2014) Ex vivo split-liver transplantation: the true right/left split. HPB (Oxford), 16: 267-274.

119. Colledan M, Segalin A, Andorno E, Corno V, Lucianetti A, Spada M, Gridelli B. (2000) Modified splitting technique for liver transplantation in adult-sized recipients. Technique and preliminary results. Acta Chir Belg, 100: 289-291.

120. Sommacale D, Farges O, Ettorre GM, Lebigot P, Sauvanet A, Marty J, Durand F, Belghiti J. (2000) In situ split liver transplantation for two adult recipients. Transplantation, 69: 1005-1007.

121. Kilic M, Seu P, Stribling RJ, Ghalib R, Goss JA. (2001) In situ splitting of the cadaveric liver for two adult recipients. Transplantation, 72: 1853-1858.

122. Humar A, Ramcharan T, Sielaff TD, Kandaswamy R, Gruessner RW, Lake JR, Payne WD. (2001) Split liver transplantation for two adult recipients: an initial experience. Am J Transplant, 1: 366-372.

123. Azoulay D, Castaing D, Adam R, Savier E, Delvart V, Karam V, Ming BY, Dannaoui M, Krissat J, Bismuth H. (2001) Split-liver transplantation for two adult recipients: feasibility and long-term outcomes. Ann Surg, 233: 565-574.

124. Winston CB, Lee NA, Jarnagin WR, Teitcher J, DeMatteo RP, Fong Y, Blumgart LH. (2007) CT angiography for delineation of celiac and superior mesenteric artery

variants in patients undergoing hepatobiliary and pancreatic surgery. Am J Roentgenol, 189: W13-9.

125. Stemmler BJ, Paulson EK, Thornton FJ, Winters SR, Nelson RC, Clary BM. (2004) Dual-phase 3D MDCT angiography for evaluation of the liver before hepatic resection. Am J Roentgenol, 183: 1551-1557.

126. Braun MA, Collins MB, Wright P. (1991) An aberrant right hepatic artery from the right renal artery: anatomical vignette. Cardiovasc Intervent Radiol, 14: 349-351.

127. Gordon DH, Martin EC, Kim YH, Kutcher R. (1978) Accessory blood supply to the liver from the dorsal pancreatic artery: an unusual anatomic variant. Cardiovasc Radiol, 1: 199-201.

128. Miyayama S, Yamashiro M, Hattori Y, Orito N, Matsui K, Tsuji K, Yoshida M, Matsui O. (2011) Angiographic evaluation of feeding arteries of hepatocellular carcinoma in the caudate lobe of the liver. Cardiovasc Intervent Radiol, 34: 1244-1253.

129. Kim HC, Chung JW, Jae HJ, Yoon JH, Lee JH, Kim YJ, Lee HS, Yoon CJ, Park JH. (2010) Caudate lobe hepatocellular carcinoma treated with selective chemoembolization. Radiology, 257: 278-287.

130. Rosero O, Nemeth K, Turoczi Z, Fulop A, Garbaisz D, Gyorffy A, Szuak A, Dorogi B, Kiss M, Nemeskeri A, Harsanyi L, Szijarto A. (2014) Collateral circulation of the rat lower limb and its significance in ischemia-reperfusion studies. Surg Today, 44(12): 2345-2353.

131. Kishi Y, Sugawara Y, Kaneko J, Akamatsu N, Imamura H, Asato H, Kokudo N, Makuuchi M. (2004) Hepatic arterial anatomy for right liver procurement from living donors. Liver Transpl, 10: 129-133.

132. Kishi Y, Imamura H, Sugawara Y, Sano K, Kaneko J, Kokudo N, Makuuchi M. (2010) Evaluation of donor vasculobiliary anatomic variations in liver graft procurements. Surgery, 147: 30-39.

133. Takatsuki M, Chiang YC, Lin TS, Wang CC, Concejero A, Lin CC, Huang TL, Cheng YF, Chen CL. (2006) Anatomical and technical aspects of hepatic artery reconstruction in living donor liver transplantation. Surgery, 140: 824-829.

134. Lee SS, Kim TK, Byun JH, Ha HK, Kim PN, Kim AY, Lee SG, Lee MG. (2003) Hepatic arteries in potential donors for living related liver transplantation: evaluation with multi-detector row CT angiography. Radiology, 227: 391-399.

135. Bogetti JD, Herts BR, Sands MJ, Carroll JF, Vogt DP, Henderson M. (2001) Accuracy and utility of 3-dimensional computed tomography in evaluating donors for adult living related liver transplants. Liver Transpl, 7: 687-692.
136. Coşkun M, Kayahan EM, Ozbek O, Cakir B, Dalgiç A, Haberal M. (2005) Imaging of hepatic arterial anatomy for depicting vascular variations in living related liver transplant donor candidates with multidetector computed tomography: comparison with conventional angiography. Transplant Proc, 37: 1070-1073.
137. Kamel IR, Kruskal JB, Pomfret EA, Keogan MT, Warmbrand G, Raptopoulos V. (2001) Impact of multidetector CT on donor selection and surgical planning before living adult right lobe liver transplantation. Am J Roentgenol, 176: 193-200.
138. De Cecco CN, Ferrari R, Rengo M, Paolantonio P, Vecchietti F, Laghi A. (2009) Anatomic variations of the hepatic arteries in 250 patients studied with 64-row CT angiography. Eur Radiol, 19: 2765-2770.
139. Koops A, Wojciechowski B, Broering DC, Adam G, Krupski-Berdien G. (2004) Anatomic variations of the hepatic arteries in 604 selective celiac and superior mesenteric angiographies. Surg Radiol Anat, 26: 239-244.
140. Covey AM, Brody LA, Maluccio MA, Getrajdman GI, Brown KT. (2002) Variant hepatic arterial anatomy revisited: digital subtraction angiography performed in 600 patients. Radiology, 224: 542-547.
141. Ugurel MS, Battal B, Bozlar U, Nural MS, Tasar M, Ors F, Saglam M, Karademir I. (2010) Anatomical variations of hepatic arterial system, coeliac trunk and renal arteries: an analysis with multidetector CT angiography. Br J Radiol, 83: 661-667.
142. Ferrari R, De Cecco CN, Iafrate F, Paolantonio P, Rengo M, Laghi A. (2007) Anatomical variations of the coeliac trunk and the mesenteric arteries evaluated with 64-row CT angiography. Radiol Med, 112: 988-998.
143. Rygaard H, Forrest M, Mygind T, Baden H. (1986) Anatomic variants of the hepatic arteries. Acta Radiol Diagn, 27: 425-427.
144. De Santis M, Ariosi P, Calò GF, Romagnoli R. (2000) Hepatic arterial vascular anatomy and its variants. Radiol Med, 100: 145-151.
145. López-Andújar R, Moya A, Montalvá E, Berenguer M, De Juan M, San Juan F, Pareja E, Vila JJ, Orbis F, Prieto M, Mir J. (2007) Lessons learned from anatomic variants of the hepatic artery in 1,081 transplanted livers. Liver Transpl, 13: 1401-1404.

146. Abdullah SS, Mabrut JY, Garbit V, De La Roche E, Olagne E, Rode A, Morin A, Berthezene Y, Baulieux J, Ducerf C. (2006) Anatomical variations of the hepatic artery: study of 932 cases in liver transplantation. Surg Radiol Anat, 28: 468-473.
147. Soin AS, Friend PJ, Rasmussen A, Saxena R, Tokat Y, Alexander GJ, Jamieson NV, Calne RY. (1996) Donor arterial variations in liver transplantation: management and outcome of 527 consecutive grafts. Br J Surg, 83: 637-641.
148. Kostelic JK, Piper JB, Leef JA, Lu CT, Rosenblum JD, Hackworth C, Kahn J, Thistlethwaite JR, Whitington PF. (1996) Angiographic selection criteria for living related liver transplant donors. Am J Roentgenol, 166: 1103-1108.
149. Pérez-Saborido B, Pacheco-Sánchez D, Barrera Rebollo A, Pinto Fuentes P, Asensio Díaz E, Labarga Rodriguez F, Sarmentero Prieto JC, Martínez Díez R, Rodríguez Vielba P, Gonzálo Martín M, Rodríguez López M, de Anta Román A. (2012) Incidence of hepatic artery variations in liver transplantation: does it really influence short- and long-term results? Transplant Proc, 44: 2606-2608.
150. Sakamoto Y, Takayama T, Nakatsuka T, Asato H, Sugawara Y, Sano K, Imamura H, Kawarasaki H, Makuuchi M. (2002) Advantage in using living donors with aberrant hepatic artery for partial liver graft arterialization. Transplantation, 74: 518-521.
151. Andraus W, Haddad LB, Ducatti L, Martino RB, Santos VR, D'Albuquerque LA. (2013) Artery reconstruction in liver transplantation: the best reconstruction of right hepatic artery variation. Arq Bras Cir Dig, 26: 62-65.
152. Walker TG. (2009) Mesenteric vasculature and collateral pathways. Semin Intervent Radiol, 26: 167-174.
153. Ishigami K, Yoshimitsu K, Irie H, Tajima T, Asayama Y, Hirakawa M, Honda H. (2006) Accessory left gastric artery from left hepatic artery shown on MDCT and conventional angiography: correlation with CT hepatic arteriography. Am J Roentgenol, 187: 1002-1009.
154. Murthy R, Nunez R, Szklaruk J, Erwin W, Madoff DC, Gupta S, Ahrar K, Wallace MJ, Cohen A, Coldwell DM, Kennedy AS, Hicks ME. (2005) Yttrium-90 microsphere therapy for hepatic malignancy: devices, indications, technical considerations, and potential complications. Radiographics, 25 Suppl 1: S41-55.

155. Johnson PB, Cawich SO, Shah S, Gardner MT, Roberts P, Stedman B, Pearce NW. (2013) Vascular supply to the liver: a report of a rare arterial variant. Case Rep Radiol, 2013: 969327.

156. Polguj M, Podgórski M, Hogendorf P, Topol M. (2014) Variations of the hepatobiliary vasculature including coexistence of accessory right hepatic artery with unusually arising double cystic arteries: case report and literature review. Anat Sci Int, 89: 195-198.

157. Chaib E, Ribeiro MA Jr, Saad WA, Gama-Rodrigues J. (2005) The main hepatic anatomic variations for the purpose of split-liver transplantation. Transplant Proc, 37: 1063-1066.

158. Fasel JH, Majno PE, Peitgen HO. (2010) Liver segments: an anatomical rationale for explaining inconsistencies with Couinaud's eight-segment concept. Surg Radiol Anat, 32: 761-765.

159. Wadhwa S, Khorwal G, Tigga SR. (2013) Retroportal proper hepatic artery with malrotated gut. Anat Sci Int, 88: 242-245.

160. Nakamura H, Uchida H, Kuroda C, Yoshioka H, Tokunaga K, Kitatani T, Sato T, Ohi H, Hori S. (1980) Accessory left gastric artery arising from left hepatic artery: angiographic study. Am J Roentgenol, 134: 529-532.

161. Kawarada Y, Isaji S, Taoka H, Tabata M, Das BC, Yokoi H. (1999) S4a + S5 with caudate lobe (S1) resection using the Taj Mahal liver parenchymal resection for carcinoma of the biliary tract. J Gastrointest Surg, 3: 369-373.

162. Chaib E, Ribeiro MA Jr, Silva Fde S, Saad WA, Cecconello I. (2008) Caudate lobectomy: tumor location, topographic classification, and technique using right- and left-sided approaches to the liver. Am J Surg, 196: 245-251.

163. Tanaka S, Shimada M, Shirabe K, Maehara S, Tsujita E, Taketomi A, Maehara Y. (2005) Surgical outcome of patients with hepatocellular carcinoma originating in the caudate lobe. Am J Surg, 190: 451-455.

164. Ikegami T, Ezaki T, Ishida T, Aimitsu S, Fujihara M, Mori M. (2004) Limited hepatic resection for hepatocellular carcinoma in the caudate lobe. World J Surg, 28: 697-701.

165. Kim HC, Chung JW, Jae HJ, Yoon JH, Lee JH, Kim YJ, Lee HS, Yoon CJ, Park JH. (2010) Caudate lobe hepatocellular carcinoma treated with selective chemoembolization. Radiology, 257: 278-287.
166. Shibata T, Maetani Y, Ametani F, Kubo T, Itoh K, Konishi J. (2002) Efficacy of nonsurgical treatments for hepatocellular carcinoma in the caudate lobe. Cardiovasc Intervent Radiol, 25: 186-192.
167. Terayama N, Miyayama S, Tatsu H, Yamamoto T, Toya D, Tanaka N, Mitsui T, Miura S, Fujisawa M, Kifune K, Matsui O, Takashima T. (1998) Subsegmental transcatheter arterial embolization for hepatocellular carcinoma in the caudate lobe. J Vasc Interv Radiol, 9: 501-508.
168. Yoon CJ, Chung JW, Cho BH, Jae HJ, Kang SG, Kim HC, Choi YH, Jeon UB, Park JH. (2008) Hepatocellular carcinoma in the caudate lobe of the liver: angiographic analysis of tumor-feeding arteries according to subsegmental location. J Vasc Interv Radiol, 19: 1543-1550.
169. Miyayama S, Yamashiro M, Yoshie Y, Nakashima Y, Ikeno H, Orito N, Yoshida M, Matsui O. (2010) Hepatocellular carcinoma in the caudate lobe of the liver: variations of its feeding branches on arteriography. Jpn J Radiol, 28: 555-562.
170. Tohma T, Cho A, Okazumi S, Makino H, Shuto K, Mochiduki R, Matsubara K, Gunji H, Ochiai T. (2005) Communicating arcade between the right and left hepatic arteries: evaluation with CT and angiography during temporary balloon occlusion of the right or left hepatic artery. Radiology, 237: 361-365.
171. Cho A, Gunji H, Koike N, Narumoto S, Asano T, Yamamoto H, Kainuma O, Ryu M, Mori C, Murakami G, Okazumi S, Ochiai T. (2007) Intersegmental arterial communication between the medial and left lateral segments of the liver. Dig Surg, 24: 328-330.
172. Gunji H, Cho A, Tohma T, Okazumi S, Makino H, Shuto K, Mochizuki R, Matsubara K, Hayano K, Mori C, Murakami G, Ochiai T. (2006) The blood supply of the hilar bile duct and its relationship to the communicating arcade located between the right and left hepatic arteries. Am J Surg, 192: 276-280.
173. Miyayama S, Matsui O, Yamashiro M, Ryu Y, Kaito K, Ozaki K, Takeda T, Yoneda N, Notsumata K, Toya D, Tanaka N, Mitsui T. (2007) Ultraselective transcatheter arterial chemoembolization with a 2-f tip microcatheter for small

hepatocellular carcinomas: relationship between local tumor recurrence and visualization of the portal vein with iodized oil. J Vasc Interv Radiol, 18: 365-376.
174. Shimada M, Matsumata T, Maeda T, Yanaga K, Taketomi A, Sugimachi K. (1994) Characteristics of hepatocellular carcinoma originating in the caudate lobe. Hepatology, 19: 911-915.
175. Vellar ID. (1999) The blood supply of the biliary ductal system and its relevance to vasculobiliary injuries following cholecystectomy. Aust N Z J Surg, 69: 816-820.
176. Marcos A, Killackey M, Orloff MS, Mieles L, Bozorgzadeh A, Tan HP. (2003) Hepatic arterial reconstruction in 95 adult right lobe living donor liver transplants: evolution of anastomotic technique. Liver Transpl, 9: 570-574.

12 Bibliography of the candidate's publications

Publications related to the subject

Nemeth K, Deshpande R, Mathe Z, Szuak A, Kiss M, Korom C, Nemeskeri A, Kobori L. (2015) Extrahepatic arteries of the human liver - anatomical variants and surgical relevancies. Transpl Int, 28(10): 1216-1226. IF: 2,599

Kiss M, Deshpande RR, Nemeskéri A, Nguyen TT, Kürti Z, Kovács S, Pápai Z, Németh K, Szuák A, Dudás I, Kóbori L. (2015) Optimal line of hepatotomy for left lateral living donor liver transplantation according to the anatomical variations of left hepatic duct system. Pediatr Transplant, 19(5): 510-516. IF: 1,441

Rosero O, Nemeth K, Turoczi Z, Fulop A, Garbaisz D, Gyorffy A, Szuak A, Dorogi B, Kiss M, Nemeskeri A, Harsanyi L, Szijarto A. (2014) Collateral circulation of the rat lower limb and its significance in ischemia-reperfusion studies. Surg Today, 44(12): 2345-2353. IF: 1,526

Rosero O, Németh K, Turóczy Zs, Fülöp A, Garbaisz D, Kiss M, Nemeskéri A, Szijártó A. (2013) Collateral circulation of the rat lower limb and its significance in ischemia-reperfusion studies
European Surgical Research, 50:(Suppl 1) 26. - Abstract

Fülöp András, Rosero Olivér, Turóczi Zsolt, Garbaisz Dávid, Dorogi Bence, Németh Károly, Nemeskéri Ágnes, Harsányi László, Szijártó Attila. (2013) A collateralis hálózat szerepe alsó végtagi ischaemiás modellek kialakítása során. Magyar Sebészet, 66: 84. - Abstract

Other publications

Tarnoki DL, Tarnoki AD, Nemeth K, Bata P, Berczi V, Karlinger K. (2013) Partial absence of superior vena cava in an adult patient - Case report and literature review. Herz, 38(7): 785-789. IF: 0,912

13 Acknowledgements

First of all I'd like to express my deepest gratitude to **Ágnes Nemeskéri MD, Ph.D.**, Head of the Clinical Anatomy Research Laboratory and Associate Professor at the Department of Human Morphology and Developmental Biology, who, by her altruistic generosity and helpfulness, has guided our research throughout the years. This work would never have come to existence without her continuous professional assistance and foresight. **Mátyás Kiss MD.**, for the innovative idea of the resin mixture, which allowed us provide new, useful results to the field of clinical anatomy and transplantation. **András Szuák MD.**, Assistant Lecturer at the Department of Human Morphology and Developmental Biology I could always rely on during all phases of our study. **István Apáthy Foundation**, which founded our endeavour to further discover the colorful anatomy of the human liver and helped us share our experiences with national and international audiences. **Zoltán Máthé MD, Ph.D.**, Director of the Department of Transplantation and Surgery, **Prof. László Kóbori MD, Ph.D.**, Deputy Director of the Department of Transplantation and Surgery and **Rahul Deshpande MD, Ph.D.** from the Department of Hepatobiliary Surgery, Manchester Royal Infirmary, who have provided the clinical data to our work, thus upholding the importance of anatomical research. **Csaba Korom MD.** radiologist, for his invaluable radiological images, reconstructions and video materials. **Prof. Kinga Karlinger MD, Ph.D.**, for allowing us to use the CT scanners. Last, but not least, I'd like to thank **Prof. Ágoston Szél MD, Ph.D., Dr. habil., D.Sc.**, Rector of Semmelweis University and Head of the Department of Human Morphology and Developmental Biology, for his extensive support and encouragement.