CHAPTER V

EXTRAHILAR LEFT PORTAL VEIN RE-VASCULARIZATION

In searching for alternative techniques for liver transplantation, we developed an *original extrahilar approach for portal re-vascularization of partial liver grafts*. Although the procedure was designed primarily for graft re-vascularization at the time of transplantation, it was first used for successful relief of extrahepatic portal hypertension related to portal vein thrombosis [81]. Later, it was used in selected cases, i.e. when a very large left lobe graft had to be transplanted in small children with severe portal vein hypoplasia.

V- 1. BACKGROUND

V- 1. 1- Thrombosis of the portal trunk is not unfrequent after liver transplantation [11,116,252,360,362,366]. It has been reported either with full size (0 to 10%) [27,181,194] or reduced grafts (0.8 to 11%) [11,27]; it is much more frequent in small children with portal vein hypoplasia [11,43,194]. Like other authors, we have stressed the importance of positioning the portal vein when a cut-down liver graft is implanted [104,207,285]. The natural position of those grafts in the right liver fossa displaces the portal stump away from the axis of the recipient portal vein. End-to-end anastomosis of the donor and the recipient portal veins entails a double bending of the portal trunk with a smooth curve of the recipient portal vein to the right and a sharper curve of the graft portal vein to the left at the entrance in the parenchyma (Figures 23, 55, 56, 57). Inappropriate positioning of the portal vein may have been responsible for the portal vein thromboses observed in our experience or in other series.

Another factor must be taken into account: the portal flow is directly and negatively influenced by the intra-abdominal pressure. Payen and Houssin observed a 26% decrease in portal flow after closure of the abdomen [263]. This is probably even more important when the graft is large or slightly oversized. In this latter situation, the problem is more critical because the vein, running between the graft and the posterior abdominal wall (Figures 29, 30), can be occluded by compression when the abdomen is closed.

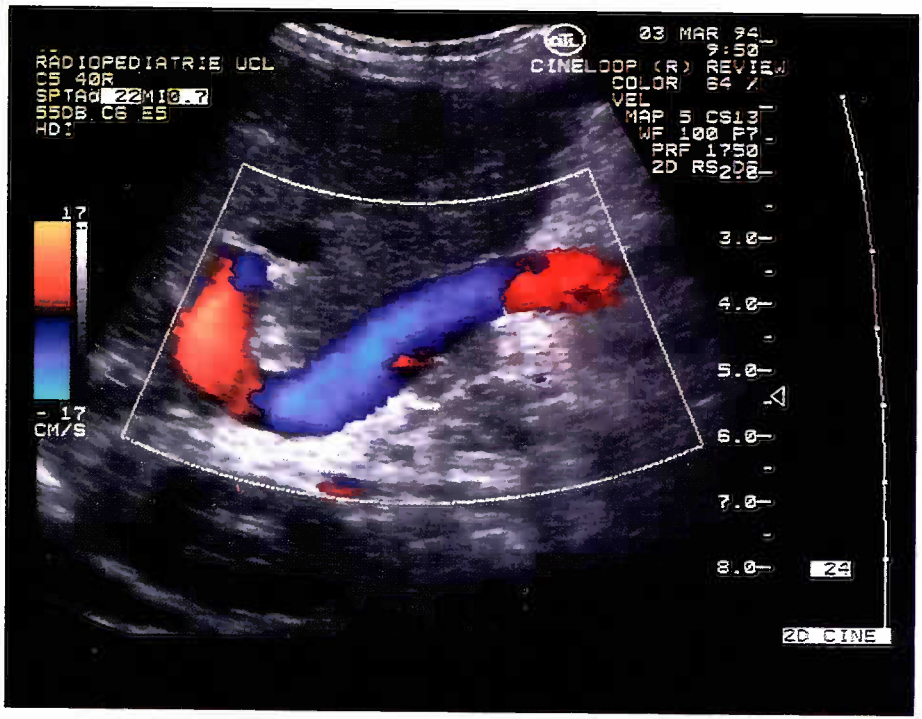

Figure 55: *Doppler Ultrasound of the portal vein in a segmental graft: the vein runs harmoniously below the graft with a right sided J shape before entering the parenchyma.*

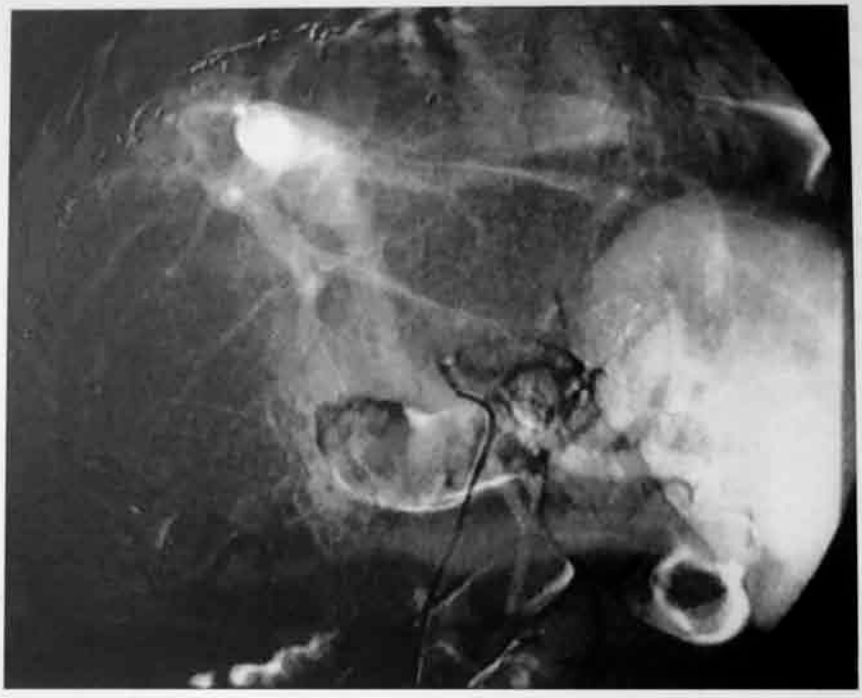

Figure 56: *Portal vein opacification (digitalised angiography): the donor portal trunk runs posteriorly until it reaches the porta hepatis which is right sided (see figure 57).*

Figure 57: *Schematic representation of figure 56. (S: splenic vein; T: portal trunk; R: Rex recessus; II and III: segment II and III portal veins)*

V- 1. 2- The formation of new collateral venous pathways restoring some portal flow to the liver has been described in patients presenting with a late thrombosis of the portal vein after transplantation [136,387,397]. No data concerning intrahepatic portal hemodynamics after PVT are however available. In our experience, in all cases except one (presenting with extensive venous thrombosis involving caval and portal systems), *the intrahepatic portal branches remained free of thrombus and portal flow was maintained probably by partial rerouting of arterial hepatic inflow*, and subsequent redistribution with inversed portal flow in several branches as suggested also by Burke in a case report [53] (Figures 58, 59). This particular aspect allows the rerouting of the portal flow to the intrahepatic portal system. Detailed Doppler investigation should be performed in all post-transplant PVT cases, including the intrahepatic branches of the portal vein, before considering any surgical decompression.

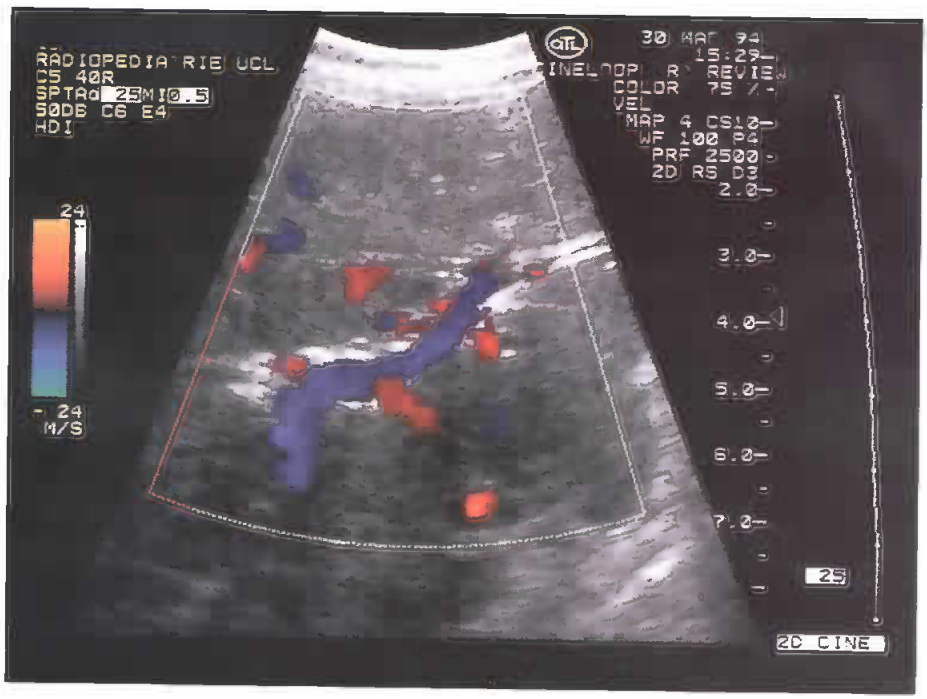

***Figure 58**: Doppler ultrasound in a child transplanted with a full-size liver graft and presenting with portal trunk thrombosis. The intrahepatic portal system is not thrombosed. The blue stream corresponds to the portal flow (from right to left: from the Rex recessus to the right portal vein). In the left portal branches there is an inversed flow which enters the right liver.*

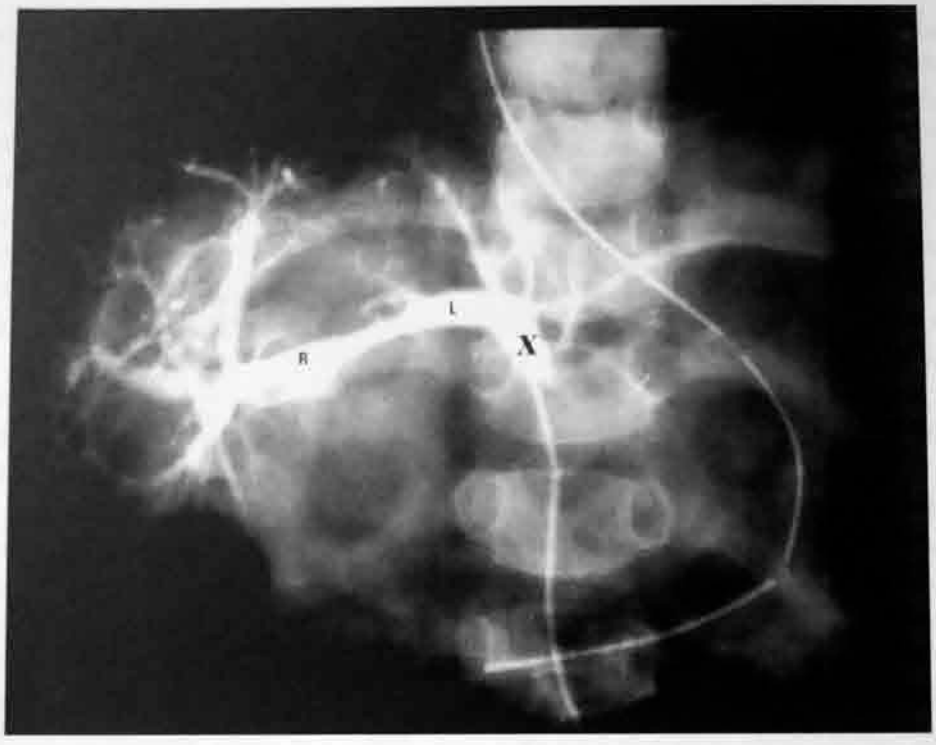

***Figure 59**: Peroperative opacification of the intrahepatic portal vein in a patient with a thrombosed portal vein: the intrahepatic portal system is patent. (R and L: right and left portal veins; X: Rex recessus).*

V- 1. 3- The possible ischemic damage of the graft, due to low portal flow, has to be taken into account when planning the portal reconstruction *in a small child receiving a large segmental graft. In such a situation, a primary re-vascularization from the superior mesenteric vein to the intrahepatic left portal vein could be a good alternative* (see below).

V- 2. EXTRAHILAR LEFT PORTAL VEIN REVASCULARIZATION IN EXTRAHEPATIC PORTAL VEIN THROMBOSIS

V- 2. 1- Advantages

Although portal vein thrombosis is not a common post-transplant complication, it is reported in several series. Few data are however available in the literature concerning its treatment.

Thrombosis of the portal vein has been reported as a cause of graft failure [33,34,58,136,194,259,322]. Splenectomy, sclerotherapy and portosystemic shunts have been performed to treat chronic secondary portal hypertension in liver transplanted

patients [212,219,259,271,282,342,387]. Re-operation for portal thrombectomy is feasable when the thrombosis is diagnosed very early [27,181]. But when the diagnosis is delayed, thrombectomy is unsuccessful, furthermore is contra-indicated by the risk of bleeding because of cavernomatous transformation of the liver hilum. It also requires a direct and anterior access to the portal trunk which is very difficult because the biliary reconstruction is usually performed in children using a bilio-jejunal anastomosis, with the Roux-en-Y loop lying in front of the portal trunk. On the contrary, **the extrahilar approach in the umbilical fissure allows dissection in a territory free of portal hypertension or cavernomatous transformation, and at some distance of the main arterial and biliary graft structures** [81].

Bypassing the liver spontaneously or by any kind of surgical shunt interferes with the liver physiology and is potentially harmful in the long term [6,110,128,130,149,179,271,275,276,291,348,369,376,378]. The extrahilar approach to the left portal vein **allows the portal blood to be rediverted freely to the liver** and is more physiological than portal blood diversion away from the liver [317,321]. It cures the portal hypertension and its pathological consequences, but also re-vascularizes the liver [20,21,24,97,111,115,117,124,126,128,171,261,348,352,377].

Because of its cost and its risk, re-transplantation of the liver should be strictly limited to cases where the portal thrombosis is associated with other complications which, in their own right, justify a re-transplantation.

V- 2. 2- Indications

The procedure is indicated in cases of extrahepatic portal hypertension after liver transplantation, when:

1- the intrahepatic portal branches (or only the left portal vein) remain patent,

2- the liver histology is normal (absence of other cause of portal hypertension),

3- complications of portal hypertension occurred, or the patient is at high risk of bleeding.

V- 2. 3- Technique [81]

After portal hypertension has been confirmed by direct pressure gradient measurement (atrium/mesenteric vein), the umbilical vein is recanalized, allowing confirmation of a normal pressure gradient between intrahepatic portal branches and the right atrium (to rule out an intrahepatic block). A venography through the recanalized umbilical vein is helpful to confirm patency and anatomy of the left portal vein (Figure 59). The umbilical remnant is then further dissected, as well as the distal part of the left portal vein (Rex recessus) and its branches for segments III and IV. Careful dissection provides excellent exposure of the vein on a 3-4 cm length without any damage to biliary or arterial branches (Figure 60). A small vascular clamp, laterally positioned, provides appropriate clamping of the vein and all its branches. After section of the umbilical remnant at its junction with the left portal vein, this terminal

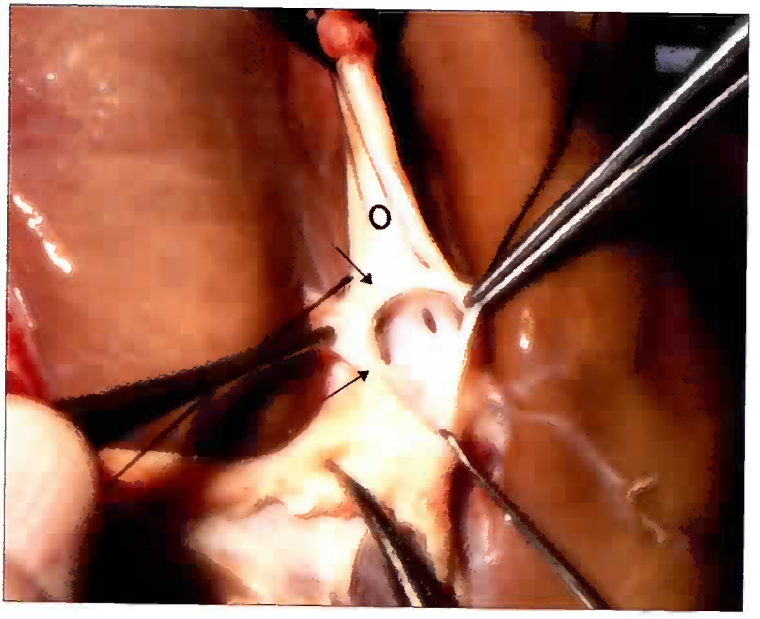

***Figure 60**: Ex-situ liver graft: dissection of the anterior part of the Rex recessus (arrows), which is open on its ventral aspect. The ostia of portal branches are easily identified (O: umbilical remnant)*

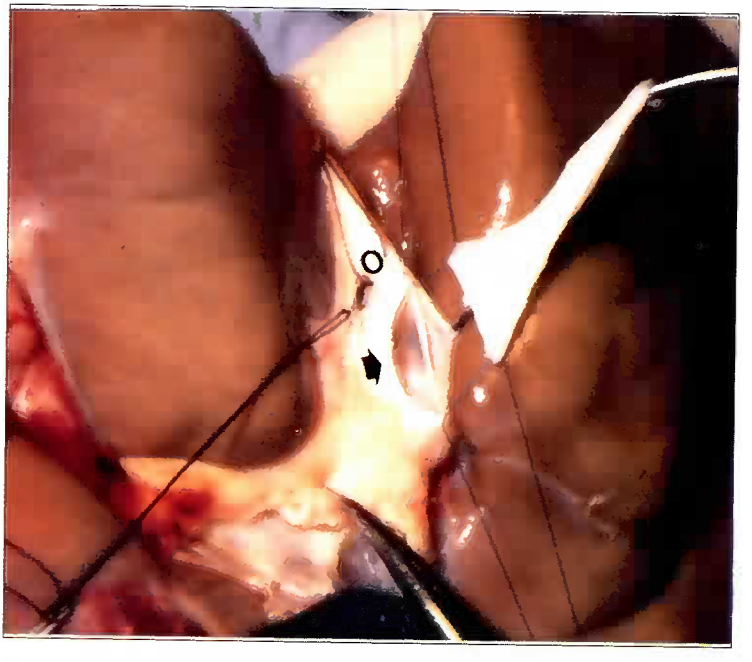

***Figure 61**: Ex-situ liver graft: view before anastomosing a venous homograft to the Rex recessus (arrows). (O: umbilical remnant)*

venotomy is longitudinally prolonged on its ventral aspect. A venous graft (own jugular vein or venous homograft) is anastomosed end-to-side to this termino-lateral opening (Figure 61). The venous graft is brought in front of the duodenum and through the transverse mesocolon to reach the superior mesenteric vein, to which it is connected in an end-to-side fashion (Figure 62).

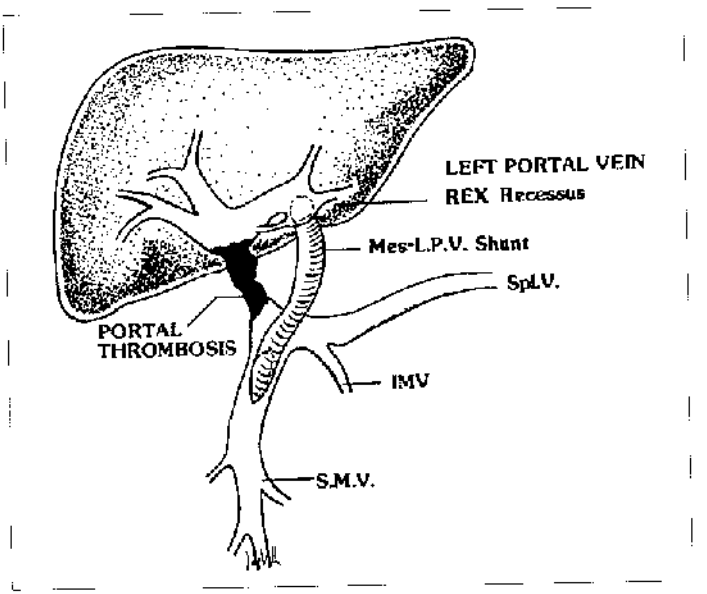

Figure 62: Schematic and general aspect of the mesenterico-Rex bypass. (Spl V: splenic vein, SMV: superior mesenteric vein, IMV: inferior mesenteric vein).

V- 2. 4- Results

Out of 382 liver transplants performed using conventional portal re-vascularization, ***20 portal vein obstructive type complications*** were diagnosed.

In two cases, an anastomotic stenosis was diagnosed in the late follow-up: in both cases, splenomegaly and hypersplenism were present. Treatment consisted in balloon transluminal angioplasty, either percutaneously [57,281] or during laparotomy after venotomy of the inferior mesenteric vein [10].

Four patients presented with a thrombosis of the portal trunk related to another cause. In one case, it was caused by local sepsis and terminal liver failure. In another one, it was diagnosed at re-transplantation for secondary biliary cirrhosis (graft hepatic artery thrombosis related biliary strictures). Two patients re-transplanted for primary non function, were found to have an occlusion of both the artery and the portal vein of their reduced graft, which seemed to have occurred in the terminal phase.

In the other 14 cases, obstruction of the portal vein occurred as a primary complication. Anatomical or technical factors that could have precipitated this problem in some way are detailed in table 27:

PEDIATRIC ORTHOTOPIC LIVER TRANSPLANTATION: UCL EXPERIENCE **TABLE 27**

PORTAL VEIN THROMBOSIS **AFTER CLASSICAL PORTAL VEIN RECONSTRUCTION**

N = 14 out of 383 liver transplantations

case	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	*11*	*12*	*13*	*14*
YEAR	86	86	87	87	88	89	90	91	91	91	92	92	92	93
OCCURENCE														
first 4 weeks			•	•						•		•	•	•
> 1 month	•	•			•	•	•	•	•		•			
Preservation solution														
Euro-Collins	•	•	•	•	•									
UW solution						•		•	•	•	•	•	•	•
HTK							•							
Donor age < 1 year					•									
Recipient Weight < 8 kg		•			•							•	•	
D/R WEIGHT > 4							•	•			•	•	•	
Full-size + D/R>1.5	•	•							•					
TYPE of GRAFT														
FULL-SIZE	•	•		•	•	•			•	•				
CUT-DOWN			•				•	•			•	•	•	•
Type of anastomosis														
Porto-portal	•		•			•	•	•	•		•	•	•	•
Porto-Confluence		•			•									
Porto-Mesenteric				•						•				
Venous homograft interposed				•						•				
Technical problem			Twist							Twist			Twist	
Anatomical factor														
Portal vein hypoplasia		•		•	•	•	•			•		•		
Preduodenal vein						•								
Situs Inversus													•	
Re-transplantation											•			
Portal vein phlebitis								•						

1) *In six cases, PVT was diagnosed in the early post-operative course* (first four weeks). A re-operation was performed, consisting in four redo and two re-vascularizations from the superior mesenteric vein. Four of those grafts were lost, due to death of the patient in two cases.

2) *Two cases were diagnosed during the fifth week*: the first presented with refractory ascites and was the first to benefit from the mesenterico-left intrahepatic portal vein bypass (MRS) [81]. In the second patient, PVT was diagnosed at one month follow-up and he was re-operated on day 35: the left portal vein was re-vascularized using a conventional approach, but he was re-transplanted two months later because of persistence of severe ischemic sequellae and cholangitis related to intrahepatic biliary sludge.

3) *In the six remaining cases, diagnosis was made in the late follow-up* and surgical correction performed 6, 24, 34 and 38 months, or 5 and 8 years after transplantation. All presented with splenomegaly and oesophageal varices. Five patients had proven gastrointestinal hemorrhage and/or chronic anemia. One other patient was treated conservatively during four years because no complications were observed. With time, high risk of hemorrhage developed (oesophageal varices grade 4 with red spots, gastric varices) and he was then operated before complications occurred. In all, normal liver histology was confirmed by pre-operative biopsy and patency of intrahepatic portal branches was proved by Doppler ultrasound. *For these last six patients, a mesenterico-Rex bypass* was also performed using the previously described extrahilar approach.

Overall, seven patients benefited thus from a mesenterico-Rex bypass: a venous homograft from an unrelated ABO identical cadaveric donor was used for bypassing in three patients, and the patients' own left jugular vein for the other four. In one case, extrinsic compression caused an absence of flow and was diagnosed immediately after operation: it was successfully corrected at re-laparotomy. *Sclerosis of the venous homograft* led to obstruction of the bypass in two other cases, in which a redo using a jugular autograft was successful (in one case, percutaneous transhepatic balloon dilatation for stenosis was followed by delayed thrombosis) (Figures 63, 64). An immunological process could have caused these thromboses [370]; this hypothesis is supported by the following facts. All venous autografts and only one homograft (implanted during the 5th post-operative week) were successful; all are currently patent. On the contrary, the two homografts that were implanted in the late follow-up (6 and 33 months after the transplantation) were unsuccessful. The major difference between early or late implantation of a vascular homograft, is the amount of immunosuppression. When unmatched homografts were used during the transplantation itself for other types of reconstruction, they achieved usually good results: in our experience, we observed only one stenosis out of 21 mismatched homografts implanted during transplantation. Heffron et al. reported also a 100% patency in cryopreserved allografts when used at the time of graft implantation [135]. The throm-

bosis of two MRS was thus probably not related to the technique itself but was likely caused by the use of mismatched homografts in patients receiving a low immunotherapy.

The effectiveness of this innovative decompressive technique was controlled peroperatively and confirmed by clinical evolution. During surgery, there was a *drop in the mesenterico-atrial pressure gradient* (median before and after bypass: 14 and 7 mm Hg, respectively); in four cases, peroperative angiography and/or flow measurement showed a good flow through the bypass. One patient had a ^{99m}TC-sulfur colloidal scintigraphy in order to evaluate liver perfusion two days before and one week after surgery: this showed an increase in hepatic blood flow and liver volume, and a decrease in splenic volume (Figure 65) (personal communication: Dr S Pauwels, UCL).

All seven patients who benefited from a mesenterico-Rex secondary portal re-vascularization are alive and well with their original graft. In five patients, the follow-up is currently longer than three years and *permanent relief of clinical symptoms* (disappearance of splenomegaly and variceal or gastrointestinal bleeding) was correlated with bypass patency at Doppler ultrasound (Figure 66). Two other cases are clinically well and their shunt is patent, two and three months after operation.

V- 3. PRIMARY PORTAL RE-VASCULARIZATION of LIVER GRAFTS THROUGH the EXTRAHILAR APPROACH of THE LEFT PORTAL VEIN

V- 3. 1- Technique

We recommend to plan the bypass before the liver graft implantation, selecting cases where it seems appropriate. This can be decided by comparison of the respective volumes of the abdomen of the recipient and the final volume of the graft after reduction. Transplant surgeons know that making that large grafts fit correctly in small abdomens can be a challenge; appropriate positioning of the graft and the portal vein is essential to prevent compression. When comparing respective volumes, the surgeon must take into account the increase in volume at reperfusion of the graft. From our experience, we would recommend to consider this optional technique if the donor/recipient weight ratio is greater than 10. We also think that in such cases, re-vascularization from the superior mesenteric vein can help to avoid portal vein compression.

Performing the implantation of a venous graft on the Rex recessus is very easy when done ex-situ during the back-table work. The first step consists in removing the portal trunk up to its bifurcation; it can be used as a venous graft for bypassing. After dissection of the anterior part of the Rex recessus, the distal part of the venous homograft is anastomosed end-to-side on the recessus; its proximal part is anastomosed end-to-side to the superior mesenteric vein when the graft is in situ. Implantation of another segment of venous homograft end-to-side on the superior mesenteric vein, before hepatectomy of the recipient, helps to perform portal re-vascularization more quickly and easily.

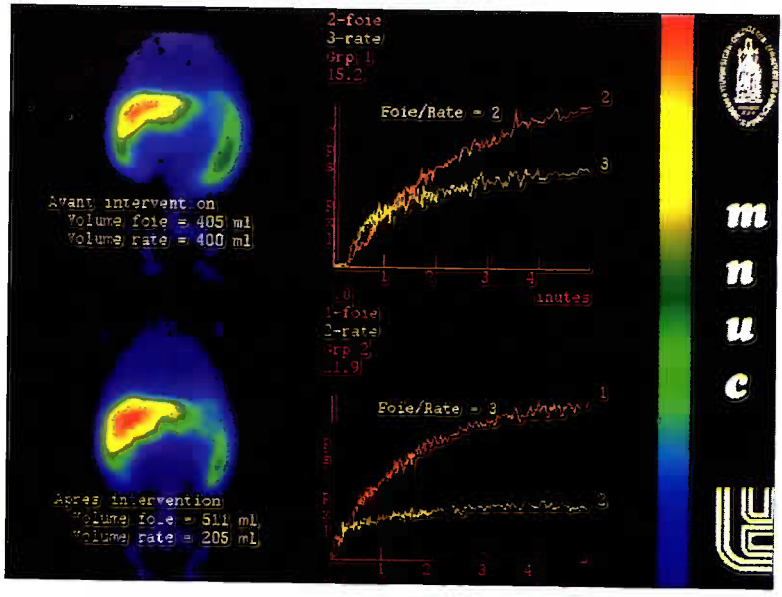

***Figure 65**: Hepatic scintigraphy (99 mTC sulfur colloidal) performed 2 days before and 1 week after mesenterico-Rex bypass for extrahepatic portal hypertension. It showed an increase in hepatic volume and in the hepatic/splenic blood flow ratio, as well as a decrease in the volume of the spleen.*

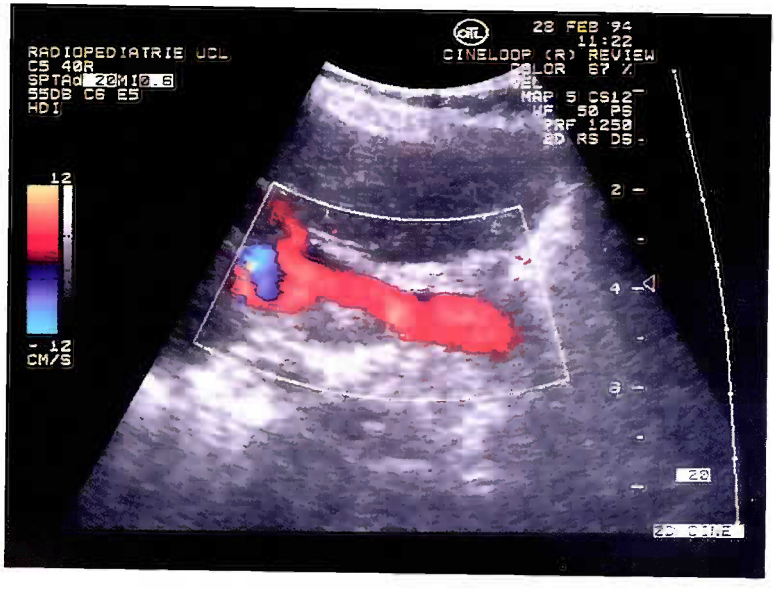

***Figure 66**: Doppler ultrasound of a mesenterico-Rex bypass entering the Rex recessus in a more direct manner than via classic revascularization (see figure 55): the portal flow is normal and well diverted into the intrahepatic venous collaterals.*

V- 3. 2- Results

In six patients receiving their first graft for biliary atresia cirrhosis, the portal re-vascularization was primarily performed using a bypass between Rex recessus and superior mesenteric vein. Out of these six cases, three were hospital-bound at the time of transplantation, and five were less than 15 months old and weighed less than 8 kg . The Dw/Rw was greater than 8.5 in five patients (all donors weighed more than 70 kg), and all received a segmental graft (segments II+III). Graft artery was implanted onto the aorta in all. A prosthetic (temporary) closure of the abdomen was needed in three of them. In one patient, thrombosis of this bypass was diagnosed three years after transplantation. He presented two months after diagnosis with intractable and massive intestinal bleeding. A new mesenterico-Rex bypass was successfully performed, using his own jugular vein.

V- 4. CONCLUSION

V- 4. 1- From this experience, we can conclude that portal vein thrombosis is well tolerated when occurring lately. This contrasts with the progressive deterioration of other cases in which the diagnosis is made very early after transplantation, or when occlusion of the portal vein is associated with another graft pathology [136,385]. When an histologically normal liver is associated with symptomatic chronic extrahepatic portal hypertension, the extrahilar mesenterico-left intrahepatic portal vein bypass seems to be a good alternative to the conventional porto-systemic shunt or to long-term sclerotherapy (Figures 67, 68). We strongly recommend the use of the patients' own jugular vein as autograft for the cases treated late after transplantation.

V- 4. 2- Using large donors for small children is technically more demanding. The use of the SMV-Rex bypass can be helpful for successful portal re-vascularization when the portal vein is hypoplastic and/or when a very large graft must be implanted in a small abdomen, avoiding compression of the vein and a subsequent decrease in portal flow and thrombosis.

V- 4. 3- We recommend transplant surgeons to *add this innovative approach* to the conventional technical armamentarium, being aware that its use is to be restricted to selected situations.

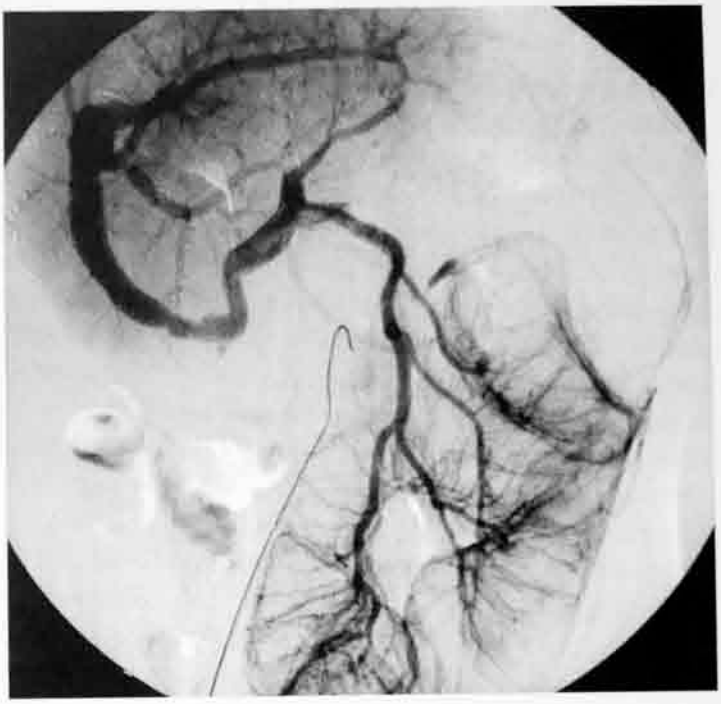

***Figure 67**: Opacification of the inferior mesenteric vein and the portal system in a child 2 days after a mesenterico-Rex bypass, showing a reversed flow in the superior mesenteric vein until it reaches the bypass and correct perfusion of the liver through the Rex recessus (see corresponding schematic view on figure 68).*

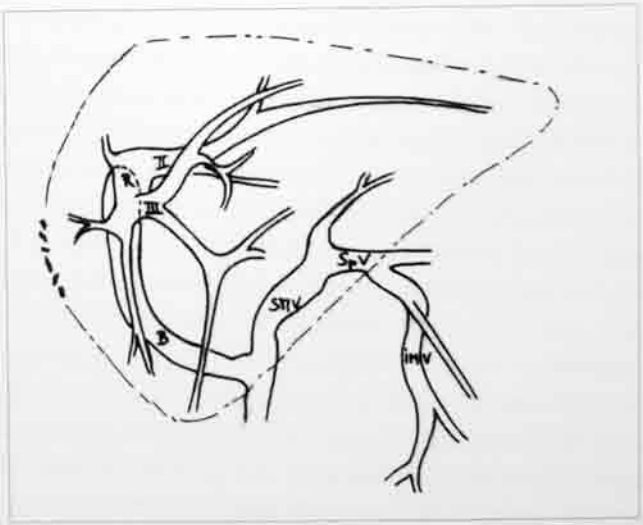

***Figure 68**: Schematic view of the portography described in figure 67 (IMV and SMV: inferior and superior mesenteric veins; SpV: splenic vein; B: jugular venous bypass; R: Rex recessus; II and III: segment II and III portal veins).*