Morphometric and Simulation Analyses of Right Hepatic Vein Reconstruction in Adult Living Donor Liver Transplantation Using Right Lobe Grafts

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The incidence of clinically significant right hepatic vein (RHV) stenosis after adult living donor liver transplantation has been higher than expected. In this study, an assessment of the risk factors for the development of RHV stenosis in this context was undertaken. Hepatic anatomy, surgical techniques, and the incidence of RHV stenosis 1 year after transplantation were evaluated retrospectively in 225 recipients of right lobe grafts. These patients underwent independent RHV reconstruction, which was facilitated by the application of computed tomography morphometry and computational simulation analyses. Three types of preparation of the orifice of the graft RHV and 7 types of preparation for venoplasty of the recipient RHV were used. The frequency of high, middle, and low sites of RHV insertion into the inferior vena cava (IVC) was 56.0%, 36.4%, and 7.6%, respectively, for donors, and 26.7%, 58.7%, and 14.7%, respectively, for recipients. Nine patients (4%) developed RHV stenosis of early onset that required stent insertion during the first 2 postoperative weeks; in 12 patients (5.3%), RHV stenosis of delayed onset occurred. Inappropriate matching of RHV sites of insertion correlated with the incidence of stenosis of early onset ($P = 0.039$). Technical refinements to avoid adverse consequences of inappropriate ventrodorsal matching of RHV sites of insertion include making the recipient RHV orifice wide and enlarging the recipient IVC by a customized incision and patch venoplasty after anatomical assessment of the RHV and IVC of the graft and recipient.


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A graft of the right lobe (RL) of the liver allows satisfactory matching of the sizes of the graft and the recipient liver. Such partial liver grafts are used most commonly in adult living donor liver transplantation (LDLT). Successful implantation of an RL graft requires effective reconstruction of sufficiently large outflow veins, including the right hepatic vein (RHV), the middle hepatic vein (MHV), and the inferior RHV. Reconstruction of the RHV is an important procedure during RL graft implantation with respect to both surgical technique and recovery of graft function. The surgical techniques that have been used for RHV reconstruction are direct anastomosis, incision/excision venoplasty, and patch venoplasty.1-11 Although these techniques have been described in detail, relatively little is known about the outcomes for each of

Abbreviations: 3-DR, 3-dimensional reconstruction; CSA, cross-sectional area; CT, computed tomography; DICOM, Digital Imaging and Communication in Medicine; IVC, inferior vena cava; LDLT, living donor liver transplantation; MELD, Model for End-Stage Liver Disease; MHV, middle hepatic vein; RHV, right hepatic vein; RL, right lobe; SD, standard deviation.

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On the basis of our experience with more than 1000 LDLT procedures using RL grafts, we recognize that the incidence of clinically significant RHV stenosis has been higher than expected. However, there have been few studies of the risk factors for the development of RHV stenosis after LDLT. We have applied 3-dimensional reconstruction (3-DR) morphometric analysis of computed tomography (CT) images and computational simulation modeling to assess risk factors for the occurrence of RHV stenosis after LDLT to facilitate the development of technical refinements aimed at preventing or minimizing this problem.

**PATIENTS AND METHODS**

**Patient Selection**

Between May 2007 and April 2008, 280 LDLT procedures were performed in adult patients at the Asan Medical Center. 245 (87.5%) were RL grafts, which included extended RL and right posterior segment grafts. To focus on the assessment of the patency of RHV reconstruction alone, we excluded the following patients: 3 who died within 2 weeks of LDLT (2 from overwhelming sepsis and 1 from encephalopathy), 14 with combined reconstruction of the RHV and MHV, and 3 who underwent a salvage LDLT procedure after a major hepatic resection. The remaining 225 patients were included in this study; the clinicopathological profiles of these patients are summarized in Table 1.

**Models That Simulate RHV Reconstruction**

To investigate the underlying mechanisms of RHV stenosis after LDLT, simulation models that incorporated hemodynamic principles were constructed. To assess the effectiveness of vascular reconstruction, a simulation model should include essential information about the inflow vessel (the size of the intrahepatic RHV), the outflow vessel [the size of the retrohepatic inferior vena cava (IVC)], and their connection (the size and shape of the RHV anastomosis).

For 3-DR morphometric analysis, stereoscopic images of RHV anastomoses were visualized with commercial 3-DR software (Lucion, Infinitt Co., Seoul, 640 HWANG ET AL. LIVER TRANSPLANTATION.DOI 10.1002/lt. Published on behalf of the American Association for the Study of Liver Diseases
the IVC and the mathematical constant verse diameters of the RHV at its site of insertion into the product of multiplying the longitudinal and trans-sectional area (CSA) was considered to be ovoid. The gradient across RHV anastomoses.

The Cavalieri principle was applied to calculate the CSA: the product of multiplying the longitudinal and transverse diameters of the RHV at its site of insertion into the IVC and the mathematical constant \( \pi \) was calculated and divided by 4. The severity of RHV stenosis was expressed as the unoccluded anastomotic CSA as a proportion of the original CSA on a donor CT scan before transplantation (eg, RHV stenosis equivalent to 33% occlusion of the original CSA). The effective diameter of an RHV anastomosis was expressed as a fraction of the original diameter of the RHV after the conversion of the ovoid CSA to a circular CSA (eg, an effective diameter fraction of 1/1.41 for 33% occlusion of the original CSA). The baseline RHV CSA was obtained from the donor CT scan before transplantation, and the recipient RHV CSA was determined from recipient CT scans after transplantation. The outflow blood volume across the RHV anastomosis was calculated by the multiplication of the RHV CSA by the mean RHV outflow velocity.

Single-Blind Acquisition of Imaging Data to Mimic a Prospective Study

Although this study was designed retrospectively, we tried to mimic a prospective study design, as far as possible, by adopting a single-blind approach: there was complete separation of the processes for acquiring images and the assessments of clinical outcomes after transplantation. CT images of both recipients and donors before transplantation were converted into Digital Imaging and Communication in Medicine (DICOM) format, in which patient information was removed and identification numbers were allocated after randomization. Similar blinding procedures were adopted for recipient CT images obtained 1 week, 1 month, and 1 year after transplantation. Data in DICOM form were converted to various sectional and 3-DR CT images with commercial software.

Determination of the Relative Site of Insertion of the RHV into the IVC

Our previous experience had suggested that the development of RHV stenosis may be related to the extent to which the site of insertion of the graft RHV into the recipient IVC is not optimal. Accordingly, the concept of the relative ventrodorsal position of the insertion of the graft RHV into the recipient IVC was introduced.

CT images before transplantation were used to determine the relative location of the RHV. To determine the site of insertion of the RHV into the donor IVC, its ventrodorsal diameter was divided into 3 equal parts: high, middle, and low. The site of insertion of the RHV was considered to be the geometric center of the RHV CSA. Thus, RL grafts had 3 levels of insertion of the RHV: high, middle, and low (Fig. 1).

In contrast, examination of the CT images of recipients obtained before transplantation showed that the ventrodorsal diameter of the recipient IVC varied appreciably in different patients. In addition, an apparently small IVC, buried within a shrunken hepatic parenchyma and/or an enlarged caudate lobe, usually became noticeably larger after its isolation during LDLT surgery. The relative position of the recipient RHV was defined arbitrarily from CT images before transplantation. If the recipient liver did not exhibit definite shrinkage of the parenchyma and the diameter of the retrohepatic IVC at the level of insertion of the donor RHV was equal to or greater than that of the infrarenal IVC (ie, normal or mildly shrunken IVC), insertions of the recipient RHV were divided into the same 3 categories as those for the donors. If the diameter of the IVC was greater than 1 cm but less than that of the infrarenal IVC (ie, moderately shrunken IVC), the same 3 categories as those for the donors were used because the IVC would become noticeably larger after its isolation. If, however, the IVC diameter was less than 1 cm (ie, severely shrunken IVC), the upper and lower halves were divided as middle and low levels, respectively, because division into 3 categories was often not
feasible on account of poor visualization of the intra-hepatic RHV on CT scans before transplantation and the size of the IVC being insufficiently large even after meticulous dissection (Fig. 1). As with donor grafts, the relative sites of insertion of the RHV into the recipient IVC were also categorized as high, middle, and low. Morphological configurations of the various combinations of sites of insertion of the graft and recipient RHV are shown in Fig. 2.

Selection of the Technique for RHV Reconstruction

Various techniques for RHV reconstruction have been applied at our institution; during this study, 2 were used most commonly (Fig. 3). Four surgeons, who had experience with more than 200 RHV reconstructions before this study, undertook the RHV reconstructions. Accordingly, the influence of variations in technical dexterity was not taken into account. To prepare the orifice of the graft RHV, 3 techniques (types 1-3) were used, and to prepare the orifices of the recipient RHV and IVC, 7 techniques (types A-G) were used. The method of reconstruction used for each patient was expressed as a combination of the types of preparation for the graft and recipient. Deep side clamping of the IVC was undertaken in all recipients for RHV reconstruction; none of the patients in this series underwent IVC cross-clamping.

Evaluation of RHV Patency and Treatment of RHV Stenosis

In accordance with the local management protocol for LDLT, Doppler ultrasonography to assess graft inflow to and outflow from the RHV was performed intraoperatively, daily for the first week after surgery, and then weekly until discharge from hospital. As a routine, dynamic CT follow-up was undertaken weekly while the patient was in hospital and at 1 month, 3 months, and 1 year after transplantation. Patients with hepatocellular carcinoma underwent CT scans more frequently for cancer surveillance.

Significant RHV stenosis was diagnosed when there was appreciable weakness of pulsatility or changes in the waveform from a triphasic pattern to a biphasic or monophasic pattern on Doppler ultrasonography and there was concurrent noticeable narrowing of the RHV at the anastomosis within the liver graft or at the IVC on CT scans. Hepatic venous congestion was indicated by changes in parenchymal perfusion on CT scans, with or without increases in serum levels of hepatic enzymes. A CT scan was undertaken promptly when serum levels of hepatic enzymes (aspartate/alanine aminotransferase) increased rapidly to values >500 IU/L at any time after transplantation.

Patients who developed definite RHV stenosis (pressure gradient >5 mm Hg) underwent stent insertion soon after the detection of the stenosis, even if their hepatic function was not appreciably impaired. Zilver stents (Cook, Bloomington, IN) were inserted through an internal jugular vein and across the RHV anastomosis. Other than routine treatment with an antiplatelet agent for the first 3 months after stent insertion, no specific anticoagulation therapy was administered after this procedure.
Rapid regeneration of RL grafts occurred for only a few weeks after transplantation. The timing of stenosis was classified as early onset (<2 weeks) or delayed onset (>2 weeks). Stenosis of early onset may be directly related to the anastomosis itself, whereas stenosis of delayed onset may be related more to graft regeneration-induced torsion of the RHV anastomosis. Stent insertion for diffuse intrahepatic RHV stenosis due to extrinsic parenchymal compression, which is usually associated with sustained graft dysfunction, was classified as intrahepatic RHV stenosis. RHV patency 1 year after transplantation was not evaluated in this study because clinically significant RHV stenosis occurs only rarely after such a long interval.

**Institutional Review Board and Statistics**

The study protocol was approved by the institutional review board of our institution. All numerical data are reported as means and standard deviations or as medians and ranges. Incidence rates were compared with the $\chi^2$ test or Fischer’s exact test. Mean values were compared with the Student t test. Actuarial survival rates were estimated with the life table method. Correlations were expressed by the explanation power ($r^2$) and $P$ value. A $P$ value <0.05 was considered to imply statistical significance.

**RESULTS**

**Anatomy of the RHV and IVC in Donors and Recipients**

The native RHV site of insertion into donor IVCs was categorized according to CT images as high [126 (56.0%)], middle [82 (36.4%)], or low [17 (7.6%)]. In recipient CT images before transplantation, retrohepatic IVCs in the vicinity of the RHV were subdivided into 3 categories on the basis of their size: normal or mildly shrunken [63 (28.0%)], moderately shrunken [82 (36.4%)], and severely shrunken [80 (35.6%)].

The native RHV site of insertion into the recipient IVC was initially categorized as high [83 (36.9%)], middle [109 (48.4%)], or low [33 (14.7%)], even when the IVC was severely shrunken. These proportions differed from those for donors. After correction for the small size of a severely shrunken IVC, the native RHV site of insertion into the recipient IVC was finally categorized as high [60 (26.7%)], middle [132 (58.7%)], or low [33 (14.7%); Table 2].

**Simulation of the Anastomotic Configuration According to Matching of the Site of Insertion of the Graft and Recipient RHV**

The morphological configuration of the RHV anastomosis in every possible combination of graft and recipient RHV sites immediately after RL graft implantation is depicted in simplified simulation models (Fig. 2). If the RL graft size is large with respect to the depth of the right subphrenic fossa, the recipient IVC is relatively small, and/or rapid graft regeneration occurs early after transplantation, then suture-line indentation and angulation/buckling deformity of the RHV anastomosis, which are illustrated in the simulation models, will become exaggerated because of ventromedial deviation of the graft RHV and graft compression of the recipient IVC.

An analysis of CT images taken 1 and 2 weeks after transplantation showed that most of the configurations of RHV anastomoses were similar to those in our simulation models. Patients exhibiting definite evidence of RHV stenosis underwent stent insertion (Table 2).

CT scans taken 1 month after LDLT showed a widening of the RHV anastomosis and smoothing of acute-angle anastomotic margins in most patients. However, a small number of patients exhibited exaggerated intravascular folding of anastomotic margins that led to functional narrowing of the RHV anastomosis. Stents were inserted when significant RHV stenosis was diagnosed on follow-up CT scans (Table 3). By 1 year after transplantation, most RHV anastomoses had become streamlined after gradual remodeling of the RL graft and the retrohepatic IVC.

**Reconstruction Techniques and RHV Stenosis**

The surgical techniques used for RHV reconstruction are illustrated in Fig. 3. The preparation of the orifice of the graft RHV was type 1 in 185 patients (82.2%), type 2 in 31 (13.8%), and type 3 in 9 (4.0%). The preparation of the recipient IVC was typed A in 8 patients (3.6%), type B in 5 (2.2%), type C in 9 (4.0%), type D in 77 (34.2%), type E in 10 (4.4%), type F in 11 (4.9%), and type G in 105 (46.7%). Chronologically,
the recipient techniques were introduced in the order of A to G.

The autologous greater saphenous vein was the most common source for patch plasty in both RL grafts and recipients. The sources of vein patches included the cryopreserved iliac vein/artery and the autologous recipient portal vein. There was no difference in the rates of stenosis associated with the different types of vein grafts.

The overall incidences of RHV stenosis (ie, the sum of stenoses of early and late onset) according to the matching status of three-step RHV sites of insertion between the right lobe graft and the recipient IVC are summarized at Table 3. The relative location of RHV sites of insertion between RL grafts and recipient IVCs was closely associated with the early onset of RHV stenosis (Fig. 2): its incidence was 1.6% (2 of 129) in patients with a higher or equal recipient-side level and 7.9% (7 of 96) in patients with a lower recipient-side level (P = 0.039; Table 2). All patients who received stent insertion have, so far, survived for more than 1 year.

RHV stenosis of delayed onset that required stent insertion between 2 weeks and 1 year after transplantation occurred in 12 of 225 patients (5.3%). Stent insertion for stenosis of early onset effectively prevented later stenosis, so the true incidence of stenosis of delayed onset was 5.6% (12 of 216 patients). A discrepancy in the location of RHV sites of insertion between the RL graft and recipient IVC had no influence on the incidence of RHV stenosis of delayed onset; this incidence was 3.9% (5 of 129) in patients with a higher or equal recipient-side level and 7.3% (7 of 96) in patients with a lower recipient-side level (P =

### TABLE 3. Overall One-Year Incidence of Right Hepatic Vein Stenosis Requiring Stent Insertion According to the Matching Status of Three-Step Right Hepatic Vein Sites of Insertion Between the Right Lobe Graft and the Recipient Inferior Vena Cava

<table>
<thead>
<tr>
<th>Recipient</th>
<th>High</th>
<th>Middle</th>
<th>Low</th>
<th>Subtotal</th>
</tr>
</thead>
<tbody>
<tr>
<td>High</td>
<td>3/38 (7.9%)</td>
<td>1/18 (5.6%)</td>
<td>0/4 (0%)</td>
<td>4/60 (6.7%)</td>
</tr>
<tr>
<td>Middle</td>
<td>7/71 (9.9%)</td>
<td>3/50 (6.0%)</td>
<td>0/11 (0%)</td>
<td>10/132 (7.6%)</td>
</tr>
<tr>
<td>Low</td>
<td>3/17 (17.6%)</td>
<td>4/14 (28.6%)</td>
<td>0/2 (0%)</td>
<td>7/33 (21.2%)</td>
</tr>
<tr>
<td>Subtotal</td>
<td>13/126 (10.3%)</td>
<td>8/82 (9.8%)</td>
<td>0/17 (0%)</td>
<td>21/225 (9.3%)</td>
</tr>
</tbody>
</table>

### TABLE 4. Incidence of Right Hepatic Vein Stenosis with Respect to the Methods of Reconstruction

<table>
<thead>
<tr>
<th>Recipient</th>
<th>Onset Timing</th>
<th>RHV Type 1</th>
<th>RHV Type 2</th>
<th>RHV Type 3</th>
<th>Stenosis Incidence</th>
<th>Subtotal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type A</td>
<td>Early</td>
<td>1/7 (14.3%)</td>
<td>0</td>
<td>0/1 (0%)</td>
<td>1/8 (12.5%)</td>
<td>8 (3.6%)</td>
</tr>
<tr>
<td></td>
<td>Delayed</td>
<td>0/7 (0%)</td>
<td>0/1 (0%)</td>
<td>0/5 (0%)</td>
<td></td>
<td>5 (2.2%)</td>
</tr>
<tr>
<td>Type B</td>
<td>Early</td>
<td>0/5 (0%)</td>
<td>0/1 (0%)</td>
<td>0/5 (0%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Delayed</td>
<td>0/5 (0%)</td>
<td>0/1 (0%)</td>
<td>0/5 (0%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type C</td>
<td>Early</td>
<td>0/4 (0%)</td>
<td>0/1 (0%)</td>
<td>0/5 (0%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Delayed</td>
<td>1/4 (25%)</td>
<td>0/1 (0%)</td>
<td>0/5 (0%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type D</td>
<td>Early</td>
<td>0/48 (0%)</td>
<td>2/27 (7.4%)</td>
<td>1/2 (50%)</td>
<td>7/77 (9.1%)</td>
<td>77 (34.2%)</td>
</tr>
<tr>
<td></td>
<td>Delayed</td>
<td>3/48 (6.3%)</td>
<td>1/27 (3.7%)</td>
<td>0/2 (0%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type E</td>
<td>Early</td>
<td>0/8 (0%)</td>
<td>1/2 (50%)</td>
<td>0</td>
<td>3/10 (30.0%)</td>
<td>10 (4.4%)</td>
</tr>
<tr>
<td></td>
<td>Delayed</td>
<td>1/8 (12.5%)</td>
<td>1/2 (50%)</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type F</td>
<td>Early</td>
<td>0/11 (0%)</td>
<td>1/2 (50%)</td>
<td>0</td>
<td>1/11 (9.1%)</td>
<td>11 (4.9%)</td>
</tr>
<tr>
<td></td>
<td>Delayed</td>
<td>1/11 (9.1%)</td>
<td>1/2 (50%)</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type G</td>
<td>Early</td>
<td>0/2 (0%)</td>
<td>0/2 (0%)</td>
<td>0/1 (0%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Delayed</td>
<td>3/10 (2.9%)</td>
<td>0/2 (0%)</td>
<td>0/1 (0%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stenosis incidence</td>
<td>Overall</td>
<td>11/185 (5.9%)</td>
<td>5/31 (16.1%)</td>
<td>5/9 (55.6%)</td>
<td>21/225 (9.3%)</td>
<td>225 (100%)</td>
</tr>
<tr>
<td>Subtotal</td>
<td>Case number</td>
<td>185 (82.2%)</td>
<td>31 (13.8%)</td>
<td>9 (4.0%)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Timing of Onset of RHV Stenosis

Appreciable RHV stenosis of early onset that required stent insertion occurred in 9 of 225 patients (4%) during the first 2 weeks after transplantation (Table 2 and Fig. 4). The relative location of RHV sites of insertion between RL grafts and recipient IVCs was closely associated with the early onset of RHV stenosis (Fig. 2); its incidence was 1.6% (2 of 129) in patients with a higher or equal recipient-side level and 7.9% (7 of 96) in patients with a lower recipient-side level (P = 0.039; Table 2). All patients who received stent insertion have, so far, survived for more than 1 year.

RHV stenosis of delayed onset that required stent insertion between 2 weeks and 1 year after transplantation occurred in 12 of 225 patients (5.3%). Stent insertion for stenosis of early onset effectively prevented later stenosis, so the true incidence of stenosis of delayed onset was 5.6% (12 of 216 patients). A discrepancy in the location of RHV sites of insertion between the RL graft and recipient IVC had no influence on the incidence of RHV stenosis of delayed onset; this incidence was 3.9% (5 of 129) in patients with a higher or equal recipient-side level and 7.3% (7 of 96) in patients with a lower recipient-side level (P =
The overall incidence of RHV stenosis 1 year after transplantation was 9.3% (21 of 225; Table 3). Subtraction of the incidence of stenosis of early onset (Table 2) from the overall incidence of stenosis (Table 3) gives the incidence of stenosis of delayed onset. Three patients with stenosis of delayed onset died: 1 from pneumonia and 2 from progressive graft failure associated with intrahepatic RHV stenosis.

Analysis of RHV Stenosis Using a Simulation of Computational Fluid Dynamics

The effects of RHV stenosis were simulated with a model of computational fluid dynamics that had been calibrated with reference data consisting of independently obtained relevant measurements (Fig. 5). When RHV stenosis was equivalent to 75% of the original CSA (25% occlusion), the pressure gradient across the RHV anastomosis increased to 130% of the initial value, the mean outflow velocity increased to 114% of the initial value, and the outflow volume decreased to 85.5% of the initial value. Thus, RHV stenosis in which there was 25% occlusion reduced RHV outflow by 14.5%. Such changes were associated with gentle linear slopes of hemodynamic changes up to 50% of the original CSA (50% occlusion); at this level of occlusion, the pressure gradient increased to 163%, the outflow velocity increased to 147%, and the outflow volume decreased to 73.5%. For higher degrees of occlusion, the slopes of hemodynamic changes became steeper; the values for the pressure gradient and outflow volume were 217% and 61.7% at 67% occlusion, 290% and 51.2% at 80% occlusion, 372% and 40.3% at 90% occlusion, and 483% and 25.6% at 95% occlusion, respectively.

Risk Factors for RHV Stenosis

It was not possible to determine the independent risk factors for RHV stenosis from the results of this study, other than inappropriate matching of RHV sites of insertion. There was no difference in the graft-recipient weight ratio between patients with RHV stenosis and patients without RHV stenosis (1.05 ± 0.20 versus 1.10 ± 0.21, P = not significant).

Patients who underwent concurrent reconstruction of the inferior RHV had a higher overall incidence of RHV stenosis (12.1%), probably because a large proportion of the patients had received a small RHV graft; however, this incidence did not differ significantly from that in patients who did not have inferior RHV reconstruction (6.8%, P = not significant).

Selection of Methods for RHV Reconstruction and Technical Refinements to Minimize the Risk of RHV Stenosis

The general approach to decreasing the risk of RHV stenosis caused by mismatching of RHV sites of insertion and graft regeneration is to make a large anastomotic CSA with concurrent enlargement of the retrohepatic IVC. Statistically significant differences for outcomes between various methods of RHV reconstruction were not found, primarily because only 2 methods (types G and D) were used preferentially.

RHV reconstruction using graft type 1/recipient type G seemed to be the most reliable technique; it was the most commonly applied technique in the late
DISCUSSION

In adult LDLT using an RL graft, most surgeons have preferred to use the preexisting RHV orifice at the recipient IVC for RHV reconstruction. A caudal shift of the anastomotic site has been described for the conjoined reconstruction of the RHV and MHV of the graft.\(^2\)\(^3\) We have rarely attempted such a longitudinal shift instead of a caudal extension, except when there is overt longitudinal mismatching of the RHV orifices. The relative location of the RHV orifice of the graft in the recipient is determined by the RHV anatomy of the donor and the relationship between the depth of the right subphrenic fossa and the size of the graft. In contrast, the location of the RHV orifice of the recipient is fixed at the IVC from birth, although the development of cirrhosis can result in shrinkage of the IVC. If the ventrodorsal sites of RHV orifices are accurately matched, successful reconstruction can be achieved after a wide anastomotic orifice is made. Otherwise, techniques to offset site discrepancies should be considered to minimize the risk of RHV stenosis.

It appears that none of the techniques for reconstructing the RHV currently available is associated with complete prevention of anastomotic stenosis. In this study, during reconstruction of the RHV, the anastomotic suture line was made at least 30% larger than the orifice of the RHV of the graft. The introduction of this procedure may lead to a lower incidence of anastomotic stricture of early onset, as suggested by an incidence of only 1.8% in this study. Many RHV stenoses were found to be associated with anastomotic dislocation caused by inappropriate matching of the sites of insertion of the RHV of the graft and recipient or by ventromedial shifting of the RHV of the graft due to rapid regeneration of the graft. This type of anastomotic distortion occurred in 33.3% of patients with stenosis of early onset and in 75% of patients with stenosis of delayed onset. These findings indicate that the underlying causes of anastomotic distortion include graft regeneration-associated angulation/buckling deformities of the graft.

Bulging of the ventral part of an RHV anastomosis has been regarded as beneficial in accommodating the effects of regeneration of the graft.\(^1\)\(^2\)\(^4\)\(^2\)\(^5\) However, a localized redundancy associated with a shrunken IVC occasionally has led to adverse intraluminal folding of the redundant portion rather than radial expansion. Such intravascular folding reduces the CSA of an RHV anastomosis and hence results in a substantial increase in the pressure gradient and decrease in the outflow volume. Even without intravascular folding, distortion of the IVC may induce functional obstruction to the outflow of the RHV. The simulation models provide explanations of the mechanisms underlying anastomotic dislocation. If the graft-recipient RHV sites of insertion are the same or if the site for the graft is lower than that for the recipient (eg, mid-graft and high-recipient sites), the discrepancy and graft regeneration will be accommodated without induction of severe distortion of the RHV anastomosis. If, however, the graft RHV site is high and the recipient site is middle or low, graft regeneration will exacerbate the discrepancy, and functional obstruction of the RHV can occur, as illustrated by the results of this study.

Although its effectiveness has already been established, stent insertion does not appear to be indicated unless the pressure gradient exceeds 5 mm Hg. An RL graft with RHV occlusion \(\leq 50\%\) seemed to be well tolerated; partial compensation for the associated decrease in the CSA occurred because of an increase in the outflow velocity that was driven by an increased pressure gradient. On the other hand, RHV occlusion \(\geq 67\%\) may induce clinically significant hepatic venous congestion; such a degree of stenosis is sufficiently severe that an effective increase in the outflow volume cannot occur by an increase in the outflow velocity. Thus, guidelines for the management of RHV stenosis may be summarized as follows: if RHV occlusion is \(\leq 50\%\) and serum biochemical liver test results are satisfactory, no intervention is indicated; if RHV occlusion is 50% to 67%, stent insertion should be seriously considered; and if RHV occlusion is \(\geq 67\%\), regardless of the results of serum biochemical liver tests, stent insertion should be undertaken. Balloon
angioplasty alone may be considered instead of balloon dilatation followed by stent insertion, but in our experience, the former procedure has been less efficacious.

A weak correlation between a decrease in the CSA and the pressure gradient was found; this relationship was influenced appreciably by baseline pressures, such as the intrahepatic RHV pressure and the central venous pressure. Subtle changes in these pressures may induce complex effects on the whole venous drainage system. These considerations may explain, at least in part, why no reliable computational simulation model of venous reconstruction has yet been proposed. In contrast, such models do exist for aortic/arterial stenoses and their reconstruction.14-16

To develop a simulation model in this study, correlation curves that incorporated raw data from several simplified fluid-structure interaction models were meticulously calibrated; the principles of fluid dynamics were applied. The simulation model that was developed showed that pressure gradients of about 5, 6 to 10, and >10 mm Hg were associated with 50%, 67% to 80%, and >90% occlusion of the RHV, respectively. The reliability of this simulation model has not yet been fully evaluated. More experience with applying it in additional prospective studies is necessary before its clinical application can be recommended.

A problem in applying this quantitative simulation model in further studies is the measurement of the size of the CSA because a severe stenosis often results in pre-anastomotic intrahepatic dilatation in an RL graft, as occurs in portal vein stenosis. Arbitrarily, a quantitative assessment was made of the baseline RHV CSA obtained from the donor CT scan before transplantation and of the recipient RHV CSA obtained from CT scans after transplantation. A noticeable RHV stenosis was usually accompanied by RHV occlusion >67% of the original CSA.

RHV stenosis of early onset is closely associated with technical faults or mismatching of RHV sites of insertion; in contrast, RHV stenosis of delayed onset is often associated with graft regeneration. Surgeons are unable to match RHV sites of insertion intentionally, and graft regeneration is unavoidable after adult LDLT. New innovative surgical techniques are needed to overcome these unavoidable anatomical and physiological issues.

The implications of this study can be summarized as follows. In patients in whom matching of RHV sites of insertion is appropriate, most current techniques that enable the formation of a wide anastomosis will be acceptable. If the graft RHV site of insertion is higher, the ventral wall of the RHV anastomosis should not be excessively redundant, but if the graft RHV site of insertion is lower, some redundancy of the ventral wall may be permitted. Sufficient enlargement of the IVC to accommodate anatomical discrepancies and graft regeneration-related torsion is most important. RHV venoplasty should be undertaken not only to widen the anastomosis but also to enlarge the IVC to a size similar to that of a normal IVC.

Currently, 3 techniques for reconstruction of the RHV are considered to be reliable in clinical practice: types G, I, and H. The current method of choice for RHV reconstruction would appear to be type G. If the size of the IVC is insufficiently large and the graft RHV site of insertion is high, type I is preferred. If a sufficiently long vein patch is not available, type H may be used after a deep incision is made in the recipient RHV within the hepatic parenchyma to generate a wider RHV stump.

In conclusion, inappropriate ventrodorsal matching of graft-recipient RHV sites of insertion was found to be a significant risk factor for the development of RHV stenosis. Technical refinements to lessen this risk factor include widening the orifice of the recipient RHV and enlarging the recipient IVC by customized incisions and patch venoplasty.

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REFERENCES


