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# Flow and Pressure during Liver Preservation under ex situ and in situ Perfusion with University of Wisconsin Solution and Histidine-Tryptophan-Ketoglutarate Solution

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**Key Words**
Flow/pressure, liver preservation · Liver preservation, ex situ/in situ perfusion · University of Wisconsin solution · Histidine-tryptophan-ketoglutarate solution · Pressure perfusion · Ischemic-type biliary lesions

**Abstract**
Effective preservation of liver grafts is the first essential step for successful liver transplantation. Insufficient perfusion leads to ischemic-type biliary lesions after transplantation. Perfusion of the graft can be performed either in situ or ex situ, with gravity flow or pressure-controlled. Mainly University of Wisconsin (UW) and histidine-tryptophan-ketoglutarate (HTK) solutions are used widespread in clinical liver transplantation. Due to a persistent lack of data, we performed this systematic investigation of in situ and ex situ perfusion of liver grafts with HTK (low-viscous) and UW (high-viscous) solutions at different pressure steps on the perfusion solution (gravity flow, 50, 100, 150, and 200 mm Hg). End points were perfusion flow and pressure in the hepatic artery. A pig model was used with n = 8 pigs randomized to each (HTK and UW) group. In situ perfusion was ineffective for both solutions at any pressure on the perfusate bag. Ex situ perfusion showed significantly improved flow and pressure in the hepatic artery and, therefore, was highly effective. No major differences between HTK and UW solutions could be detected. Therefore, an additional ex situ perfusion of the hepatic artery should be mandatory in every liver procurement.


## Introduction

Liver transplantation is currently the only therapeutic approach for end-stage liver disease. Effective preservation of the donor liver is the first essential step to perform a successful liver transplantation. The two main principles of organ preservation include flushing of the graft with preservation solution and rapid cooling of the graft down to 4°C. Although many preservation solutions were introduced in the past, University of Wisconsin (UW) solution [1] and histidine-tryptophan-ketoglutarate (HTK) solution [2] are commonly used for clinical transplantation in Germany. However, other solutions such as the Collins solution, the Euro-Collins solution or the Marshall solution are used in clinical liver transplantation as well.

Besides major differences in the composition of UW and HTK solutions, one fundamental difference with regard to the technical aspects of perfusion is in the vis-

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cosity: the UW solution is a highly viscous solution with a fourfold viscosity as compared with the less viscous HTK solution [2]. Some surgeons prefer the HTK solution because of the potentially better distribution in the graft during preservation due to the low viscosity. In experimental rat liver preservation, the HTK solution showed a better washout of blood cells during preservation [3]. However, prospective clinical trials comparing UW and HTK solutions failed to show any differences between the two solutions so far [4, 5].

Clinical liver preservation can be performed in many technical variants. The perfusion can either be performed in situ in the donor body before hepatectomy or ex situ after hepatectomy as a back-table procedure. In order to get access to as many donor organs as available, multiorgan procurement with an in situ preservation through the distal aorta of the donor is mostly performed [6]. Isolated ex situ back-table perfusion is only used in living-related donor liver transplantation.

A simple but effective approach to increase the in situ circulation of the preservation solution in the donor is to add pressure on the solution bag which was introduced by the Berlin group [7]. Because of lack of data concerning the pressure in perfusion, there is no standard. Some centers prefer pressure during preservation, while others perform preservation with gravity flow only.

Our own group [8] could demonstrate a significant reduction of diffuse biliary strictures (ischemic-type biliary lesions) after orthotopic liver transplantation under UW preservation with an additional ex situ pressure perfusion. However, the application of our ex situ perfusion technique in Germany is still limited. Centers in many other countries already prefer ex situ perfusion as well.

We also showed a significant reduction of ischemia-reperfusion damage under ex situ pressure perfusion [8]. Ischemic-type biliary lesions are also clinically present under HTK perfusion [9].

Since there is still a lack of theoretical background of liver preservation under different perfusion techniques (in situ versus ex situ), preservation pressures (gravity pressure vs. high pressure), and preservation solutions (HTK vs. UW/low viscosity vs. high viscosity), we initiated the current experimental study. The aim of this study was to determine the preservation technique that is most effective in flushing the donor organ with special regard to flow and pressure of the preservation solution in the hepatic artery of the graft. We therefore created a model that was set exactly similar to the clinical procedure that is used for organ procurement. Our aim was to obtain data on flow and pressure conditions during organ procurement. We did not aim at a transplantation of the livers (since there is no animal model that generates ischemic-type biliary lesions) nor at measuring any microcirculatory or posttransplant parameters, since these were not end points of our study.

## Materials and Methods

Multiorgan procurement using the technique described by Starzl et al. [6] was performed in male pigs. The study was performed according to the guidelines of the German animal protection law and was approved by the local committee for animal welfare under the title 'In-situ-Druckmessung bei Organkonservierung', No. 1.5 177-07/051-15.

German Landrace pigs were premedicated with the sedative azaperone (7.5 mg/kg i.m.). Anesthesia was initiated with an intravenously administered sodium thiopental bolus (5 mg/kg) and then maintained by intravenous infusion of sodium thiopental (10 mg/kg/h). After intubation, the pigs were mechanically ventilated with a Dräger (Lübeck, Germany) respirator Servo 900b (oxygen-air: $FiO_2$ 0.35, $pCO_2$ controlled), and arterial and central venous lines were introduced via femoral artery and vein. Prior to femoral artery preparation, a 7.5-mg bolus of the analgetic piritramide was given intravenously and infused continuously (0.25 mg/kg/h). Heart rate and oxygen saturation were continuously measured with ECG, pulse oximetry, and capnometry. The ventilation was adjusted according to repeated blood gas analysis. Infusion therapy with NaCl 0.9% at 10 ml/kg was performed during the operation.

The animals were randomized either to a UW group or to a HTK group prior to surgery. The procedure was performed exactly in the same way in both groups. The abdominal cavity was opened by a midline incision. The hepatic artery was isolated in the hepatoduodenal ligament, and a baseline flow measurement was performed. The flow (ml/min) was measured by a flowmeter model CM 4008 with a 5-mm probe (CardioMed Supplies, Lindsay, Ont., Canada). The distal aorta was exposed by dissection of the retroperitoneum, and an 18-Fr perfusion cannula was placed in the aorta. One centimeter cranial to the perfusion cannula, an arterial pressure probe was inserted into the aorta using the Seldinger technique to measure the blood pressure before and the perfusion pressure during preservation. An ultrasound probe was also placed at this site of the aorta to measure the flow during the procedure. Another arterial pressure probe was inserted surgically into the gastroduodenal artery to measure blood and perfusion pressures at the site of the liver artery.

Cardiac arrest was induced by intravenously administered KCl. Cross-clamp of the aorta was performed at the diaphragm. Immediately after cardiac arrest, the organs and the body cavity were cooled down to 4°C by pouring ice-cold Ringer solution into the body cavity and by cooling the preservation solution (either HTK or UW) to 4°C prior to perfusion. In situ perfusion was started into the aortic cannula by gravity flow, and flow and pressure were measured in the aorta and in the liver artery. The pressure of the preservation solution was increased stepwise by adding pressure to the solution bag with a pressure gauge. The follow-

**Table 1.** Baseline measurements of systolic flow and pressure in the aorta and in the hepatic artery before perfusion under physiological conditions in the heart-beating pig

| | Flow aorta ml/min | Pressure aorta mm Hg | Flow A. hepatica ml/min | Pressure A. hepatica mm Hg |
|---|---|---|---|---|
| HTK group | 1,243.7 ± 195.8 | 82.2 ± 5.7 | 87.3 ± 41.9 | 69.2 ± 16.1 |
| UW group | 1,245.0 ± 203.6 | 83.6 ± 7.1 | 86.0 ± 33.3 | 76.1 ± 4.7 |
| p | 0.990 | 0.677 | 0.943 | 0.265 |

Mean values ± SD are shown (Student's t test).

**Table 2.** Pressure and flow in aorta and hepatic artery during in situ and ex situ perfusion with HTK solution and UW solution

| | Gravity flow | 50 mm Hg | 100 mm Hg | 150 mm Hg | 200 mm Hg |
|---|---|---|---|---|---|
| *Pressure aorta in situ, mm Hg* | | | | | |
| HTK | 24 ± 10 | 23 ± 15 | 28 ± 19 | 27 ± 27 | 25 ± 15 |
| UW | 25 ± 10 | 23 ± 8 | 27 ± 12 | 32 ± 15 | 37 ± 18 |
| p | 0.940 | 0.984 | 0.928 | 0.650 | 0.195 |
| *Flow aorta in situ, ml/min* | | | | | |
| HTK | 626 ± 323 | 694 ± 454 | 831 ± 556 | 886 ± 611 | 902 ± 601 |
| UW | 283 ± 94 | 275 ± 188 | 347 ± 168 | 443 ± 249 | 667 ± 475 |
| p | **0.018** | **0.030** | **0.035** | 0.079 | 0.4 |
| *Pressure A. hepatica in situ, mm Hg* | | | | | |
| HTK | 16 ± 12 | 13 ± 9 | 16 ± 9 | 20 ± 19 | 19 ± 15 |
| UW | 19 ± 10 | 13 ± 10 | 19 ± 15 | 19 ± 18 | 27 ± 14 |
| p | 0.675 | 0.980 | 0.589 | 0.903 | 0.847 |
| *Flow A. hepatica in situ, ml/min* | | | | | |
| HTK | 8 ± 6 | 8 ± 9 | 9 ± 10 | 8 ± 9 | 8 ± 11 |
| UW | 7 ± 12 | 4 ± 6 | 9 ± 16 | 8 ± 11 | 14 ± 18 |
| p | 0.817 | 0.360 | 0.957 | 0.946 | 0.427 |
| *Pressure A. hepatica ex situ, mm Hg* | | | | | |
| HTK | 38 ± 20 | 60 ± 27 | 83 ± 33 | 93 ± 27 | 118 ± 45 |
| UW | 22 ± 8 | 33 ± 19 | 56 ± 18 | 68 ± 22 | 77 ± 33 |
| p | 0.089 | **0.044** | 0.092 | 0.139 | 0.086 |
| *Flow A. hepatica ex situ, ml/min* | | | | | |
| HTK | 52 ± 46 | 83 ± 54 | 125 ± 74 | 119 ± 81 | 148 ± 120 |
| UW | 23 ± 27 | 48 ± 49 | 54 ± 46 | 81 ± 77 | 79 ± 73 |
| p | 0.153 | 0.220 | **0.042** | 0.390 | 0.233 |

Mean values ± SD are shown. $p < 0.05$ printed in bold.

ing pressure steps were used: 50, 100, 150, and 200 mm Hg. After the in situ preservation was completed, a hepatectomy with preservation of the whole celiac trunc was performed. The liver was put into a dish with ice-cold Ringer solution back-table in order to keep the temperature at 4°C. All dissected side vessels of the celiac trunc were closed by ligature. Ex situ perfusion was performed back-table via the hepatic artery at gravity pressure and using the same pressure steps as during in situ perfusion. Flow and pressure in the hepatic artery were measured during the procedure.

## Results

Eight animals were randomized to each group. The mean weight was 22.7 kg in the UW group and 23.1 kg in the HTK group. All animals survived the anesthetic and surgical procedures until perfusion without complications. The blood loss was <100 ml in all cases, and hemodynamic stability could be confirmed by continuous monitoring. Baseline heart beat measurements showed

**Table 3.** Differences in flow and pressure in the hepatic artery during in situ and ex situ perfusion with HTK solution

| | Flow A. hepatica in situ, ml/min | Flow A. hepatica ex situ, ml/min | p (flow in situ vs. ex situ) | Pressure A. hepatica in situ, mm Hg | Pressure A. hepatica ex situ, mm Hg | p (pressure ex situ vs. in situ) |
|---|---|---|---|---|---|---|
| Gravity flow | 8.2 ± 7.1 | 52.4 ± 46.5 | **0.038** | 15.8 ± 13.1 | 38.5 ± 20.2 | 0.126 |
| 50 mm Hg | 9.0 ± 10.2 | 83.0 ± 54.7 | **0.006** | 14.4 ± 9.6 | 83.0 ± 54.7 | **0.019** |
| 100 mm Hg | 9.8 ± 10.83 | 125.7 ± 74.9 | **0.004** | 17.3 ± 11.1 | 83.6 ± 33.6 | **0.014** |
| 150 mm Hg | 6.0 ± 5.51 | 119.3 ± 81.2 | **0.020** | 22.8 ± 14.4 | 119.3 ± 81.2 | **0.044** |
| 200 mm Hg | 4.3 ± 4.4 | 148.1 ± 120.9 | **0.035** | 22.6 ± 17.3 | 118.5 ± 45.3 | **0.011** |

Mean values ± SD are shown; $p < 0.05$ printed in bold (Student's t test).

**Table 4.** Differences in flow and pressure in the hepatic artery during in situ and ex situ perfusion with UW solution

| | Flow A. hepatica in situ, ml/min | Flow A. hepatica ex situ, ml/min | p (flow in-situ vs. ex situ) | Pressure A. hepatica in situ, mm Hg | Pressure A. hepatica ex situ, mm Hg | p (pressure ex situ vs. in situ) |
|---|---|---|---|---|---|---|
| Gravity flow | 7.2 ± 12.7 | 22.0 ± 29 | **0.0001** | 19.3 ± 11.5 | 21.6 ± 9.3 | 0.435 |
| 50 mm Hg | 4.5 ± 6.3 | 48.2 ± 49.6 | **0.001** | 13.4 ± 11.4 | 33.5 ± 19.6 | 0.438 |
| 100 mm Hg | 9.5 ± 16.5 | 54.2 ± 46.6 | **0.001** | 19.4 ± 16.9 | 56.2 ± 18.9 | 0.856 |
| 150 mm Hg | 8.3 ± 11.9 | 81.2 ± 77.4 | 0.082 | 19.2 ± 20.1 | 68.7 ± 22.1 | 0.698 |
| 200 mm Hg | 10.4 ± 15.8 | 79.4 ± 73.2 | 0.088 | 21.0 ± 15.8 | 77.2 ± 33. 5 | 0.851 |

Mean values ± SD are shown; $p < 0.05$ printed in bold (Student's t test).

comparable results for pressure and flow in the HTK and UW groups and are shown in table 1. Although the number of 8 pigs per group is low, the investigation made a systematic analysis of perfusion flow and pressure possible.

Table 2 gives an overview about mean pressure and flow during perfusion with HTK and UW solutions. All investigated vessels (aorta in situ, hepatic artery in situ, and hepatica artery ex situ) are described. Pressure and flow in the abdominal aorta were only measured during in situ preservation, since the integrity of this vessel is only present before hepatectomy. With both solutions, HTK and UW, it was impossible to reach baseline pressure or baseline flow during perfusion, even when pressure was added on the solution bag. The aortic pressure did not exceed 30 ± 19 or 37 ± 18 mm Hg during HTK perfusion or UW perfusion, respectively. Increasing the pressure on the solution bag to 200 mm Hg did not result in a significant increase of the aortic pressure ($p = 0.957$ for the HTK and $p = 0.175$ for the UW group). The perfusion flow was limited in the aorta as well. Although there was a tendency for an increasing flow with increasing pressure, significant changes were absent. During the HTK perfusion, the flow increased from 626 ± 323 ml/min (gravity perfusion) to 902 ± 601 ml/min (200 mm Hg) with $p = 0.272$. The UW solution showed an impaired flow as compared with the HTK solution. At gravity flow and 50 and 100 mm Hg pressure on the perfusion bag, the HTK solution showed a significantly better flow as compared with the UW solution ($p = 0.018$ for gravity flow, $p = 0.030$ for 50 mm Hg, and $p = 0.035$ for 100 mm Hg). The aortic flow was comparable between HTK and UW groups at 150 mm Hg ($p = 0.079$) and 200 mm Hg ($p = 0.4$). During UW perfusion, we observed a gravity flow of 283 ± 94 ml/min with an increase to up to 667 ± 475 ml/min at 200 mm Hg ($p = 0.057$).

The insufficient flow and pressure states at the site of the aorta resulted in extremely insufficient perfusion in the hepatic artery during in situ perfusion. Independent of perfusion solution and perfusion pressure, the in situ flow in the hepatic artery never exceeded 20 ml/min and was mostly as low as 10 ml/min. The perfusion pressure in the hepatic artery was always lower than the perfusion pressure in the aorta. Increasing the perfusion pressure

to up to 200 mm Hg did not change the perfusion result at all. Baseline flow and pressure were never reached during in situ perfusion.

Ex situ perfusion of the hepatic artery led to a significant change in perfusion flow and perfusion pressure with both solutions as compared with in situ perfusion. Tables 3 and 4 illustrate the results for the comparison between ex situ and in situ perfusion for pressure and flow in HTK and UW perfusion, respectively.

Ex situ perfusion with HTK solution (table 3) resulted in a significant increase of perfusion flow and perfusion pressure at any pressure on the perfusion bag. A linear increase in perfusion flow and pressure with increasing perfusion pressure could be demonstrated by this approach. The hepatic artery flow went up to 148 ± 120 ml/min (as compared with 4.3 ± 4.4 ml/min during in situ perfusion; $p = 0.035$) and thereby reached baseline values. The hepatic artery pressure went up to 118 ± 45 mm Hg (as compared with 22.6 ± 17.3 ml/min during in situ perfusion; $p = 0.011$) at 200 mm Hg on the perfusion bag and reached baseline as well. Ex situ perfusion was extremely effective with the HTK solution.

Under UW solution (table 4), ex situ perfusion was effective as well. Ex situ perfusion resulted in a significant increase in flow in the hepatic artery at gravity ($p = 0.0001$) and 50 mm Hg ($p = 0.001$) and 100 mm Hg ($p = 0.001$) on the bag of the perfusion solution. Although there was a tendency to higher perfusion pressures during ex situ perfusion, the perfusion pressure in the hepatic artery did not differ statistically significantly between ex situ and in situ perfusion. Ex situ perfusion with UW solution resulted in a maximum flow of 81.2 ± 77.4 ml/min and a maximum pressure of 77.2 ± 33.5 mm Hg in the hepatic artery. Baseline was met under UW perfusion for both pressure and flow. The increase of flow and pressure under the stepwise increase of pressure on the solution bag was linear up to 100 mm Hg and then plateaued.

A final comparison of the different perfusion modalities (in situ vs. ex situ, different pressure steps) between HTK and UW solutions showed mostly nonsignificant differences, with only two exceptions: at 50 mm Hg on the solution bag, the HTK solution showed a significantly higher perfusion pressure during ex situ perfusion (83.6 ± 33.6 vs. 33.5 ± 19.6 mm Hg; $p = 0.044$), and at 100 mm Hg on the solution bag, the HTK solution showed a significantly better flow in the hepatic artery during ex situ perfusion (125.7 ± 74.9 vs. 54.2 ± 46.6 ml/min; $p = 0.042$).

## Discussion

Insufficient preservation of the liver graft prior to transplantation has a negative impact on the postoperative result. Besides primary nonfunction [10], delayed graft function [11], increased ischemia-reperfusion injury, and ischemic-type biliary lesions [12, 13] are major long-term complications related to ineffective preservation of the graft. Many approaches to influence ischemia-reperfusion damage pharmacologically were published during the last decades. However, there is almost no literature on how to improve the distribution of the preservation solution in the graft [14], although this approach is much easier than other forms of preconditioning.

Under physiological conditions, the blood circulation is determined by the blood pressure and the resistance of the system. Other circulatory impediments and elasticity of the vessels contribute to the dynamics of this complex system. The blood is propulsed by the heart which raises the blood pressure at its ventricular outlets and thereby keeps the blood running. A normal cardiac function provided, these essentials guarantee an adequate perfusion of the periphery.

If the blood volume is reduced by events such as hemodynamic shock, the cardiovascular pressure decreases, and the heart rate increases. The same state occurs if the peripheral resistance of the system decreases, e.g., during sepsis. Adequate filling of the cardiocirculatory system and adequate resistance are required to maintain normal cardiac output, normal mean blood pressure, and, finally, normal hemocirculation of any organ. In clinical practice, circulatory impairment during pathological conditions is recognized by sequelae, such as an increase in serum lactate and organ failure. During the procedure of multiorgan preservation in the deceased donor, many essentials of a normal circulation are nonfunctional: the cardiovascular pressure drops to zero, since the blood is evacuated from the vessels, and the peripheral resistance declines due to functional denervation.

As mentioned above, organ preservation is usually performed by in situ perfusion via the aorta. Thereby, a perfusion system is inserted into the abdominal aorta, and about 10 liters of HTK solution or 6 liters of UW solution are given within 10–15 min. This means that the perfusion 'output' maximum is 1 liter/min. Even if the pressure is increased within the perfusion bag, the flow may increase, but it is limited due to impediments within the perfusion line. The smallest diameter in the perfusion line is usually the cannula to be introduced into the perfusate bag. The low volume load of the aorta prevents suf-

ficient intravascular pressure. As a consequence, perfusion of peripheral organs comparable to the physiological circulation is impossible. This has been demonstrated by our study. In addition, the peripheral resistance may be assumed to be extremely low. As a consequence, escape of the perfusate from the aorta into the periphery prefers vessels with favorable hydrodynamic conditions due to its location and is not equally distributed. The perfusion of organs to be retrieved can hardly be controlled. Together with the cooling of the abdominal cavity by rinsing with ice-cold solution, this might suffice for organ cooling, but it is insufficient for providing a homogeneous preservation.

By introducing the technique of arterial back-table pressure perfusion [8], we could demonstrate improved results after human orthotopic liver transplantation. We saw a significant reduction of postoperative peak transaminases and a significant reduction of ischemic-type biliary lesions. However, we had a lack of theoretical background with the method, since we did not study the mechanism in this technique in detail. Although we always speculated about improved perfusate flow under ex situ perfusion, we were unable to differentiate between the effect of pressure in in situ perfusion as compared with ex situ perfusion. 't Hart et al. [15] also identified the importance of the initial blood washout during organ procurement as a key determinant of liver injury after preservation and reperfusion. Therefore, we aimed at a controlled insight into perfusion pressure and perfusion flow using different perfusion techniques. Because of the ongoing discussion, with improved results and protective effects for the bile ducts under perfusion with low-viscous solutions [2, 16, 17], we included both HTK and UW in this investigation.

As expected, in situ perfusion was insufficient in reaching physiological flow or pressure values in the hepatic artery, independent of the pressure on the perfusion bag. Both HTK and UW failed as well as gravity pressure as compared with high-pressure perfusion. In contrast, ex situ perfusion was highly effective: for both solutions normal flow and pressure in the hepatic artery could be demonstrated.

Although we investigated different pressures on the perfusate bag during in situ and ex situ perfusion and, therefore, received extensive data of the perfusion conditions of the liver, it is difficult to determine the best technique for a single solution or to compare the technique for different solutions.

For the HTK solution, during in situ perfusion, the pressure on the perfusion bag was not beneficial, and therefore the initial flush can be performed at gravity flow. During ex situ perfusion with the HTK solution, pressure on the perfusion bag led to a significant increase in pressure and flow, and, therefore, we suggest any pressure on the solution bag as being effective. In the clinical setting, we use a physiological pressure of 120 mm Hg in order not to induce arterial injuries due to a high pressure.

The highly viscous UW solution was more reliable in high-pressure perfusion. We, therefore, suggest a high-pressure in situ perfusion at 200 mm Hg on the solution bag during UW perfusion. Clinical data underlying the effectiveness of this approach were also reported earlier by Langrehr et al. [7]. For the ex situ setting, the situation is more difficult. Although a significant improvement in the perfusion flow between ex situ and in situ perfusion was only obtained at perfusion pressures of up to 100 mm Hg, further increasing the pressure led to further increases in perfusion flow. We, therefore, suggest an optimal physiological perfusion pressure of 120 mm Hg for the ex situ perfusion of the liver artery, too.

A comparison between UW and HTK solutions did not show major differences. We were surprised about the fact that the UW solution showed a higher pressure in the donor vessel during in situ perfusion. Although many transplant surgeons suggest that the HTK solution has superior flow conditions as compared with the UW solution, we could not demonstrate this effect at the site of the liver artery.

In conclusion, ex situ perfusion of the liver is significantly more effective than in situ perfusion through the aorta, independent of the viscosity of the preservation solution. Therefore, an additional ex situ perfusion of the liver artery after an initial in situ flush should be mandatory in liver procurement. Further investigation must show whether the main flush with a high volume of the perfusion solution should be performed in situ or ex situ.

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