Assessment of hepatosplanchnic pathophysiology during thoracoabdominal aortic aneurysm repair using visceral perfusion and shunt

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Abstract

Objective: Despite the recognition of importance to avoid visceral ischemia during thoracoabdominal aortic aneurysm (TAAA) repair, the methodology of visceral perfusion seems still controversial and its pathophysiology has not been clearly understood. We investigated hepatosplanchnic metabolism during visceral perfusion/shunt in TAAA repair. Methods: Seventeen patients (10 male, 64 ± 15 years old) who underwent elective TAAA repair using visceral perfusion/shunt under mild hypothermic distal aortic perfusion were retrospectively enrolled. Their aneurysm extension was type I and II in eight patients. In seven patients, four visceral arteries were perfused through a side-arm of distal aortic perfusion, while they were perfused by an independent pump in another five patients. In four of these 12 (two in each technique), visceral perfusion was converted into selective shunt after completion of aortic anastomosis. In the remaining five patients, four branches were initially perfused through a side-arm of distal aortic perfusion, and aortic perfusion was subsequently stopped after completion of aortic anastomosis. Hepatic venous oxygen saturation (ShO2), oxygen and lactate extraction ratio (OER, LER), and arterial ketone body ratio (AKBR) were measured at six time points. Results: There was no mortality, liver/renal dysfunction, or spinal cord injury. Two patients required re-exploration for bleeding. Fourteen patients were extubated within 24 h postoperatively. Mean intensive care unit stay was 2.3 ± 1.7 days. During visceral perfusion, OER raised (31 ± 13% to 68 ± 21%, p = 0.0012) and ShO2 decreased (67 ± 12% to 34 ± 24%, p = 0.0026) significantly. They recovered to baseline at skin closure. During the same period, LER (41 ± 22% to −1 ± 34%, p = 0.0035) and AKBR (0.47 ± 0.13 to 0.20 ± 0.08, p = 0.0012) significantly decreased. AKBR recovered to baseline at skin closure, but LER did not. ShO2 (R2 = 0.483, p = 0.0257) and LER (R2 = 0.774, p = 0.0018) at skin closure and LER after initiation of partial cardiopulmonary bypass (R2 = 0.427, p = 0.0211) had significant correlation with postoperative peak serum bilirubin level. AKBR after initiation of partial cardiopulmonary bypass had significant correlation with postoperative peak serum alanine aminotransferase level (R2 = 0.289, p = 0.0476). Conclusions: Visceral perfusion/shunt in TAAA repair may avoid critical irreversible hepatosplanchnic ischemia but provide unphysiological blood flow to the liver and thus should be shortened.

Keywords: Thoracoabdominal aortic aneurysm: Visceral perfusion: Hepatic venous oxygen saturation: Oxygen extraction ratio: Lactate extraction ratio: Arterial ketone body ratio

1. Introduction

In the last two decades, the main interest of aortic surgeons with regard to thoracoabdominal aortic aneurysm (TAAA) repair seemed to be focused on prevention of spinal cord injury [1,2]. However, intestinal ischemia and reperfusion injury may play another key role in influencing patient outcome in this type of operation [3]. It is well known that increasing intestinal permeability caused by longer visceral ischemia is implicated in the genesis of coagulopathic bleeding [4]. Furthermore, this condition may modulate elevated serum levels of proinflammatory cytokines that leads to remote organ injury (i.e. the lung) [5]. Pioneers in the treatment of TAAA recognized the danger of visceral ischemia and have sought to avoid it with various techniques [6]. Nevertheless, uniform methodology of visceral perfusion or shunt has not been established and its pathophysiology has not been clearly understood.

We sought to elucidate hepatosplanchnic metabolic change during visceral perfusion/shunt in TAAA repair by means of optic catheter introduced in the hepatic vein.

1.1. Materials and methods

Forty-nine patients underwent elective TAAA repair between April 1998 and February 2000 and between August

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2004 and November 2006 (almost a 4-year period) in our hospital, of whom 27 patients underwent selective visceral perfusion/shunt. Seventeen patients who were monitored for hepatic venous oxygen saturation (ShO2) stably were retrospectively enrolled in this study (Table 1). We obtained approval from the institutional review board and written informed consent was given by all patients for measuring ShO2, collecting blood samples, and anonymous use of their data. There were ten male and seven female patients, and their age ranged from 26 to 77 years (mean, 64 ± 15). The underlying etiology of aneurysm was chronic dissection in seven patients including two with Marfan syndrome, and non-dissection degenerative aneurysm in ten patients. The distribution of aneurysm lesion according to classification by Crawford and Safi [2] was as follows: type I in three patients, type II in five patients, type III in four patients, type IV in four patients, and type V in one patient. Mean body weight of all patients was 59 ± 10 kg and mean body surface area was 1.6 ± 0.2 m². Eight patients had a history of previous aortic surgery and their details are shown in Table 1.

1.2. Surgical techniques

Our routine operative methods for TAAA repair were described previously in detail [7]. All patients underwent distal aortic perfusion using mild hypothermic partial cardiopulmonary bypass (CPB) (femoro–femoral bypass). The employed system for CPB consisted of a heparin-coated circuit (Hepaface™, Terumo, Tokyo, Japan), a centrifugal pump (Capiox™, Terumo, Tokyo, Japan), and a membrane oxygenator (Capiox™, Terumo, Tokyo, Japan) with a heat exchanger. Before November 2002, open CPB circuit was used (n = 5) and thereafter semi-closed CPB system with soft reservoir bag was introduced in the remaining 12 patients. We administered 1–2 mg/kg heparin (mean, 1.4 ± 0.5) and cardiotomy sucker was used in all patients except one. Since November 2002, we have tried to reduce the dosage of heparin from 2 to 1 mg/kg in most cases. A cell-saving device (Cell Saver 5™, Haemonetics, Braintree, MA) was used in all patients. Five mg/kg of betamethasone was given to both the priming solution of CPB circuit and patients intravenously before starting partial CPB. Mean lowest rectal temperature was 34 ± 1 °C. Deep hypothermic circulatory arrest was not applied in the subjects of current study. Multi-segmental sequential repair technique was used as far as possible. We performed either proximal or distal anastomosis first and, subsequently, reimplantation of the intercostal arteries under evoked spinal cord potential guidance and reconstruction of the right renal artery. We preferred to perform another aortic anastomosis prior to the reattachment of the remaining three visceral branches. Therefore, there was a substantial time difference between partial CPB time and aortic cross-clamping time. This duration was mostly used for reattachment of the visceral branches and extracorporeal circuit was used for visceral perfusion alone. Visceral or intercostal branches were reattached individually in most cases using separate 8 or 10 mm side-arm tube grafts. In three patients, the celiac axis and superior mesenteric artery were included in the proximal or distal anastomosis using beveling technique and only the latter was reattached in the same manner in two patients. Cerebrospinal fluid drainage and continuous intravenous infusion of naloxone hydrochloride at a dose of 1 µg/kg/h were used routinely.

1.3. Visceral perfusion/shunt

Our standard protocol for visceral perfusion/shunt was fundamentally consistent over time. We routinely perfuse every four visceral arteries from their ostia using 12–16 F Clini™ nephrostomy catheter (Create Medico Co., Ltd. Yokohama, Japan). For the celiac axis, 12 F, 14 F, and 16 F catheters were used in 10, 4, and 1 patient, respectively. The celiac axis was not perfused in one patient due to severe stenosis. For the superior mesenteric artery, 12 F, 14 F, and 16 F catheters were used in 10, 5, and 1 patient, respectively. One patient had a common origin of the celiac axis and superior mesenteric artery, which was perfused with 14 F catheter. For perfusion, we use our original heparin-coated circuit made with 10 mm tube with four 6 mm side-arm branches [8]. In seven patients, visceral arteries were perfused through a side-arm of distal aortic perfusion using a centrifugal pump, which was converted into selective shunt in two patients after completion of both aortic anastomoses. In five patients, visceral arteries were selectively perfused using an independent roller pump, which was converted into a selective shunt in two patients after completion of both aortic anastomoses. An initial flow rate of selective perfusion was set at 150 ml/min/branch and then regulated according to ShO2 data or urine output in the range of 150–200 ml/min/branch. In the remaining five patients, visceral arteries were initially perfused through a side-arm of distal aortic perfusion, and aortic perfusion was subsequently stopped after completion of aortic anastomosis (Fig. 1). Perfusion pressure was not measured.

1.4. ShO2 monitoring and blood sampling

Using a percutaneous retrograde approach, a 4 F oximetric catheter (OPTICATH™, Abbott laboratories, Abbott Park, IL) was placed in the right hepatic vein under fluoroscopic guidance through a 5.5 F sheath placed in the right internal jugular vein. ShO2 was continuously monitored and recorded throughout the operation after in vivo calibration. We

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**Table 1**

Baseline characteristics of patients studied.

<table>
<thead>
<tr>
<th>Age (years old)</th>
<th>64 ± 15 (26–77)</th>
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<tbody>
<tr>
<td>Gender</td>
<td>Male 10, female 7</td>
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<tr>
<td>Etiology</td>
<td>Dissection 7 (Marfan syndrome 2)</td>
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<td></td>
<td>Non-dissection 10 (Takayasu arteritis 1)</td>
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<tr>
<td>Crawford/Safi’s classification</td>
<td>I/II/III/IV/V 3/5/4/4/1</td>
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<tr>
<td>Body weight (kg)</td>
<td>59 ± 10 (40–76)</td>
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<tr>
<td>Body surface area (m²)</td>
<td>1.6 ± 0.2 (1.3–2.0)</td>
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<tr>
<td>Previous aortic surgery (n = 8)</td>
<td>Aortic root repair/replacement 4</td>
</tr>
<tr>
<td></td>
<td>+hemiarch replacement 1</td>
</tr>
<tr>
<td></td>
<td>+total arch replacement 2</td>
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<tr>
<td>Total arch replacement</td>
<td>2</td>
</tr>
<tr>
<td>Descending aorta replacement</td>
<td>1</td>
</tr>
<tr>
<td>Infrarenal abdominal aorta replacement</td>
<td>1</td>
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collected samples of whole heparinized blood from both the radial artery and the right hepatic vein at the following six time points: just before and after the initiation of partial CPB (T0, T1), just after the initiation of selective visceral perfusion (T2), just after the initiation of selective visceral shunt (n = 4, T3), just after the conclusion of selective visceral perfusion or shunt (T4), just after skin closure (T5). Oxygen contents and lactate levels in blood samples were measured immediately with a Radiometer ABL520TM pH/oxygen contents and lactate levels in blood samples were measured immediately with a Radiometer ABL520TM pH/ blood gas analyzer (Copenhagen, Denmark). Oxygen extraction ratio (OER), an index of hepatosplanchnic metabolic function, was calculated according to the following equation:

\[ \text{OER} = \frac{\text{CaO}_2 - \text{ChO}_2}{\text{CaO}_2} \times 100 \]

where CaO₂ is arterial oxygen content and ChO₂ is hepatic venous oxygen content.

Lactate extraction ratio (LER) was also calculated in the same manner. Acetoacetate and 3-hydroxybutyrate levels in arterial blood samples were also measured afterward and arterial ketone body ratio (AKBR), an index of redox state of liver mitochondria, was calculated according to the following equation:

\[ \text{AKBR} = \frac{\text{Acetoacetate}}{3\text{-hydroxybutyrate}} \]

Preoperative and postoperative serum levels of total-bilirubin (T-Bil), alanine aminotransferase (ALT), aspartate aminotransferase (AST), creatinine (Cr), and blood urea nitrogen (BUN) were measured according to our routine program until discharge.

1.5. Statistical analysis

All values are expressed as mean ± standard deviation. Statistical analysis was performed using StatView™ 5.0 (SAS Institute Inc., NC, USA). Wilcoxon’s signed rank test with a Bonferroni correction was performed in analyses between control and intraoperative values (T1-5) of ShO₂, OER, LER, and AKBR. Thus a p value less than 0.01 was considered statistically significant in these analyses. Otherwise, Mann–Whitney test was used for comparison of the continuous variables and Fisher’s exact test was used for comparison of frequencies between the groups. A linear correlation analysis was used to test the relationship between metabolic and clinical laboratory data. A p value less than 0.05 was considered statistically significant in these analyses.

2. Results

2.1. Clinical outcome

All patients survived the operation. No patient required postoperative plasmapheresis or hemofiltration except one who was in regular hemodialysis preoperatively. No patient developed spinal cord injury. Postoperative laboratory data are shown in Table 2. Two patients required re-exploration for bleeding (11.8%). However, both of them underwent their operation before we introduced semi-closed CPB system with soft reservoir bag. All but three patients were extubated within 24 h after surgery (82%). One patient was extubated on the 2nd postoperative day and the remaining two on the 4th. Mean intensive care unit stay was 2.3 ± 1.7 days. Intraoperative demographic data are demonstrated in Table 2.

2.2. Metabolic data

During visceral perfusion/shunt, ShO₂ decreased to half of the baseline (T0: 67 ± 12%, T2: 34 ± 24%, T3: 29 ± 27%) (Fig. 3). In the same way, OER raised more than twice compared with preoperative value (T0: 31 ± 13% to T2: 68 ± 21%, T3: 71 ± 27%) (Fig. 3). OER (P = .0012) and ShO₂ (p = 0.0026) at T2 were significantly different from those at T0. They recovered to preoperative value at skin closure. During the same period, LER (T0: 41 ± 22% to T2: −1 ± 34%, T3: 13 ± 36%) and AKBR (T0: 0.47 ± 0.13 to T2: 0.20 ± 0.08, T3: 0.24 ± 0.15) also decreased (Figs. 4 and 5). LER (p = 0.0035) and AKBR (p = 0.0012) at T2 were significantly different from those at T0. AKBR recovered to the preoperative value at skin closure, but LER remained

Table 2

<table>
<thead>
<tr>
<th>Intraoperative demographic data and postoperative laboratory data.</th>
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<tr>
<td>Operation time (min)</td>
<td>537 ± 182 (330—907)</td>
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<tr>
<td>Partial cardiopulmonary bypass time (min)</td>
<td>157 ± 60 (86—299)</td>
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<tr>
<td>Aortic cross-clamping time (min)</td>
<td>106 ± 66 (23—243)</td>
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<tr>
<td>Estimated visceral perfusion time (min)</td>
<td>82 ± 39 (15—147)</td>
</tr>
<tr>
<td>Minimum rectal temperature (°C)</td>
<td>34 ± 1 (30—35)</td>
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<tr>
<td>Distal perfusion flow (l/min)</td>
<td>2.7 ± 0.5 (1.6—3.3)</td>
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<tr>
<td>Visceral perfusion flow (ml/min)</td>
<td>610 ± 145 (400—800)</td>
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<tr>
<td>Postoperative peak serum creatinine level (mg/dl)</td>
<td>3.3 ± 3.0 (1.0—12.3)</td>
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<tr>
<td>T-Bil (total-bilirubin) level (mg/dl)</td>
<td>180 ± 333 (61—1462)</td>
</tr>
<tr>
<td>AST level (IU/I)</td>
<td>92 ± 63 (26—255)</td>
</tr>
<tr>
<td>ALT level (IU/I)</td>
<td>3.0 (1.0—12.3)</td>
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significantly lower as compared with the baseline level. When the influence of perfusion technique to the visceral organs on LER at T2 was considered, independent pump perfusion technique provided better LER (12.1 ± 26.4%) than side-arm perfusion technique (−1.4 ± 39.2%), although it had no statistical power ($p = 0.90$). When balloon catheter intro
duced into the superior mesenteric artery was considered, a 12 F balloon catheter was associated with lower LER at T2 ($n = 10$, $−9.0 \pm 39.3\%$) compared with greater one ($n = 6$, $20.7 \pm 25.6\%$) without statistical power ($p = 0.07$). No other perioperative variables were associated with abnormal metabolic data during visceral perfusion such as ShO$_2 < 30\%$ 30\% ($n = 9$), LER < 0\% ($n = 7$), OER > 75\% ($n = 7$), AKBR < 0.2 ($n = 9$) at T2. Perioperative factors entered into the univariate analysis of predictors for abnormal metabolic data were as following; age, gender, body weight, height, Crawford/Safi classification, etiology, use of side-arm visceral perfusion, use of independent pump for visceral perfusion, use of visceral shunt, operation time, partial CPB time, aortic cross-clamping time, estimated visceral perfu
sion time, minimal rectal temperature, size of catheter used for the celiac axis or superior mesenteric artery. Multivariate logistic regression analysis to identify abnormal metabolic data was not performed because of small number of events.

2.3. Clinical data

When the relationships between postoperative liver function and metabolic data were tested, ShO$_2$ at T5 ($R^2 = 0.483$, $p = 0.0257$), LER at T1 ($R^2 = 0.427$, $p = 0.0211$), and LER at T5 ($R^2 = 0.774$, $p = 0.0018$) had significant correlation with postoperative peak serum T-Bil level. AKBR at T2 had significant correlation with postoperative peak serum ALT level ($R^2 = 0.289$, $p = 0.0476$). Postoperative peak serum AST level had no correlation with any of the measured parameters.

3. Discussion

Patients involved in the current study developed neither intraoperative catastrophic coagulopathy nor clinically relevant liver or renal dysfunction in spite of relatively longer duration of the procedure. Therefore visceral perfusion/shunt may be clinically useful to prevent critical visceral end-organ damage and mandatory when longer duration in reattachment of the visceral branches is anticipated. However, metabolic data during visceral perfusion/shunt suggest that it cannot provide physiologically adequate blood flow. Thus duration of visceral perfusion/shunt should possibly be minimized. These findings seem consistent with a recent study by Hanssen and associates that showed selective visceral perfusion was associated with intestinal mucosal injury caused by intestinal hypoperfusion [9]. The great difference between two studies may be that hypoperfusion to the hepatosplanchnic system was not prolonged in our study (except for LER), whereas significant elevated plasma levels of intestinal- and liver-type fatty acid binding proteins persisted up to 6 h postoperatively in their study. Maybe we measured more sensitive and real-time markers than protein that can be easily affected by wash-out effect; or steroid given to our patients might attenuate sustained ischemia and reperfusion injury.

In the last two decades most aortic surgeons seemed to struggle mainly against prevention of spinal cord injury after TAAA repair because it appears the most devastating morbidity [1,2]. However, the more important consequence to patients, i.e. mortality, did not prove to be affected by spinal cord injury itself but by other end-organ injury such as the kidney or lung. Those who have championed in TAAA repair have repeatedly reported renal insufficiency as a
predictor for mortality [1]. In addition, considerable clinical and experimental evidences suggest that visceral ischemia/ reperfusion injury is associated with remote organ dysfunction, most frequently pulmonary injury [5]. Its underlying mechanisms are interpreted as increased intestinal permeability and subsequent bacterial translocation that mediates increased levels of proinflammatory cytokines [3,5].

Pioneers in the treatment of TAAA recognized the importance of preventing visceral ischemia, and distal shunt was already used a half century ago [6]. Then trends have changed in favor of distal perfusion using either left heart bypass or partial CPB. Currently, most experienced aortic surgeons seem to minimize visceral ischemic time with the use of more aggressive selective continuous visceral perfusion/shunt. To date, various modifications have been made in the technique of visceral perfusion/shunt, but widespread consensus in its methodology has not been achieved.

Selective visceral shunt from proximal thoracic aorta or prosthesis after proximal anastomosis was liberally used because of its simplicity and the unneccessity of systemic heparinization [8]. However, this mode of perfusion is obviously affected by systemic arterial pressure, oxygenation achieved by the native lung, visceral vascular resistance, and diameter of the catheters used.

We initiate selective visceral perfusion at a flow rate of 150 ml/min per branch that is identical with the report by Kuniyoshi and co-workers [10]. Their group showed that renal function showed good recovery when kidneys were perfused at a flow rate of 25% of native flow in a canine experiment [11]. Our flow rate is also supported by the findings of decreased whole body oxygen consumption up to 30% at 32 °C [12]. On the other hand, Coselli’s group, using a lower flow rate of 400 ml/min for four branches at normothermia, reported that intermittent cold crystalloid perfusion provides superior renal protection than selective perfusion [13]. Safi and Colleagues [14], using a lower flow rate of 200—600 ml/min for four branches at moderate hypothermia, also reported no beneficial effect of selective perfusion on renal function. However, both of them reported no clinically deleterious effect of their perfusion protocol on liver function. These findings may be interpreted by the fact that OER of the liver and small intestine is flow dependent, whereas the kidney is a pressure dependent organ [12,15]. Currently Coselli’s group perfuses the celiac and superior mesenteric arteries at 200—300 ml/min under mild hypothermia [16], which seems similar to ours. Jacobs and co-workers advocated the pressure-controlled perfusion for better outcome in visceral function [17]. They perfuse visceral branches at a flow of 60—210 ml/min per branch and the renal arteries at 200—280 ml/min, which also seems not far different from our protocol. On the other hand, it was pointed out that too much flow through a small diameter catheter might lead to jeopardized rheological properties of the blood [12,17]. We reported that our original heparin-coated 10 mm tube circuit with four 6 mm side-arm branches could provide a free flow of 230 ml/min at 60 mmHg and 335 ml/min at 100 mmHg through a 12 F balloon [8]. Therefore, we believe that our protocol does not deteriorate rheological properties of the blood. However, the small size of balloon catheter used for the superior mesenteric artery was associated with decreased LER at T2 without statistical power. Thus possibly a greater catheter should be used for rheological and metabolic advantage.

ShO₂ can provide real-time information on low invasive liver oxygenation and has been used mainly in the field of liver surgery or intensive care for two decades. In 2000, we reported its usefulness during TAAA repair and have continued applying it and have become confident with its clinical safety and efficacy [18]. However, to our best knowledge, no clinical report to apply ShO₂ systematically in TAAA repair has been published. It has been suggested that ShO₂ lower than 25—30% is associated with liver dysfunction [19,20]. Our current results of ShO₂ during visceral perfusion/shunt seem acceptable but may be borderline. It has been reported that intestinal and liver OER is normally less than 25% and 40% and increases up to 80% and 95% with hypoperfusion, respectively [15]. Elevated OER in our study means inadequate blood flow to hepatosplanchnic vascular bed but seems within acceptable limit. Therefore, either perfusion flow should be increased or temperature should be lowered when either ShO₂ is further decreased or OER is more increased than our findings.

It seems worth measuring LER of the liver in critical condition, because the liver accounts for half of total body lactate clearance [21]. It was observed that liver lactate consumption initially could prevent splanchnic lactate release, despite the increase in intestinal lactate production in hypoxic condition. However, in further hypoxic condition, these compensatory mechanisms may fail as liver lactate clearance may be deteriorated. One drawback of our study is lack of sampling of the portal vein that has two and a half times more blood flow than the proper hepatic artery. Therefore, it seems unclear whether decreased LER during selective visceral perfusion was caused by increased lactate production in the intestine or the liver, or decreased lactate clearance in the liver, or a combination of them. At least, decreased LER suggests that some extent of hypoxia might occur in hepatosplanchnic area. The fact that persistent decrease in LER at skin closure had significant correlation with postoperative peak T-Bil level may indicate that LER is an important marker of liver dysfunction. Strictly, these effects should be excluded to retrieve concrete conclusions.

AKBR has been used as a classical clinical marker that may reflect redox state of liver mitochondria mainly in the field of liver surgery and intensive care since the early 1980s [22]. Then in the early 1990s it has been frequently investigated in estimating the effects of CPB on liver function [19,23]. However, it has not been assessed systematically in TAAA repair except our report in 2000 [18]. Its cut-off value for uneventful postoperative outcome is estimated at 0.7. AKBR below 0.4 is presumed to be critical zone and that below 0.25 appears lethal [24]. It is well known that CPB itself lowers AKBR to critical level, followed by gradual recovery to baseline level within a few days [23]. Thus delay in recovery of AKBR has great implication in postoperative prognosis [25]. In the current study, visceral perfusion/shunt was associated with lethal levels of AKBR, but they returned to preoperative value at skin closure. Therefore, our protocol of selective visceral perfusion/shunt seems less than optimal. When more pronounced reduction of AKBR is observed, increase in perfusion flow is preferable to lowering the temperature.
because hypothermia is associated with lower AKBR levels than normothermia [25]. Lower levels of AKBR in our patients before introduction of partial CPB seem due to accelerated free fatty acid metabolism influenced by fasting status, hypovolemia, surgical stress-induced endogenous insulin, and beta-adrenergic agents. Of interest, it has been emphasized that changes of AKBR occur immediately after hypoperfusion induced by CPB [23] or partial CPB [25], whereas lactate reaction is supposed to be relatively late. Therefore, AKBR seems to be a potential sensitive and noninvasive marker of hepatosplanchnic ischemia during TAAA repair in the future.

3.1. Study limitations

Several potentially confounding influences should be discussed. First, the current investigation was not a randomized trial but a cohort study with selected patients. A prospective randomized control trial would provide definitive data. However, it seems unethical to perform extended TAAA repair without visceral protection. To confirm usefulness of our protocol, an experimental study will be mandatory.

Second, the detail of our visceral perfusion/shunt technique was not uniform. Two different modes of perfusion were employed and the diameter of cannula was also variable. Perfusion flow was also not quite equivalent among the patients and perfusion pressure was not monitored in our cases. In addition, we measured neither perfusion pressure nor blood flow of each visceral branch, so that each branch blood flow depended on the resistance of the corresponding vessel. However, the major aim of this study was not to compare various visceral perfusion techniques to each other but to disclose usefulness of continuous perfusion to visceral organs regardless of technique to avoid critical visceral organ ischemia. Therefore, we suppose that minor heterogeneity of patients and methodology does not affect the main results. Indeed, our current results that patients were associated with neither critical end-organ dysfunction (liver, kidney, and lung) nor catastrophic coagulopathy might justify our protocol in preventing hepatosplanchnic ischemia-reperfusion injury.

4. Conclusion

Visceral perfusion/shunt in TAAA repair may avoid critical irreversible hepatosplanchnic ischemia but provide unphysiological blood flow to the liver. Therefore, duration of visceral perfusion/shunt should possibly be shortened. Hepatic venous oximetric catheter provides useful information of hepatosplanchnic pathophysiology during TAAA repair.

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References


