The impact of intraoperative vasopressin infusion in complex neonatal cardiac surgery

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Abstract

OBJECTIVES: Although recent advances have led to a better understanding of the beneficial effects of vasopressin on haemodynamics in paediatric cardiac surgery, not much information is available on the adverse effects. The objective of this study was to assess the impact of intraoperative vasopressin infusion on postoperative liver, renal and haemostatic function and lactate levels in neonates undergoing cardiac surgery.

METHODS: We reviewed data from 34 consecutive neonates who had undergone complex cardiac surgery. The cohort was divided into two groups according to the use of vasopressin. Seventeen patients received vasopressin [vasopressin (+) group], and 17 patients did not [vasopressin (−) group].

RESULTS: No differences between the groups in terms of age, weight, cardiopulmonary bypass time, Risk Adjustment for Congenital Heart Surgery-1 score or the comprehensive Aristotle score were seen. No differences in the systolic or diastolic arterial blood pressures, heart rate or inotropic score upon admission to the intensive care unit were observed between the groups. No adverse effects on the aminotransferase levels were seen. The vasopressin (+) group had higher urea and creatinine levels. All the patients except one received peritoneal dialysis on the day of surgery. Thirteen patients in the vasopressin (+) group and 7 patients in the vasopressin (−) group continued to require peritoneal dialysis on postoperative day 5 (POD 5) (P = 0.04). The platelet count had decreased to a significantly lower level in the vasopressin (+) group on POD 5 [97 × 10³/mm³ (range: 40–132 × 10³/mm³)]. A tendency toward a high lactate concentration was seen in the vasopressin (+) group. In comparison with the vasopressin (−) group, the number of patients whose lactate level remained above 2.0 mmol/l was higher in the vasopressin (+) group on PODs 2 and 3 (17 patients vs 8 patients, P < 0.01 and 15 patients vs 7 patients, P = 0.01, respectively).

CONCLUSIONS: These findings suggest that the intraoperative use of vasopressin extends the period of peritoneal dialysis, reduces platelet counts and delays the recovery of the lactate concentration. Intraoperative vasopressin infusion should not be used routinely, but only in catecholamine-refractory shock.

Keywords: Vasopressin • Neonate • Congenital heart disease • Cardiopulmonary bypass • Postoperative complication

INTRODUCTION

Severe hypotension associated with systemic vasodilatation can occur after cardiopulmonary bypass (CPB). Vasodilatory shock after cardiac surgery is occasionally refractory to conventional inotropes and vasopressors such as epinephrine, dopamine, dobutamine and norepinephrine, especially in neonates who have undergone complex cardiac surgery with a long bypass duration or using a deep hypothermic technique.

Recent investigations have suggested that vasopressin deficiency is one of the important causes of vasodilatory shock after cardiac surgery and that a low-dose continuous infusion of vasopressin is very effective in increasing the blood pressure under such conditions [1–3].

Although the beneficial effects of vasopressin on haemodynamics have been widely recognized in paediatric cardiac surgery, concerns have been raised about its potential adverse effects on other vital organs. Several reports have indicated that liver dysfunction, thrombocytopenia, elevated serum urea and creatinine levels and a decreased urine output may occur [4–6]. However, whether these changes have clinical relevance, especially after neonatal cardiac surgery, remains unclear.

The objective of this retrospective study was to assess the clinical impact of intraoperative vasopressin use on early postoperative liver, renal and haemostatic function and lactate levels in neonatal cardiac patients who underwent surgery while under CPB.
MATERIALS AND METHODS

Patients

All 34 consecutive neonates with congenital heart disease who had undergone complex cardiac surgery while under CPB at Saitama International Medical Center, Saitama, Japan, between August 2007 and March 2011 were enrolled in this study. Patients who underwent a simple systemic-pulmonary shunt, such as a Blalock-Taussig shunt without intracardiac repair, were excluded. In addition, patients placed on extracorporeal membrane oxygenation perioperatively were not enrolled in the present study. The patients were allocated to two groups, depending on the intraoperative use of vasopressin.

In the vasopressin (+) group (n = 17), the continuous intravenous infusion of vasopressin was started to increase the blood pressure during the period of modified ultrafiltration (MUF) just after weaning from CPB. Based on our standard practice, the anesthesiologists selected the most appropriate inotropic therapy to achieve mean arterial blood pressure: 40–50 mmHg, central venous pressure: 8–12 cmH2O, heart rate < 170/min, SaO2 – SvO2 < 25%, rsO2 > 50% in univentricular repair and >70% in biventricular repair. In general, vasopressin infusion was reserved for vasodilatory and hypotensive states refractory to the conventional inotropes, such as epinephrine and dobutamine. Transesophageal echocardiography (TEE) was performed in all the patients. In cases where the TEE indicated poor cardiac wall motion, vasopressin was not infused to avoid afterload mismatching. Ultimately, the use of vasopressin was left to the discretion of the anesthesiologists. In the vasopressin (–) group (n = 17), vasopressin was not administered perioperatively.

A comprehensive review of the medical records was performed to collect patient characteristics (age, body weight at surgery, details of surgical procedures and CPB time) and perioperative laboratory data (the day before surgery, the day of surgery and the first 5 days after surgery), including aspartate aminotransferase (AST), alanine aminotransferase (ALT), blood urea nitrogen (BUN), creatinine, platelet counts, prothrombin time-international normalized ratio (PT-INR), blood sugar and lactate concentration. The daily urine output and platelet transfusion volume were abstracted from the intensive care charts. The surgical complexity of each procedure was classified using the Risk Adjustment for Congenital Heart Surgery (RACHS-1) score [7]. Furthermore, the preoperative status was assessed in terms of the comprehensive Aristotle score [http://www.aristotelinstitute.org]. Haemodynamic data, including systolic and diastolic arterial pressures, heart rate, inotropic score and vasopressin requirements, were obtained upon admission to the intensive care unit (ICU). Inotropic scores were calculated using a method described by Rosenzweig et al. [1]. Postoperative clinical parameters assessed were mortality, the length of ICU stay and the duration of mechanical ventilation. Owing to the retrospective nature of this study, individual consent was waived, and Institutional Review Board approval was granted.

Surgery and postoperative management

A single surgeon performed all the operations. For cooling and re-warming, the pH-stat management was applied. A low-flow regional cerebral perfusion technique was used while reconstructing the aortic arch. MUF was used in all the cases. A peritoneal dialysis (PD) catheter was placed intraoperatively at the discretion of the cardiac surgeon in anticipation of the need for adjunctive fluid removal in high-risk neonates. The indications for PD catheter placement included inadequate urine output, a need for high-dose catecholamine treatment at the end of the CPB and physical evidence of severe oedema after MUF.

Patients were transferred to the ICU after surgery. All patients received our standard ICU care by cardiac surgeons and paediatric cardiologists. Blood transfusion therapy, such as platelet transfusion, inotropic and vasopressor therapy including vasopressin infusion, and PD management, was performed at their discretion. Blood samples were obtained according to routine clinical practices in our ICU.

Statistical analysis

Continuous variables are presented as the median with range. Categorical data are presented as a count. Differences between the groups were tested using a Mann-Whitney U-test or a chi-square test, as appropriate. Differences among the groups were assessed using the Kruskal-Wallis test followed by Dunn’s multiple comparison test.

Statistical analyses were performed using GraphPad Prism 5 (GraphPad Software, Inc., San Diego, CA, USA). All the analyses were performed with a level of significance of 5%.

RESULTS

Patient characteristics and haemodynamic data upon admission to the intensive care unit

The patient characteristics and haemodynamic data upon admission to the ICU of the study groups are shown in Table 1. No differences between the groups in terms of age, body weight at surgery or CPB time were seen. We found no difference in the surgical complexity and the preoperative status between the groups according to the RACHS-1 category and the comprehensive Aristotle score (P = 0.69 and P = 0.82, respectively). No differences in the systolic or diastolic arterial blood pressures, heart rate or inotropic score upon admission to the ICU were observed between the groups. The median dose of vasopressin upon admission to the ICU was 0.5 mU/kg/min (range: 0.2–1.0 mU/kg/min) in the vasopressin (+) group. During the study period, there were one death in the vasopressin (+) group (cause: circulatory failure) and two deaths in the vasopressin (–) group (causes: methicillin-resistant Staphylococcus aureus infection and Acinetobacter baumannii infection) (P = 0.55). No differences in the length of ICU stay and the duration of mechanical ventilation were found between groups (P = 0.58 and P = 0.17, respectively).

Aspartate aminotransferase and alanine aminotransferase

The perioperative AST and ALT values are presented in Fig. 1. The AST values were elevated on the day of surgery and on postoperative day 1 (POD 1) in both groups, compared with the preoperative value. We observed statistically higher ALT values in the vasopressin (+) group than in the vasopressin (–) group on PODs 1, 2, 3 and 4.
BUN, creatinine and urine output

The perioperative BUN, creatinine levels and urine output are presented in Fig. 2. A steady elevation in the BUN level was seen. The BUN level in the vasopressin (+) group was higher than that in the vasopressin (-) group on PODs 4 and 5. We observed statistically higher creatinine values on PODs 2, 3, 4 and 5, compared with the preoperative values in both groups, although every value was below 2.0 mg/dl throughout the study period. The creatinine level in the vasopressin (+) group was statistically higher than that in the vasopressin (-) group on POD 3. Between the groups, there was no significant difference in the urine output.

Platelet counts, platelet transfusion and PT-INR

The perioperative platelet counts and PT-INR values are presented in Fig. 3. In the vasopressin (+) group, the platelet counts tended to decrease after POD 1, reaching $97 \times 10^3$/mm$^3$ (range: $40-423 \times 10^3$/mm$^3$) on POD 5. In the vasopressin (-) group, the platelet counts progressively decreased to a level lower than the preoperative value. The platelet counts in the vasopressin (+) group were significantly lower than that in the vasopressin (-) group on the day before surgery and on POD 5. During the study period, platelet concentrates were transfused according to our standard practice. Sixteen patients in the vasopressin (+) group received 20 ml/kg (range: 5–47 ml/kg) of platelet concentrates and 12 patients in the vasopressin (-) group received 15 ml/kg (range: 8–62 ml/kg) on the day of surgery ($P = 0.28$). More patients (15 patients) in the vasopressin (+) group had platelet transfusion than those of the vasopressin (-) group (8 patients) on POD 1 ($P = 0.01$), although we could not find the difference between the groups in terms of transfused volume (24 ml/kg (range: 7–79 ml/kg) vs 27 ml/kg (range: 7–49 ml/kg), respectively. $P = 0.72$). After POD 2, 1 or 2 patients had platelet transfusion sporadically, and no statistically significant difference was found between the groups. The PT-INR values were stationary during the study period in both groups, although a clinically meaningless difference was seen on POD 3.

Lactate concentration

The postoperative blood sugar and lactate levels were presented in Table 2. No differences in blood sugar levels between the groups were seen for each time point. The highest lactate values were observed on the day of surgery in both groups, with no significant difference seen between the groups. The lactate levels
were significantly higher in the vasopressin (+) group than in the vasopressin (−) group on POD 2 ($P = 0.02$) and POD 3 ($P = 0.02$). The number of patients whose initial lactate level upon admission to the ICU was elevated by more than 6.0 mmol/l was 15 of 17 patients in the vasopressin (+) group and 10 of 17 patients in the vasopressin (−) group, with no significant difference seen between the groups ($P = 0.12$). However, in comparison with the vasopressin (−) group, the number of patients whose lactate level remained above 2.0 mmol/l was higher in the vasopressin (+) group on PODs 2 and 3 (17 patients vs 8 patients, $P < 0.01$; and 15 patients vs 7 patients, $P = 0.01$, respectively). The differences between the groups in terms of lactate concentration and the number of high lactate (>2.0 mmol/l) patients were resolved by POD 4 or 5.

**DISCUSSION**

The present results indicate that intraoperative vasopressin infusion (dose: 0.2–1.0 mU/kg/min upon admission to the ICU) for neonates who undergo complex congenital heart surgery may deteriorate renal function, prolong the length of PD therapy, reduce the platelet count and delay the normalization of the lactate concentration after surgery, although not powered to assess for clinical outcomes, such as mortality, the length of ICU stay and the duration of mechanical ventilation.

**Liver function**

An increase in the aspartate aminotransferase (AST) level was observed on the day of surgery and on POD 1 in both groups, with no significant differences observed between the groups. In contrast, the ALT level did not increase significantly throughout the early postoperative period. Most of the ALT values were within the normal limit according to the reference values in neonates (AST: 25–75 IU/l, ALT: 13–45 IU/l [8]). These findings agree with those of other reports [9, 10] depicting changes in the AST and ALT levels in children after cardiac surgery. The mild elevation of AST ($\leq 100$–500 IU/l) and the stability of ALT ($\leq 100$ IU/l), which is more specific than AST for liver, suggest that serious liver cell injury did not occur in either group during the study period.

We could not find any remarkable adverse effect of intraoperative vasopressin infusion on AST and ALT. Statistically significant, but clinically negligible differences in ALT were observed between the groups on POD 1–4 (Fig. 1). Although vasopressin infusion has been associated with the worsening of liver function
tests in response to the vasopressin-mediated reduction in hepatic blood flow [11], the preservation of splanchnic blood flow by adequate repletion of the intravascular volume during vasopressin infusion may prevent the deterioration of liver function [5, 11]. In this study, the postoperative lactate levels decreased progressively to an acceptable range at POD 4 or POD 5 in both groups (Table 2), and the urine output was maintained with the support of PD (Fig. 2). These findings could reflect an adequately replete intravascular volume of patients as the central venous pressures were continuously monitored, possibly indicating the maintenance of splanchnic perfusion in our patients.

Severe liver dysfunction did not appear to be induced by the intraoperative use of vasopressin in neonates undergoing complex cardiac surgery in the present study, although in cases with rapid increases in bilirubin or aminotransferase levels, the discontinuation of vasopressin should be considered.

Renal function

The reported incidence and mortality rate of acute renal insufficiency after paediatric CPB surgery range between 10 and 30% and between 20 and 50%, respectively [12–15]. The immaturity of the neonatal kidney makes it more vulnerable to haemodynamic stress and ischaemia-reperfusion injury. Therefore, it is important to limit risk factors of renal injury, such as a low cardiac output and hypoxic ischaemic injury and toxic insults caused by drugs. A longer duration of vasopressin infusion has been associated with higher urea and creatinine levels and a lower urine output in sick paediatric patients [5]. In contrast, low doses of vasopressin infusion may increase diuresis and possibly prevent acute renal failure and increases in plasma creatinine concentrations through its effect on increasing the arterial pressure without incurring detrimental effects on mesenteric, renal, iliac or carotid blood flows [16]. In our study, a higher postoperative creatinine level and BUN level tended to be seen in the vasopressin (+) group, although we did not observe any patients with severe renal dysfunction in either group, such as patients with a urine output of less than 0.5 ml/kg/h or a creatinine level greater than 2.0 mg/dl (reference value in neonates: 0.3–1.0 mg/dl [8]).

All the patients except one received prophylactic PD. In 17 of the 17 patients in the vasopressin (+) group and 16 of the 17 patients in the vasopressin (−) group, PD therapy was started prophylactically upon admission to the ICU. Our rationale for

### Table 2: Blood sugar, lactate levels and the number of patients whose lactate level was elevated above 6.0 mmol/l or above 2.0 mmol/l

<table>
<thead>
<tr>
<th>Vasopressin (+) (n = 17)</th>
<th>Blood sugar (mg/dl)</th>
<th>Lactate (mmol/l)</th>
<th>&gt;6.0; &gt;2.0</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ope</td>
<td>380 (252–566)</td>
<td>8.1 (4.4–13.5)</td>
<td>15; 17</td>
</tr>
<tr>
<td>POD 1</td>
<td>272 (102–627)</td>
<td>5.8 (2.7–10.9)</td>
<td>8; 17</td>
</tr>
<tr>
<td>POD 2</td>
<td>192 (98–369)</td>
<td>3.0 a,b (2.1–10.3)</td>
<td>2; 17 c</td>
</tr>
<tr>
<td>POD 3</td>
<td>142 (86–250)</td>
<td>2.7 a,b (1.8–7.6)</td>
<td>1; 15 c</td>
</tr>
<tr>
<td>POD 4</td>
<td>138 (76–232)</td>
<td>2.0 a (1.5–5.9)</td>
<td>0; 9</td>
</tr>
<tr>
<td>POD 5</td>
<td>133 (83–223)</td>
<td>2.0 a (1.1–4.2)</td>
<td>0; 7</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Vasopressin (−) (n = 17)</th>
<th>Blood sugar (mg/dl)</th>
<th>Lactate (mmol/l)</th>
<th>&gt;6.0; &gt;2.0</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ope</td>
<td>391 (177–580)</td>
<td>6.4 (2.9–16.6)</td>
<td>10; 17</td>
</tr>
<tr>
<td>POD 1</td>
<td>272 (117–560)</td>
<td>3.8 (2.0–14.2)</td>
<td>4; 17</td>
</tr>
<tr>
<td>POD 2</td>
<td>155 (94–272)</td>
<td>2.1 a,b (1.5–4.7)</td>
<td>0; 8 c</td>
</tr>
<tr>
<td>POD 3</td>
<td>138 (94–345)</td>
<td>2.0 a,b (1.1–3.2)</td>
<td>0; 7 c</td>
</tr>
<tr>
<td>POD 4</td>
<td>115 (67–219)</td>
<td>1.8 a (1.0–3.3)</td>
<td>0; 5</td>
</tr>
<tr>
<td>POD 5</td>
<td>108 (60–235)</td>
<td>1.5 a (0.9–3.3)</td>
<td>0; 4</td>
</tr>
</tbody>
</table>

Data are presented as the median (range) or number of patients. Ope: the day of surgery; POD X: postoperative day X.

aSignificant difference vs the value on the day of surgery (Ope).

bSignificant difference of the value between the groups on the same day.

cSignificant difference of the number of patients between groups on the same day.
prophylactic and early institution of PD is that the benefit of PD lies in the favourable influence of fluid balance, besides being an effective and safe method for the treatment of ARF in infants after open heart surgery [14, 17].

It is worthwhile to note that more patients in the vasopressin (+) group required PD throughout the study period, and a significant difference in the number of patients requiring PD on POD 5 was seen (13 patients in the vasopressin (+) group vs 7 patients in the vasopressin (−) group, P = 0.04).

Taken together, these results suggest that renal impairment was induced by intraoperative vasopressin infusion in neonatal cardiac surgery patients and postoperative renal function should be monitored strictly.

Haemostatic function

Significantly lower platelet counts were observed in the vasopressin (+) group compared with the vasopressin (−) group on POD 5, but the platelet counts never reached levels less than \(30 \times 10^3/mm^3\). These findings are compatible with those reported by Dünser et al. [4, 6], who reported a significant reduction in the platelet count following vasopressin infusion in patients with vasodilatory septic and post-cardiectomy shock, while a similar incidence of severe thrombocytopenia was seen between vasopressin-treated and norepinephrine-treated patients. Jerath et al. [5] also reported a significant reduction in the platelet counts in patients admitted to a multidisciplinary tertiary paediatric critical care unit. Vasopressin acts via V1 receptors of vascular smooth muscle to elevate blood pressure. In addition, it acts via V1 receptors expressed on platelets to aggregate platelets via thromboxane release [18]. This V1 receptor-mediated platelet aggregation is considered to be one of the mechanisms of thrombocytopenia related to vasopressin infusion in our study.

Despite the proper use of platelet transfusion therapy in both groups according to our standard practice, the platelet counts were significantly reduced in the vasopressin (+) group on POD 5. Although we could not exclude the influence of postoperative platelet transfusion therapy, Dünser et al. reported the occurrence of thrombocytopenia during vasopressin infusion [6, 11] and confirmed that factors other than a significant difference in platelet transfusion (e.g. vasopressin-induced platelet aggregation) were responsible for the reduced platelet counts in patients treated with vasopressin [6].

Physiologically, V2 receptor stimulation induces haemostatic effects through the liberation of the von Willebrand factor, factor VIII and plasminogen activator, thereby promoting platelet aggregation and coagulation. Jerath et al. reported the normalization of PT-INR values during and after vasopressin infusion in paediatric patients after cardiac surgery [5]. On the other hand, Dünser et al. did not observe any effects on the prothrombin time, factor VIII level or von Willebrand factor level in adult patients with severe multiple organ dysfunction syndrome [6]. In our study, all the PT-INR values were within the normal limits in both groups (reference value in neonates: 0.9–2.7 [19]). Although our standard practice, such as aggressive transfusion therapy (fresh frozen plasma, red blood cell and platelet) may affect the PT-INR values, our results suggest that intraoperative vasopressin infusion did not affect the PT-INR values adversely.

We surmised that perioperative vasopressin administration facilitated platelet aggregation and resulted in moderate thrombocytopenia (platelet count >30 × 10^3/mm^3) after neonatal congenital heart surgery. The clinical significance of thrombocytopenia, such as an increase in the need for postoperative red blood cell and platelet transfusion, could not be assessed in this study.

Lactate levels

Blood lactate concentrations can be used to detect low systemic output and tissue hypoxia during the early postoperative period. Hyperlactatemia upon admission to the ICU after congenital heart surgery has been recognized as a potentially sensitive indicator of an adverse outcome [20, 21]. Initial postoperative lactate levels >6.0 mmol/l and >4.2 mmol/l have been associated with a positive predictive value for mortality of 32 and 100%, respectively [20, 22]. In addition, the Lactime, the time during which the lactate level remains >2.0 mmol/l, has been shown to be a useful predictor of mortality among children undergoing the repair or palliation of congenital cardiac defects under CPB [21, 23].

The intraoperative use of vasopressin for neonates may decrease tissue perfusion, especially splanchnic perfusion, and may subsequently lead to hyperlactatemia as a result of profound vasoconstriction. Dünser et al. showed an improvement in lactate levels after the cessation of vasopressin infusion in postcardiac surgery patients [4]. In our study, the serum lactate concentrations were highest on the day of surgery and decreased progressively, with similar trends seen in both groups. There were 15/17 patients in the vasopressin (+) group and 10/17 patients in the vasopressin (−) group who exhibited lactate levels higher than 6.0 mmol/l; no significant difference was seen between the groups (P = 0.12). However, the lactate concentrations were significantly higher in the vasopressin (+) group than in the vasopressin (−) group at PODs 2 and 3. In addition, the number of patients whose lactate levels remained above 2.0 mmol/l (Lactime) on PODs 2 and 3 was significantly higher in the vasopressin (+) group than in the vasopressin (−) group (P < 0.01 and P = 0.01, respectively). These findings suggested that the normalization of the lactate level (≤2.0 mmol/l) after neonatal cardiac surgery was delayed in the vasopressin (+) group.

Limitations

This study should be considered in light of its retrospective nature, with clinical and surgical data limited to those documented in the patient medical records. First, the use of vasopressin was not randomized. In order to avoid afterload mismatching, we limited the use of vasopressin to hypotensive neonates in whom cardiac contractility, as evaluated using TEE, was not severely impaired. Vasopressin was infused as an adjunct to conventional inotropic and vasopressor drugs for refractory hypotension. Therefore, vasopressin use itself did not reduce the requirement for them in our patients. Thus, the patients who received vasopressin infusion intraoperatively had the confounding factor of being sicker at baseline than the patients who received conventional inotropic and vasopressor therapy. Second, the postoperative care, including the decision to stop vasopressin infusion, platelet transfusion and PD therapy, was determined at the discretion of the ICU team caring for the patient. Although the cardiac surgeons and paediatric
cardiologists were aware of whether vasopressin was being used in each case, their postoperative management was based on the standard practices of our institution, and not on vasopressin use. Third, vasopressin was used and dosed at the discretion of the attending anesthesiologists. We administered vasopressin at 0.2–1.0 mU/kg/min. Although the effective and safe use of vasopressin (0.3–2.0 mU/kg/min) has been shown in children after cardiac surgery [1, 3, 24], future clinical trials should define when vasopressin should best be started and what dose should best be infused in children, especially neonates.

CONCLUSION

We found that intraoperative vasopressin infusion prolonged the need for PD, reduced the platelet count and delayed the normalization of the lactate concentration after neonatal complex cardiac surgery. Although these adverse effects of vasopressin infusion may not be serious from a clinical point of view, we recommend that intraoperative vasopressin infusion be used cautiously with strict postoperative monitoring of the platelet counts, the renal function and functional recovery of the kidney and the lactate level. Intraoperative vasopressin infusion should not be used routinely but only in catecholamine-refractory shock.

Conflict of interest: none declared.

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