Pediatr Surg Int (1999) 15: 78–84 

MAIN TOPIC

**S. Fukuda · M. Aoyama · Y. Yamada · N. Saitoh**
**T. Honjoh · T. Hasegawa · M. Futamura · J. Katoh**
**N. Niimi · S. Tanaka · M. Nagaya**

# Comparison of venoarterial versus venovenous access in the cerebral circulation of newborns undergoing extracorporeal membrane oxygenation

**Abstract** This study was designed to compare venoarterial (VA) with venovenous (VV) access in the cerebral circulation of newborn infants during extracorporeal membrane oxygenation (ECMO). Among 14 infants with VA ECMO, 7 had no intracranial complications (group 1), while the others (group 2) developed intracranial hemorrhage (ICH). In contrast, among 19 infants with VV ECMO, only 1 developed ICH. Serial echocardiograms were performed before and after 1, 6, 12, and 24 h and 2 and 3 days of ECMO. The mean cerebral blood flow (CBF) velocities were measured in the anterior cerebral artery (ACA), right and left internal carotid arteries (Rt, Lt-ICA), basilar artery (BA), and right and left middle cerebral arteries (Rt, Lt-MCA). Ejection fraction (EF), cardiac output (CO), and stroke volume (SV) were also measured using standard echography. The velocity levels in the ACA, Rt-MCA, and Lt-MCA in VA ECMO were lower than those in VV ECMO, while those in the Lt-ICA and BA in VA ECMO were higher than those in VV ECMO. The EF, CO, and SV were lower in cases of VA ECMO than in VV ECMO. In cases of VA ECMO, there were no differences between groups 1 and 2 in velocities in the ACA, Rt-ICA, or Lt-ICA. However the velocities in group 2 in the BA, Rt-MCA, and Lt-MCA were lower than those in group 1 before and during ECMO. Similarly, the EF, CO, and SV were lower in group 2 (12.0%–31.0%, 0.10–0.32 l/min, and 0.66–1.55 ml, respectively) than in group 1 (29.5%–49.3%, 0.25–0.63 l/min, and 2.15–3.85 ml) during ECMO. However, in the infants on VV ECMO the CBF was either maintained or gradually increased before and during ECMO. Their cardiac parameters were: EF 46.1%–53.0%, CO 0.43–0.52 l/min, and SV 2.72–3.84 ml during ECMO. It is concluded that in VA ECMO CBF velocities, particularly in infants who developed ICH, decreased after the onset of ECMO in association with poor cardiac function, while in VV ECMO they were stable, probably due to normal systemic hemodynamics and cardiac function.

**Key words** Extracorporeal membrane oxygenation · Cerebral blood flow velocity · Cerebral hemorrhage

S. Fukuda (✉) · M. Aoyama · Y. Yamada · N. Saitoh
T. Honjoh · T. Hasegawa · M. Futamura
Department of Neonatology, Central Hospital,
Aichi Prefectural Colony, 713-8 Kagiya, Kasugai,
Aichi 480-0392, Japan

J. Katoh · N. Niimi · S. Tanaka · M. Nagaya
Department of Pediatric Surgery, Central Hospital,
Aichi Prefectural Colony, 713-8 Kagiya, Kasugai,
Aichi 480-0392, Japan

## Introduction

Extracorporeal membrane oxygenation (ECMO) is an important strategy in the treatment of neonates with severe cardiorespiratory failure, including persistent pulmonary hypertension of the newborn (PPHN) [4, 18]. When venoarterial (VA) ECMO is employed, the right internal carotid artery (Rt-ICA) is cannulated and ligated. Although the VA mode is effective in supporting gas exchange, it is likely to be associated with serious complications, especially intracranial hemorrhage (ICH) [8]. Decreased blood flow and embolism to the right middle cerebral artery (MCA) is believed to be one of the main causes of ICH [21]. Recently, venovenous (VV) ECMO has been used by many authors. In contrast to the VA method, VV ECMO does not require carotid arterial ligation and seems to have few negative effects on cerebral blood flow (CBF) [9]. Furthermore, VA ECMO sometimes induces an ischemic cardiomyopathy due to an inadequate myocardial oxygen supply [7].

The purpose of this study was to evaluate the cerebral hemodynamics and cardiac function during VA and VV ECMO.

## Patients and methods

The records of patients treated with ECMO at the Central Hospital, Aichi Prefectural Colony, Japan over the past 4 years were

reviewed. Patients were selected for ECMO if they had severe respiratory distress unresponsive to maximal medical management and met our entry criteria [18]. Diagnoses included meconium aspiration syndrome, sepsis, and congenital diaphragmatic hernia. No patient had evidence of ICH before ECMO. The clinical data of the patients and ECMO are summarized in Table 1.

Fourteen neonates were placed on VA ECMO, mainly before 1994; 7 of them developed an ICH. In contrast, of 19 neonates placed on VV ECMO from 1994 only 1 developed an ICH. The patients on VA ECMO were classified into two groups: those with (group 2) and without (group 1) ICH. The clinical and ECMO data of the two groups are shown in Table 2.

In VA ECMO, infants were treated by cannulation of the Rt common carotid artery (CCA) and jugular vein. Venous blood was drained from the right atrium into the circuit and pumped by a roller pump (HAD-101, Senko Medical Instrument Co., Japan) through a membrane oxygenator and heat exchanger (Mera Silox-SHSO-0.5, MSH-51, Senko Medical Instrument Co., Japan). Oxygenated blood was returned to the aortic arch. In 14 of 19 infants with VV ECMO, a double-lumen cannula was introduced into the Rt internal jugular vein to the midportion of the Rt atrium with supplemental insertion of another catheter to the cephalic portion. In the 5 other cases, after failure to cannulate the double-lumen catheter, a thin-wall catheter was inserted into the Rt jugular vein as a drainage route and another into the umbilical vein as a reinfusion route.

No attempt was made to repair the vessels after ECMO. Systemic heparin was titrated to an activated clotting time (ACT) of 180 to 220 S (Hemochron, International Technidyne Corporation, Edison, NJ). Blood and platelets were transfused to maintain hemoglobin concentrations greater than 10 g/dl and platelet counts greater than 100,000/mm$^3$.

Serial cranial and cardiac ultrasonography (US) was carried out before cannulation and 1, 6, 12, and 24 h and 2 and 3 days after ECMO using a EUB-565 (Hitachi Medico Co., Tokyo). Pulsations were obtained as color flow in many parts of the brain; the anterior cerebral artery (ACA), both ICAs, the basilar artery (BA), and both MCAs. Using a built-in analyzer, the mean cerebral blood velocities were calculated on representative tracings of the ACA, Rt and Lt-ICA, BA, and Rt and Lt-MCA. The cardiac performance indexes examined were cardiac output (CO), stroke volume (SV), and ejection fraction (EF) of the left ventricle. Systolic arterial blood pressure (SBP) was measured at the descending aorta.

Variable values are presented as mean ± one standard deviation (SD) for each group. Group differences for continuous data were examined by means of independent Student's *t*-test. The critical $\alpha$ level for all tests was 0.05.

## Results

### Cerebral blood flow velocity (CBFV)

The mean CBFV in the ACA in VV cases tended to be higher than in VA cases up to 3 days of ECMO, and was statistically significant at 6 and 12 h of ECMO (Fig. 1). The mean CBFV in the Lt-ICA was significantly increased in VA compared with VV cases, particularly at 1 h of ECMO (Fig. 2). Before and during ECMO, the mean CBFV in the BA in VA cases tended to be higher

**Table 1** Clinical data of patients with venoarterial (VA) and venovenous (VV) ECMO (*n.s.* not significant)

| | VA ECMO | VV ECMO | *P* value |
|---|---|---|---|
| No. of patients | 14 | 19 | |
| Male | 8 | 16 | |
| Female | 6 | 3 | |
| Gestational age | 38 weeks 5 days ± 15 days | 39 weeks 4 days ± 7 days | n.s. |
| Birth weight (g) | 3,090 ± 405 | 3,220 ± 499 | n.s. |
| Diagnoses | | | |
| Meconium aspiration syndrome | 2 | 7 | |
| Congenital diaphragmatic hernia | 9 | 7 | |
| Sepsis | 3 | 4 | |
| Persistent pulmonary hypertention newborn | | 1 | |
| Intracranial hemorrhage | 7 | 1 | |
| Alive | 7 | 15 | |
| Dead | 7 | 4 | |
| Onset of ECMO (h) | 17 ± 11 | 31 ± 26 | <0.05 |
| Duration (h) | 127 ± 108 | 137 ± 115 | n.s. |

**Table 2** Clinical data of patients in VA ECMO groups 1 and 2 (*n.s.* not significant)

| | Group 1 | Group 2 | *P* value |
|---|---|---|---|
| No. of patients | 7 | 7 | |
| Male | 3 | 5 | |
| Female | 4 | 2 | |
| Gestational age | 40 weeks 1 day ± 6 days | 37 weeks 1 day ± 13 days | <0.01 |
| Birth weight (g) | 3,190 ± 331 | 2,990 ± 471 | n.s. |
| Diagnoses | | | |
| Meconium aspiration syndrome | 2 | 0 | |
| Congenital diaphragmatic hernia | 3 | 6 | |
| Sepsis | 2 | 1 | |
| Intracranial hemorrhage | 0 | 7 | |
| Alive | 7 | 0 | |
| Dead | 0 | 7 | |
| Onset of ECMO (h) | 19 ± 10 | 16 ± 12 | n.s. |
| Duration (h) | 58 ± 17 | 195 ± 119 | <0.05 |

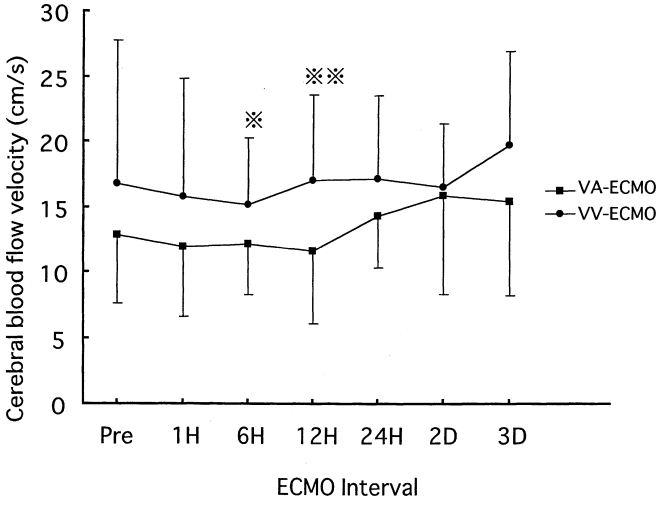


**Fig. 1** Mean cerebral blood flow velocities in anterior cerebral artery before and during venoarterial (VA) and venovenous (VV) ECMO (※:$P < 0.05$, ※※:$P < 0.01$)

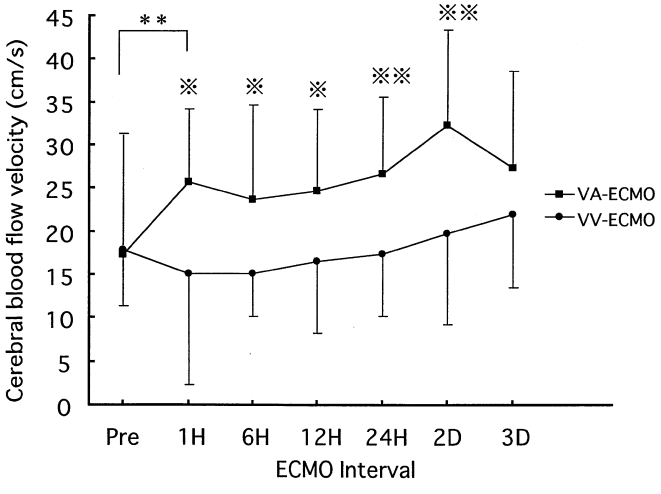


**Fig. 2** Mean cerebral blood flow velocities in left internal carotid artery before and during venoarterial (VA) and venovenous (VV) ECMO (※$P < 0.05$, ※※$P < 0.01$, **$P < 0.01$ [VA-ECMO])

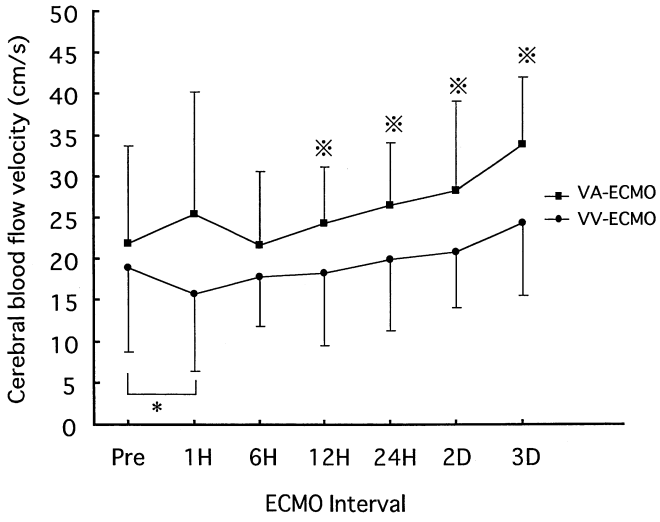


**Fig. 3** Mean cerebral blood flow velocities in basilar artery before and during venoarterial (VA) and venovenous (VV) ECMO (※:$P < 0.05$, *:$P < 0.05$ [VV-ECMO])

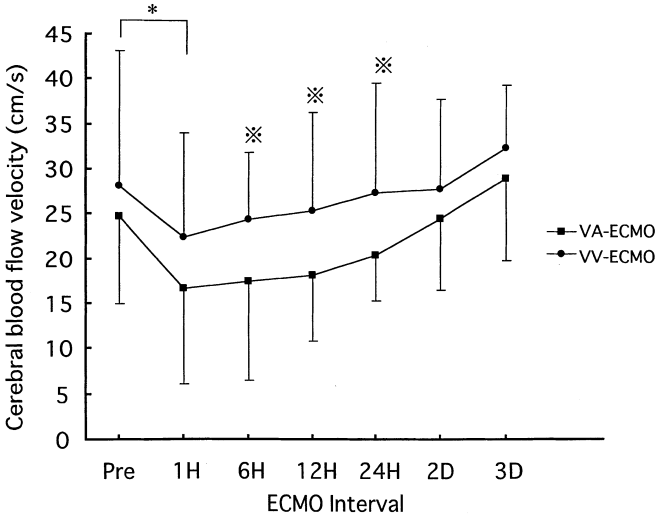


**Fig. 4** Mean cerebral blood flow velocities in right middle cerebral artery (Rt-MCA) before and during venoarterial (VA) and venovenous (VV) ECMO (※:$P < 0.05$, *: $P < 0.05$ [VV-ECMO])

than in VV cases, and was significantly higher at 12 and 24 h and 2 and 3 days of ECMO (Fig. 3). The mean CBFV in the Rt-MCA in VA cases tended to be lower than in VV cases, and was significantly lower at 6, 12, and 24 h of ECMO. In particular, the CBFV in the Rt-MCA in VA cases was less than 20 cm/s from 1 to 12 h of ECMO (Fig. 4). Although mean CBFV in the Lt-MCA also had tended to be lower in VA than in VV cases before and during ECMO, there were no statistically significant differences. The CBFV in the Lt-MCA in VA cases was less than 20 cm/s at 6 and 12 h of ECMO (Fig. 5).

The CBFs was not significantly different between groups 1 and 2 in the ACA and Rt and Lt-ICA (Figs. 6–8), but was significantly increased in the Lt-ICA at 1 h of ECMO in both groups. The mean CBFV in the BA was significantly higher in group 1 than group 2 at 6, 12, and 24 h and 3 days of ECMO; in particular, it was significantly increased in group 1 and decreased in group 2 at 1 h of ECMO (Fig. 9). The same tendencies were observed in the Rt and Lt-MCAs, in particular, the CBFV in group 2 did not exceed 20 cm/s from 1 to 24 h of ECMO (Figs. 10 and 11).

## Cardiac function

The EFs in VA cases were significantly lower than those in VV cases before and during ECMO. The CO in VA cases tended to be lower than in VV cases before and during ECMO, and was statistically significant at 6, 12,

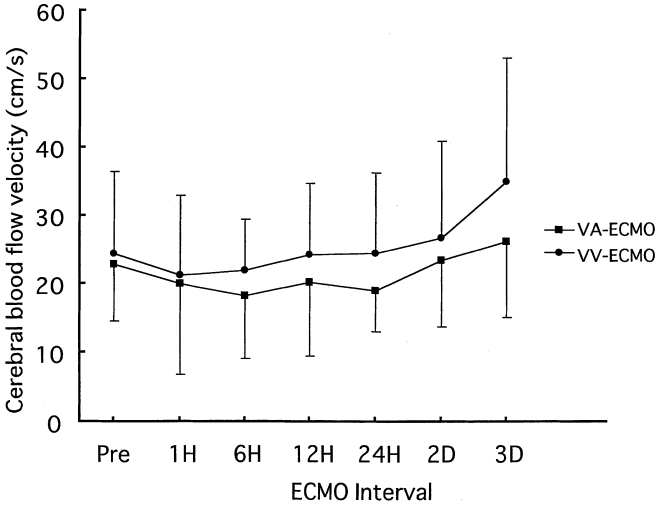


**Fig. 5** Mean cerebral blood flow velocities in left middle cerebral artery before and during venoarterial (VA) and venovenous (VV) ECMO

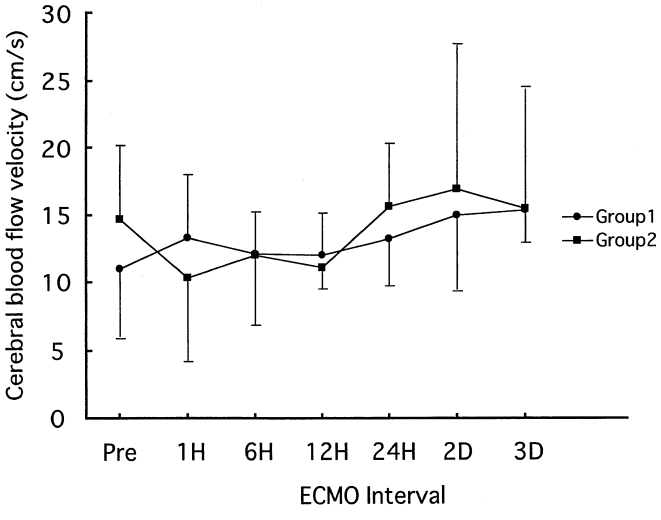


**Fig. 6** Mean cerebral blood flow velocities in anterior cerebral artery before and during venoarterial ECMO in groups 1 and 2

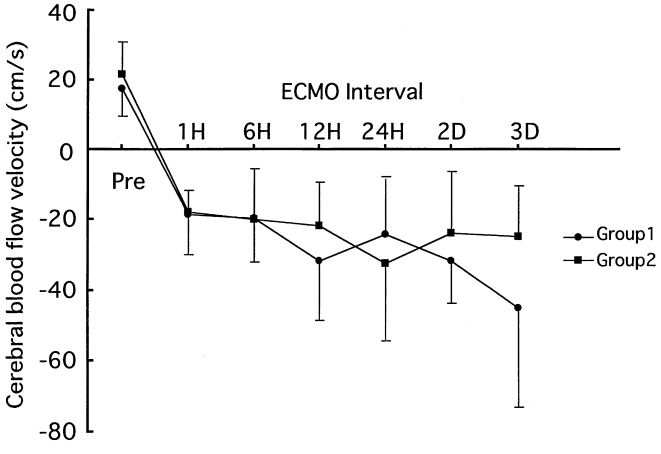


**Fig. 7** Mean cerebral blood flow velocities in right internal carotid artery before and during venoarterial ECMO in groups 1 and 2

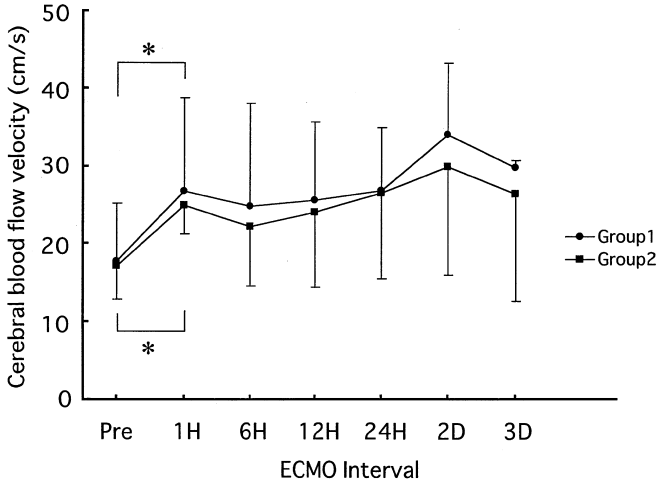


**Fig. 8** Mean cerebral blood flow velocities in left internal carotid artery before and during venoarterial ECMO in groups 1 and 2 (*: $P < 0.05$)

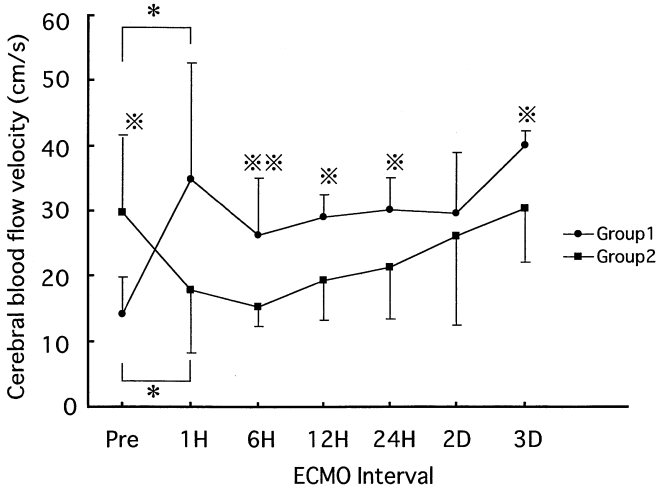


**Fig. 9** Mean cerebral blood flow velocities in basilar artery before and during venoarterial ECMO in groups 1 and 2 (※: $P < 0.05$, ※※: $P < 0.01$, *: $P < 0.05$)

and 24 h and 2 and 3 days of ECMO. The same tendency was observed in SVs. ECMO flows in VV cases were significantly higher than those in VA cases at 12 and 24 h and 2 days of ECMO. There were no differences in SBP between the VA and VV groups before and during ECMO. These data are summarized in Table 3.

The EFs in group 1 tended to be higher than those in group 2 during ECMO, becoming significantly higher at 6, 12, and 24 h and 2 days of ECMO. The CO in group 1 showed the same trend, but no significant differences were found between the groups. The SVs in group 1 showed a similar tendency. There were no differences in SBP between the two groups before and during ECMO. These data are summarized in Table 4.

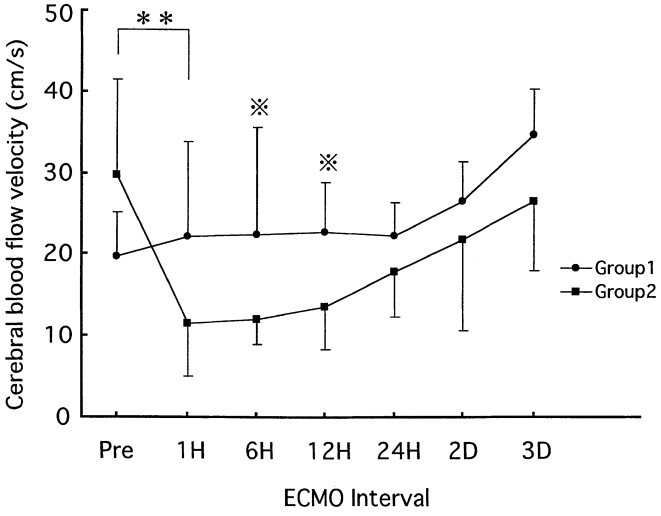


**Fig. 10** Mean cerebral blood flow velocities in right middle cerebral artery before and during venoarterial ECMO in groups 1 and 2 (※: $P < 0.05$, **: $P < 0.01$ [Group 2])

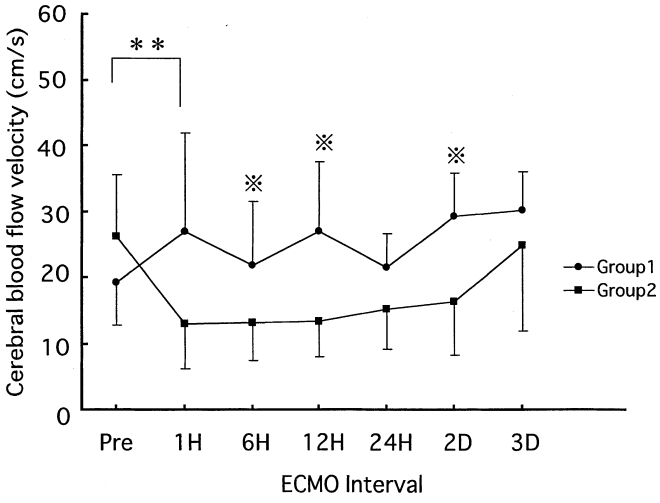


**Fig. 11** Mean cerebral blood flow velocities in left middle cerebral artery before and during venovenous ECMO in groups 1 and 2 (※: $P < 0.05$, **: $P < 0.01$ [Group 2])

## Discussion

ECMO has been recognized to be effective for neonates with severe respiratory failure; however, it is not without risks [12]. Factors predisposing to complications especially ICH, are believed to include hypoxia, hypercapnia, systemic asphyxia, high ventilator requirements before ECMO, systemic heparinization, and alterations in CBF due to ligation of the Rt-ICA during ECMO [16, 23]. Many patients have cardiorespiratory distress refractory to maximal conventional ventilatory and medical support before ECMO. Furthermore, induction of ECMO may result in significant fluctuations in systemic hemodynamics, particularly in the cerebral circulation [14]. Doppler US offers noninvasive and continuous bedside investigation of CBFV [27].

Ligation of the right (CCA) is necessary for standard VA ECMO. There are a few reports on the effects of VA ECMO on brain hemodynamics [3, 31]. CBFs of survivors evaluated using US at 1 to 11 years of age [13, 29] suggested that collateral circulation was established after the CCA was ligated, but the authors did not express opinions as to a risk between ligation of the CCA and ICH during and just after ECMO. A study done just after ligation of the CCA in a healthy lamb suggests that the initiation of VA bypass under normothermic conditions did not alter CBF or oxygen metabolism [25].

**Table 3** Comparison of cardiac function in venoarterial (VA) and venovenous (VV) ECMO (*n.s.* not significant)

| | Ejection fraction (%) | | | Cardiac output (l/min) | | |
|---|---|---|---|---|---|---|
| | VA ECMO | VV ECMO | *P* value | VA ECMO | VV ECMO | *P* value |
| Pre-ECMO | 26.5 ± 27.0 | 44.6 ± 22.0 | n.s. | 0.30 ± 0.37 | 0.46 ± 0.03 | n.s. |
| 1 h | 34.1 ± 15.3 | 46.9 ± 16.7 | <0.05 | 0.49 ± 0.28 | 0.43 ± 0.22 | n.s. |
| 6 h | 22.5 ± 16.8 | 46.1 ± 14.4 | <0.001 | 0.25 ± 0.27 | 0.49 ± 0.33 | <0.05 |
| 12 h | 32.4 ± 22.7 | 47.9 ± 18.3 | <0.05 | 0.30 ± 0.23 | 0.48 ± 0.23 | <0.05 |
| 24 h | 23.3 ± 17.7 | 49.2 ± 19.1 | <0.001 | 0.26 ± 0.20 | 0.45 ± 0.26 | <0.05 |
| 2 days | 24.8 ± 13.4 | 47.4 ± 13.9 | <0.001 | 0.25 ± 0.20 | 0.52 ± 0.24 | <0.01 |
| 3 days | 26.2 ± 20.2 | 53.0 ± 14.1 | <0.01 | 0.26 ± 0.10 | 0.50 ± 0.23 | <0.05 |

| Stroke volume (ml) | | | ECMO flow (ml/kg · min) | | | Systolic blood pressure (mmHg) | | |
|---|---|---|---|---|---|---|---|---|
| VA ECMO | VV ECMO | *P* value | VA ECMO | VV ECMO | *P* value | VA ECMO | VV ECMO | *P* value |
| 1.62 ± 1.92 | 3.03 ± 1.79 | n.s. | – | – | | 64 ± 20 | 63 ± 14 | n.s. |
| 2.52 ± 1.92 | 2.72 ± 1.43 | n.s. | 88 ± 13 | 94 ± 24 | n.s. | 65 ± 8 | 62 ± 13 | n.s. |
| 1.65 ± 2.08 | 3.44 ± 2.23 | <0.05 | 84 ± 14 | 96 ± 25 | n.s. | 63 ± 12 | 68 ± 12 | n.s. |
| 2.04 ± 1.96 | 3.33 ± 1.53 | n.s. | 80 ± 11 | 100 ± 27 | <0.01 | 70 ± 7 | 72 ± 13 | n.s. |
| 1.76 ± 1.83 | 3.38 ± 1.85 | <0.05 | 72 ± 8 | 95 ± 25 | <0.01 | 75 ± 7 | 73 ± 14 | n.s. |
| 1.86 ± 1.80 | 3.84 ± 2.01 | <0.01 | 62 ± 19 | 88 ± 24 | <0.01 | 79 ± 12 | 73 ± 14 | n.s. |
| 1.72 ± 1.33 | 3.72 ± 2.01 | <0.01 | 71 ± 24 | 88 ± 27 | n.s. | 80 ± 19 | 75 ± 12 | n.s. |

**Table 4** Comparison of cardiac function in groups 1 and 2 (*n.s.* not significant)

| | Ejection fraction (%) | | | Cardiac output (l/min) | | |
|---|---|---|---|---|---|---|
| | Group 1 | Group 2 | *P* value | Group 1 | Group 2 | *P* value |
| Pre-ECMO | 23.7 ± 19.7 | 26.0 ± 33.6 | n.s. | 0.50 ± 0.45 | 0.10 ± 0.05 | n.s. |
| 1 h | 34.2 ± 8.5 | 31.0 ± 20.9 | n.s. | 0.63 ± 0.26 | 0.32 ± 0.24 | n.s. |
| 6 h | 30.7 ± 17.1 | 12.0 ± 9.8 | <0.05 | 0.39 ± 0.33 | 0.10 ± 0.07 | n.s. |
| 12 h | 49.3 ± 19.8 | 15.7 ± 6.3 | <0.01 | 0.43 ± 0.27 | 0.18 ± 0.13 | n.s. |
| 24 h | 29.5 ± 22.9 | 14.5 ± 6.2 | <0.05 | 0.33 ± 0.26 | 0.18 ± 0.11 | n.s. |
| 2 days | 31.1 ± 12.5 | 16.8 ± 8.8 | <0.05 | 0.35 ± 0.24 | 0.20 ± 0.11 | n.s. |
| 3 days | 34.8 ± 22.5 | 26.9 ± 21.2 | n.s. | 0.25 ± 0.13 | 0.30 ± 0.23 | n.s. |

| Stroke volume (ml) | | | ECMO flow (ml/kg · min) | | | Systolic blood pressure (mmHg) | | |
|---|---|---|---|---|---|---|---|---|
| Group 1 | Group 2 | P value | Group 1 | Group 2 | *P* value | Group 1 | Group 2 | *P* value |
| 2.62 ± 2.40 | 0.62 ± 0.36 | n.s. | | | | 69 ± 5 | 59 ± 30 | n.s. |
| 3.85 ± 1.91 | 1.19 ± 0.47 | <0.05 | 88 ± 15 | 89 ± 12 | n.s. | 63 ± 5 | 68 ± 10 | n.s. |
| 2.65 ± 2.66 | 0.66 ± 0.50 | n.s. | 88 ± 13 | 78 ± 15 | n.s. | 62 ± 8 | 64 ± 17 | n.s. |
| 3.17 ± 2.52 | 1.14 ± 0.79 | n.s. | 86 ± 11 | 72 ± 4 | <0.05 | 71 ± 3 | 70 ± 11 | n.s. |
| 2.37 ± 2.49 | 1.15 ± 0.69 | n.s. | 77 ± 23 | 65 ± 7 | n.s. | 78 ± 7 | 73 ± 7 | n.s. |
| 2.77 ± 2.26 | 0.95 ± 0.34 | n.s. | 57 ± 22 | 68 ± 15 | n.s. | 82 ± 9 | 73 ± 17 | n.s. |
| 2.15 ± 1.53 | 1.55 ± 1.39 | n.s. | 87 ± 25 | 63 ± 24 | n.s. | 85 ± 6 | 78 ± 24 | n.s. |

However, candidates may be exposed to prolonged hypoxia and hypercapnia before ECMO [1].

Schumacher et al. reported eight survivors of neonatal ECMO with varying degrees of right hemispheric brain injury, and indicated that right-sided brain injury might be associated with Rt-CCA ligation for ECMO, but they also suggested that the risk factors before ECMO could not be ignored, because some subjects needed cardiopulmonary resuscitation due to severe hypoxia before ECMO [22]. Kirkpatrick et al. reported a similar opinion [12]. Taylor et al. claimed that the risk for cerebrovascular injury associated with ECMO is multifactorial and not necessarily related to the ligation of the Rt-CCA [28]. These analyses in the literature indicate that brain injuries occurring during ECMO may be due to both pre-ECMO situations as well as ECMO practices [6].

Campbell et al. reported decreased blood flow in the Rt-MCA during VA ECMO [5]. Therefore, a transient decrease in CBF may play a role in brain injury. Some of our patients undergoing VA ECMO showed a transient decrease in SBP with rapid recovery, and others showed poor or almost no recovery of CBFV, especially in the ACA and the Rt and Lt-MCAs. These arteries are so-called distal arteries compared with proximal Rt and Lt-ICAs and the BA, whose flow velocities increased after the onset of ECMO. Especially in VA ECMO group 2, the mean CBFV in both MCAs was under approximately 20 cm/s during ECMO, and the infants showed subsequent intracranial complications. These data suggest that a decrease in CBFV in the Rt and Lt-MCAs throughout ECMO may be followed by ICH.

Autoregulation of the cerebral circulation is an important homeostatic mechanism in neonates [15, 17]. Normal term autoregulation is believed to maintain constant perfusion pressures to the brain in spite of varying arterial pressures. Severely high-risk infants can have disrupted cerebral autoregulation, however, leaving the cerebral microcirculation vulnerable to alterations in systemic blood pressure [30], which may result in fluctuations in cerebral hemodynamics [20]. Hypotension can cause ischemic cerebral damage and hypertension can cause cerebral hyperemia, all of which pose risks for ICH [24]. From our data correlating cardiac failure and low CBFV in VA ECMO, we infer that hypoxia prior to ECMO was accompanied by a loss of cerebral autoregulation, especially in cases of ICH.

VA ECMO is associated with an initial decrease in left ventricular ejection phase indices [11]. Although left ventricular preload may be decreased with the initiation of VA ECMO because a large portion of blood is bypassing the heart, cardiac dysfunction might be induced by an ischemic cardiomyopathy. The so-called stunned myocardium during ECMO may be caused by hypovolemia in the cardiopulmonary circulation and ischemic cardiomyopathy [7, 10]. Infants in the present study who developed ICH had low CBFV associated with disturbed cardiac function. Thus, we deduce that a decrease in CBF secondary to myocardial failure can result in cerebral hemorrhage. When the mean CBFV in the MCA fell successively below 20 cm/s, the infant had a high risk of ICH.

A double-lumen catheter for neonates has recently been developed [2]. It was reported that supplemental cephalic jugular venous drainage during neonatal ECMO appeared to be safe, and might decrease the incidence of ICH [19]. Several cardiopulmonary vascular benefits might be expected to result from VV as opposed to VA ECMO [26]. VV ECMO could be advantageous since it perfuses oxygenated blood into not only the

pulmonary, but also the coronary vessels. In the present study, all infants had normalization and no infant had a deterioration in cardiac function as a result of VV ECMO. It has been suggested that VV ECMO does not have deleterious effects on cardiac performance [26]. In our only case of cerebral hemorrhage on VV ECMO, the velocity in each of the cerebral arteries was higher compared with the cases without cerebral complications, suggesting a loss of cerebral autoregulation before the onset of ECMO. This infant also suffered from severe asphyxia before ECMO, with an almost flat and irreversible EEG.

In conclusion, neonatal patients with severe pulmonary failure can be effectively supported by VV ECMO. In addition to stable hemodynamics of the brain compared with VA ECMO, it has advantages in myocardial and pulmonary vascular oxygenation, resulting in favorable cerebral hemodynamics.

## References


1. von Allmen D, Babcock D, Matsumoto J, et al (1992) The predictive value head ultrasound in the ECMO candidate. J Pediatr Surg 27: 36–39
2. Anderson HL III, Snedecor SM, Otsu T, et al (1993) Multicenter comparison of conventional venoarterial access versus venovenous double-lumen catheter access in newborn infants undergoing extracorporeal membrane oxygenation. J Pediatr Surg 28: 530–535
3. Andrews AF, Nixon CA, Cilley RE, et al (1986) One- to three-year outcome for 14 neonatal survivors of extracorporeal membrane oxygenation. Pediatrics 78: 692–698
4. Bartlett RH, Roloff DW, Cornell RG, et al (1985) Extracorporeal circulation in neonatal respiratory failure: a prospective randomized study. Pediatrics 76: 479–487
5. Campbell LR, Bunyapen C, Holmes GL, et al (1988) Right common carotid artery ligation in extracorporeal membrane oxygenation. J Pediatr 113: 110–113
6. Campbell LR, Bunyapen C, Gangarosa ME, et al (1991) Significance of seizures associated with extracorporeal membrane oxygenation. J Pediatr 119: 789–792
7. Cater G, Lotze A, Miller M, et al (1988) Stunned myocardium in an infant treated with extracorporeal membrane oxygenation. J Pediatr Surg 23: 1011–1013
8. Cilley RE, Zwischenberger JB, Andrews AF, et al (1986) Intracranial hemorrhage during extracorporeal membrane oxygenation in neonates. Pediatrics 78: 699–704
9. Cornish JD, Heiss KF, Clark RH, et al (1993) Efficacy of venovenous extracorporeal membrane oxygenation for neonates with respiratory and circulatory compromise. J Pediatr 122: 105–109
10. Dickson ME, Hirthler MA, Simoni J, et al (1990) Stunned myocardium during extracorporeal membrane oxygenation. Am J Surg 160: 644–646
11. Kimball TR, Daniels SR, Weiss RG, et al (1991) Changes in cardiac function during extracorporeal membrane oxygenation for persistent pulmonary hypertension in the newborn infant. J Pediatr 118: 431–436
12. Kirkpatrick BV, Krummel TM, Mueller DG, et al (1983) Use of extracorporeal membrane oxygenation for respiratory failure in term infants. Pediatrics 72: 872–876
13. Krummel TM, Greenfield LJ, Kirkpatrik BV, et al (1984) The early evaluation of survivors after extracorporeal membrane oxygenation for neonatal pulmonary failure. J Pediatr Surg 19: 586–590
14. Liem KD, Hopman JCW, Oeseburg B, et al (1995) Cerebral oxygenation and hemodynamics during induction of extracorporeal membrane oxygenation as investigated by near infrared spectrophotometry. Pediatrics 95: 555–561
15. Lou HC, Lassen NA, Friis-Hansen B (1979) Impaired autoregulation of cerebral blood flow in the distressed newborn infant. J Pediatr 94: 118–121
16. McManus ML, Kevy SV, Bower LK, et al (1995) Coagulation factor deficiencies during initiation of extracorporeal membrane oxygenation. J Pediatr 126: 900–904
17. Milligan DWA (1980) Failure of autoregulation and intraventricular haemorrhage in preterm infants. Lancet 26: 896–898
18. Nagaya M, Tsuda M, Futamura M (1993) The efficacy of extracorporeal membrane oxygenation (ECMO) in newborns with septic shock. Pediatr Surg Int 8: 298–302
19. O'Connor TA, Haney BH, Grist GE, et al (1993) Decreased incidence of intracranial hemorrhage using cephalic jugular venous drainage during neonatal extracorporeal membrane oxygenation. J Pediatr Surg 28: 1332–1335
20. Pape KE (1989) Etiology and pathogenesis of intraventricular hemorrhage in newborns. Pediatrics 84: 382–385
21. Schumacher RE, Barks JDE, Johnston MV, et al (1988) Right-sided brain lesions in infants following extracorporeal membrane oxygenation. Pediatrics 82: 155–161
22. Schumacher RE, Palmer TW, Roloff DW, et al (1991) Follow-up of infants treated with extracorporeal membrane oxygenation for newborn respiratory failure. Pediatrics 87: 451–457
23. Sell LL, Cullen ML, Whittlesey GC, et al (1986) Hemorrhagic complications during extracorporeal membrane oxygenation: prevention and treatments. J Pediatr Surg 21: 1087–1091
24. Sell LL, Cullen ML, Lerner GR, et al (1987) Hypertension during extracorporeal membrane oxygenation: cause, effect, and management. Surgery 102: 724–730
25. Short BL, Walker LK, Gleason CA, et al (1990) Effect of extracorporeal membrane oxygenation on cerebral blood flow and cerebral oxygen metabolism in newborn sheep. Pediatr Res 28: 50–53
26. Strieper MJ, Sharma S, Dooley KJ, Cornish JD, Clark RH (1993) Effects of venovenous extracorporeal membrane oxygenation on cardiac performance as determined by echocardiographic measurements. J Pediatr 122: 950–955
27. Taylor GA, Short BL, Glass P, et al (1988) Cerebral hemodynamics in infants undergoing extracorporeal membrane oxygenation: further observations. Radiology 168: 163–167
28. Taylor GA, Short BL, Fitz CR (1989) Imaging of cerebrovascular injury in infants treated with extracorporeal membrane oxygenation. J Pediatr 114: 635–639
29. Towne BH, Lott IT, Hicks DA, et al (1985) Long-term follow-up of infants and children treated with extracorporeal membrane oxygenation (ECMO): a preliminary report. J Pediatr Surg 20: 410–414
30. Tweed A, Cote J, Lou H, et al (1986) Impairment of cerebral blood flow autoregulation in the newborn lamb by hypoxia. Pediatr Res 20: 516–519
31. Wildin SR, Landry SH, Zwischenberger JB (1994) Prospective, controlled study of development outcome in survivors of extracorporeal membrane oxygenation: the first 24 months. Pediatrics 93: 404–408