

Original Article

## ***Hematological Abnormalities in Neonatal Patients Treated with Extracorporeal Membrane Oxygenation (ECMO)***

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### **ABSTRACT**

The physical process of extracorporeal membrane oxygenation (ECMO) results in derangement of the hemostatic mechanism, which may lead to increased morbidity, secondary to the disease process. The purpose of this study was to evaluate the hematological status of neonates undergoing ECMO therapy, and to evaluate coagulation tests in predicting hemorrhagic risk.

Following Institutional Review Board approval, 30 patients undergoing ECMO treatment were retrospectively entered into this study. Medical records were reviewed and indicators of hemostasis, transfusion, morbidity, and outcomes recorded. Assessment of coagulation was determined through serial analysis of platelet count, fibrinogen concentration, prothrombin time (PT), activated partial thromboplastin time (aPTT), antithrombin III, fibrin split products, D-dimers, plasma free hemoglobin, activated clotting time, ionized calcium, and thrombelastography (TEG).

Median total transfusion requirements for all patients were 1.79 ml/kg/ECMO hr. Fifty-seven percent of the 30 patients were diagnosed as coagulopathic according to Extracorporeal Life Support Organization standards. Patients were separated into either a hemorrhagic group (HEM, >2.0 ml/kg/ECMO hr, n=13) or a nonhemorrhagic group (N-HEM, n=17), with HEM patients requiring twice the transfusion volume of N-HEM ( $p<0.0001$ ). Hemorrhagic complications were reported in 53.8% of the HEM patients vs. 35.3% in the N-HEM group. HEM patients were transfused with significantly greater quantities of platelets on days 1,3,5, and 8 and packed red blood cells on day 7 when compared to N-HEM ( $p<0.05$ ). TEG determination showed significant differences between groups on days 3 and 6 ( $p<0.005$ ), and 8 ( $p<0.05$ ).

Derangements in hemostasis resulting from ECMO are profound, with methods of assessing coagulation complicated by both the variability in patient condition and lack of specificity of laboratory tests. Interpretation of TEG data has shown to be a valuable supplement for managing this challenging patient population.

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## INTRODUCTION

Bartlett and his associates reported the first successful clinical use of neonatal extracorporeal membrane oxygenation (ECMO) for the treatment of respiratory failure in 1976 (1). The primary diagnosis of neonates treated with ECMO includes meconium aspiration syndrome, respiratory distress syndrome/hyaline membrane disease, other aspiration syndromes, perinatal asphyxia, congenital pneumonia/sepsis and congenital diaphragmatic hernia, with persistent pulmonary hypertension almost always a major contributing factor (2,3). Nearly 8,000 cases of neonatal ECMO were reported to the Extracorporeal Life Support Organization (ELSO) between 1973 and 1994, with a survival rate to the time of discharge of 81%. It is estimated that without ECMO, only 20% of those children would have survived (4). Eighty-nine percent of the 120 neonates reported by Keszler (3) from 1985-1990 survived their ECMO course to be discharged home, with 88% of the 198 neonates reported by Nagaraj et al. (5) from 1985-1991 surviving to the time of discharge.

Despite these successes, however, ECMO is extremely invasive and there are inherent risks associated with the extracorporealization of blood. Mechanical complications can occur within the extracorporeal circuit including clot development, cannula problems, or oxygenator failures (4). Other potential complications include internal hemorrhage (particularly intracranial hemorrhage), cerebral infarction, renal failure, seizures, and even the accidental introduction of an air embolism (3). Thirty-five percent of the patients reported to ELSO between 1973 and 1994 suffered neurological complications, 43% suffered cardiopulmonary complications, 32% metabolic, 25% renal, and 9% infectious (4). It is poorly understood whether the complications are a direct result of ECMO, the underlying disease process, or both.

One of the most serious complications seen with ECMO, and one in which a direct relationship with extracorporeal circulation is known, is hemorrhage (3, 6, 7). Zwischenberger et al. (4) reported bleeding complications in 35% of ECMO cases. Shanley et al. (2) reported 102 instances of intracranial infarct, intracranial hemorrhage, or other major bleeding in 460 ECMO patients treated over 20 years at one institution. Nagaraj et al. (5) reported a significantly higher ( $p < 0.001$ ) mortality rate in their ECMO patients with hemorrhagic or thoracic complications compared to those without the complications. The most serious form of hemorrhage is intracranial bleeding, with an occurrence rate in ECMO patients reported between 9.7 and 16% (3, 5, 8). Recent improvements in the methods of ECMO treatment have reduced overall hemorrhagic morbidity. In cases reported to the ELSO Registry between 1988-1991, the incidence of intracranial hemorrhage in premature infants ( $\leq 34$  weeks) decreased from nearly 100%, previously, to 37%, and the survival rate increased from 25% to 63% (9).

It is known that ECMO causes platelet consumption and decreased platelet function, probably due to the "whole body

inflammatory reaction" associated with extracorporeal circulation, and the relatively large priming volume of the extracorporeal circuit (7,10). The hemostatic mechanism is altered during ECMO due to a number of factors, including systemic heparinization, intravascular and extracorporeal thrombogenicity, qualitative and quantitative platelet disorders, hyperfibrinolysis, enhanced coagulation factor consumption, and disseminated intravascular coagulation (6,10). Because of the increased bleeding with ECMO, patients treated with the modality often receive significant transfusions of blood products, higher than other critically ill patients, which increases the risk of transfusion-related complications (11). Understanding the etiologies of hematological abnormalities and formulating effective intervention strategies can decrease the need for blood transfusions, decrease morbidity, and possibly extend the indications for ECMO. Since hematological complications are directly related to increased morbidity and mortality, the present study retrospectively reviewed the hematological status of patients treated with ECMO for respiratory failure at the University of Nebraska Medical Center between 1992 and 1997.

## MATERIALS AND METHODS

### POPULATION

Thirty neonatal patients undergoing standard ECMO therapy at the University of Nebraska Medical Center between 1992 and 1997 for respiratory failure were studied following Institutional Review Board approval. University of Nebraska Medical Center ECMO entrance requirements were similar to those reported elsewhere (3, 12) and included: body weight  $> 2$  kg, reversible lung or cardiac disease, and an 80% chance of mortality without ECMO based upon respiratory criteria.

### ECMO CIRCUIT AND MANAGEMENT

The ECMO circuit was similar to that described by Shanley et al. (2), consisting of siphon drainage from a venous cannula to a collapsible bladder, a roller pump, a coiled silicone membrane blood gas exchange device<sup>a</sup>, and a heat exchanger<sup>a</sup>. The pump was servo-regulated by a microswitch sensor in contact with the venous bladder. The priming volume of the circuit was approximately 600 mL, and consisted of 220 mL of fresh red blood cells ( $< 7$  days old), 50 mL of salt poor albumin, 330 mL of a balanced electrolyte solution and 100 IU bovine heparin. Blood flow rates ranged from 100 to 125 mL/kg/min, with adequate perfusion determined by venous oxyhemoglobin saturations and blood gas values. Blood flow was decreased in timed intervals during ECMO weaning. The patient was given a trial without ECMO once ECMO was providing minimal support. If the trial off was successful, the cannulas were removed.

### HEMOSTATIC CONTROL

Daily hemostatic monitoring included the following: acti-

a AVECOR, Plymouth, MN 55440

vated clotting time (every hr), platelet count (4 hrs), fibrinogen concentration, prothrombin time (PT), activated partial thromboplastin time (aPTT), antithrombin III, fibrin(ogen) split products (FSP), D-dimers, and thrombelastography<sup>b</sup> (TEG) (24 hrs). In addition, tests were performed when coagulopathies or increased transfusions were experienced.

Thrombelastographs were performed using heparinase, an enzyme used to degrade heparin. Parameters measured or calculated by the TEG include: R time, K time, alpha angle, maximum amplitude and TEG index. The R time marks the time until the initiation of clot. The K time and alpha angle represent clot growth kinetics and are mainly indicative of platelet function. The maximum amplitude of the wave form represents clot strength, and it is related to both platelet and clotting factor function and their interaction. The TEG index is calculated using the measured parameters and is indicative of overall coagulability. A TEG index < -2 indicates a hypocoagulable state and a TEG index > +2 indicates a hypercoagulable state. Thrombelastograph interpretation has been previously described (10).

Patients were given a 100 IU/kg body weight loading dose of heparin, then titrated with heparin to maintain an activated clotting time (ACT) between 180-220 seconds. Platelets were transfused to keep the platelet count above 100,000/mm<sup>3</sup>, fresh frozen plasma and cryoprecipitate to maintain a fibrinogen concentration 100-150 mg/dL, and packed red blood cells (PRBC's) to keep the hematocrit greater than 40%.

**GROUP CLASSIFICATION**

An earlier study (10) grouped patients into a hemorrhagic group if the total transfusion requirement over their ECMO course exceeded 1.0 mL/kg body weight/ECMO hour. Significant differences were found in packed red blood cell transfusions between the hemorrhagic group and the rest of the patients. Ninety percent of our patients received over 1.0 mL/kg/hr in total transfusions, while 13 of 30 neonates received over 2.0 mL/kg/hr. With the incidence of bleeding complications nationwide reported to be around 35% by the ELSO Registry (4), it was felt that using a cut-off of 1.0 mL/kg/hr to designate a high-risk group at a greater risk of complications might be too inclusive, with 2.0 mL/kg/hr more appropriate. Therefore, those patients receiving over 2.0 mL/kg/hr in total transfusions over their ECMO course were designated as a hemorrhagic (HEM) group, and those receiving less in transfusions were termed the nonhemorrhagic (N-HEM) group.

**DATA COLLECTION**

Mechanical and patient complications were identified by the ECMO Director (LW), who was blinded to the classification of patients according to quantifiable transfusions, and recorded according to ELSO Registry guidelines. Although all patients had some degree of hemorrhage, a hemorrhagic complication was

**Table 1: Patient demographics**

Parameter	HEM(n=13)	N-HEM (n=17)
Male/Female	5/8	13/4
Birthweight (kg)	3.10±0.53	3.26±0.72
Gestational age (wks)	37.54±8.40	39.06±3.30
Age at ECMO initiation (hrs)	46.69±75.55	58.60±69.97*
ECMO duration (hrs)	139.95±97.89	162.13±88.69
ECMO type		
VA	9	14
VV	1	3
VV to VA	3	0

\* = excludes two patients 10 and 16 weeks of age at the time of ECMO initiation; VA = venoarterial; VV = veno-venous

only assigned to a patient if they had one of the following conditions: significant gastrointestinal, cannulation site, or other surgical site bleeding requiring transfusion or reexploration or significant hemolysis recorded with plasma free hemoglobin levels exceeding 50 mg/dL. Transfusions, platelet count, and TEG data were blocked into 24 hour periods for quantification purposes.

**DATA ANALYSIS**

Data was loaded onto a personal computer in spreadsheet format. Statistical analyses on parametric data were performed using a one-way analysis of variance, with significant p values found on a multiple comparison test. A Fischer's protected least significant difference multiple comparison test was used on a commercially prepared statistics program<sup>c</sup>. Statistical analyses on nonparametric data were performed using a Pearson's chi square test. Statistical significance was accepted at the p≤0.05 level. All parametric data are presented as the mean ± the standard deviation of the mean.

**RESULTS**

The patients included 18 males and 12 females with an average birthweight of 3.19 ± 0.64 kg. The average ECMO duration was 150.5 ± 90.7 hrs. Twenty-three patients underwent veno-arterial ECMO, four underwent veno-venous ECMO, and three were converted from veno-venous to veno-arterial. A comparison of the demographics between HEM and N-HEM patients can be found in Table 1.

The most common primary diagnosis was meconium aspiration syndrome, occurring in nine of the 30 patients, with primary pulmonary hypertension noted as a secondary diagnosis in 18 cases. Table 2 breaks down the primary diagnosis of each group. No significant difference was found between groups in

b C3000, Haemoscope Corporation, Skokie, IL 60077  
 c SuperANOVA, Abacus Concepts, Inc., Berkeley, CA 94704

**Table 2: Primary diagnosis**

Diagnosis	HEM(n=13)	N-HEM(n=17)
MAS	3	6
Pneumonia/Sepsis	2	6
CDH	5	2
PPHN	3	1
RDS	0	1
Cardiac	0	1

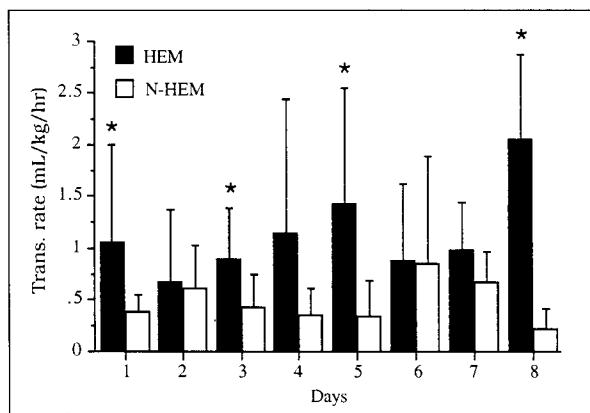
CDH = congenital diaphragmatic hernia; MAS = meconium aspiration syndrome; PPHN = primary pulmonary hypertension; RDS = respiratory distress syndrome

**Table 3: ELSO complications of UNMC patients**

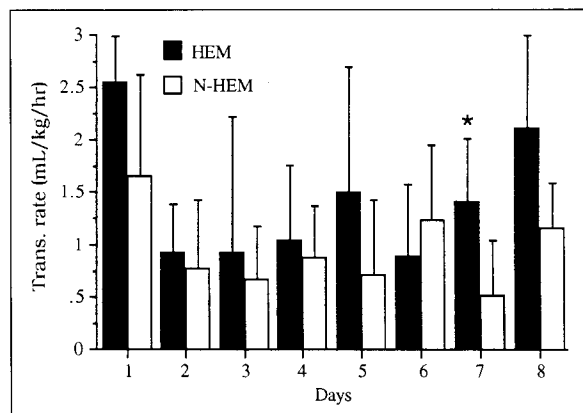
Complication	HEM(%)	N-HEM(%)
Cardiopulmonary	53.8	47.1
Neurologic	46.1	35.3
Hemorrhagic	53.8*	35.3
Mechanical	38.5	35.3
Metabolic	30.8	41.2
Renal	15.4	17.6
Infectious	15.4	17.6
Pulmonary	7.6	5.9

\*p > 0.05 vs. N-HEM

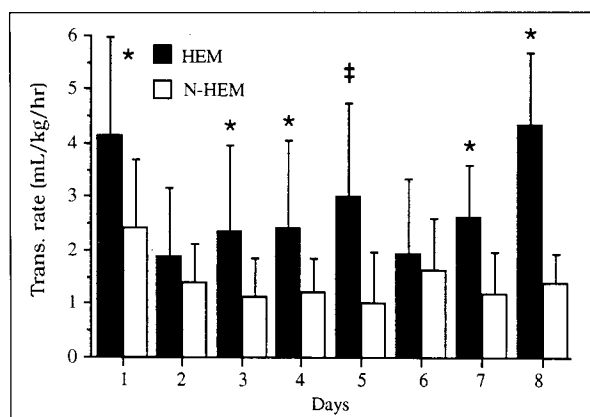
**Figure 1: (a) Platelet Transfusions (HEM vs. N-HEM); (b) PRBC Transfusions (HEM vs. N-HEM); (c) Total Transfusions (HEM vs. N-HEM)**



(a) \* = p < 0.05 vs. N-HEM



(b) PRBC = packed red blood cells; \* = p < 0.05 vs. N-HEM



(c) \* = p < 0.05 vs. N-HEM; ‡ = p < 0.001 vs. N-HEM

patient demographics or prevalence of primary diagnosis.

FSP's and D-dimers were elevated during all periods of the ECMO course, indicating enhanced fibrinolysis. Activated clotting times, PTs, and aPTTs failed to follow any trend and were not found to correspond with increased transfusions or TEG values. Mean 24 hour fibrinogen concentrations ranged from 110

mg/dL to 155 mg/dL. Plasma free hemoglobin values varied considerably, and a trend could not be identified. An insufficient number of antithrombin-III values were recorded to analyze statistically

Cardiopulmonary complications resulting from ECMO occurred in 50.0% of patients, with hemorrhagic complications occurring in 43.3%. Table 3 lists the prevalence of individual complications within each group. The incidence of hemorrhagic complications was significantly higher in the HEM group versus the N-HEM (p < 0.05). The survival rate to the time of discharge was 70.0%. Two deaths occurred while on ECMO, five additional deaths occurred within 30 days post ECMO and two patients died greater than 30 days post ECMO. The survival rate of the HEM group was 61.5%; while the survival rate of the N-HEM was 76.5%. The mean transfusion rates over the entire ECMO course, in mL blood/kg body weight/ECMO hour were as follows: platelets, 0.73 ± 0.58 mL/kg/hr, PRBC's, 1.16 ± 0.55 mL/kg/hr, fresh frozen plasma, 0.13 ± 0.16 mL/kg/hr, cryoprecipitate, 0.05 ± 0.06 mL/kg/hr and total, 2.06 ± 1.10 mL/kg/hr. The median total transfusion requirement for all patients was 1.79 mL/kg/hr.

The HEM group averaged  $2.94 \pm 1.07$  mL/kg/hr in total transfusions, while the N-HEM group averaged  $1.40 \pm 0.48$  mL/kg/hr ( $p < 0.0001$ ). HEM patients received almost three times the platelet transfusions,  $1.11 \pm 0.69$  versus  $0.44 \pm 0.20$  mL/kg/hr and almost double the PRBC's,  $1.55 \pm 0.51$  versus  $0.86 \pm 0.36$  mL/kg/hr, as N-HEM patients. Figure 1(a-c) shows significant differences in platelet, PRBC, and total transfusion requirements between the HEM group and the N-HEM group at different periods of the ECMO course.

The HEM group received a greater amount of platelets on day 8 when compared to days 1, 2, 3, and 6; while the N-HEM group had a significantly higher transfusion rate of platelets on day 6 when compared to other ECMO days ( $p < 0.05$ ). Both the HEM and N-HEM groups were transfused significantly greater amounts of PRBCs on day 1 when compared to other days ( $p < 0.05$ ). The HEM group had a significantly higher total transfusion rate on days 1 and 8 when compared to days 2, 3, 4, and 6, with the N-HEM group receiving significantly higher amounts in total transfusions on day 1 when compared to days 2, 3, 4, 5, and 7 ( $p < 0.05$ ). Mean 24-hour platelet counts ranged from 93,000 to 123,000/mm<sup>3</sup>. There was no significant difference found in platelet counts between the HEM and N-HEM groups.

Thrombelastographic determination of clot formation and stability was assessed at serial times during ECMO. All indices for clot function (R time, K time, alpha angle, maximum amplitude) declined rapidly during the first several hours of ECMO as a result of the hemodilution and extracorporealization of blood. Significant differences were found between groups in TEG values on days 3 and 6 ( $p < 0.005$ ) and 8 ( $p < 0.05$ ) (Figure 2 a-e).

The HEM group's R time was significantly prolonged on day 3 versus days 1, 2, and 5 ( $p < 0.05$ ) and the K time was significantly prolonged on days 3 and 6 versus all other days except day 8 ( $p < 0.005$ ). The alpha angle was significantly diminished on days 3, 6, and 8 versus day 1 ( $p < 0.05$ ), and the maximum amplitude was significantly decreased on day 3 versus day 1 and day 6 versus days 1, 2, 3, and 5 ( $p < 0.05$ ).

## DISCUSSION

Treatments for neonatal respiratory failure include ECMO (1, 3), mechanical ventilation (13, 14), high frequency jet and oscillatory ventilation (14, 15), pharmacological alkalization (13), nitric oxide administration (16, 17), surfactant therapy (18, 19), and liquid ventilation (20). Neonatal ECMO has been very successful since its use was first reported by Bartlett and his associates in 1976 (1). Eighty-one percent of the nearly 8,000 neonates reported to ELSO who were treated with the modality have survived to be discharged home (4). Seventy percent of the neonates in our study survived to the time of discharge.

Walsh-Sukys et al. (21) compared the survival and health outcomes of 74 neonates treated over a 24 month period at Case Western Reserve University in Cleveland, Ohio, for respiratory failure with and without ECMO. Ninety percent of the 48 pa-

tients treated with ECMO survived to 20 months, compared to only 69% of the patients treated conventionally ( $n=26$ ). While the neurodevelopmental outcomes of both groups were similar, the conventionally treated group had significantly worse pulmonary outcomes and more hospitalizations than the ECMO group. Another study (3) reported a lower mortality rate (10% to 80%), lower mean hospital stay (29 days to 108 days), and a lower mean cost of initial hospitalization (\$57,000 to \$135,000) in patients treated with ECMO versus conventional treatments at their institution.

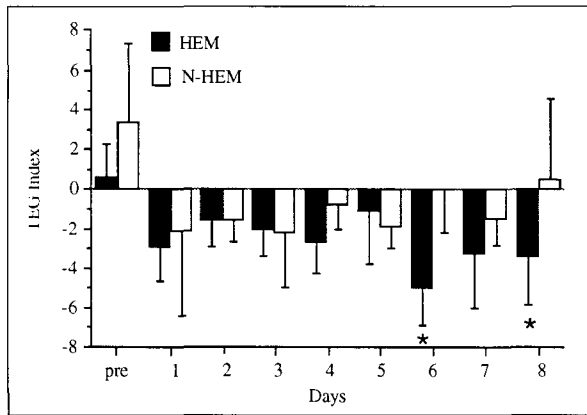
However, ECMO is extremely invasive and is associated with a number of complications. The major complication seen with the modality is hemorrhage, reported in 35% of ELSO cases 1973-1994 and 43.3% of the University of Nebraska Medical Center's cases 1992-1997. Hemorrhagic complications have been associated with increased morbidity and mortality (2, 5). Hemorrhagic complications occurred in 53.8% of HEM patients versus 35.3% of N-HEM patients. Indeed, the survival rate of the HEM group was 61.5%, while the survival rate of N-HEM patients was 76.5%.

Neonates are especially prone to coagulopathies because of their immature coagulation function. Not all clotting factors exist at adult levels, and platelet function has been shown to be decreased in neonates compared to that of adults (22,23). Additional derangements of the hemostatic mechanism that occur with ECMO include systemic heparinization, intravascular and extracorporeal thrombogenicity, qualitative and quantitative platelet disorders, hyperfibrinolysis, enhanced coagulation factor consumption and disseminated intravascular coagulation (6, 10). Because of those derangements, ECMO patients are transfused with greater quantities of blood products than other critically ill patients. The mean donor exposure of 21 consecutive neonates treated with ECMO at one institution was found to be  $22.8 \pm 9.5$  donors per ECMO course (24). With increased transfusions and donor exposures, ECMO patients are at an increased risk of transfusion-related complications.

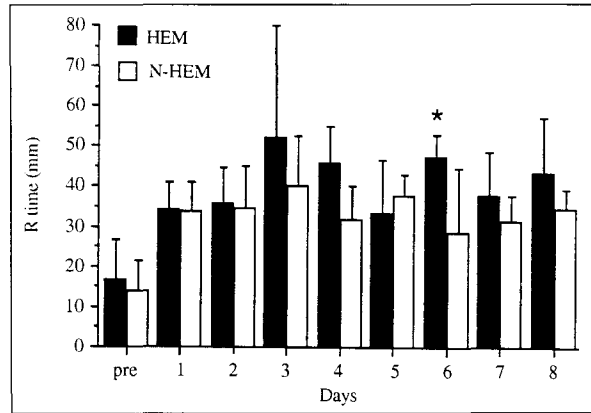
The patients in this study were transfused  $2.06 \pm 1.10$  mL/kg/hr in total transfusions while on ECMO. The HEM group received more than twice the blood products of N-HEM,  $2.94 \pm 1.07$  to  $1.40 \pm 0.48$  mL/kg/hr. HEM patients received almost three times the platelet transfusions and almost double the PRBCs than N-HEM. Significant differences were found in the transfusion requirements of the two groups on ECMO days 1, 3, 4, 5, 7, and 8 [Figure 1(c)]. The HEM group received a significantly greater amount of platelets on days 1, 3, 5, and 8 [Figure 1(a)] and PRBCs on day 7 versus N-HEM [Figure 1(b)].

Both groups received a significantly higher amount of PRBCs on ECMO day 1 when compared to other ECMO days ( $p < 0.05$ ), probably as a result of the hemodilution that occurs with the initiation of extracorporeal circulation or blood loss during and after cannulation. The HEM group seemed to be especially prone to increased transfusions on days 1 and 8 (Figure 2). In addition, they received a significantly greater amount of platelets on day 8 versus days 1, 2, 3, and 6 (Figure 2).

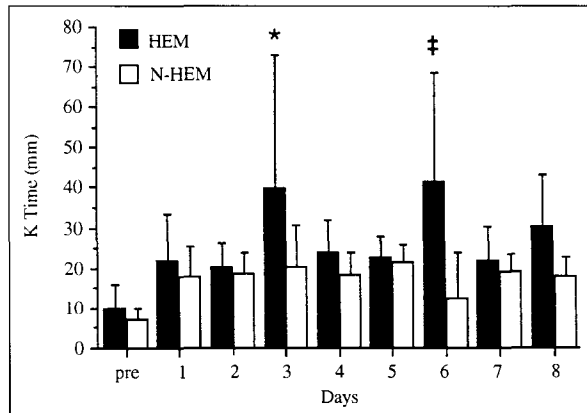
Figure 2: (a) TEG Index (HEM vs. N-HEM); (b) R Time (HEM vs. N-HEM); (c) K Time (HEM vs. N-HEM); (d) Alpha Angle (HEM vs. N-HEM); (e) Maximum Amplitude (HEM vs. N-HEM)



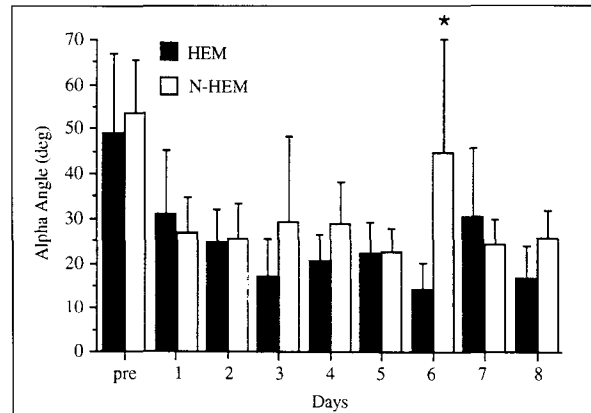
(a) \*= p<0.05 vs. N-HEM



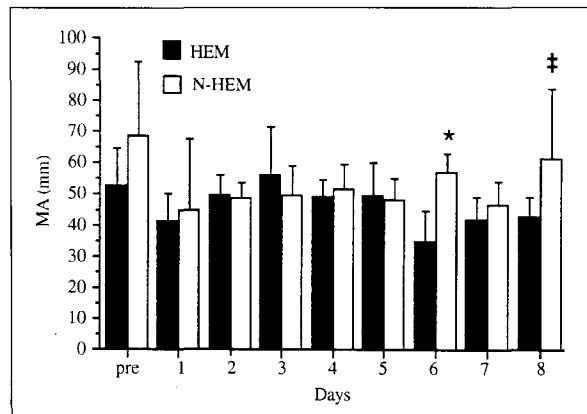
(b) \*= p<0.05 vs. N-HEM



(c) \*= p<0.005 vs. N-HEM; ‡= p<0.001 vs. N-HEM



(d) \*= p<0.001 vs. N-HEM



(e) \*= p<0.01 vs. N-HEM; ‡= p<0.05 vs. N-HEM

Plotz (25) observed two periods of blood activation during ECMO, one within the first 24 hours of treatment and one after 3 days of treatment. The first period of activation was probably a result of the initial blood-extracorporeal circuit interaction, and was characterized by activation of both the complement system

and the contact phase of the coagulation pathway. The contact activation leads to decreased platelet function and altered hemostasis. The patients in this study also suffered impaired hemostasis on day 1, receiving increased amounts of platelets.

The late activation period, in which they observed altered hemostasis without an activation of the complement system, is harder to explain, although the circuit is probably the source of the derangement. Late periods of altered hemostasis were seen in this study and appeared to be related to decreased platelet function.

Extracorporeal membrane oxygenation is known to be associated with platelet consumption and decreased platelet function (6, 10). In a study published by Stammers et al. in 1995 on 17 ECMO patients treated at the University of Nebraska Medical Center in Omaha, Nebraska, the most common cause of hemorrhage was platelet dysfunction (10). Sell et al. found an increased risk of hemorrhage with ECMO associated with platelet counts less than 100,000/ $\mu\text{L}$  for  $\geq 75\%$  of the day (6). Another group separated ECMO patients into two groups, maintaining the platelet count of one at 100,000/ $\text{mm}^3$  and the other

at 200,000/mm<sup>3</sup>. A significant difference was found in the incidence of hemorrhagic complications between the two groups, even though the volume of platelets received by the two groups was similar (26). At this institution, platelets are transfused to maintain the platelet count above 100,000/mm<sup>3</sup>. Mean 24 hour platelet counts ranged from 93,000/mm<sup>3</sup> to 123,000/mm<sup>3</sup> during the course of ECMO treatment.

However, platelet counts fail to show the whole picture. A platelet count quantitatively assesses number, but does not identify function. Likewise, individual coagulation tests are often inconsistent and fail to be specific. The TEG is a whole-blood coagulation monitor which measures the viscoelastic properties of blood as it clots. In that way, a profile of the interaction of clotting factors and platelets over time can be developed (27). This institution has previously reported (10) the use of heparinase TEGs with neonates undergoing ECMO. By analyzing indices of clot function, rather specific coagulopathies have been diagnosed.

The HEM group's TEG index was more hypocoagulable than N-HEM during the entire ECMO course. Thrombelastographic determination showed significant differences between HEM and N-HEM groups on days 3 and 6 ( $p < 0.005$ ) and 8 ( $p < 0.05$ ), indicating periods where the HEM group appeared to be at an increased risk for bleeding than N-HEM [Figure 2(a-e)]. In addition, the HEM group's R time was significantly prolonged on day 3 versus days 1, 2, and 5 ( $p < 0.05$ ). Their K time was significantly prolonged on days 3 and 6 versus all other days except day 8 ( $p < 0.005$ ). The alpha angle was significantly diminished on HEM days 3, 6 and 8 versus day 1 ( $p < 0.05$ ). The maximum amplitude was significantly decreased in HEM patients on day 3 versus day 1 and day 6 versus days 1, 2, 3, and 5 ( $p < 0.05$ ). The decrease in platelet function in those periods of HEM patient's ECMO course seems to be related to days in which they received significantly greater quantities of platelets than N-HEM patients (days 3, 5, and 8) [Figure 1(a)].

In conclusion, decreased platelet function determined by the TEG was seen during late periods of increased transfusions in our patients. The TEG could prove to be a valuable supplement in managing a very challenging patient population. It may help to identify periods of the ECMO course where patients may be at an increased risk of hemorrhage. In the future, prospective studies with the TEG may help further characterize late periods of activation and identify subsets of the population that may be at an increased risk for hemorrhagic complications. That would enable clinicians to more accurately direct their transfusion therapy, decreasing hemorrhagic-related morbidity and mortality. Furthering our understanding of the etiologies of hematological abnormalities and formulating effective intervention strategies can decrease the need for blood transfusions and may extend the indications for ECMO.

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