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THE BIOCHEMICAL AND ECHOCARDIOGRAPHIC EFFECTS OF ANAEMIA OF PREMATURITY AND RED CELL TRANSFUSION ON CARDIAC FUNCTIONS IN THE VLBW INFANTS

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Aim: We investigated myocardial contractility as measured by Echocardiographic parameters of Tissue Doppler Imaging (TDI), Myocardial Performance Index (MPI), Ejection Fraction (EF) and Biochemical markers in VLBW infants before and after red cell transfusion.

Method:Prospective, observational study on VLBW infants of < 34 weeks gestation and >2 weeks of age. Blood sampling and echocardiogram performed in 12 hours before and between 3 to 7 days after transfusion. Infants with congenital heart lesions were excluded. Data was analysed using paired t-test, Wilcoxon signed rank and Kendall's Tau to compare the differences and to see correlation between pre and post values.

Results: 74 pre and post transfusion (Pst) studies were performed on 28 infants. Pre-transfusion (Prt) haematocrit (Hct) ranged between 0.20% and 0.29%.Significant improvement in each biochemical and echocardiographic parameters were seen after transfusion:

TDI (cm/sec)		Prt (Mean)	SD	Pst (Mean)	SD
Left Ventricle	Systolic	5.15	0.79	5.57	0.81
	Diastolic	5.89	1.18	6.74	1.16
Left Ventricle(MPI)		0.395	0.1622	0.248	0.1627
Biochem-istry (pg/ml)					
NTproBNP		1013	718.40	681	491.65
Troponin-T		54	17.04	46	13.50

*[Measurements and values]*P value < 0.001 for all readings

There was also a significant improvement in EF, septal and right ventricular contractility as assessed by TDI and MPI.

Conclusion:Assessmentofmyocardialperformance by echocardiography and biochemical markers demonstrate significant improvement following transfusion which is not proportional to the severity of the anaemia.

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CEREBRAL AUTOREGULATORY CAPACITY DURING HYPOVOLAEMIA IN NEWBORN PIGLETS

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Background and aims: Hypovolaemia constitutes a stress to systemic circulation. The place of volume expansion in ill newborn infants with low arterial blood pressure (ABP) is uncertain. Our hypothesis was that hypovolaemia would further reduce cerebral autoregulatory capacity (CA-capacity) at low ABP.

Methods: Piglets (n=7, age 1-2 days) were anaesthetized with propofol and randomized to normovolaemia- hypovolaemia or hypovolaemia-normovolaemia. Hypovolaemia was induced by removal of ⅓ of the estimated blood volume. Normovolaemia was re-established by re-infusing the removed blood volume. A resting period of 30 minutes was interpolated after removal and re-infusion of the blood volume to enable recovery of cerebral autoregulation. CA-capacity was estimated from changes in cortical flux (laser-Doppler probe) to induced rises in ABP by inflation of a thoracic aorta balloon for 30 seconds. Subsequently, CA-capacity was calculated as %ΔCVR/%ΔABP (i.e. percentage of full autoregulatory capacity), where CVR was estimated as ABP/Doppler flux.

Results: A total of 104 and 94 rises in ABP were analyzed during normo-and hypovolaemia, respectively. Hypovolaemia reduced mean ABP from 54±4 to 39±5 mmHg (P=0.02) and CA-capacity from 62±9 to 37±8% (P=0.05) (mean ±SEM). CA-capacity was 56±3 at ABP above 40 mmHg compared to 33± 3 below 40 mmHg (P=0.00005). Analysis of variance demonstrated an interaction between hypovolaemia and ABP such