that paracetamol could be used as an alternative for infants who are either ibuprofen-resistant or for whom ibuprofen is contraindicated. Further prospective randomized-controlled trials are needed to evaluate the efficacy of paracetamol for the closure of PDA.

### Abstracts

**1122**

**THE EFFECT OF HEPARIN INFUSION ON DEVELOPMENT OF THROMBOSIS SECONDARY TO CARDIAC CATHETERIZATION**

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**Background and Aim** Cardiac catheterization is an important diagnostic tool. In this study, the frequency and the factors affecting the development of thrombosis were prospectively evaluated in neonates who were subjected to diagnostic and interventional cardiac catheterization.

**Method** 29 patients were enrolled in this study. Blood samples were taken for complete blood count, prothrombin, activated partial thromboplastin time, INR ratio, mutation of factor V Leiden, prothrombin 20210 A, MTHFR C667 and A1298 before the procedure. 50 U/kg bolus of heparin was infused during catheterization and 20 U/kg/hour infusion of heparin was given to patients with clinically suspected thrombosis. Doppler was performed in all patients within 6 hours after catheterization.

**Results** Arterial catheterization in 16 cases, venous catheterization in 7 cases and both were applied in 6 cases. Arterial thrombosis in 7 patients and venous thrombosis in two patients was detected. It’s observed that infusion of 20U/kg/h heparin had no effect on the development of arterial thrombosis. On the development of arterial and venous thrombosis, patient age, gender, diagnosis, treatments received prior to catheterization, hemoglobin and platelet count, PT, aPTT and INR values and Factor V Leiden, prothrombin 20210 A, MTHFR C667, A1298 mutations were found as not impacting.

**Conclusion** There are no exact protocols for the prevention of thrombosis during and after catheterization. In this study, 50 unit/kg heparine bolus during catheterization and 20 unit/kg/hour heparine infusion after catheterization did not prevent the development of thrombosis. Extensive studies are needed to determine the appropriate drugs and/or doses of preventive treatments.

**1123**

**DO PATENT DUCTUS ARTERIOSUS AND ITS TREATMENT WITH ORAL IBUPROFEN AFFECT RENAL AND MESENTERIC TISSUE OXYGENATION IN PRETERM INFANTS?**

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**Background/Aim** Patent ductus arteriosus (PDA) in preterm infants can result in serious hemodynamic changes causing respiratory, gastrointestinal and renal morbidities if not treated within the first week of life. We investigated the impact of PDA and its treatment with oral ibuprofen on regional renal and mesenteric oxygen saturation.

**Methods** 13 infants with PDA (gestational age <32 weeks), subsequently treated with oral ibuprofen, were monitored for mean arterial blood pressure, arterial oxygen saturation, near-infrared spectroscopy-determined regional renal and mesenteric oxygen saturation. The patients with PDA were matched for gestational age, birth weight, postnatal age, and severity of respiratory distress syndrome with infants without PDA, who served as control subjects.

**Results** Median renal and mesenteric oxygen saturation were similar in infants with PDA before and up to 12 hours after the start of ibuprofen therapy (renal oxygen saturation: 50% (25th–75th percentile: 29.5–65.5%) vs. 54% (25th–75th percentile: 56–72.5%), p=0.556; respectively); mesenteric oxygen saturation: 31% (25th–75th percentile: 19–38%) vs. 32% (25th–75th percentile: 23.5–46%), p=0.239; respectively). Median renal and mesenteric oxygen saturation in control infants were also not significantly different in infants with PDA before and up to 12 hours after the start of ibuprofen therapy.

**Conclusions** A hemodynamically significant PDA and its adequate treatment with oral ibuprofen seem not to affect the renal and mesenteric perfusion and subsequent oxygen delivery in very low birth weight preterm infants.

**1124**

**LEVOSIMENDAN RESCUE THERAPY IN NON-CARDIOSCIRUGICAL NEONATES WITH HEART FAILURE: A CASE SERIES**

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**Background and Aims** To report the successful use of the new inodilator levosimendan in 4 critically ill neonates with refractory heart failure. At present, no data are available on the use of levsimendan in newborns outside the cardiosurgical setting.

**Methods** Clinical chart review.

**Results** Neonates described in Table 1 were given LS due to severe refractory heart failure when standard treatment was ineffective and/or complications occurred. LS was administered as a continuous i.v. infusion (0.2 mcg/kg/min over the first 24 hrs). LS addition resulted in an improvement and/or stabilisation of hemodynamic status, with nearly normal restoration of heart function in 2/4 infants. Patients with PH and RV enlargement could be weaned off from pulmonary vasoilators.

**Conclusions** In these full-term neonates with post-ischaemic low cardiac output/pulmonary hypertension and heart failure of infectious or metabolic origin, refractory to first line inotropic drugs, LS was a potent inotropic agent and a possible add-on therapy. As suggested, in the neonatal period LS may represent an ideal drug for immature myocardium characterized by a much more calcium dependent contractility than adults. Further studies are needed to evaluate the role of LS in refractory HF.