**BLOOD PRESSURE AND CAPILLARY MICROCIRCULATION AT BIRTH IN INFANTS BORN TO MOTHERS WITH A HYPERTENSIVE DISORDER OF PREGNANCY**

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**Background:** Offspring of mothers with hypertensive disorders of pregnancy (HDP) are at increased future risk of hypertension, cardiovascular disease and stroke. The pathophysiological mechanisms for this association are not well understood, but microvascular abnormalities and in particular capillary rarefaction (CR) have been implicated. In a previous pilot study, we found that infants born pre-term to HDP mothers have significant CR at birth.

**Methods:** We studied 111 infants born to mothers with an HDP (90 born at term (T-HDP) and 21 born pre-term (PT-HDP) and compared them to 278 normal birth weight infants born at term to normotensive mothers (control group). We used intravital capillary microscopy to measure basal i.e. functional (BCD) and maximal i.e. structural (MCD) capillary densities and BP at birth, 3 months, 6 months and 12 months. We report here BP and capillary density results at birth.

**Results:** PT-HDP infants had a significantly higher BCD (mean difference 12.62 cap/field; p=0.001) and (mean difference 17.26 cap/field; p<0.001) compared to T-HDP infants and controls respectively. T-HDP infants also had a significantly higher BCD (mean difference 4.64, cap/field; p=0.012) compared to control infants). PT-HDP infants also had higher MCD (mean difference 10.53 cap/field; p=0.003) and (mean difference 13.67 cap/field; p<0.001) respectively compared to T-HDP and control infants. Systolic BP was significantly higher in PT-HDP infants compared to control infants (mean difference 7.19 mmHg; p=0.032). There were no significant differences in diastolic BP.

**Conclusions:** PT-HDP infants have a significantly higher systolic BP at birth compared to infants of normotensive mothers. Furthermore, PT-HDP have, contrary to our pilot study, a significantly higher BCD and MCD compared to NBW of normotensive mothers. Further follow-up studies of these infants are required to investigate the crucial role of the microcirculatory abnormalities in the pathogenesis of future hypertension.

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