**Introduction**: Ischemic necrosis of the femoral head is one of the most serious complications that can arise following an injury or treatment of the pediatric hip. It can also arise from a common idiopathic condition called Legg-Calve-Perthes disease. In many instances, ischemic necrosis produces permanent femoral head deformity that can lead to premature degenerative arthritis in adulthood. Although it is generally agreed that the deformity following ischemic necrosis is produced by interaction of mechanical and biological factors, little is known about the changes in the mechanical properties of the growing femoral head as it undergoes complex tissue changes due to ischemic injury and repair. The purpose of this study was to determine the alteration of indentation stiffness of the growing femoral head following ischemic injury and to correlate the changes with radiographic and histopathologic changes observed in the femoral head as it develops deformity. We hypothesized that a significant decline in the overall aggregate mechanical properties of the infarcted femoral head precedes the development of the deformity and contributes to biomechanical failure resulting in the deformity.

**Methods**: The study was approved by the local IACUC. A well-established, immature swine model of ischemic necrosis was used (1,2). Ischemic necrosis was induced in 18 animals by surgically placing a ligature around the femoral neck to disrupt the blood supply to the right femoral head. Animals were sacrificed 2, 4, and 8 weeks following the operation (6 per time period). Non-operated femoral heads from the contralateral leg served as controls. Eight additional animals received sham operations in which all procedures were performed except for tightening of the ligature (sham controls). The study also included 6 animals sacrificed at time point 0. From all animals, proximal femora were dissected out and measured for femoral head size and weight and femoral head size.

**Experimental results**: The indentation stiffness of the femoral head at the time of surgery averaged 3.49 ± 0.56 MPa/mm (Fig. 1, 0 wk). Indentation stiffness of the contralateral control femur increased to 5.46 ± 1.53 MPa/mm at 2 weeks and to 7.03 ± 1.11 MPa/mm at 4 weeks. Between 4 and 8 weeks, the control indentation stiffness declined to 4.05 ± 1.59 MPa/mm. This drop in stiffness correlated with an substantial increase in growth of the piglets measured by weight and femoral head size.

The mean indentation stiffness of sham-operated femoral heads decreased by 18 and 20% compared to non-operated, contralateral femoral heads at 2 and 8 weeks, respectively (data not shown). The sham-operated femoral heads showed no histological or radiographic evidence of ischemic necrosis. The epiphyseal cartilage was significantly stiffer on the sham-operated side (2.09 ± 0.13 mm) compared to the non-operated side (1.80 ± 0.12 mm) at 2 weeks (p < 0.01) but not at 8 weeks.

The mean indentation stiffness of the infarcted femoral heads at 2, 4, and 8 weeks after surgery was significantly lower than the mean indentation stiffness of the contralateral non-infarcted femoral heads at all time points (P < 0.001) (Fig. 1). At 2 weeks, a 52% reduction in the mean indentation stiffness was observed in the infarcted heads compared to the controls. Radiographically, the infarcted heads had smaller bony epiphyses, however, the mean epiphyseal quotient (0.42 ± 0.06) was similar to the controls (0.41 ± 0.05) consistent with the absence of deformity. No histological evidence of repair or revascularization was observed in the infarcted heads at 2 weeks. At 4 weeks, the mean indentation stiffness was reduced by 74% as compared to the contralateral controls (p<0.001). The mean head quotient of the infarcted side was slightly lower but the difference was not statistically significant. A variable degree of revascularization and repair was observed in the infarcted heads ranging from none to extensive repair. At 8 weeks, the mean indentation stiffness was reduced by 72% compared to the controls (p < 0.001). The infarcted heads were severely deformed with a mean epiphyseal quotient of 0.22 ± 0.07 compared to the control side which had a mean of 0.41 ± 0.02 (p < 0.05). Large areas of revascularization and bone resorption with areas of new bone formation were observed. The epiphyseal cartilage was significantly thicker on the infarcted side at 2 and 4 weeks, however, the cartilage thickness only had a weak correlation with the indentation stiffness (R²=0.55).

**Results**: The results showed a significant decrease in the indentation stiffness of the infarcted femoral head prior to radiographic appearance of femoral head deformity and histological evidence of repair. The decrease could not be explained solely by the increase in the cartilage thickness. A further decrease in the indentation stiffness was associated with revascularization and repair of the infarcted bony epiphysis. Although indentation stiffness provides a reliable measure of the relative properties of the femoral head as a whole, it does not discriminate between the specific contributions of cartilage and bony epiphysis to the aggregate mechanical properties. Studies are currently underway measuring the material properties of isolated specimens of cartilage and bone from this animal model.

In conclusion, the indentation properties of growing femoral head were significantly affected by ischemic injury and the repair process that ensued. From this study, we conclude that load limiting treatment should be instituted at an early stages of ischemic necrosis even before the appearance of femoral head deformity.

**Acknowledgments**: Funded by the Pediatric Orthopaedic Society of North America and by the Shriners Hospitals for Children.