HIP

New insights into the biomechanics of Legg-Calvé-Perthes’ disease

THE ROLE OF EPiphySEAL SKELETAL IMMaturity IN VASCULAR OBSTRUCTION

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Objectives
Legg–Calvé–Perthes’ disease (LCP) is an idiopathic osteonecrosis of the femoral head that is most common in children between four and eight years old. The factors that lead to the onset of LCP are still unclear; however, it is believed that interruption of the blood supply to the developing epiphysis is an important factor in the development of the condition.

Methods
Finite element analysis modelling of the blood supply to the juvenile epiphysis was investigated to understand under which circumstances the blood vessels supplying the femoral epiphysis could become obstructed. The identification of these conditions is likely to be important in understanding the biomechanics of LCP.

Results
The results support the hypothesis that vascular obstruction to the epiphysis may arise when there is delayed ossification and when articular cartilage has reduced stiffness under compression.

Conclusion
The findings support the theory of vascular occlusion as being important in the pathophysiology of Perthes disease.

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Keywords: Perthes’ disease, Vessel obstruction, Juvenile hip, Biomechanics, Finite element analysis

Article focus
This article explores potential mechanisms of vascular occlusion within the epiphysis that may lead to Perthes’ disease of the hip.
These mechanisms are investigated with a high-resolution finite element model of the juvenile hip incorporating the retinacular vessels.

Key messages
Vascular obstruction at the epiphysis may arise when there is both delayed ossification and articular cartilage that has compromised compressive stiffness.
Initial obstruction of the blood supply may lead to alterations in bone properties, increasing the likelihood of further femoral head collapse.
A vascular mechanism for Perthes’ disease seems possible in a child with skeletal immaturity, in combination with an acute cartilage insult.

Strengths and limitations
This study uses a unique high-resolution finite element model of the juvenile hip incorporating the retinacular vessels.
The study is hypothesis-generating, and experimental models of disease are required to formally test these hypotheses.

Introduction
Legg–Calvé–Perthes’ disease (LCP or Perthes’ disease) is an idiopathic osteonecrosis of the femoral head that most frequently occurs between four and eight years old. Although described more than 100 years ago by four independent studies, the aetiology of Perthes’ disease remains poorly understood. LCP is between four and five times more common in boys than in girls, and children...
with this disease have delayed skeletal development of
between one and two years.\textsuperscript{7,8} Approximately 90% of
LCP cases are unilateral.\textsuperscript{1} In the most severe cases, LCP
can lead to the permanent flattening of the femoral head,
articular cartilage degeneration and early osteoarthritis.\textsuperscript{9}

An impairment or obstruction of the blood supply to
the developing femoral head is thought to be the most
likely mechanism for the disease. Factors leading to this
vascular obstruction are unclear and are the subject to
debate. During development, the epiphyseal blood sup-
ply is almost exclusively provided by the deep branch of
the medial femoral circumflex artery (MFCA).\textsuperscript{10} Both
imaging and histological studies have shown a partial or
complete loss of blood flow\textsuperscript{11,12} and the development of
ischaemic necrosis\textsuperscript{13} of the femoral head in Perthes’ dis-
ease. Furthermore, there is evidence of abnormal periph-
eral vasculature in children with LCP.\textsuperscript{14}

We have previously proposed five mechanisms for the
pathology of Perthes’ disease,\textsuperscript{15} and the implications for
the morphology of the juvenile hip and the biomechanics
of the hip joint.\textsuperscript{16} From these we suggested that fracture
and collapse of the femoral head, even in an immature
epiphyseal cartilage, is unlikely. We now hypothesize that morpho-
logical changes of the femoral head, together with an
alteration in the mechanical properties of the epiphyseal
cartilage, may cause compression or occlusion of the reti-
nacular vessels. Using high-resolution finite element (FE)
models, we have investigated the simulated forces acting
on the MFCA under a number of experimental conditions
to determine if this hypothesis is plausible.

\textbf{Materials and Methods}

FE modelling has been increasingly used for implant bio-
mechanical optimization,\textsuperscript{17,18} custom-implant develop-
ment,\textsuperscript{19,20} and tissue engineering.\textsuperscript{21} We have previously
developed an FE model of a healthy 7.9-year-old male hip
from CT data.\textsuperscript{19} Several variations of this healthy model
were developed to understand the impact of hip mor-
phology in the loading of the bony epiphysis. Delayed
ossification was modelled by offsetting the normal ep-
iphysis inwards by 2.3 mm according the method of Kitoh
et al.,\textsuperscript{22} whereas the cartilage thickness of the femoral
head was defined by offsetting the healthy epiphyseal
surface outwards by 2.0 mm.\textsuperscript{23} Static muscle optimization
was performed in each model to determine the loading
conditions across the hip. The thigh muscles were
modelled with simplified lines of action (Fig. 1a), and the
physiological cross-sectional areas (PCSA) scaled to a
body weight (BW) of 23 kg.\textsuperscript{24} Muscle forces were obtained
by considering the 24 muscles acting around the hip,
using non-linear optimization.\textsuperscript{16} The model was opti-
mized for single-leg stance and predicted a hip joint reac-
tion forces (HJR) of 3.36BW, which compares well with
previously reported values for juvenile subjects.\textsuperscript{25}

In this current work, two high-resolution FE sub-
models were derived from the original (normal) and
offset models (Fig. 1b). The main retinacular vessels that
branch from the MFCA into the developing epiphysis
were included in these sub-models of the femoral head
(Fig. 1c). Here, in addition to the delay in epiphyseal ossi-
faction, other types of joint tissue immaturities were
evaluated. Biomechanical immaturity of the bony epiph-
sis was modelled with different bone volume density
(bone volume divided by total volume; BV/TV) and asso-
ciated mechanical properties, whereas the degree of car-
tilage maturation was modelled by varying its stiffness in
compression. The loading conditions obtained with the
full models were mapped onto the two higher-resolution
sub-models (HRSM) to assess the likelihood of vessel con-
striction for each combination of epiphyseal ossification,
BV/TV, and cartilage stiffness.

Both the full models and the sub-models were meshed
using a voxel-based FE mesher that uses the isosurface
stuffing algorithm,\textsuperscript{26} and consisted of approximately
3.0 million and 5.0 million quadratic tetrahedral elements,
respectively. The mechanical properties for cortical bone
were derived from a series of compressive tests using
bone from juvenile subjects (12 children with ages rang-
ing between four and 15 years).\textsuperscript{27} Thus, an elastic modu-
lus (Young’s modulus) $E_{\text{cortical}} = 11880 \text{ MPa}$ was used,
with yield and ultimate strains of 1.11% and 2.31%,
respectively. In addition to delayed ossification,\textsuperscript{9} Perthes’
patients may also have delayed bone and cartilage matu-
ration. Different values of BV/TV\textsuperscript{28} and cartilage stiffness
were considered across the juvenile epiphysis. The BV/TV
values considered were \{0.06, 0.10, 0.19, 0.28\}, which
correspond to a trabecular bone apparent modulus

$$
E_{\text{trabecular}} = 1.240 E_{\text{tissue}} \left( \frac{BV}{TV} \right)^{1.8}
$$

\text{(1)}

where $E_{\text{tissue}}$ was set equal to $E_{\text{cortical}}$. The corresponding
Young’s modulus for the target BV/TV values were then
$E_{\text{epiphysis}} = \{100, 250, 750, 1500\} \text{ MPa}$, respectively. In
the analysis, only the mechanical properties of the epiphysis
were changed; the other trabecular bone properties were
kept constant ($E_{\text{trabecular}} = 1500 \text{ MPa}$). The articular car-
tilage modulus was also varied between the following val-
ues, $E_{\text{cartilage}} = \{0.50, 1.00, 1.50, 2.00\} \text{ MPa}$.

The geometry of the vessels was derived from an anal-
ysis of the anatomical descriptions found in the litera-
ture.\textsuperscript{30,31} The MFCA and the three main retinacular vessels
were modelled as solid tubular structures with an outer
diameter of 1.60 mm and 0.80 mm, respectively, and
included in the two HRSM (Fig. 1b). The properties of the
retinacular vessels were derived from the literature.\textsuperscript{32}
Vessel obstruction was then assessed by comparing the
loaded and unloaded cross-sectional areas of the retinac-
lular vessels. The general shape of the distorted cross-
sections was also assessed by computing the deviation of
the outer cross-sectional geometry from the (ideal) circular shape through the elliptical eccentricity index:

$$r = \sqrt{1 - \frac{b^2}{a^2}}$$  \hspace{1cm} (2)

where $0 \leq r \leq 1$ is a dimensionless parameter (with $r = 0$ for a circle), and $a$ and $b$ are the lengths of the semi-major and the semi-minor axis of the ellipse, respectively. According to Shilo and Gefen,\textsuperscript{33} who studied capillary behaviour under large compressive and shear tissue deformations, complete collapse of blood vessels occurs when $r \geq 0.88$. The Young’s modulus and Poisson’s ratio of the vessels were assumed to be $E_{\text{vessel}} = 0.11 \text{ MPa}$ and $\nu = 0.490$, respectively.

All materials were modelled as linear elastic, isotropic and homogenous, and all model simulations were carried out in ANSYS v15 (ANSYS Inc., Canonsburg, Pennsylvania). The full details of the material properties used are summarized in Table I. The ultimate strain for the trabecular bone was defined as 2.31% and considered constant across different BV/TV. Equivalence between the loading of the two models was obtained by mapping the nodal displacements from the full to the HRSM.

This research was part of a larger study investigating the diagnosis and prevention of Perthes’ disease. Ethical approval for the work was given by the Office for Research Ethics Committees Northern Ireland (ORECNI), REF 13/NI/0139.

### Table I. Material properties considered in the juvenile hip model\textsuperscript{16,32}

<table>
<thead>
<tr>
<th>Material properties</th>
<th>Cortical bone</th>
<th>Trabecular bone</th>
<th>Epiphyseal plate</th>
<th>Epiphyseal cartilage</th>
<th>Pubic symphysis</th>
<th>Acetabular labrum</th>
<th>Retinacular vessels</th>
</tr>
</thead>
<tbody>
<tr>
<td>Young’s modulus (MPa)</td>
<td>11 880</td>
<td>100 to 1500</td>
<td>0.75</td>
<td>0.50 to 2.00</td>
<td>5.00</td>
<td>5.00</td>
<td>0.11</td>
</tr>
<tr>
<td>Poisson’s ratio</td>
<td>0.300</td>
<td>0.300</td>
<td>0.495</td>
<td>0.495</td>
<td>0.450</td>
<td>0.495</td>
<td>0.490</td>
</tr>
</tbody>
</table>
Results

Both the full model and sub-model of the developing femoral epiphysis were simulated under single-leg stance. The strain values across the bony epiphysis were uniformly very low, and only for low BV/TV values were there significant portions of the femoral epiphysis above the ultimate strain. Figure 2 shows the proportion of epiphyseal volume above the ultimate strain of 2.31%, for healthy and sample immature models with BV/TV = 0.10, .06, considering different cartilage properties. In the immature epiphysis, the volume above the ultimate strain is up to 7.3% of the overall ossified volume for $E_{\text{epiphysis}} = 250 \text{ MPa}$ (BV/TV = 0.06) and $E_{\text{epiphysis}} = 100 \text{ MPa}$ (BV/TV = 0.10) with different cartilage properties. (Values for $E=250\text{MPa}$ are < 0.1%)

Figures 3a and 3b presents the displacement of the femoral head with healthy and immature proximal femoral epiphysis with $E_{\text{epiphysis}} = 1500 \text{ MPa}$ and $E_{\text{cartilage}} = 0.50 \text{ MPa}$. In the healthy epiphysis, the overall shape of the epiphysis is preserved. A softer epiphyseal cartilage leads to an increase in its lateral displacement; nevertheless, this is limited by the ossified epiphysis (Fig. 3a). In contrast, in the small/skeletally immature head, there is more lateral displacement of the cartilage, and this becomes more pronounced as the stiffness of the cartilage decreases (Fig. 3b).

The lateral displacement of the cartilage causes the blood vessels to be stretched as they pass through the articular cartilage (Fig. 3b). For a small epiphysis, reducing the stiffness of the articular cartilage leads to an increase in the lateral displacement and stretching of the blood vessels, irrespective of the bone density of the femoral epiphysis (all models are shown in supplementary Fig. a). Table II and Table III list the minimum and maximum displacements for each combination of $E_{\text{epiphysis}}$ and $E_{\text{cartilage}}$ for the healthy and small femoral epiphysis, respectively. For instance, for the healthy epiphysis and a $E_{\text{epiphysis}} = 1500 \text{ MPa}$, vessel displacement remains relatively unchanged as the stiffness of the cartilage is reduced (with a maximum vessel stretch of 1.12 mm for $E_{\text{cartilage}} = 0.50 \text{ MPa}$, Table II and supplementary Fig. b), whereas for the small epiphysis and $E_{\text{cartilage}} = 0.50 \text{ MPa}$ the blood vessel stretch is 2.75 mm (Table III and Fig. a). The vessel cross-sectional distortion also increases (Table IV) and the cross-sectional area decreases (Table V) as cartilage becomes softer. For $E_{\text{cartilage}} \leq 1.0 \text{ MPa}$ there is an almost complete collapse of the blood vessels (with $r = 0.88$), whereas the reduction in the cross-sectional area ranges from approximately 22% to 68%. Figure 4 shows the change in the cross-sections of the three blood vessels (anterior, medial, and posterior) across the regions of highest distortion. Vessel twisting was also observed, especially in the posterior vessel (Figs 4a to 4d).

Discussion

We have shown that significant vessel obstruction can occur when there is delayed ossification of the bony epiphysis and reduced stiffness of the articular cartilage, under simple single-leg stance loading conditions. The obstruction of the blood supply may lead to further reduction in bone and articular cartilage biomechanical properties, increasing the likelihood of further femoral head collapse.

Under the loading conditions considered, collapse of the femoral epiphysis due to overload is unlikely to occur, unless the BV/TV of the trabecular bone is extremely low (Fig. 2). Overloading occurs for very low values of $BV/TV = 0.06$, which corresponds to a skeletal immaturity greater than the one to two years normally observed.
Displacements of the juvenile hip and three main terminal branches of the deep mFCa for a) the healthy femoral epiphysis with $E_{\text{epiphysis}} = 1500$ MPa and $E_{\text{cartilage}} = 0.50$ MPa, and b) the skeletally immature epiphysis with $E_{\text{epiphysis}} = 1500$ MPa and $E_{\text{cartilage}} = 0.50$ MPa.

**Table II.** Minimum and maximum blood vessel displacements (mm) as a function of $E_{\text{epiphysis}}$ and $E_{\text{cartilage}}$ with the healthy epiphysis (see supplementary information Fig. b)

<table>
<thead>
<tr>
<th>$E_{\text{epiphysis}}$ (MPa)</th>
<th>BV/TV</th>
<th>$E_{\text{cartilage}}$ (MPa) 2.00, range</th>
<th>$E_{\text{cartilage}}$ (MPa) 1.50, range</th>
<th>$E_{\text{cartilage}}$ (MPa) 1.00, range</th>
<th>$E_{\text{cartilage}}$ (MPa) 0.50, range</th>
</tr>
</thead>
<tbody>
<tr>
<td>1500</td>
<td>0.28</td>
<td>1.55 to 2.50</td>
<td>1.73 to 2.72</td>
<td>2.02 to 3.06</td>
<td>2.63 to 3.75</td>
</tr>
<tr>
<td>750</td>
<td>0.19</td>
<td>1.55 to 2.51</td>
<td>1.73 to 2.73</td>
<td>2.02 to 3.08</td>
<td>2.63 to 3.77</td>
</tr>
<tr>
<td>250</td>
<td>0.10</td>
<td>1.55 to 2.57</td>
<td>1.73 to 2.78</td>
<td>2.03 to 3.13</td>
<td>2.63 to 3.81</td>
</tr>
<tr>
<td>100</td>
<td>0.06</td>
<td>1.56 to 2.68</td>
<td>1.74 to 2.90</td>
<td>2.04 to 3.24</td>
<td>2.64 to 3.92</td>
</tr>
</tbody>
</table>

**Table III.** Minimum and maximum blood vessel displacements (mm) as a function of $E_{\text{epiphysis}}$ and $E_{\text{cartilage}}$ with the small/immature epiphysis (see supplementary information Fig. a)

<table>
<thead>
<tr>
<th>$E_{\text{epiphysis}}$ (MPa)</th>
<th>BV/TV</th>
<th>$E_{\text{cartilage}}$ (MPa) 2.00, range</th>
<th>$E_{\text{cartilage}}$ (MPa) 1.50, range</th>
<th>$E_{\text{cartilage}}$ (MPa) 1.00, range</th>
<th>$E_{\text{cartilage}}$ (MPa) 0.50, range</th>
</tr>
</thead>
<tbody>
<tr>
<td>1500</td>
<td>0.28</td>
<td>2.84 to 4.46</td>
<td>3.29 to 5.03</td>
<td>4.04 to 6.07</td>
<td>5.65 to 8.40</td>
</tr>
<tr>
<td>750</td>
<td>0.19</td>
<td>2.82 to 4.47</td>
<td>3.27 to 5.04</td>
<td>4.03 to 6.08</td>
<td>5.68 to 8.41</td>
</tr>
<tr>
<td>250</td>
<td>0.10</td>
<td>2.76 to 4.51</td>
<td>3.20 to 5.08</td>
<td>3.96 to 6.12</td>
<td>5.67 to 8.45</td>
</tr>
<tr>
<td>100</td>
<td>0.06</td>
<td>2.69 to 4.58</td>
<td>3.12 to 5.15</td>
<td>3.86 to 6.19</td>
<td>5.55 to 8.51</td>
</tr>
</tbody>
</table>

These observations support the results of our previous work. Even with such significantly reduced bone stiffness, only 7.3% of the epiphysis was overloaded.
In an MRI study of adults, it was found that necrosis of more than 30% of the epiphyseal volume was necessary to cause the collapse of the femoral head.\(^{35}\) In a similar radiological study, it was found that femoral head collapse depended on the size of the necrotic region and its position relative to the medial weight-bearing area but, for example, a failure rate of only 4.5% was reported for necrotic volumes < 30%.\(^{36}\)

For a healthy epiphysis, vessel distortion was negligible irrespective of the BV/TV and cartilage stiffness values considered (Fig. 3a), whereas for a small epiphysis, significant vessel distortion was observed even for moderately reduced levels of cartilage stiffness (Fig. 3b; Tables III and IV). Hence, the combination of epiphyseal size and stiffness of the cartilage has a more critical impact on vessel constriction than bone maturation (reflected by BV/TV) in our modelling.

An early study in LCP suggested that damage to the articular cartilage might precede the degeneration of the secondary ossification centre.\(^{37}\) Ischaemic-induced necrosis of the femoral epiphyses in piglets has also been shown to cause both structural changes often observed in LCP,\(^{38}\) as well as the injury to the deep layer of epiphyseal cartilage around the secondary ossification centre.\(^{39}\) Kim et al\(^{40}\) observed that the biomechanical properties of cartilage and bone significantly decrease in the infarcted femoral head. These changes to both bone and cartilage of the developing femoral head have been modelled in this study. We have demonstrated that vascular obstruction may occur from a combination of delayed ossification and reduced stiffness of the articular cartilage. The results suggest that cartilage injury and growth arrest may be established prior to the occlusion of the MFCA for Perthes’ disease to develop. Both factors, independently, seem to be insufficient to cause the collapse of the retinacular vessels.

Both the FE models investigated included several simplifications. For loading, the muscles were simplified to single lines of action.\(^{16}\) All materials were modelled as homogeneous with linear elastic properties, which is a simplification of the more complex non-isotropic and nonlinear behaviour of both bone and cartilage. Finally, the retinacular vessels’ dimensions and overall geometry were taken from a single X-ray study.\(^{31}\) The modelled vessels may not accurately represent the juvenile MFCA and retinacular vessels. The vessel diameter was kept constant (0.80 mm) for the three retinacular arteries, which may be slightly oversized compared to other values reported for juvenile subjects.\(^{30,31}\) Despite these simplifications, the results are still interesting and provide useful insights into the potential mechanisms that could lead to LCP.

The underlying cause of reduced cartilage stiffness is unclear. During maturation of articular cartilage, there is known to be an increase in tensile and compressive stiffness.\(^{41}\) Proteoglycan and collagen fibres are the major contributors to stiffness under compression.\(^{42}\) Any variation in collagen or proteoglycan makeup will affect the biomechanical properties. Cartilage injury may release proteoglycan fragments into the synovial fluid and has been reported in LCP. The macroscopic modulus of degenerative articular cartilage has been found to be as low as 0.28 MPa to 0.50 MPa in adult osteoarthritic joints.\(^{44}\) In the current study, values of 0.50 MPa and 1.00 MPa were found to lead to reductions in vessel cross-sectional area of 68% and 40% respectively, during single-leg stance, which is a normal loading pattern. Articular cartilage injury and release of degradation products into the synovial fluid has a synergistic effect on the

**Table IV.** Cross-sectional eccentricity (\(\epsilon\)) of each retinacular vessel as they pass through the epiphyseal cartilage for the skeletally immature epiphysis (BV/TV values for the trabecular bone displayed in brackets)

<table>
<thead>
<tr>
<th>(E_{\text{epiphysis}}) (MPa)</th>
<th>(E_{\text{cartilage}}) (MPa) 2.00</th>
<th>(E_{\text{cartilage}}) (MPa) 1.50</th>
<th>(E_{\text{cartilage}}) (MPa) 1.00</th>
<th>(E_{\text{cartilage}}) (MPa) 0.50</th>
</tr>
</thead>
<tbody>
<tr>
<td>1500 (0.28)</td>
<td>0.72 0.74 0.73</td>
<td>0.78 0.80 0.78</td>
<td>0.85 0.87 0.84</td>
<td>0.94 0.96 0.92</td>
</tr>
<tr>
<td>750 (0.19)</td>
<td>0.72 0.75 0.74</td>
<td>0.78 0.80 0.81</td>
<td>0.85 0.87 0.86</td>
<td>0.94 0.96 0.94</td>
</tr>
<tr>
<td>100 (0.06)</td>
<td>0.73 0.75 0.79</td>
<td>0.79 0.80 0.83</td>
<td>0.86 0.87 0.89</td>
<td>0.94 0.96 0.95</td>
</tr>
</tbody>
</table>

ant., anterior; med., medial; pos., posterior

**Table V.** Variation of the cross-sectional area (%) for each retinacular vessel as they pass through the epiphyseal cartilage for the skeletally immature epiphysis (BV/TV values for the trabecular bone displayed in brackets)

<table>
<thead>
<tr>
<th>(E_{\text{epiphysis}}) (MPa)</th>
<th>(E_{\text{cartilage}}) (MPa) 2.00</th>
<th>(E_{\text{cartilage}}) (MPa) 1.50</th>
<th>(E_{\text{cartilage}}) (MPa) 1.00</th>
<th>(E_{\text{cartilage}}) (MPa) 0.50</th>
</tr>
</thead>
<tbody>
<tr>
<td>1500 (0.28)</td>
<td>-21.9 -22.2 -13.9</td>
<td>-26.1 -28.2 -19.1</td>
<td>-32.9 -39.4 -21.6</td>
<td>-45.5 -67.7 -31.6</td>
</tr>
<tr>
<td>750 (0.19)</td>
<td>-21.9 -22.1 -14.8</td>
<td>-26.1 -28.1 -17.9</td>
<td>-32.8 -39.3 -22.7</td>
<td>-45.5 -67.6 -32.7</td>
</tr>
<tr>
<td>250 (0.10)</td>
<td>-21.8 -21.8 -17.2</td>
<td>-26.0 -27.8 -20.7</td>
<td>-32.7 -38.9 -26.0</td>
<td>-45.5 -67.3 -36.6</td>
</tr>
<tr>
<td>100 (0.06)</td>
<td>-21.8 -21.2 -20.0</td>
<td>-26.1 -27.2 -24.1</td>
<td>-32.8 -38.2 -30.5</td>
<td>-45.9 -66.6 -42.8</td>
</tr>
</tbody>
</table>

ant., anterior; med., medial; pos., posterior
inflammatory response of the synovium, causing further cartilage breakdown, joint effusion, stiffness, and pain. While this study explores the potential mechanisms of Perthes disease, the aetiology remains uncertain. One possible explanation that could fit the hypothesized mechanism combines delayed development and transient synovitis. Skeletal immaturity or ‘skeletal standstill’ is well described in Perthes’ disease and this may be
related to societal exposures, such as worsening socioeconomic deprivation. An at risk skeletally immature hip may then suffer a relatively benign injury to the cartilage, and through a process such as transient synovitis, these events may be enough to precipitate the onset of LCP through vascular occlusion.

The two high-resolution finite element models of the juvenile hip suggest that significant vessel obstruction can occur when there is delayed ossification of the femoral head epiphysis and there is reduced stiffness of the articular cartilage, under simple single-leg stance loading. The continued obstruction of the blood supply may lead to further reduction in bone stiffness and strength, increasing the likelihood of progressive femoral head collapse.

**Supplementary material**

Figures showing the displacements of the blood vessels for values of trabecular bone Young’s modulus and cartilage stiffness for the normal and immature epiphysis are available alongside this article at www.bjr.boneandjoint.org.uk

**References**

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Author Contribution

- M. Pinheiro: Modelling, finite element analysis, and development of the manuscript.
- C. A. Dobson: Project conception, results analysis and development of the manuscript.
- D. Perry: Clinical advice and development of the manuscript.
- M. J. Fagan: Project conception, results analysis and development of the manuscript.

Conflict of Interest Statement

None declared

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