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Review

Finite element analysis of mechanical behavior of human dysplastic hip joints: a systematic review

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SUMMARY

Developmental dysplasia of the hip (DDH) is a common condition predisposing to osteoarthritis (OA). Especially since DDH is best identified and treated in infancy before bones ossify, there is surprisingly a near-complete absence of literature examining mechanical behavior of infant dysplastic hips. We sought to identify current practice in finite element modeling (FEM) of DDH, to inform future modeling of infant dysplastic hips. We performed multi-database systematic review using PRISMA criteria. Abstracts ($n = 126$) fulfilling inclusion criteria were screened for methodological quality, and results were analyzed and summarized for eligible articles ($n = 12$). The majority of the studies modeled human adult dysplastic hips. Two studies focused on etiology of DDH through simulating mechanobiological growth of prenatal hips; we found no FEM-based studies in infants or children. Finite element models used either patient-specific geometry or idealized average geometry. Diversities in choice of material properties, boundary conditions, and loading scenarios were found in the finite-element models. FEM of adult dysplastic hips demonstrated generally smaller cartilage contact area in dysplastic hips than in normal joints. Contact pressure (CP) may be higher or lower in dysplastic hips depending on joint geometry and mechanical contribution of labrum (Lb). FEM of mechanobiological growth of prenatal hip joints revealed evidence for effects of the joint mechanical environment on formation of coxa valga, asymmetrically shallow acetabulum and malformed femoral head associated with DDH. Future modeling informed by the results of this review may yield valuable insights into optimal treatment of DDH, and into how and why OA develops early in DDH.

Introduction

Developmental dysplasia of the hip (DDH) is a common anatomic deformity leading to hip dysfunction and osteoarthritis (OA). Present in 1–3/1000 live births[1], DDH accounts for one-third of hip replacement surgeries in patients under 60 years old[2]. Untreated dysplastic hips are associated with mechanical instability, limited mobility, muscle imbalance, abnormal joint load, increased cartilage contact pressure (CP), subluxation[3–5], and OA[3,5–7]. In addition to age, trauma, activity, weight, and genetics, OA in DDH relates to anatomic hip abnormalities[5–7]. Since the mechanical environment strongly affects bone growth and development[6,8–13], understanding hip mechanical behavior can provide insight into how premature OA occurs[6,7,14], and help optimize treatment[10,15–18].

Hip mechanical behavior has been studied through experimental measurements, theoretical models, and computational methods including multibody dynamics (MBD), discrete element analysis (DEA), and finite element modeling (FEM)[19–21]. Experimental studies[14,22–27] measure hip CP directly using sensors, but these are invasive and it is difficult to maintain physiological conditions during measurement[20,21]. Theoretical models[6,14,28–33] estimate articular surface CP with fundamental methodological simplifications limiting their predictive validity[20,34]. MBD[35,36] can

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only estimate joint reaction force (JRF), not CP. DEA[34,37–41] and FEM[19,20] are both capable of simulating articular surface CP, contact area, and JRF. Although more computationally intensive, FEM can also model the sliding contact mechanism inside the hip joint, cartilage deformations in all directions, bone deformations, material anisotropy, and stresses inside tissue layers[19,20].

Mechanical behavior of adult normal hips has been well studied through FEM, with generally good agreement with experimental outcomes[19–21,27]. In contrast, FEM-based studies on dysplastic hips have not been specifically reviewed. A dedicated review on this complex topic can clarify the diverse methods in use to study DDH from prenatal initiation to adult morbidity. Notably we could find no published FEM of infant (post-birth) dysplastic hips. Since the most important time to diagnose and treat DDH to prevent OA is in infancy, this is an important gap in the literature. The objective of this systematic review was to determine the state-of-the-art in FEM of human dysplastic hips, with a view to making informed recommendations for future FEM of infant dysplastic hips.

Materials and methods

Search strategy

We performed a systematic review, searching PubMed, Medline and Elsevier (ScienceDirect) for relevant peer-reviewed articles with English-language abstracts published from 1946 to June 25th, 2016 based on these keywords: ("dysplasia" OR "hip dysplasia" OR "dysplastic hip") AND ("finite element" OR "computational" OR "computer simulation" OR "mechanics"). Reference lists of relevant publications were also reviewed to avoid missing relevant articles.

Selection criteria

Titles and abstracts of all potentially relevant articles were reviewed. Article selection was performed by an engineer/research associate (BV) and confirmed in consensus with a radiologist/biomedical engineer (JJ). Full-text articles were included when they (1) demonstrated mechanical behavior of the human dysplastic hip through FEM of full or partial three-dimensional (3D) geometries of natural and/or post-acetabular-osteotomy dysplastic hip joints; and/or (2) employed FEM to study formation of human dysplastic hip morphology. We excluded articles related to total hip arthroplasty, and those in which hip mechanics were investigated by methods other than FEM, i.e., theoretical models, MBD, and DEA. However, noting that the closest alternative to FEM is DEA and to capture merits of DEA in this area, we included a brief review on applications of DEA for studying human dysplastic hips at the end of this paper.

To better understand potential biases, we stratified studies by data source (patient-specific or virtual) and structures modeled. The summary measures reviewed were differences in model outputs between normal and dysplastic hips.

Results

We found 76 unique articles. 29 merited full-text review, and 12 met eligibility criteria (Fig. 1).

The included studies, published between 2004 and 2016, fell into three groups. The first group[42–46] compared mechanical behavior of dysplastic vs normal hips (Tables I–III). The second group compared the behavior of dysplastic hips before and after physical[16] or virtual[15,17,46–48] acetabular osteotomy (Tables I–III). The third group[8,11] investigated DDH etiology by mechanobiological prediction of patterns of prenatal hip growth (Table IV). When an article also included FEM of joints with other pathology

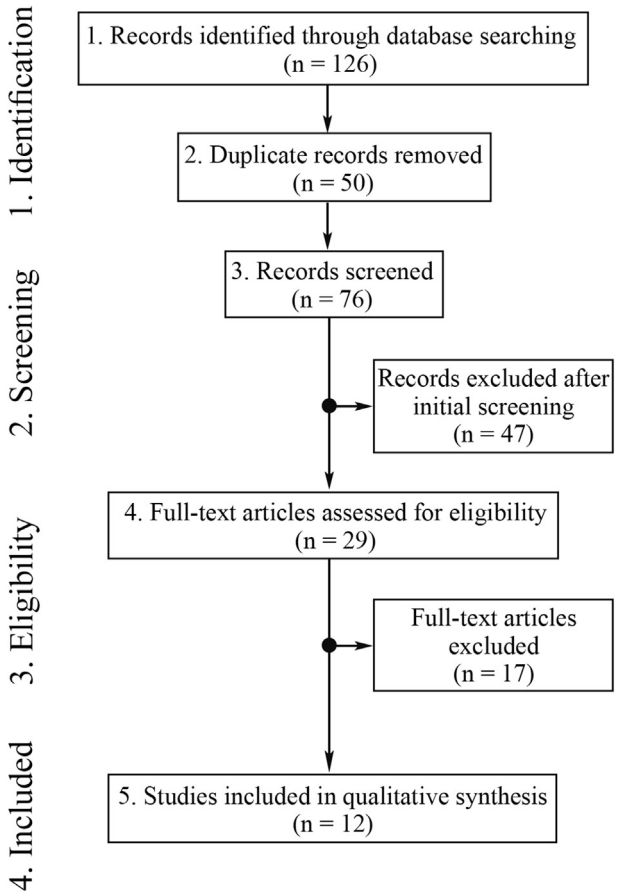

Fig. 1. Study selection flow chart.

(protrusio, femoroacetabular impingement[44,46]), we focused on the results obtained in dysplastic hips. All studies contained FEM of either adult hips or simplified prenatal hips. We found no studies regarding post-birth pediatric hips.

Discussion

The following sections summarize methods and results from the reviewed studies modeling dysplastic hips.

Geometry

Hip FEM requires computational geometries for the pelvis and proximal femur (PF), from either (1) patient-specific volumetric data, usually from computed tomography (CT) scan, or (2) idealized computer-aided design (CAD) models (Table I). Patient-specific CT data for both normal and dysplastic hips were employed in several studies[42,43,45]. Others had only access to either normal[17] or dysplastic[15,47,48] patient-specific CT data and generated models of dysplastic or close-to-normal hips by deforming the acetabular rim or changing morphological parameters of the acetabulum through virtual osteotomy. One study employed patient-specific CT data of dysplastic hips pre- and post-osteotomy[16].

CT-based models demonstrate bony anatomy well, but generating accurate geometries of soft tissues (cartilage and ligaments) is difficult since these structures are poorly seen on CT images. Some studies[42,43,45,48] used CT arthrography (CTA), which is invasive but clearly demonstrates articular cartilage (AC) boundaries, to directly model cartilage thickness (0.5–2.8 mm[45], 0.8–2.0 mm[42,43]). Non-