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An Integrated Concept Explaining for Risk Factors Related to the Onset of Developmental Dysplasia of the Hip Joint

Graf R[1], Hammer N[2,3,4] and Matthiesen D[5]*

[1]*Department of Orthopedics, General and Orthopedic Regional Hospital Stolzalpe, Austria*

[2]*Institute of Macroscopic and Clinical Anatomy, University of Graz, Austria*

[3]*Department of Orthopedic, University of Leipzig, Germany*

[4]*Division of Medical Technology, Fraunhofer Institute for Machine Tools and Forming Technology, Germany*

[5]*Annette Alle 24, Germany*

Abstract

Purpose: A broad body of risk factors exists, predisposing the onset of DDH. Literature is sparse on the subject of combining these risks into a single integrated concept. Revisiting microscopic anatomy of the growing hip joint may help determine such concepts.

Methods: Based on knowledge derived from contemporary hip sonography, it has been shown that shearing forces directed caudo-cranially deform the hyaline cartilage in the preformed acetabular roof in a typical sequence. This acetabular epiphysis region comprising of the chondro-osseous junction between the growth plate and the bony acetabular roof was assessed microscopically. Sections of non-dysplastic and non-dislocated joints were compared with the different types of dislocated joints.

Results: Cartilaginous cell columns in the acetabular epiphysis at the region of the chondro-osseous junction were deformed in a typical sequence, depending on the extent of loading: oblique, oblique and partially damaged or fully damaged.

Conclusion: Shearing forces exerted caudo-cranially *via* the femur not only deform the cartilaginous acetabular roof, but also the chondro-osseous junction in a typical manner, thereby causing damage and subsequent necrosis of the cartilaginous cell columns. The mechanisms causing the femur to press against the acetabular roof and are likely related to a size disproportion of the fetus in utero or fetal positions exerting high pressures to the cartilage. Sonography should be performed as early as possible to detect borderline type IIc joints, which may have no clinical signs to prevent or minimize disease progression related to cartilage column death in the infant's hip joint.

Keywords: Risk factors; Histology; DDH; Graf sonography; Secondary dysplasia of hip joints

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Correspondence:
Graf R, Department of Orthopedics, General and Orthopedic Regional Hospital Stolzalpe, Stolzalpe, Austria, E-mail: reinhardgraf@gmx.at

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Introduction and Background

Risk factors related to the onset of Developmental Hip Dysplasia (DDH) have been studied extensively over the last five decades. Without presenting all of these findings individually, an encompassing summary can be found in the book by Tönnis D [1]. Common risk factors include: Genetics, a family history of DDH, socio-cultural background, high birth weight, breech presentation, female sex, certain hormones such as relaxin and the intrauterine position of the fetus prior to birth [2].

However, it remains unknown to date whether a common mechanism exists, combining these risk factors into a single pathophysiological cluster which results in DDH postpartum. Based on the clinical knowledge of hip sonography [3] elucidating that DDH-related hip dislocation is a developing pathology with typical pathomorphological deformity signs [3,4] (Figure 1), a first attempt has been made to establish a possible an integrated concept. This considers all the risk factors associated with DDH and how these may jointly cause an onset of dysplasia-related dislocation. The chondro-osseous junction of the acetabular roof forms a key structure responsible for the ossification. This area seems to influence the course of DDH in two ways, either resulting in dislocation or secondary dysplasia, or following early treatment with excellent primary results [4]. Histology of the growth and ossification of the cartilage roof seems well known [5-7]. The influences

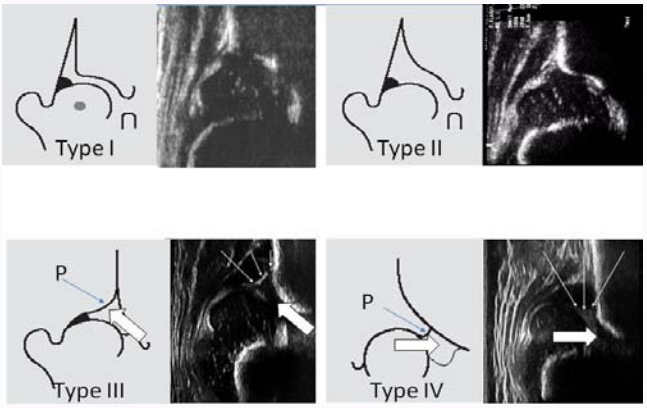

Figure 1: The four sonographic types with the typical deformation of the cartilaginous roof in type III/IV. Arrows show the cartilage roof. In type III most of the cartilage roof is pressed upwards, in type IV downwards. P-Perichondrium.

of mechanical loading, exerted pressure and shearing forces as well as the influence to the growing sites at the border between bony and cartilage acetabular roof have been described [7-9]. However, the literature is sparse on the integration of the morphological findings into concepts explaining the underlying mechanisms resulting in DDH.

We hypothesized that there might be a common sign or cell deformity and histology in the chondro-osseous junction of the acetabular roof, explaining how a majority of risk factors may result in DDH. Histology of the hip region of fetuses and young infants were assessed for this purpose.

Materials and Methods

Histological sections aligned in the frontal plane of acetabular roofs in healthy, non-luxated, 4 to 8-week-old infants where compared with frontal histology sections of luxated hip joints in cadavers (n=5), as well as with micro biopsies obtained during open reductions (n=11). Especially the chondro-osseous junction between the bony and hyaline cartilage roof were observed and compared with histological sections from the literature [5-8]. Additionally, histology in normal and in the deformed acetabulum roof in luxated hip joints was checked microscopically. These sections have been retrieved, fixed and embedded according to standard techniques [10].

Results

The chondro-osseous junction on the acetabular roof in non-dislocated joints yielded typical morphological features, comprising of the following features:

- A resting zone of cartilage.
- A proliferative zone where chondrocytes form cellular columns, aligned in parallel rows perpendicular to the ossification zone.
- A hypertrophic cartilage zone where the ossification starts with increasing infarction of the cartilage cells, and finally,
- An ossification zone, where enchondral bone tissue is formed (Figure 2).

Additionally, vascular sinuses are shown in the roof of the acetabular cartilage, which may become compressed.

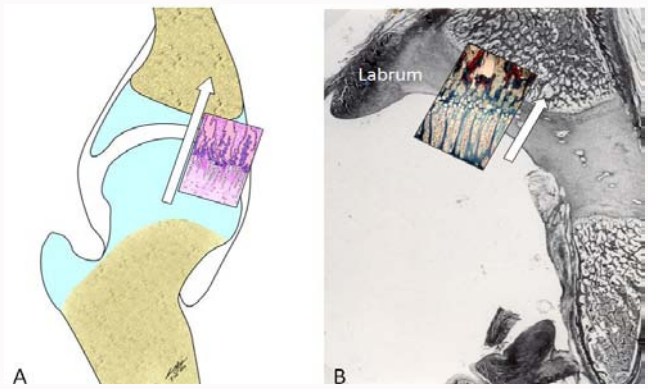

Figure 2: Frontal section which a microscopical inlay to demonstrate the histology on the epiphysial junction in a normal acetabulum: L Labrum. Cell columns are straight ahead to the ossification area (arrow).

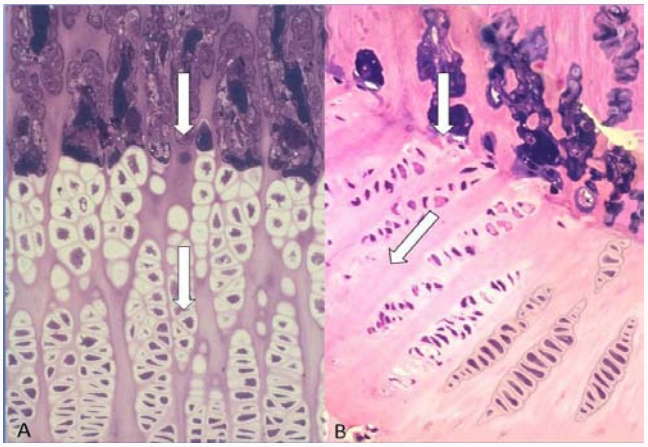

Figure 3: A) Normal chondro-osseous junction, the cells columns are aligned in the same direction as the ossified part. B) Caudo-cranial shearing forces made the obliquity of cell columns.

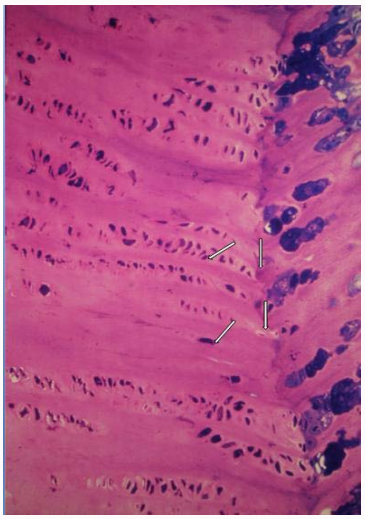

Figure 4: Obliquity of the cell columns (arrows). Additionally, disturbed columns can be seen.

Acetabula of dislocated hip joints show the following features:

- **First stage**: The proliferation zone where those chondrocyte columns, normally aligned perpendicular to the ossification zone

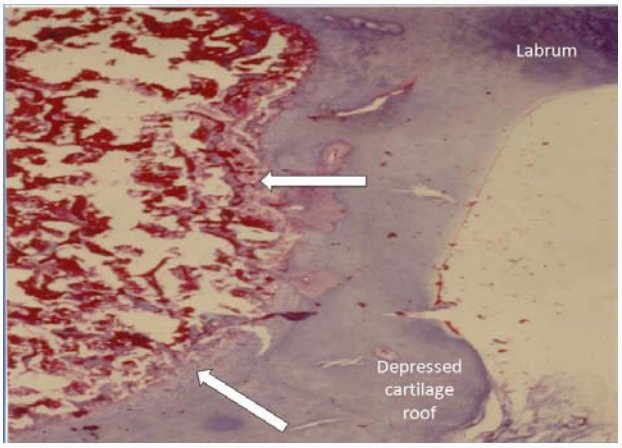

Figure 5: Completely disturbed chondro-osseous junction. The depressed part of the cartilage roof equals the so called neolimbus according to Ortolani [22].

under healthy conditions, become oblique (Figure 3).

- **Second stage**: In some dislocations, the chondrocyte columns become increasingly oblique, and become partially to fully destroyed (Figure 4).

- **Third stage**: Finally, in the last stage, the chondrocyte columns become permanently and irreversibly damaged. The typical cell structure and alignment cannot be seen anymore (Figure 5).

Discussion

In fact, caudo-cranial shearing forces [3] press the cartilage roof upwards deform the anatomical of the growing acetabulum in a typical way [3,5]. Well seen in arthrograms [1], and even better in sonograms [3] (Figure 1).

Shearing forces directed caudo-cranially deform the cartilaginous acetabular roof and are likewise exerted by the proximal femur. These shearing forces increase as a consequence of the increasing obliquity of the osseous roof [4] and are well reflected in the histology of the cartilage (sonographic type IIIb, where the hyaline cartilage becomes fibro-cartilaginous) [3,7], as well as to the chondro-osseous junction between the osseous and the cartilage roof. **Preliminary statement**: Excluded "congenital malformations" (also considered teratologic) or endogenous dysplasia [4], which has its onset in the embryonic period [8], DDH seems to be predominantly a mechanical problem [8,9] with progressive deformation of an initially normally formed acetabulum and a progressive deformation of its chondro-osseous junction [3]. Because of the bending of the cell columns in the chondro-osseous junction, the ossification is impaired and finally stops [6,7].

It remains valuable to understand under which conditions caudo-cranial shearing forces may damage the chondro-osseous junction in the different stages, and as a consequence, what may happen to the morphology and functionality of the joint. A summary of the existing causes and risk factors is listed below, and supplemented in Figure 6.

Less place in utero, some examples

- Higher birth weight: Females tend to have body weight at birth [11]. Weight is related to body size, and in consequence the lack of space in utero may cause more shearing to the hip joint.

- Oligohydramnios [11]: As shown for 1.1, results in lacking space of the fetus when compared to the surrounding uterus, and in consequence, increasing pressure to the hip exerted by the femur.

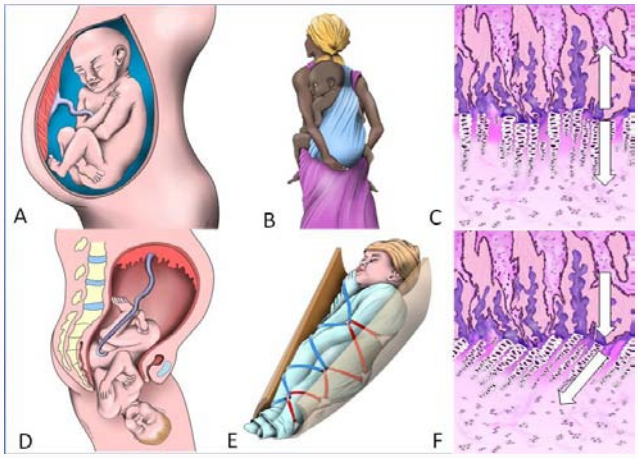

Figure 6: A) Breech presentation with flexed legs with minimal caudo-cranial stress exerted to the acetabulum. B) Frog like carrying mode of infants. C) Cell columns at the chondrosseous junction with normal appearance. D) The infant's left hip joint is pressed against the mother's sacrum. E) Sketch of a device to carry the babies as often used by the Navajo natives of America, resulting in caudo-cranial stress with subsequent shearing forces to the infant's hip joint. F) Exemplified results of caudo-cranial stress with deformed cell columns.

Figure 7: Images of devices to carry infants used by the A) Navajo natives of America, and B) infants in Mongolia, both with extended legs and increasing caudo-cranial shearing forces to the acetabulum.

- Vaginally delivered babies have more decentered joints than those delivered by C-sections (4.7% *vs.* 1.1%) [12,13]. Again, the space limitation of the birth canal may cause shearing to the acetabulum.

Laxity and seasonal aspects

Zarrow et al. [2] have shown that relaxin, a hormone involved in peripartum laxity of the pelvic ring with effects on type I and II collagens, influences ligamentous laxity. Maximum serum levels are reached a few days before birth. This laxity makes it much easier for the shearing forces to produce a dislocation because of increasing instability in the fetus. This stands in correlation with the fact that a seasonal peak for DDH is in correlation with the seasonal variations of hormone induced laxity [14,15].

Breech position with extended knee joints

Given the adductors are crossing both the knee joints and the

hip joints; with flexed knees the muscles remain long enough to not press excessively the femoral head cranially by means of passive muscle traction. Other muscle groups (e.g. muscle iliopsoas) may be equally involved in this situation; however, this has not been quantified to date. *Vice versa*, with knees extended, the muscles become tightened passively and therefore shorten the distance of the femoral head towards the acetabulum in a cranial-dorsal direction. These mechanisms have been further confirmed by Quan et al. [16], who found that preterm infants with extended legs have a higher incidence of DDH when compared to preterm infants with flexion of hips and knees.

Socio-cultural causes

Coleman et al. [17] reported a high incidence of DDH in Apache and Navaho Indians (Figure 7). In contrast, in black Africans DDH rates are extremely low [18]. A likely explanation is the mode of carrying the infants. While in Africa babies are carried in the frog-like position, red Indians such as the Navajo carry them with extended and adducted legs in special wooden devices (Figure 7).

Hereditary components

Vogel [19] found family occurrence as much as 30%. Similar results were found in a number of other studies [20]. However, for DDH, no chromosomal abnormalities have been found to date [8]. A possible explanation may be that DDH may not be an alteration in genes related to the development of the healthy hip joint but to those responsible for body constitution, with effects on the femur in utero pushing cranially into the immature acetabulum. These hereditary components, combined with nutrition, are however inseparably merged with socio-cultural factors.

One exception to the above-mentioned factors is teratologic dislocation of the hip joint. In contrast, DDH seems to be a biomechanically-induced problem, caused by caudo-cranial forces causing shearing at the immature acetabular roof. The shearing forces are not only deforming the hyaline-cartilaginous roof, but also deform the cartilaginous cell columns at the chondro-osseous junction. This can be observed on a micro-structural level. As a primary summary it can be concluded, that the combined mechanisms involved with all risk factors identified so far lead to increasing caudo-cranial loading of the acetabular roof, causing shearing forces resulting in DDH.

Simplified in 3 stages

First stage: The cartilaginous cell columns are only bent by shearing forces: e.g. sonographic type IIc stable/unstable [3]. When treated immediately, there is a high likelihood to reduce the shearing forces, the cell columns become rectified, - with excellent results in clinical treatment.

Second stage: While some columns only become bent, others become partially destroyed (e.g., decentered hip joints). Following treatment interventions, the acetabular roof is partly restored. However, with time a secondary dysplasia is likely to arise since the cartilaginous cell columns will not regain function. In consequence, in spite of the excellent primary results of the treatment at this stage, it is expressly recommended to follow up on these cases to prevent "secondary dysplasia".

Third stage: Cartilaginous columns become fully destroyed in decentered joints (e.g., late diagnosed, type IV). Despite excellent treatment, an acetabular dysplasia will still develop, with the needs for surgical correction [21,22].

Conclusion and Clinical Recommendations

Shearing forces not only deform the cartilaginous roof of the developing acetabulum, they also destroy the chondro-osseous junction, which is responsible for impaired ossification and growth of the acetabulum in three stages. These three stages may provide an explanation for excellent treatment results, secondary dysplasia or the primary need of an immediate surgical correction of the acetabulum. Infant hip joints should be checked sonographically as early as possible after birth, deploying an adequate, standardized technique according the maturation curve of the joints until the end of the 5th week postpartum at the latest. It is vital to determine those joints with a critical range (sonographic types IIc or worse), and to minimize all effects on the joint that increase the shearing forces at the chondro-osseous junction. Otherwise the chondro-osseous junction as the site of ossification is permanently destroyed. Decentered hip joints, even with excellent treatment results following a primary treatment, may be partially damaged concerning the cartilaginous cell columns, and should therefore be followed-up in certain instances to detect a potential onset of secondary dysplasia.

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