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The John Charnley Award: Redefining the Natural History of Osteoarthritis in Patients With Hip Dysplasia and Impingement

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Abstract

Background Structural hip deformities including developmental dysplasia of the hip (DDH) and femoroacetabular impingement (FAI) are thought to predispose patients to degenerative joint changes. However, the natural history of these malformations is not clearly delineated.

Questions/purposes (1) Among patients undergoing unilateral THA who have a contralateral hip without any radiographic evidence of hip disease, what is the natural history and progression of osteoarthritis in the native hip based on morphological characteristics? (2) Among patients undergoing unilateral THA who have a contralateral hip without any radiographic evidence of hip disease, what are the radiographic parameters that predict differential rates of degenerative change?

Methods We identified every patient 55 years of age or younger at our institution who received unilateral primary THA from 1980 to 1989 (n = 722 patients). Preoperative radiographs were reviewed on the contralateral hip and only hips with Tönnis Grade 0 degenerative change that had minimum 10-year radiographic followup were included. A total of 172 patients met all eligibility criteria with the following structural diagnoses: 48 DDH, 74 FAI, and 40 normal morphology, and an additional 6% (10 of the 172 patients) met all eligibility criteria but were lost to followup before the 10-year minimum. Mean age at the time of study inclusion was 47 years (range, 18–55 years), and 56% (91 of 162) of the patients in this study were female. Mean followup was 20 years (range, 10–35 years). Radiographic metrics, in conjunction with the review of two experienced arthroplasty surgeons, determined the structural hip diagnosis as DDH, FAI, or normal morphology. Every available followup AP radiograph was reviewed to determine progression from Tönnis Grade 0 to 3 until the time of last followup or operative intervention with THA. Survivorship was analyzed by Kaplan-Meier methodology, hazard ratios, and multistate modeling. Thirty-five patients eventually

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Each author certifies that his or her institution approved the human protocol for this investigation, that all investigations were conducted in conformity with ethical principles of research, and that informed consent for participation in the study was obtained.

This work was performed at the Mayo Clinic, Rochester, MN, USA.

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underwent THA: 16 (33%) DDH, 13 (18%) FAI, and six (15%) normal morphology.

Results Degenerative change was most rapid in patients with DDH followed by FAI and normal morphology. Among patients who recently developed Tönnis 1 degenerative change, the probability of undergoing THA in 10 years based on hip morphology was approximately one in three for DDH and one in five for both FAI and normal morphology hips, whereas the approximate probability at 20 years was two in three for DDH and one in two for both FAI and normal morphology hips. The likelihood of radiographic degeneration was increased in patients with the following findings: femoral head lateralization > 8 mm, femoral head extrusion index > 0.20, acetabular depth-to-width index < 0.30, lateral center-edge angle $< 25°$, and Tönnis angle $> 8°$.

Conclusions Degenerative change occurred earliest in patients with DDH, whereas the natural history of patients with FAI was quite similar to structurally normal hips. However, patients with cam deformities and concomitant acetabular dysplasia developed osteoarthritis more rapidly. Although the results of this study cannot be directly correlated to highly active patients with FAI, these findings suggest that correction of FAI to a normal morphology may only minimally impact the natural history, especially if intervention takes place beyond Tönnis 0. Analysis of radiographic parameters showed that incremental changes toward dysplastic morphology increase the risk of degenerative change.

Level of Evidence Level III, prognostic study.

Introduction

The relationship of femoral head coverage by the acetabulum exists along a spectrum with undercoverage leading to developmental dysplasia of the hip (DDH) and overcoverage resulting in femoroacetabular impingement (FAI) [8, 17]. Both of these hip morphologies have long been thought to provide a structural predisposition that potentiates premature development of osteoarthritis [3, 4, 7–9, 19]. Nevertheless, which factors predispose certain hips to eventual degenerative change remain uncertain [1, 2]. Importantly, the overall natural history of these conditions remains largely undescribed. Understanding how morphological characteristics alter the rate of degenerative change would have substantial implications for prognostic assessment and joint preservation patient selection [18].

Two prominent reports of which we are aware have examined a form of this question. Hartofilakidis and colleagues [10] described a cohort of 96 hips with FAI morphology yet no degenerative changes on initial plain films. After a mean 18.5-year followup, 17 hips (17.7%) developed radiographic evidence of osteoarthritis with 12 patients (12.5%) undergoing THA. This report lacked a control group and also did not describe progression of degenerative change over time [10]. A natural history of the skeletally mature dysplastic hip has been reported by Murphy and colleagues [15]. This study retrospectively evaluated 286 young patients who had undergone THA for dysplasia, placing focus on the contralateral nonoperative hip. Ultimately, 115 of these patients developed severe osteoarthritis in the contralateral hip before 65 years of age. These patients also had statistically greater derangement of all seven evaluated radiographic features of dysplasia. This investigation lacked a control group and did not describe progression of osteoarthritis over time; furthermore, a substantial portion of patients had signs of mild osteoarthritis at the time of inclusion [15]. Thus, an important current deficit in our knowledge is documentation of degenerative change over time in a cohort representing the spectrum of hip morphologies with no signs of osteoarthritis at study initiation and similar prognostic risk. Additionally, there is still a need for better identification of the radiographic parameters that may pose a worse prognosis in regard to radiographic progression of arthritis.

We sought to answer the following questions: (1) Among patients undergoing unilateral THA who have a contralateral hip without any radiographic evidence of hip disease, what is the natural history and progression of osteoarthritis in the native hip based on morphological characteristics? (2) Among patients undergoing unilateral THA who have a contralateral hip without any radiographic evidence of hip disease, what are the radiographic parameters that predict differential rates of degenerative change?

Patients and Methods

After obtaining institutional review board approval, we used our total joint registry to retrospectively review all patients at our institution who underwent index unilateral THA from 1980 to 1989. The primary goal was to identify patients with Tönnis Grade 0 degenerative change in the contralateral hip at the time of index THA and follow progression of the native hip through time with attention to radiographic characteristics.

Initial inclusion criteria were 55 years of age or younger at the time of index unilateral THA performed in the 1980s for primary osteoarthritis, which identified 722 patients for full chart review. Patients were then excluded based on the following findings in the medical record: prior operation or documentation of degenerative change on the contralateral hip at the time of index THA (n = 274); THA performed for inflammatory arthritis, septic arthritis, osteonecrosis, uncommon congenital disorders (eg, dwarfism), or neoplasms (n = 147); and death < 10 years after index THA (n = 10). This process resulted in 291 patients who received full review of available hip and pelvis radiographs. Eligibility

criteria at this stage of patient identification were minimum 10-year radiographic followup and Tönnis Grade 0 degenerative change in the hip under consideration at the time of index THA on the contralateral hip. On radiographic review of the 291 patients, 119 patients (41%) had evidence of degenerative change, leaving 172. Ten of the 172 patients (6%) had incomplete radiographs, yielding 162 patients who met comprehensive study criteria. Thus, all patients in the final cohort were 55 years of age or younger, had Tönnis Grade 0 degenerative change in their hip at the time of contralateral THA, and had long-term serial radiographic followup. This created a group with a controlled prognostic risk at the time of study inclusion for longitudinal study of natural history based on hip morphology.

The 162 eligible patients had the following structural diagnoses: 48 patients with DDH, 74 patients with FAI, and 40 patients classified as having normal morphology. Mean age at the time of study inclusion was 47 years (range, 18–55 years), and 56% were female. Mean followup was 20 years (range, 10–35 years). Thirty-five patients eventually underwent THA (16 DDH, 13 FAI, six normal morphology).

Preoperative AP pelvis radiographs were used to measure the following parameters on the nonoperative hip as previously described: lateral center-edge angle, Tönnis angle, acetabular version (anteverted or retroverted), acetabular depth (normal, coxa profunda, or protrusio acetabuli), femoral head lateralization (mm), femoral head extrusion index, and acetabular depth-to-width index [5]. Previously published cutoffs were used for these metrics to document association with normal morphology, DDH, or impingement hip morphology as follows: lateral center-edge angle ($< 25°$ = DDH; 25°–40° = normal morphology; $> 40°$ = impingement), Tönnis angle ($< 0°$ = impingement; 0°–10° = normal morphology; $> 10°$ = DDH), femoral head lateralization ($>$ 10 mm = dysplasia), femoral head extrusion index (> 0.25 = dysplasia), acetabular depth (coxa profunda/protrusio acetabuli = impingement), acetabular depth-to-width index (< 0.38 = dysplasia) [5, 13, 14, 20–22]. AP radiographs were the sole view reviewed, because that was the only view available for each patient.

The radiographs were then read independently by two senior arthroplasty surgeons (RTT, RJS) and the hip was classified into one of three of the following morphological categories: normal morphology, DDH, or FAI. Each surgeon was blinded to the other's initial read. Overall agreement between the surgeons was classified "very good" as measured by a kappa statistic of 0.8 (95% confidence interval [CI], 0.5–1.0). All disagreements were handled with a simultaneous reread and consensus opinion. In a few examples, patients displayed features of cam deformity combined with mild acetabular dysplasia. The senior arthroplasty surgeons were in agreement that cam morphology was more prominent in each of these patient's images, leading to their eventual inclusion with the FAI cohort. After final morphology classification, each subsequent available AP pelvis radiograph was then read with the hip assessed on the Tönnis scale from 0 to 3. The Tönnis grade for each available AP pelvis radiograph was recorded and used in subsequent analyses. Followup occurred until all AP pelvis radiographs were reviewed or until THA was performed in the hip under study. A total of 1519 radiographs of the 162 patients were included in the analysis with a median of nine radiographs per patient (range, 3–25) (Appendix 1 [Supplemental materials are available with the online version of *CORR*®.]). Importantly, the median duration of time between successive radiographs was 1.2 years, suggesting excellent followup. Indeed, 75% of the patients had time intervals between images of 3.7 years or less (Appendix 1).

All data were reported descriptively using appropriate summary statistics such as mean and range for continuous variables and count and percentage for categorical variables. Where appropriate, 95% CIs were also reported. Estimates of the probability of progression from Tönnis Grade 0 to Tönnis Grades 1, 2, 3, or THA were generated using the Kaplan-Meier method; rates were reported for the 10- and 20-year estimates along with their corresponding 95% CIs. Cox proportional hazards regression models were used to evaluate the association of potential risk factors with progression from Tönnis 0 to more advanced stages or THA. Associations of five continuous variable radiographic measurements (femoral head lateralization, femoral head extrusion index, acetabular depth-to-width index, lateral center-edge angle, and Tönnis angle) with the study outcomes were examined graphically by fitting smoothed versions of these measurements in Cox models using smoothing splines. All traditionally have been used to identify DDH with specific cutoffs; however, lateral center-edge angle and Tönnis angle have also been used to indicate FAI with values on the other end of the spectrum [20]. These variables were analyzed to determine if incremental changes impacted the risk of disease progression beyond the traditional binary or triphasic categorization. Further analysis was undertaken using multistate Markov models based on all available radiographs (Fig. 1). These models provided estimates of the probability of transitioning from one state to another, where the states are Tönnis Grade 0, 1, 2, 3, and THA (as an absorbing state). In addition to the transition probabilities, the multistate models provided estimates of the average amount of time spent in each state as well as hazard ratios for the risk of transitioning from one state to the next. As a sensitivity analysis, the same multistate models were generated for three other hypothetically enriched or depleted data sets (Appendix 1). These data sets represented situations of either consistent yearly followup or more sparsely obtained data every 2 years or 5 years.

Results of these sensitivity analyses demonstrated that the model based on our actual data set of all available radiographic records is robust. The estimates from both extremes of the model were almost identical to those based on the observed available data with regard to both point estimates and CIs, enhancing credibility of the analytical approach and subsequent results (Appendix 1). All analysis was conducted using SAS Version 9.4 (SAS Institute Inc, Cary, NC, USA) and R version 3.1.1 (R Core Team, R Foundation for Statistical Computing, Vienna, Austria, 2014).

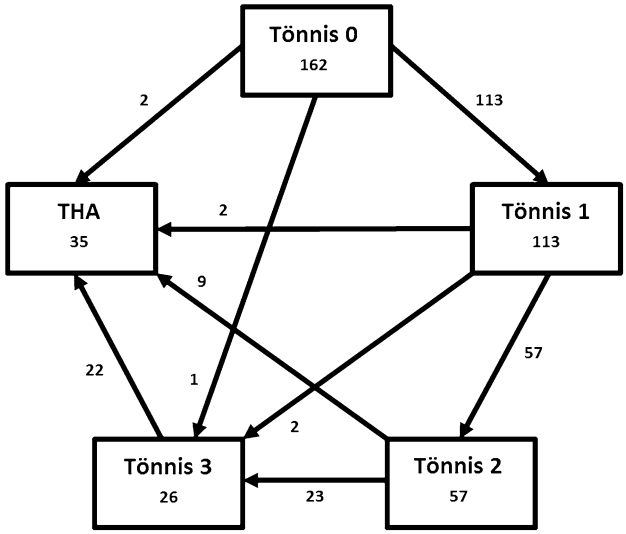

Fig. 1 This diagram summarizes all 162 patients in the study and their observed transitions through the various stages of degenerative change over long-term followup. These transitions served as the basis for subsequent multistate Markov modeling.

Results

Progression of Osteoarthritis as a Function of Morphology

Generally, progression from Tönnis 0 to Tönnis 3 or THA, as demonstrated by Kaplan-Meier analysis, was most rapid in patients with DDH followed by FAI and normal morphology (Fig. 2). There were no differences for early stages of progression (Tönnis 0 to Tönnis 1 or 2) among the three morphologies (Table 1). However, patients with DDH had increased risk of progression from Tönnis 0 to Tönnis 3 and Tönnis 0 to Tönnis 3 or THA compared with patients with normal morphology patients (hazard ratio [HR], 5.0, 95% CI, 1.1–22.1, p = 0.036; HR,

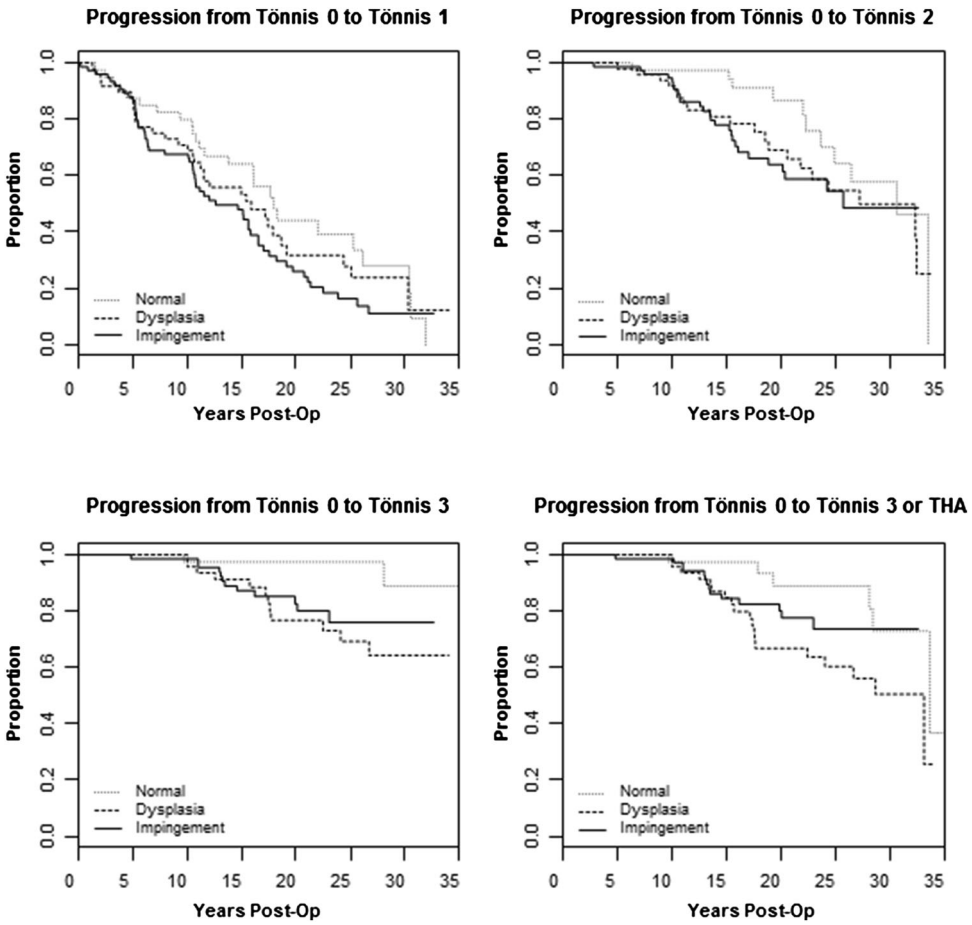

Fig. 2 These Kaplan-Meier (KM) plots demonstrate native hip survival by hip morphology. In general, patients with DDH progressed most rapidly followed by FAI with normal morphology hips progressing the slowest. This did not reach significance at early stages; however, patients with dysplasia had significantly worse survival compared with structurally normal morphology hips from Tönnis 0 to Tönnis 3 and Tönnis 0 to Tönnis 3 or THA.

Table 1. Kaplan-Meier analysis of patient and radiographic factors with stages of degenerative change

Patient or radiographic factor	Level	Tönnis 0-Tönnis 1		Tönnis 0-Tönnis 2		Tönnis 0-Tönnis 3		Tönnis 0-Tönnis 3 or THA	
		KM rates (95% CI)		KM rates (95% CI)		KM rates (95% CI)		KM rates (95% CI)	
		10-year	20-year	10-year	20-year	10-year	20-year	10-year	20-year
Overall	Overall	71 (64–78)	32 (25–41)	94 (91–98)	71 (63–79)	98 (95–100)	84 (78–91)	98 (95–100)	78 (71–85)
Morphological diagnosis	Normal	80 (69–93)	44 (30–66)	98 (93–100)	87 (75–100)	98 (93–100)	98 (93–100)	98 (93–100)	89 (78–100)
	DDH	69 (57–83)	32 (19–51)	92 (84–100)	69 (56–85)	96 (90–100)	77 (65–92)	96 (90–100)	67 (54–83)
	FAI	68 (58–79)	26 (17–40)	95 (90–100)	64 (53–78)	99 (96–100)	83 (74–93)	99 (96–100)	80 (71–91)
Gender	Female	71 (63–81)	34 (24–47)	92 (87–98)	74 (64–85)	97 (93–100)	84 (76–93)	97 (93–100)	79 (70–89)
	Male	70 (61–82)	30 (20–44)	97 (93–100)	67 (56–81)	99 (96–100)	85 (75–95)	99 (96–100)	76 (65–89)
Acetabular	Anteversion	71 (63–79)	32 (24–42)	94 (90–98)	72 (64–81)	97 (94–100)	83 (76–90)	97 (94–100)	75 (67–84)
Version	Retroversion	74 (58–94)	33 (18–62)	100 (100–100)	66 (46–93)	100 (100–100)	95 (85–100)	100 (100–100)	95 (85–100)
Femoral head lateralization	≤ 10 mm	73 (66–81)	35 (27–46)	96 (92–99)	74 (66–83)	99 (96–100)	86 (79–93)	99 (96–100)	81 (74–89)
	> 10 mm	62 (47–83)	19 (8–43)	90 (79–100)	58 (40–84)	93 (84–100)	79 (63–98)	93 (84–100)	63 (46–86)
Femoral head extrusion index	≤ 0.25	75 (67–83)	36 (28–48)	95 (91–99)	74 (66–84)	98 (95–100)	88 (82–95)	98 (95–100)	84 (76–92)
	> 0.25	61 (48–77)	21 (11–41)	93 (86–100)	63 (49–81)	98 (93–100)	74 (60–91)	98 (93–100)	62 (48–80)
Socket	Normal	70 (63–78)	29 (22–39)	93 (89–98)	70 (62–79)	97 (94–100)	83 (77–91)	97 (94–100)	76 (68–84)
	Coxa profunda or protrusio acetabuli	76 (61–95)	52 (33–82)	100 (100–100)	79 (62–100)	100 (100–100)	90 (78–100)	100 (100–100)	90 (78–100)
Acetabular depth-to-width index	≤ 0.38	62 (47–81)	23 (12–47)	88 (78–100)	61 (44–83)	94 (87–100)	75 (60–94)	94 (87–100)	65 (49–86)
	> 0.38	73 (66–82)	35 (26–45)	96 (93–100)	74 (66–83)	98 (96–100)	87 (80–94)	98 (96–100)	81 (74–89)
Lateral center-edge angle	Dysplasia (< 25)	75 (67–84)	37 (28–50)	96 (92–100)	79 (70–88)	99 (97–100)	88 (81–96)	99 (97–100)	84 (76–93)
	Impingement (> 40)	62 (49–77)	25 (14–45)	92 (84–100)	62 (47–80)	94 (87–100)	73 (60–90)	94 (87–100)	62 (48–80)
	Normal (25–40)	71 (51–100)	16 (5–57)	93 (80–100)	48 (26–89)	100 (100–100)	92 (77–100)	100 (100–100)	85 (68–100)
Tönnis angle	Dysplasia (> 10)	76 (68–86)	37 (27–50)	97 (93–100)	82 (73–91)	99 (97–100)	90 (83–98)	99 (97–100)	84 (75–93)
	Impingement (< 0)	61 (49–77)	22 (11–41)	90 (82–99)	57 (43–75)	94 (87–100)	72 (58–88)	94 (87–100)	64 (51–81)
	Normal (0–10)	70 (53–93)	36 (19–67)	95 (86–100)	59 (40–88)	100 (100–100)	89 (76–100)	100 (100–100)	84 (70–100)

KM = Kaplan-Meier; CI = confidence interval; DDH = developmental dysplasia of the hip; FAI = femoroacetabular impingement.

Table 2. Cox proportional hazards regression of patient and radiographic factors with stages of degenerative change

Patient factor analysis	Level	Number	Tönnis 0-Tönnis 1			Tönnis 0-Tönnis 2			Tönnis 0-Tönnis 3			Tönnis 0-Tönnis 3 or THA		
			Events	HR (95% CI)	p value	Events	HR (95% CI)	p value	Events	HR (95% CI)	p value	Events	HR (95% CI)	p value
Morphological diagnosis	DDH	48	32	1.2 (0.7–2.1)	0.414	20	1.5 (0.7–3.2)	0.254	12	5.0 (1.1–22.1)	0.036	19	2.8 (1.1–7.0)	0.029
	FAI	74	56	1.6 (1.0–2.6)	0.056	26	1.8 (0.9–3.7)	0.104	12	4.0 (0.9–17.8)	0.073	14	1.8 (0.7–4.8)	0.238
	Normal	40	25	1.0		11	1.0		2	1.0		6	1.0	
Gender	Male	71	51	1.1 (0.8–1.6)	0.642	23	1.1 (0.6–1.8)	0.808	10	1.0 (0.4–2.1)	0.896	15	1.0 (0.5–1.9)	0.968
	Female	91	62	1.0		34	1.0		16	1.0		24	1.0	

Categorical radiographic parameter analysis	Level	Number	Tönnis 0-Tönnis 1			Tönnis 0-Tönnis 2			Tönnis 0-Tönnis 3			Tönnis 0-Tönnis 3 or THA		
			Events	HR (95% CI)	p value	Events	HR (95% CI)	p value	Events	HR (95% CI)	p value	Events	HR (95% CI)	p value
Acetabularversion	Retroversion	23	17	1.2 (0.7–2.0)	0.468	8	1.1 (0.5–2.3)	0.842	3	0.8 (0.2–2.7)	0.735	3	0.6 (0.2–1.9)	0.348
	Anteversion	139	96	1.0		49	1.0		23	1.0		36	1.0	
Femoral head lateralization	> 10 mm	29	23	1.5 (0.9–2.3)	0.108	12	1.5 (0.8–2.8)	0.224	7	2.1 (0.9–5.0)	0.103	11	2.2 (1.1–4.4)	0.034
	≤ 10 mm	133	90	1.0		45	1.0		19	1.0		28	1.0	
Femoral head extrusion index	> 0.25	43	34	1.5 (1.0–2.2)	0.063	19	1.4 (0.8–2.4)	0.294	12	2.5 (1.2–5.4)	0.02	18	2.5 (1.3–4.7)	0.005
	≤ 0.25	119	79	1.0		38	1.0		14	1.0		21	1.0	
Socket	Coxa profunda or protrusio acetabuli	25	13	0.7 (0.4–1.2)	0.14	6	0.7 (0.3–1.6)	0.347	2	0.5 (0.1–1.9)	0.275	2	0.3 (0.1–1.3)	0.105
	Normal	137	100	1.0		51	1.0		24	1.0		37	1.0	
Acetabular depth-to-width index	> 0.38	128	87	0.7 (0.5–1.1)	0.134	44	0.7 (0.4–1.3)	0.278	16	0.3 (0.2–0.8)	0.008	26	0.4 (0.2–0.9)	0.015
	≤ 0.38	34	26	1.0		13	1.0		10	1.0		13	1.0	
Lateral center-edge angle	Dysplasia (< 25)	47	34	1.4 (0.9–2.0)	0.155	20	1.4 (0.8–2.4)	0.265	13	2.9 (1.2–6.5)	0.013	18	2.5 (1.3–4.9)	0.008
	Impingement (> 40)	14	12	1.6 (0.9–3.0)	0.136	6	1.6 (0.7–3.9)	0.291	3	2.3 (0.6–8.2)	0.215	5	2.5 (0.9–6.9)	0.075
	Normal (25–40)	101	67	1.0		31	1.0		10	1.0		16	1.0	
Tönnis angle	Dysplasia (> 10)	49	37	1.5 (1.0–2.3)	0.039	26	2.7 (1.5–4.7)	0.001	8	3.4 (1.4–8)	0.006	20	3.0 (1.5–6.1)	0.002
	Impingement (< 0)	20	14	1.1 (0.6–2)	0.655	8	2.0 (0.9–4.5)	0.093	14	2.4 (0.7–7.9)	0.158	5	1.9 (0.7–5.3)	0.227
	Normal (0–10)	93	62	1.0		23	1.0		4	1.0		14	1.0	

Continuous radiographic parameter analysis	Level	Number	Tönnis 0-Tönnis 1			Tönnis 0-Tönnis 2			Tönnis 0-Tönnis 3			Tönnis 0-Tönnis 3 or THA		
			Events	HR (95% CI)	p value	Events	HR (95% CI)	p value	Events	HR (95% CI)	p value	Events	HR (95% CI)	p value
Lateralization	(per cm)	162	113	1.7 (1.0–3.0)	0.059	57	1.6 (0.7–3.5)	0.224	26	2.4 (0.8–7.2)	0.112	39	2.9 (1.2–7.1)	0.023
Extrusion	(per 0.1)	162	113	1.4 (1.1–1.7)	0.005	57	1.2 (1.0–1.6)	0.099	26	1.4 (1.1–1.9)	0.018	39	1.4 (1.1–1.8)	0.004
Acetabular depth	(per 0.1)	162	113	0.9 (0.7–1.1)	0.272	57	0.7 (0.5–1.1)	0.086	26	0.4 (0.2–0.8)	0.01	39	0.5 (0.3–0.8)	0.007
Lateral center-edge angle	(per 10°)	162	113	0.8 (0.6–1.0)	0.0817	57	0.9 (0.7–1.2)	0.369	57	0.7 (0.5–1.0)	0.072	57	0.8 (0.6–1.0)	0.036
Tönnis angle	(per 10°)	162	113	1.3 (1.0–1.7)	0.043	57	1.3 (0.9–1.8)	0.113	57	1.7 (1.1–2.5)	0.019	57	1.6 (1.1–2.3)	0.007

HR = hazard ratio; CI = confidence interval; DDH = developmental dysplasia of the hip; FAI = femoroacetabular impingement.

Table 3. Average years spent in each Tönnis stage based on hip morphology

Hip morphology	Mean (95% CI)
Normal	
Tönnis 0	23 (16–34)
Tönnis 1	18 (10–31)
Tönnis 2	9 (4–20)
Tönnis 3	0 (0–1)
Dysplasia	
Tönnis 0	17 (12–24)
Tönnis 1	12 (8–18)
Tönnis 2	6 (4–10)
Tönnis 3	2 (1–3)
Impingement	
Tönnis 0	15 (11–19)
Tönnis 1	13 (9–19)
Tönnis 2	10 (6–17)
Tönnis 3	3 (2–5)

CI = confidence interval.

2.8, 95% CI, 1.1–7.0, p = 0.029, respectively) (Table 2). The risk of progression for FAI compared with normal morphology revealed no differences at all stages (Table 2).

Multistate modeling demonstrated that patients with DDH or FAI spent less time in early stages of disease (Tönnis 0 or Tönnis 1) compared with patients with normal morphology. The mean number of years spent in each Tönnis stage by hip morphology was as follows: Tönnis 0: DDH = 17 years, FAI = 15 years, normal morphology = 23 years; Tönnis Stage 1: DDH = 12 years, FAI = 13 years, normal morphology = 18 years; Tönnis 2: DDH = 6 years, FAI = 10 years, normal morphology = 9 years; Tönnis 3: DDH = 2 years, FAI = 2 years, normal morphology = 0 years (Table 3). Further predictive modeling was performed to show the probability of Tönnis stage transition by hip morphology at 10- and 20-year followup based on any current Tönnis stage (Table 4). For example, among patients determined to have recently progressed to Tönnis Stage 1, the probability for their Tönnis stage in 10 years by hip morphology was as follows: DDH: Tönnis 1 = 44%, Tönnis 2 = 25%, Tönnis 3/THA = 32%; FAI: Tönnis 1 = 47%, Tönnis 2 = 31%, Tönnis 3/THA = 22%; normal morphology: Tönnis 1 = 57%, Tönnis 2 = 24%, Tönnis 3/THA = 19% (Table 4). For the same patient, the probability of their Tönnis stage in 20 years by hip morphology was as follows: DDH: Tönnis 1 = 19%, Tönnis 2 = 16%, Tönnis 3/THA = 65%; FAI: Tönnis 1 = 22%, Tönnis 2 = 26%, Tönnis 3/THA = 52%; normal morphology: Tönnis 1 = 32%, Tönnis 2 = 21%, Tönnis 3/THA = 47% (Table 4). Thus, for a patient who recently developed Tönnis 1 degenerative change, the probability of undergoing THA in 10 years based on hip morphology was approximately one in three for DDH and one in five for both FAI and normal morphology hips, whereas the approximate probability at 20 years for the same patient was two in three for DDH and one in two for both FAI and normal morphology hips (Fig. 3).

Radiographic Parameters Associated With Differential Rates of Joint Degeneration

Several categorical radiographic parameters indicative of specific hip morphologies were shown to differentially change the rate of progression (Table 2). Patients with femoral head lateralization > 10 mm had a greater risk of progression from Tönnis 0 to Tönnis 3 or THA (HR, 2.2; 95% CI, 1.1–4.4; p = 0.034) (Table 2). Patients with femoral head extrusion indices > 0.25 had increased risk of progression both from Tönnis 0 to Tönnis 3 and Tönnis 0 to Tönnis 3 or THA (HR, 2.5, 95% CI, 1.2–5.4, p = 0.02; HR, 2.5, 95% CI, 1.3–4.7, p = 0.005, respectively) (Table 2). Patients with lateral center-edge angles < 25° had increased risk of progression from Tönnis 0 to Tönnis 3 and Tönnis 0 to Tönnis 3 or THA (HR, 2.9, 95% CI, 1.2–6.5, p = 0.013; HR, 2.5, 95% CI, 1.3–4.9, p = 0.008, respectively) (Table 2). Patients with Tönnis angles > 10° had increased risk of progression from Tönnis 0 to Tönnis 1, Tönnis 0 to Tönnis 2, Tönnis 0 to Tönnis 3, and Tönnis 0 to Tönnis 3 or THA (HR, 1.5, 95% CI, 1.0–2.3, p = 0.039; HR, 2.7, 95% CI, 1.5–4.7, p = 0.001; HR, 3.4, 95% CI, 1.4–8.0, p = 0.006; HR, 3.0, 95% CI, 1.5–6.1, p = 0.002, respectively) (Table 2).

Continuous variable analysis of radiographic parameters demonstrated that incremental changes toward a dysplastic morphology increased risk of osteoarthritis progression. Transition from Tönnis 0 to Tönnis 3 or THA for these parameters was as follows: femoral head lateralization increase of 1 cm (HR, 2.9; 95% CI = 1.2–7.1; p = 0.023); femoral head extrusion index increase of 0.1 (HR, 1.4; 95% CI = 1.1–1.8; p = 0.004); acetabular depth-to-width index increase of 0.1 (HR, 0.5; 95% CI = 0.3–0.8; p = 0.007); lateral center-edge angle increase of 10° (HR, 0.8; 95% CI = 0.6–1.0; p = 0.036); and Tönnis angle increase of 10° (HR, 1.6; 95% CI = 1.1–2.3; p = 0.007) (Table 2).

Table 4. Probability of transition to various Tönnis stages based on current Tönnis stage and hip morphology

Normal-transition probabilities at 10 years					
	Tönnis 0	Tönnis 1	Tönnis 2	Tönnis 3	THA
Tönnis 0	65%	26%	6%	0%	3%
Tönnis 1		57%	24%	1%	18%
Tönnis 2			31%	1%	68%
Tönnis 3				0%	100%
THA					100%
Normal-transition probabilities at 20 years					
	Tönnis 0	Tönnis 1	Tönnis 2	Tönnis 3	THA
Tönnis 0	42%	32%	12%	0%	14%
Tönnis 1		32%	21%	1%	46%
Tönnis 2			10%	0%	90%
Tönnis 3				0%	100%
THA					100%
Dysplasia-transition probabilities at 10 years					
	Tönnis 0	Tönnis 1	Tönnis 2	Tönnis 3	THA
Tönnis 0	56%	29%	9%	2%	5%
Tönnis 1		44%	25%	6%	25%
Tönnis 2			19%	7%	74%
Tönnis 3				0%	100%
THA					100%
Dysplasia-transition probabilities at 20 years					
	Tönnis 0	Tönnis 1	Tönnis 2	Tönnis 3	THA
Tönnis 0	31%	29%	14%	4%	23%
Tönnis 1		19%	16%	4%	61%
Tönnis 2			4%	1%	95%
Tönnis 3				0%	100%
THA					100%
Impingement-transition probabilities at 10 years					
	Tönnis 0	Tönnis 1	Tönnis 2	Tönnis 3	THA
Tönnis 0	51%	33%	11%	2%	3%
Tönnis 1		47%	31%	7%	15%
Tönnis 2			36%	12%	52%
Tönnis 3				2%	98%
THA					100%
Impingement-transition probabilities at 20 years					
	Tönnis 0	Tönnis 1	Tönnis 2	Tönnis 3	THA
Tönnis 0	26%	33%	20%	5%	17%
Tönnis 1		22%	26%	8%	45%
Tönnis 2			13%	5%	83%
Tönnis 3				0%	100%
THA					100%

Table 4. continued

Impingement-transition probabilities at 20 years					
	Tönnis 0	Tönnis 1	Tönnis 2	Tönnis 3	THA
THA					100%

To use the tables for patient prediction, select the appropriate hip morphology then use the far left-hand column to select a row with the current Tönnis stage of the hip. The subsequent columns to the right in that row display the probability of the hip progressing to the indicated Tönnis stage or THA at 10 and 20 year followup.

There were noted differences between traditional diagnostic cutoffs for hip morphology and prognostic cutoffs identified in this study for osteoarthritis progression (Table 5). For femoral head lateralization, the risk of progression increased beyond approximately 8 mm for each stage, whereas > 10 mm is traditionally used as the cutoff for DDH (Fig. 4). For femoral head extrusion index, risk of progression increased beyond approximately 0.20 for each stage, whereas > 0.25 is traditionally used as the cutoff for DDH (Fig. 5). For the acetabular depth-to-width index, risk of progression increased below approximately 0.30, whereas < 0.38 is traditionally used as a cutoff for DDH (Fig. 6). For lateral center-edge angle, risk of progression increased below approximately 25°, which is the same cutoff used for DDH (Fig. 7). Lateral center-edge angle did not demonstrate increased risk of progression at larger angles, particularly above 40°, which is used as a cutoff for FAI. For the Tönnis angle, risk of progression increased above approximately 8°, whereas 10° is used as a cutoff for DDH (Fig. 8). Tönnis angle did not demonstrate increased risk for progression at lesser angles, particularly below 0°, which is used as a cutoff for FAI.

Discussion

DDH and FAI are structural hip deformities thought to potentiate premature degenerative change. However, the natural history of these conditions, particularly about those factors that exacerbate osteoarthritis, is poorly understood. The current investigation demonstrates that after mild degenerative change develops in the hip, patients with DDH have a higher probability of progressing to end-stage osteoarthritis or THA at 10- and 20-year followup compared with FAI and normal morphology. Furthermore, radiographic cutoffs are established by this work that are associated with increased risk of osteoarthritis progression with incremental changes toward a dysplastic morphology portending a worse prognosis.

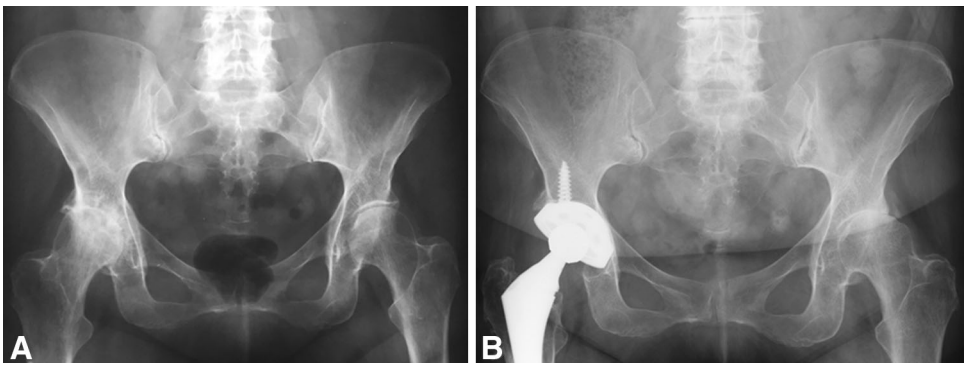

Fig. 3A–B These AP pelvis radiographs show a typical study patient with developmental dysplasia of the left hip. (**A**) This film is a preoperative radiograph for the right hip at the time of study inclusion when the patient still has Tönnis 0 degenerative change in the left hip. (**B**) This subsequent radiograph is taken 18 years later at which point the left hip has developed Tönnis 3 degenerative change.

Table 5. Comparison of diagnostic radiographic cutoffs with proposed prognostic cutoffs

Radiographic parameter	Traditional diagnostic cutoff	Proposed cutoff based on risk of OA progression
Femoral head lateralization	> 10 mm = DDH [5]	> 8 mm
Femoral head extrusion index	> 0.25 = DDH [14]	> 0.20
Acetabular depth-to-width index	< 0.38 = DDH [5]	< 0.30
Lateral center-edge angle	< 25° = DDH [22]	< 25°
	> 40° = FAI [13, 20, 21]	
Tönnis angle	> 10° = DDH [5, 14]	< 8°
	< 0° = FAI [5, 14]	

OA = osteoarthritis; DDH = developmental dysplasia of the hip; FAI = femoroacetabular impingement.

This study has a number of limitations. First, the sample size is modest for each of the three groups included in the final analysis, which likely explains why several results only trended toward significance despite point estimates of effect that actually seemed large. As such, the true effect may be underestimated or overestimated for some analyses. However, cohorts such as this have traditionally been very difficult to obtain, which was especially true for our study with more stringent eligibility criteria than previous work on the topic. Second, all measurements and categorization were based off AP pelvis radiographs. Unfortunately, this was the only view available for every patient in the study at each followup time point. It is well recognized that alternative radiographic views and three-dimensional imaging are often used to provide more complete information about hip morphology in modern practice. Third, the natural history of DDH, FAI, and normal morphology hips in patients with contralateral THA may not replicate other populations. It is possible that these patients place more stress on their native hip or that perhaps they have intrinsically poor cartilage given the young age of their first THA. However, the contralateral THA was essential to identifying a group of patients who would undergo serial radiographic followup. More importantly, it provided an excellent means of controlling prognostic risk in the native hip under study because young patients receiving THA specifically for degenerative joint disease presumably place similar demand on their native hip during ensuing decades. This could potentially be seen as a best case scenario if we assume that patients with contralateral THA are less active. Furthermore, this would likely be

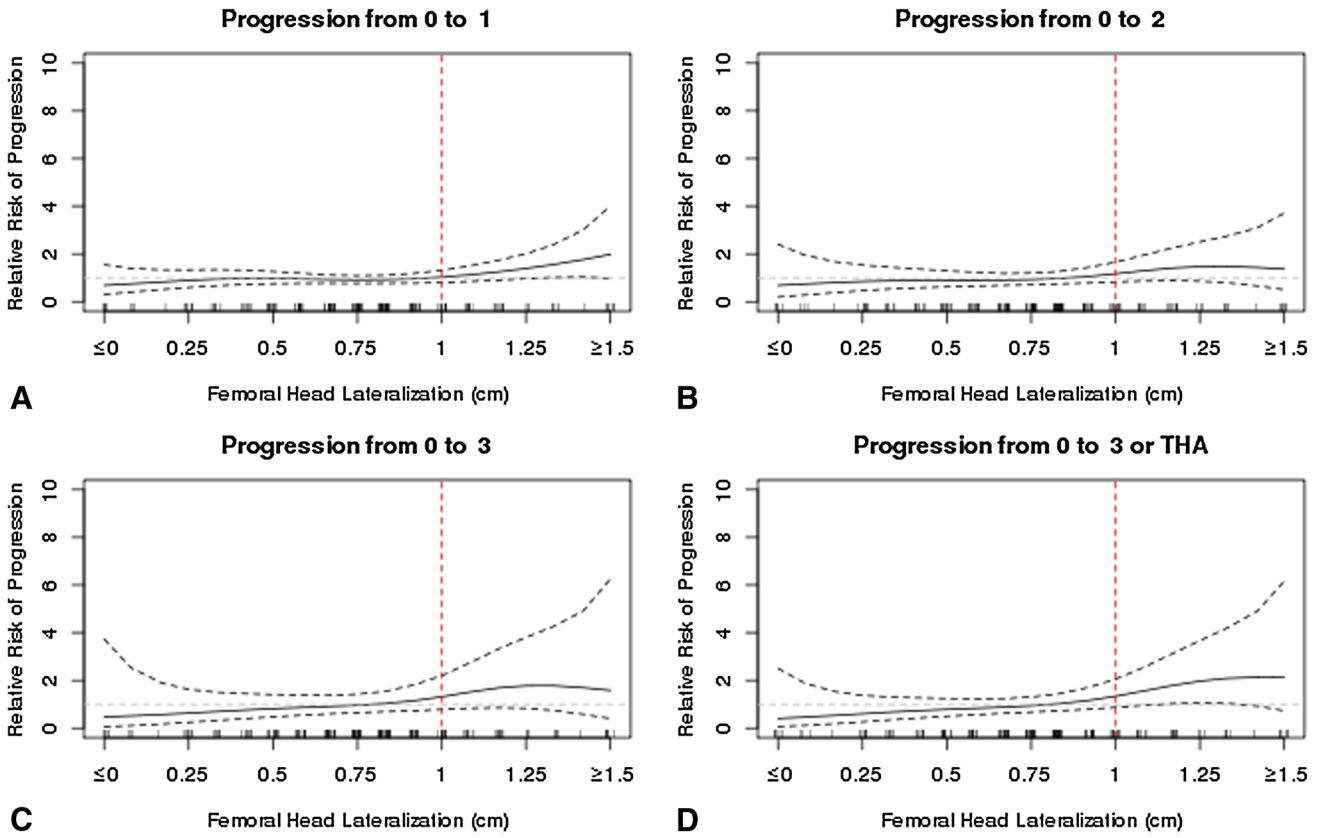

Fig. 4A–D This plot shows femoral head lateralization in a continuous fashion to describe the impact on risk of hip degeneration in the overall cohort. The horizontal dashed lines show a relative risk of 1. The red vertical dashed line at 1 cm shows the common cutoff for a morphological diagnosis of DDH (> 1 cm) versus normal morphology (< 1 cm) hips. The curvilinear solid line demonstrates the relative risk of degeneration as a function of femoral head lateralization. The curvilinear dashed lines represent the 95% CI of the relative risk. Risk of degeneration increases above 8 mm of femoral head lateralization.

more important in FAI hips in which activity and ROM resulting in impingement lead to mechanical damage of the joint. Fourth, standard radiographic followup intervals limited the precision of transition date identification. However, a sensitivity analysis of the multistate modeling was conducted with hypothetically enriched and depleted data sets. Importantly, this sensitivity analysis revealed no significant differences from our reported findings, strengthening confidence in the presented data.

Perhaps the most valuable information for surgeons comes from the multistate modeling (Table 4). It shows that for a patient who recently developed Tönnis 1 degenerative change, the probability of undergoing THA in 10 years based on hip morphology is roughly one in three for DDH and one in five for both FAI and normal morphology hips, whereas the approximate probability at 20 years for the same patient is two in three for DDH and one in two for both FAI and normal morphology hips. Thus, early joint preservation intervention on patients with DDH seems more likely to positively influence the natural history of their hip than intervention on patients with FAI, provided they do not have a large cam deformity with concomitant acetabular dysplasia. As mentioned, two reports have similarly examined the natural history of FAI and DDH. Hartofilakidis and colleagues [10] retrospectively evaluated 96 hips with radiographic evidence of FAI

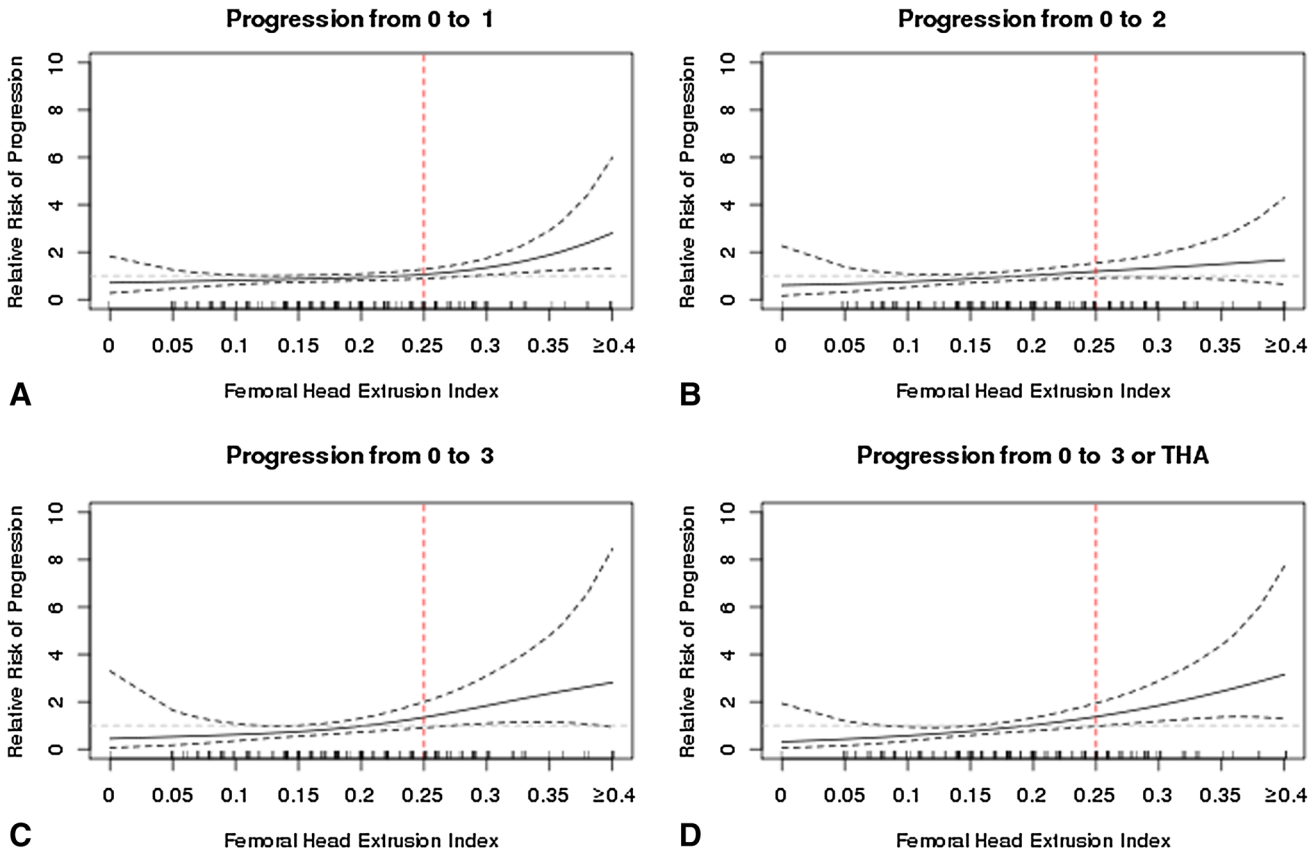

Fig. 5A–D This plot shows femoral head extrusion index in a continuous fashion to describe the impact on risk of hip degeneration in the overall cohort. The horizontal dashed lines show a relative risk of 1. The red vertical dashed line at 0.25 shows the common cutoff for a morphological diagnosis of DDH (> 0.25) versus normal morphology (< 0.25) hips. The curvilinear solid line demonstrates the relative risk of degeneration as a function of femoral head extrusion index. The curvilinear dashed lines represent the 95% CI of the relative risk. Risk of degeneration increases above a femoral head extrusion index of 0.20.

and no degenerative change in the hip. Murphy and colleagues [15] retrospectively evaluated 286 young patients who received THA for dysplasia. Both of these studies lacked a control group and did not describe progression of osteoarthritis over time; furthermore, in the Murphy et al. study, a substantial portion of included patients had signs of degenerative change in the hip under study at the time of inclusion. However, results of these two studies were quite similar to ours with respect to rates of end-stage degeneration and eventual need for THA. A total of 40.2% of Murphy et al.'s group underwent THA compared with 28.6% at 20 years and 43.3% at 30 years in our study for patients with dysplasia. The followup time is unclear in the Murphy et al. study; however, the higher percentage may be accounted for by the fact that some patients had signs of degenerative change at the time of study inclusion. Hartofilakidis et al.'s group showed a 17.7% rate of end-stage arthritis (12.5% received THA) with mean followup of 18.5 years (range, 10–40 years) compared with 19.8% at 20 years and 26.2% at 30 years with mean followup of 20 years (range, 10–35 years) in our study for patients with FAI.

Our data suggest that patients with radiographic features of dysplasia were at highest risk of progression in the entire cohort. This is in line with the report by Murphy and colleagues [15] who documented radiographic

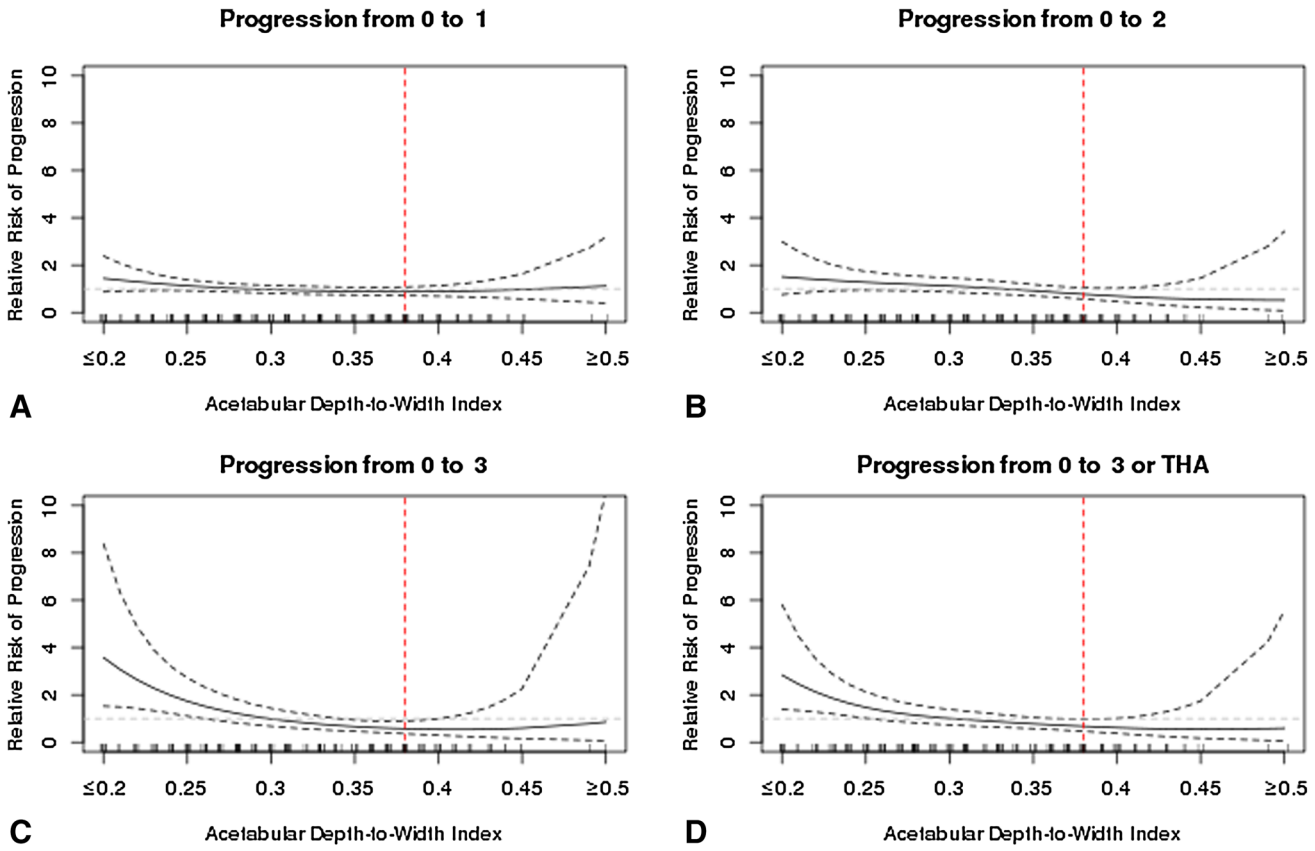

Fig. 6A–D This plot shows acetabular depth-to-width index in a continuous fashion to describe the impact on risk of hip degeneration in the overall cohort. The horizontal dashed lines show a relative risk of 1. The red vertical dashed line at 0.38 shows the common cutoff for a morphological diagnosis of DDH (< 0.38) versus normal morphology (> 0.38) hips. The curvilinear solid line demonstrates the relative risk of degeneration as a function of acetabular depth-to-width index. The curvilinear dashed lines represent the 95% CI of the relative risk. Risk of degeneration increases below an acetabular depth-to-width index of 0.30.

features consistent with more severe dysplasia in patients from their study who eventually developed osteoarthritis. At the other end of the spectrum, our data also demonstrated that among patients with FAI, an increased femoral head extrusion index was the strongest radiographic measure to portent joint degeneration. Patients classified with FAI and increased femoral head extrusion indices fit with the cam subtype of FAI. We also found that patients with cam-type FAI and concomitant low lateral center-edge angles or high Tönnis angles (both representing acetabular dysplasia) were at increased risk of osteoarthritis progression. Co-occurrence of acetabular dysplasia and FAI has been documented previously with authors positing that the combination increases the risk of intraarticular pathology [2, 6, 11, 12, 16]. Tannast and colleagues [20] recently published modified reference values for acetabular overcoverage and undercoverage based on a cohort managed with hip preservation surgery, postulating that previous cutoffs may not provide optimal accuracy. Examining this from a different perspective, we attempted to understand if current diagnostic cutoffs of radiographic parameters are predictive of osteoarthritis progression. The data for femoral head extrusion index, femoral head lateralization, acetabular depth-to-width index, and Tönnis angle suggest that risk of degeneration actually begins increasing with less extreme values than

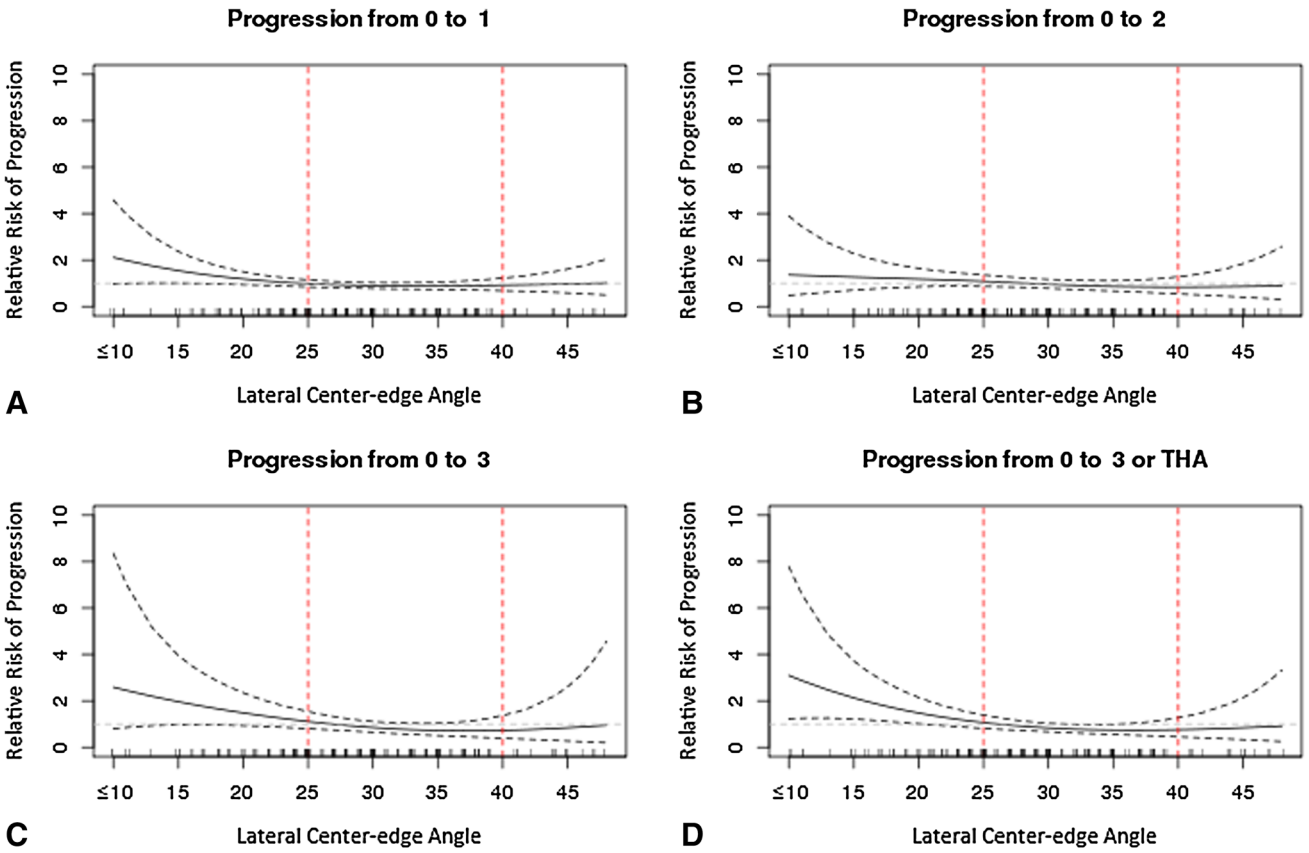

Fig. 7A–D This plot shows lateral center-edge angle in a continuous fashion to describe the impact on risk of hip degeneration in the overall cohort. The horizontal dashed lines show a relative risk of 1. The red vertical dashed lines at 25° and 40° show the common cutoffs for a morphological diagnosis of DDH (< 25°) versus normal morphology (25°–40°) versus FAI (> 40°) hips. The curvilinear solid line demonstrates the relative risk of degeneration as a function of lateral center-edge angle. The curvilinear dashed lines represent the 95% CI of the relative risk. Risk of degeneration increases below a lateral center-edge angle of 25°.

are suggestive of cutoffs for dysplasia (Table 5). These new proposed radiographic cutoffs provide an opportunity to modify natural history prognostication for patients.

In summary, these data can serve as an adjuvant prognostic tool for surgeons, enabling more informed patient counseling and decisions on how to manage disease as well as if or when to intervene. For example, although results from this study cannot be directly correlated to highly active patients with FAI, the predictive tables indicate that correction of FAI to a normal morphology may only minimally impact the natural history, especially if intervention takes place beyond Tönnis 0. Positive corrections are most likely to take place in patients with large cam lesions and shallow sockets. However, the data also indicate that correction of DDH to normal morphology at early Tönnis stages seems more likely to alter a patient's natural history with earlier intervention providing greater benefit. Future studies should formally evaluate these questions by using a similar multistate modeling approach between patients with structural hip deformity who did and did not receive joint preservation surgery. This study also identified radiographic parameters that predict more rapid degenerative change, both in continuous and categorical fashions, subclassified by hip morphology. Specifically, incremental changes toward dysplastic morphology portend a worse prognosis. Although this study provides new information, perhaps the greatest weakness is the statistical uncertainty around many of the point estimates. Similar efforts at other centers would be valuable to either validate or adjust and improve precision of these results.

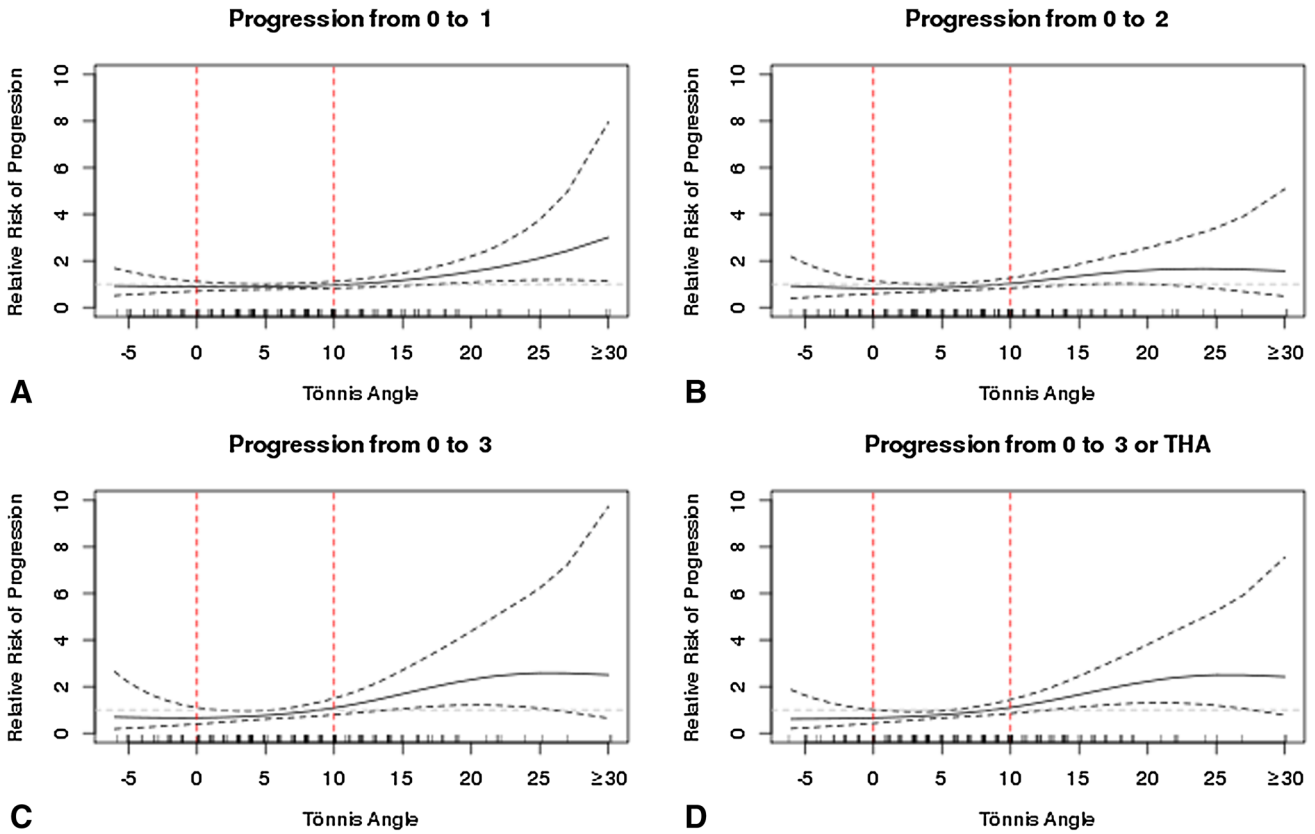

Fig. 8A–D This plot shows Tönnis angle in a continuous fashion to describe the impact on risk of hip degeneration in the overall cohort. The horizontal dashed lines show a relative risk of 1. The red vertical dashed lines at 0° and 10° show the common cutoffs for a morphological diagnosis of FAI (< 0°) versus normal morphology (0°–10°) versus DDH (> 10°) hips. The curvilinear solid line demonstrates the relative risk of degeneration as a function of Tönnis angle. The curvilinear dashed lines represent the 95% CI of the relative risk. Risk of degeneration increases above a Tönnis angle of 8°.

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