

DEA-computed Contact Stress as Computational Biomarker for OA Development in the Untreated Contralateral Hip of Individuals that Underwent Unilateral PAO to Treat Hip Dysplasia

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INTRODUCTION: While hip dysplasia is a common cause of early onset hip osteoarthritis (OA), there are many dysplastic hips that function into old age without developing OA. Dysplastic hips treated with periacetabular osteotomy (PAO) that have high joint contact stresses calculated using a technique called discrete element analysis (DEA) have been shown to have worse cartilage damage and patient-reported outcomes than hips with lower contact stress [1, 2]. This suggests that there may also be an association between elevated joint contact stress and joint failure in surgically untreated dysplastic hips, but this has not yet been shown. Therefore, the aim of this study was to use DEA modeling of the surgically untreated contralateral hip in individuals that underwent unilateral PAO for hip dysplasia, to determine if elevated contact stress may serve as a computational biomarker for future development of OA in these hips.

METHODS: Patients who underwent a unilateral PAO at our institution between 2003 and 2010 were identified from an existing cohort of patients who had both preoperative and postoperative CT scans available for generating DEA models [2]. Patients who underwent unilateral PAO were contacted to document any surgeries on either hip since their previous PAO, and to complete patient-reported outcome measures for their originally non-operated, contralateral hip. Using REDCap, patients completed the International Hip Outcomes Tool (iHOT-12), modified Harris Hip Score (mHHS), PROMIS Bank v2.0 – Physical Function, and PROMIS Bank v1.1 – Pain Interference for each hip individually. Lateral center edge angle (LCEA), Tönnis angle, and Tönnis grade of the non-operated, contralateral hip were assessed on anteroposterior radiographs acquired preoperatively for the hip treated with PAO or on early postoperative radiographs when preoperative images were unavailable. The non-operative, contralateral hip was considered dysplastic if the LCEA was $\leq 20^\circ$.

Patient-specific DEA models of the non-operative, contralateral hip were constructed from the CT scan acquired preoperatively for the original PAO. DEA was used to calculate hip joint contact stress and contact area. Patient age at follow-up, or age at total hip arthroplasty (THA) of the original contralateral hip, was used in calculation of contact stress-time exposure, a metric of cumulative exposure to contact stresses over time. Primary comparative analyses were performed between preserved and failed contralateral hips, with failure defined by either conversion to THA or a native hip with a reported mHHS <70. Secondary analyses were conducted for dysplastic and non-dysplastic contralateral hips. Between-group differences in categorical measures were evaluated using chi-square or Fisher's exact tests, as appropriate. Ordinal measures were further evaluated using Cochran-Armitage trend tests. Continuous measures were evaluated using independent *t*-tests, if normally distributed, or Wilcoxon Rank Sum tests, if not. Statistical significance was defined as $p < 0.05$.

RESULTS: 56 patients consented to participate in our study or had joint failure defined by conversion to THA noted in the medical record. Among those 56 participants, 4 participants had incomplete outcomes, 11 hips had failed by THA, and 7 were native hips defined as failed by mHHS <70, leaving the remaining 34 hips as preserved. Median follow-up was 17.5 years (IQR=16-20) for preserved contralateral hips, 16 years (IQR=16-17) for hips that failed by mHHS <70, and 14.5 years (IQR=8-17) for hips that failed by THA. Of the preserved contralateral hips, 3/34 (8.8%) had a Tönnis grade ≥ 2 at the time of the original PAO on the opposite side, as did 4/17 of the failed contralateral hips (23.5%).

Failed hips tended to have a lower median LCEA (17.6° (11° - 20.9°) vs 20.9° (16° - 24.1°)) and greater average Tönnis angle ($13.9^\circ \pm 6.8^\circ$ vs $10.5^\circ \pm 6.0^\circ$) than preserved hips, though this did not reach statistical significance ($p=0.07$). Preserved hips had lower mean contact stress (3.5 ± 0.7 vs 4.3 ± 1.5 MPa; $p=0.043$), lower peak contact stress (16.8 ± 4.0 vs 21.2 ± 7.8 MPa; $p=0.028$), and peak contact stress time exposures (34.5 vs 44.8 MPa-years; $p=0.037$) than failed hips (Table 1). Hips that failed by conversion to THA tended to have higher peak and mean contact stress than hips without THA (23.5 vs 16.8 MPa, $p=0.039$; 4.6 vs 3.5 MPa, $p=0.082$, respectively).

30/52 (57.7%) participants with complete patient-reported outcome scores had radiographically dysplastic untreated contralateral hips, of which 13 (43.3%) hips failed and 17 (56.7%) were preserved. 8 of the hips that failed, did so by conversion to THA. Among this subset of untreated dysplastic hips, both average and peak contact stress-time exposure was greater in hips that failed ($p=0.054$, Table 1).

DISCUSSION: Pathologic contact mechanics were associated with joint failure in the untreated contralateral hips of individuals who had a unilateral PAO to treat hip dysplasia at >15-year average follow-up. Not all of the initially non-operated hips were dysplastic, which would explain the reason for not performing a PAO for those individuals. A limitation to this work is that the reason for not performing PAO in individuals with dysplastic deformity on the contralateral side is unknown, but for some cases it might have been advanced OA (Tönnis grade ≥ 2).

SIGNIFICANCE: These findings highlight the potential of computational techniques to predict future joint failure in untreated dysplastic hips or in patients that underwent unilateral PAO for hip dysplasia.

REFERENCES: [1] Aitken HD, et al. *J Orthop Res.* 2022;40(11):2632-2645, [2] Aitken HD, et al. *J Orthop Res.* 2024;42(12):2773-2783.

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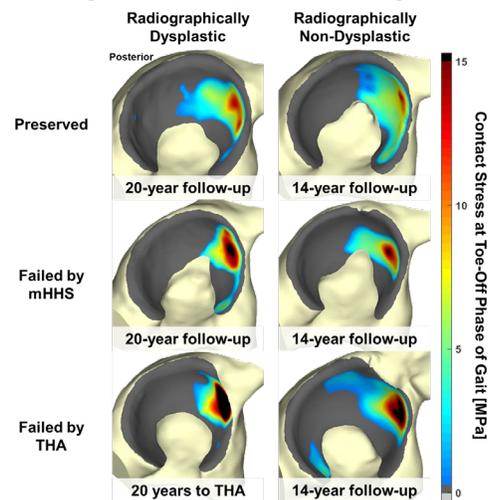


Figure 1. Example contact stress maps for two examples each from the preserved (top row), failed by mHHS (middle row), and failed by THA (bottom row) groups. Data are shown with posterior depicted to the left.

	ALL HIPs				p-value	DYSPLASTIC HIPs				
	Preserved		Failed			Preserved		Failed		
	mean±SD	median(IQR)	mean±SD	median(IQR)		mean±SD	median(IQR)	mean±SD	median(IQR)	
Average Contact Stress (MPa)	3.5±0.7		4.3±1.5		0.043	3.6±0.7		4.4±1.7		0.114
Peak Contact Stress (MPa)	16.8±4.0		21.2±7.8		0.028	17.1±4.2		21.4±9.0		0.125
Average Contact Area (mm²)	793.2±199.7		701.6±238.3		0.142	744.7±130.7		699.4±234.7		0.54
Average Stress-Time (MPa-years)		7.6(6.0-9.1)		8.6(7.7-12.2)	0.088		7.4(6.0-9.1)		9.8(7.9-12.5)	0.054
Peak Stress-Time (MPa-years)		34.5(27.1-45.6)		44.8(36.8-56.7)	0.037		35(25.9-45.0)		48.8(36.8-61.6)	0.054

Table 1. DEA-computed contact stress data for all contralateral to PAO hips (left), and contralateral to PAO hips with LCEA $\leq 20^\circ$.