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Uneven intervertebral motion sharing is related to disc degeneration and is greater in patients with chronic, non-specific low back pain. An *in-vivo*, cross-sectional cohort comparison of intervertebral dynamics using quantitative fluoroscopy

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Running head:

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Abstract

Purpose: Evidence of intervertebral mechanical markers in chronic, non-specific low back pain (CNSLBP) is lacking. This research used dynamic fluoroscopic studies to compare intervertebral angular motion sharing inequality and variability (MSI and MSV) during continuous lumbar motion in CNSLBP patients and controls. Passive recumbent and active standing protocols were used and the relationships of these variables to age and disc degeneration were assessed.

Methods: Twenty patients with CNSLBP and 20 matched controls received quantitative fluoroscopic lumbar spine examinations using a standardised protocol for data collection and image analysis. Composite disc degeneration (CDD) scores comprising the sum of Kellgren and Lawrence grades from L2-S1 were obtained. Indices of intervertebral motion sharing inequality (MSI) and variability (MSV) were derived and expressed in units of proportion of lumbar range of motion from outward and return motion sequences during lying, (passive) and standing (active) lumbar bending and compared between patients and controls. Relationships between MSI, MSV, age and CDD were assessed by linear correlation.

Results: MSI was significantly greater in the patients throughout the intervertebral motion sequences of recumbent flexion (0.29 vs 0.22, p= 0.02) and when flexion, extension, left and right motion were combined to give a composite measure (1.40 vs 0.92, p=0.04). MSI correlated substantially with age (R=0.85, p=0.004) and CDD (R=0.70, p=0.03) in lying passive investigations in patients and not in controls. There were also substantial

correlations between MSV and age (R=0.77, p=0.01) and CDD (R=0.85, p=0.004) in standing flexion in patients and not in controls.

Conclusion: Greater inequality and variability of motion sharing was found in patients with CNSLBP than in controls, confirming previous studies and suggesting a biomechanical marker for the disorder at intervertebral level. The relationship between disc degeneration and MSI was augmented in patients, but not in controls during passive motion and similarly for MSV during active motion, suggesting links between *in vivo* disc mechanics and pain generation.

Keywords: back pain, spinal injuries, kinematics, fluoroscopy, diagnosis

Background

Concepts of lumbar spine stability cover a range of complexity. Intervertebral angle change (IV-RoM) is not now thought to be very useful, due to wide population variations, although range of translation is generally preferred by spinal surgeons who assess for instability [1]. These measures, although of questionable validity, are nevertheless accessible from plain radiographs. To probe more deeply and investigate more subtle forms of instability, continuous intervertebral motion measures are needed [2-5].

For the assessment of mechanical destabilisation of the spine caused by injury, the intervertebral neutral zone is thought to be the most sensitive measure [6]. Although its measurement has been generally confined to cadaveric studies, the advancing sophistication of quantitative fluoroscopic systems (QF) is beginning to provide a surrogate *in vivo* measure in the form of slope of the intervertebral rotation-time curve, (also known as the attainment rate) [7-10].

Chronic, non-specific low back pain (CNSLBP) is widely considered to be at least partially of mechanical origin, due to its susceptibility to movement and position. It is also considered to be related to intervertebral disc degeneration [11]. However, no reliable diagnostic tool that could help a clinician to determine if a disc is the source of the pain in patients with chronic LBP is currently available [12]. Instead, relationships between trunk myoelectric activity, co-ordination and directional preference are more prevalent in the clinical biomechanics literature [13-15]. However, without an assessment of the relationship between segmental mechanics and pain, identification of biomechanical markers in CNSLBP will remain elusive.

While the *in vivo* investigation of intervertebral loading is still problematical, kinematic studies are becoming more common [16]. The lumbar spine is a kinetic chain that requires the sharing of motion between levels during bending. Various aspects of intervertebral motion sharing have been investigated in cadaveric studies [17-19] in plain radiographic studies *in vivo* [20-24] and in continuous radiographic studies [3, 5, 8, 20, 25-30]. Most of these have studied motion onset and displacement, however, two that studied displacement [4, 24] and one that studied pattern variations [29], found differences between patients with CNSLBP and controls.

Intervertebral motion pattern variation studies are of interest because they provide more information than end of range studies and can be more readily applied to contemporary concepts of spine stability. Reeves and Cholewicki [31] identified impaired restraint and performance in the passive and active intervertebral subsystems as subset measures of subtle instability, where restraint is the ability of a system to resist an imposed perturbation and performance the ability to return to the original position once the perturbation has been removed**.** In the lumbar spine, the average range of differences in the sharing of motion by each intervertebral level over the sequence reflects the inequality of restraint across levels (MSI) (Fig 1). Its variability throughout the motion (MSV), may be considered as an expression of intervertebral motion control.

Fig 1 about here

In assessing the possible role of intervertebral motion sharing in CNSLBP, Mellor et al [29] investigated the variability of recumbent intervertebral passive motion sharing and found it to be greater in patients with CNSLBP than in controls. Therefore, it was thought a possible biomechanical marker for CNSLBP. These studies only addressed motion sharing variability

(control) and not equality of restraint - and their replication has not been attempted until now. They also did not account for the effects of disc degeneration. We therefore attempted to replicate these studies, adding a measure of motion sharing inequality, investigating motion under load and incorporating disc degeneration as a possible source of interaction with these measures.

Objectives

1. To see if previous investigations of differences between patients with CNSLBP and healthy controls using passive recumbent motion could be replicated as a biomechanical marker [29].
2. To determine if these differences were also present during standing flexion motion investigations.
3. To determine the relationships between uneven motion sharing and age and disc degeneration.

Methods

We conducted two cross-sectional, prospective observational studies of intervertebral motion sharing in the lumbar spine – one during passive recumbent motion and the other during active weight bearing motion.

Participants

Forty participants were recruited. Twenty were patients who had been referred for continuous radiographic (QF) studies for CNSLBP and 20 were healthy control volunteers recruited from staff, students and visitors to our institution. Controls were matched as closely as possible to patients for age and gender. Participants were divided into two

cohorts. Cohort 1 had received passive, recumbent QF investigations in left, right, flexion and extension motion similar to the 2014 study of Mellor [29] and Cohort 2 had received active standing flexion QF investigations only. Participants could be included if they were: male or female, age 21-80, BMI <30, with no history of previous back or abdominal surgery or spondylolisthesis, no medical radiation exposure of >8mSV in the previous 2 years and no pregnancy (females). Controls had to have been free of any back pain that limited their normal activity for more than one day in the previous year and patients had to have had their back pain for longer than 3 months. All participants gave informed consent. The study received a favourable ethical opinion by the National Research Ethics Service (South West 3, REC reference 10/H0106/65).

Image acquisition and analysis

QF image acquisition and analysis of Cohort 1 were similar to that described by Mellor et al [29]. Briefly, participants lay on a movable table in which the trunk section was motorised and driven by a controller (Atlas Clinical Ltd.), which caused it to execute a bending angle of 40^{o} during separate left, right, flexion and extension motion sequences while fluoroscopic screening took place. For Cohort 2, participants stood with their right side against an upright motion frame with their pelvises secured and their arms on a projecting rest which guided them through a flexion angle of 60^{o} and back using the same controller apparatus as for the lying procedure. Thus, Cohort 1 received passive, recumbent motion in 2 planes and 4 directions and Cohort 2, active, weight bearing motion in flexion only.

The motion controllers accelerate at 6°s^{-2} for the first second followed by a uniform 6°s^{-1} thereafter. The images were collected at 15Hz using a Siemens Arcadis Avantic digital C-arm fluoroscope (Siemens GMBH). Images were exported to a computer workstation and

analysed using manual first image registration and thereafter bespoke frame to frame tracking using codes written in Matlab (V2013 – the Mathworks Inc). Anonymised image sequences were analysed by one operator (AxB) and outputted to an Excel spreadsheet in the form of frame-to frame measurements of intervertebral angular rotation throughout each motion sequence. Accuracy and repeatability for intervertebral rotations using this method have been determined as: Accuracy (side bending 0.32°, flexion-extension 0.53°)[32]. Inter and intra observer repeatability for left, right, flexion and extension recumbent motion ranged from ICC 0.74-0.99 and SEM 0.08-0.77° and for weight bearing flexion from ICC 0.94-0.96, SEM 0.23-0.61°[9, 29]. The rotational angles at each intervertebral level were transformed by a second operator (AB) into proportional motion of the segments of lumbar spine (L2-L5 in passive recumbent motion and L2-S1 in standing flexion). The proportional motion of segments refers to their relative contributions to the motion of the lumbar spine at all points in the bend[29].

Two parameters of the proportional motion sharing were extracted: Motion Sharing Inequality (MSI) and Motion Sharing Variability (MSV) (Fig 2). A full account of the derivation is given in the Appendix, but briefly, MSI was calculated as the average filtered range contributions to the motion (fRC_i) across the N image data points remaining after filtering (see Appendix).

$$MSI = \frac{\sum_{i=1}^{N} fRC_i}{N}$$

MSV was calculated as the square root of the variance of these distances across all data points in each sequence:

$$MSV = \sqrt{\frac{\sum_{i=1}^{N}(fRC_i - MSI)^2}{N}}$$

Both measures were expressed in units of proportion, with MSI being the unevenness in restraint between segments and MSV the unevenness of control**.**

Fig 2 about here

The initial lateral projection images of all sequences were assessed for disc degeneration using the Kellgren and Lawrence rating scale by a chiropractor (AB) who was trained to interpret radiographs, giving a score of 0-4 for each level [33]. These were added together to give a composite disc degeneration score (CDD) for each lumbar spine. The sum of disc degeneration scores was used in an effort to accommodate both a single point of injury and regional lumbar dysfunction as pain generators. The same images were assessed by the same operator one month later to assess reliability.

Statistical analysis

All data were inspected for normality using the Shapiro-Wilk test. Averages of non-normal data were expressed as medians and the significance of their differences and correlations calculated using the 2- way Mann-Whitney and Spearman Rank Correlation tests. Averages of normal data were expressed as means and their differences and correlations were examined using 2-way unpaired t-tests and Pearson's R for correlations. The significance of differences in proportions was calculated using the Fisher Exact Test.

For the 20 participants (Cohort 1) who received recumbent flexion, extension, left and right imaging, the MSI and MSV values were summed, as in Mellor et al [29], whereas for Cohort 2, the values for weight bearing flexion were calculated for only one plane of motion. For comparison, MSI and MSV for recumbent flexion in Cohort 1 were also calculated and compared.

Results

Image sequences of eighty-three referred patients were drawn from a group of patients with CNSLBP who had been referred for QF investigations (Fig 3). For 14 of these, referrers had requested recumbent flexion, extension, left and right examinations and in 12, weight bearing flexion. Four patients were excluded from Cohort 1 and 2 from Cohort 2 due to spondylolisthesis or previous spinal surgery.

Fig 3 about here

The characteristics of patients and controls (n=40) are shown in Table 1. Age ranges were wide, accommodating a degree of difference in disc degeneration grades, which nevertheless averaged in the lower third of the possible range. At the second assessment for disc degeneration grade one month later, one disc's grade was revised from 1 to 0 and one from 0 to 1. There were no significant differences between patients and controls in terms of age, sex or combined disc degeneration grade.

Table 1 about here

Both combined and flexion only MSIs were significantly higher in the patients than the controls in Cohort 1 (Combined MSI in patients: 1.40, controls 0.92, p=0.04: Flexion MSI in patients 0.29, controls 0.22, p=0.02), but there were no significant differences in MSV, as found by Mellor et al [29] (Table 2).

Table 2 about here

Correlations between age, combined disc degeneration (CDD), MSI and MSV are shown for each cohort examination in Table 3 and scatterplots on which these correlations are based are presented in Fig 3 (a-h).

Table 3, Fig 3(a-h) about here

There were substantial correlations between age, disc degeneration and MSI in combined passive recumbent motion in the Cohort 1 patients, but not in controls (Fig 4 a-d). In weight bearing active flexion motion in the Cohort 2 patients, there were also substantial correlations between age, disc degeneration and uneven motion, exhibited as variability of motion sharing (MSV) (Fig 4 e-h). This was also exclusive to patients with CNSLBP. (MSI appears to have been unrelated.) There was moderate correlation between MSI and MSV in both cohorts, which only reached significance in controls, although it was present in all groups except combined recumbent motion.

Discussion

In recent years, the usefulness of dynamic analysis of spinal disorders has become more apparent. For example, Shiba et al [34] found that by examining dynamic, as opposed to static global sagittal alignment at the beginning and end of a gait sequence in patients with degenerative lumbar kyphoscoliosis, loss of global sagittal alignment at the end of the gait sequence was more readily detected.

The present studies bring to three the number of cohorts in which uneven continuous motion sharing has been found to be greater in CNSLBP patients than in controls. In the present study, uneven passive restraint across the lumbar spine (MSI) was greater in patients with CNSLBP than in pain free controls. There would seem to be at least two

possible explanations for this. The first is that unequal restraint (MSI) could add to increased muscle metabolic demands during activities of daily living causing recurrent muscle pain [35]. The second may relate to the rapid accelerations associated with inadequate restraint at an injured level, which has been suggested to be a nociceptive pain generator producing a single point of pain. This could also cause motion sharing inequality [36, 37].

It is notable that age and disc degeneration were substantially correlated with uneven passive motion sharing (MSI) in the back pain patients and hardly at all in the controls in Cohort 1. This suggests that CNSLBP is linked to disc degeneration when there is uneven restraint in the passive subsystem. Barz et al put forward a new conceptual model of CNSLBP that links such structural degeneration with mechanical compensation and stability [38]. Thus, more evenly shared restraint (MSI), despite the presence of degenerative change, may be seen as the result of structural compensation that allows the individual relative freedom from symptoms.

By contrast, in weight bearing active flexion in patients, the correlations found between age, disc degeneration and increased variability of motion sharing (MSV) suggests relationships with control in the active subsystem. However, the finding that motion sharing variability (MSV) was not greater in patients during active weight bearing motion may have been due to the stabilising influence of the trunk muscles during active bending. Thus, control of MSV may be an important factor in the avoidance of CNSLBP. This possibility could be explored by future research using the above techniques in combination with electromyography [39]. However, Von Forell et al found, using finite element modelling, stresses on the spinal holding elements would generally be lower when not all lumbar discs are degenerate [40].

It is uncertain whether these relationships are causative or consequent to back pain, or both. The above suggested relationships to rapid accelerations and/or increased muscle metabolic demands are possible explanations for a causative effect. For example, in the recumbent studies, it is difficult to conceive how pain alone could have selectively affected passive segmental restraint when muscle electrical activity was minimal [7].

Recent research by Lao et al [41] supported the hypothesis of Kirkaldy-Willis[42] that disc degeneration has different effects on intervertebral motion at different stages. Contemporaneous discographic and profilometry studies have supported the hypothesis that painful discs are also usually disrupted [43]. The strong associations found here between disc degeneration and uneven intervertebral motion in patients, and but not in controls, seem consistent with this. It is also consistent with other recent research, which found that disc degeneration was associated with low back pain, especially when associated with end-plate signal change [11, 44, 45]. However, unlike the present work, these studies used MRI disc degeneration grading, while radiographic grading based on structural aspects rather than biochemical changes may be better correlated with pain when considered alongside intervertebral motion patterns.

The other two main biological generators of CNSLBP are chemical and neuroplastic. Circulating inflammatory markers have been found in such patients [46] and it may be hypothesised that the greater the unevenness of motion sharing, the greater the likely prevalence of rapid displacements during physical tasks causing the release of cytokines from failing holding elements.

Central sensitisation seems to be a consequence of many factors that are linked to the experience of having chronic pain, however the role of nociception, once it has become

embedded, is less clear [47]. Exercise in the treatment of chronic pain has neurological as well as mechanical effects, making its monitoring by brain mapping a variable of interest for comparison with changes in the above mechanical factors with treatment [48]. Future work could therefore usefully compare MSI and MSV in passive recumbent and active weight bearing motion in patients and controls to monitor their responses to physical therapies.

Limitations

The main limitation of these studies is their small numbers. However, the strength and significance of the correlations and replication of previous work suggests that the relationships found should be robust. Many clinicians would prefer weight bearing motion studies to be conducted using free bending rather than with the pelvis constrained in order to capture 'natural' motion patterns. However, this makes comparison between participant groups difficult, as it introduces sources of extraneous uncontrolled variation, including that from large hip joint motions [26]. It would also have been useful to have obtained both recumbent and weight bearing sequences for both patients and controls in Cohort 2, but this was not possible owing to missing data and will need to await future studies.

Lastly, imaging studies that use image intensifiers are associated with a radiation dose, however, image acquisition times for such studies are considerably less protracted than in clinical procedures, resulting in smaller doses. Mellor (2014), reported a mean effective radiation dose of 0.561mSv for the four sequences which were used with Cohort 1 in the present studies. This is approximately half the dose of a conventional plain radiographic examination of the lumbar spine [49].

Conclusion

This research confirms and extends the results of previous studies [8, 24, 29] that found abnormalities of shared intervertebral motion to be consistent with having CNSLBP and suggests possible mechanisms for this. It also suggests routes to improved understanding of the role of disc degeneration in common back pain in which degeneration may be considered a pain source when it is associated with uneven motion sharing and end plate signal change. The results open a route to the study of motion sharing as a moderator of outcomes and of prognosis in clinical studies and its role among other known biological factors, such as muscle metabolic demands and chemical markers. However, further confirmatory work is still needed.

Conflict of interest. The authors declare that they have no conflicts of interest.

Word count 2970

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Figure

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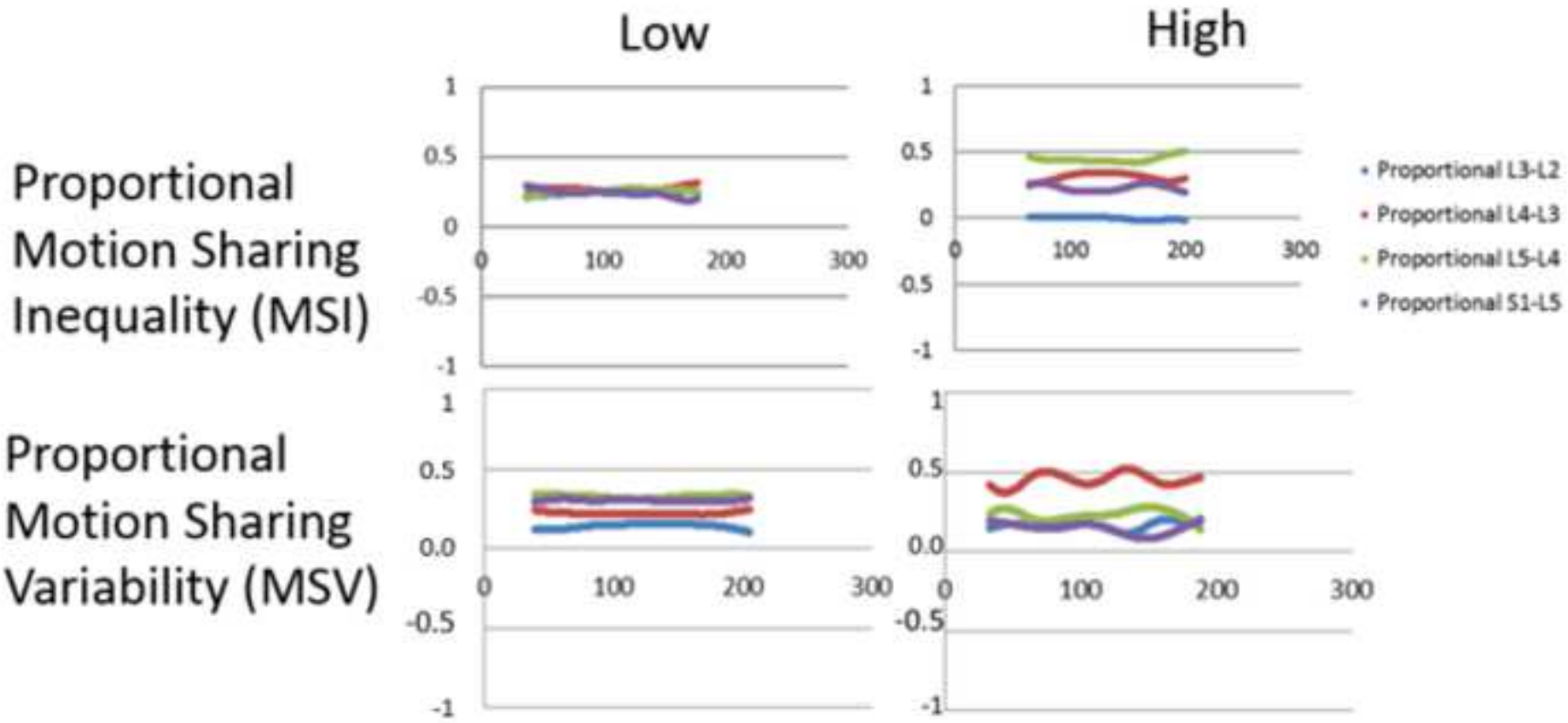

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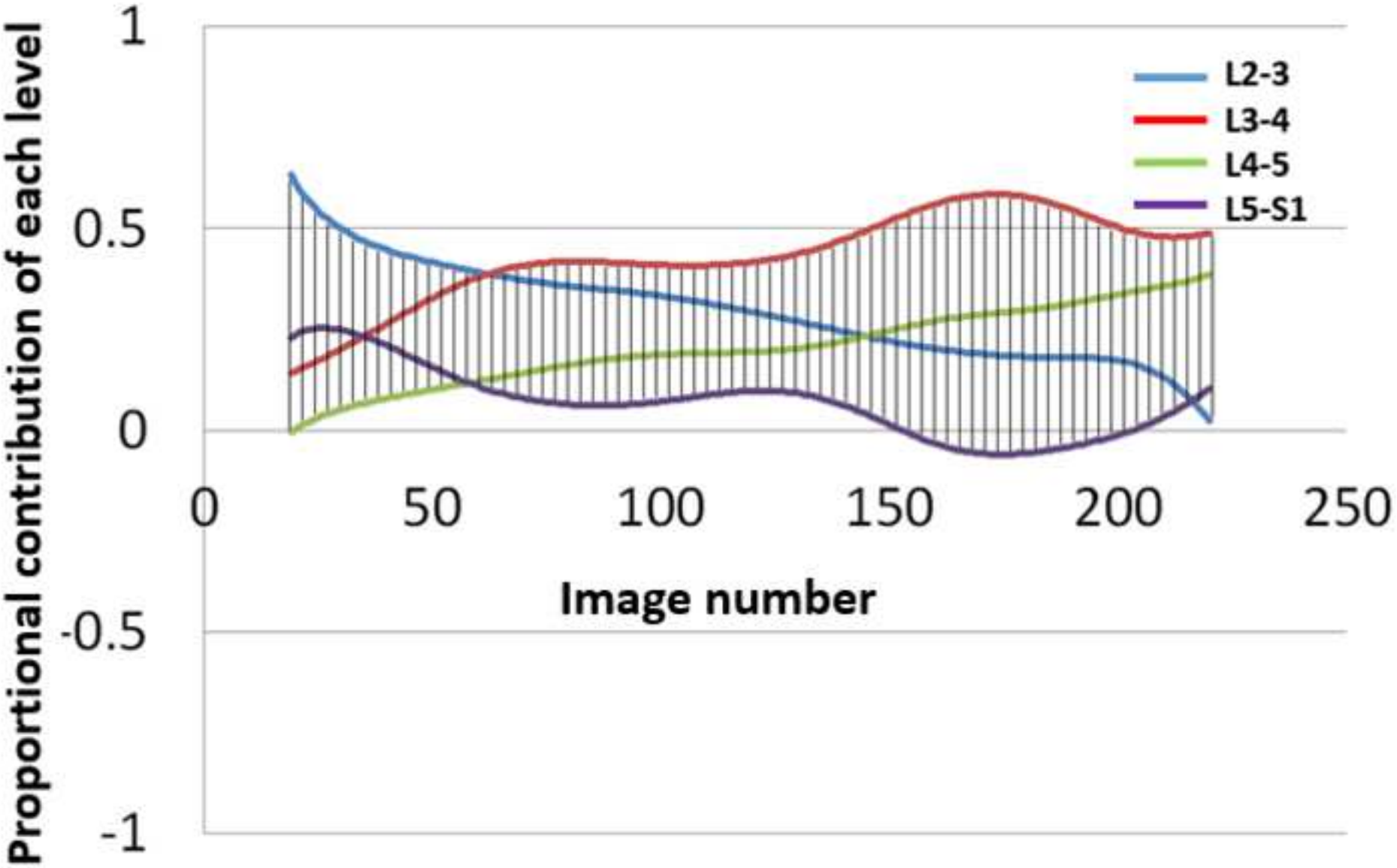

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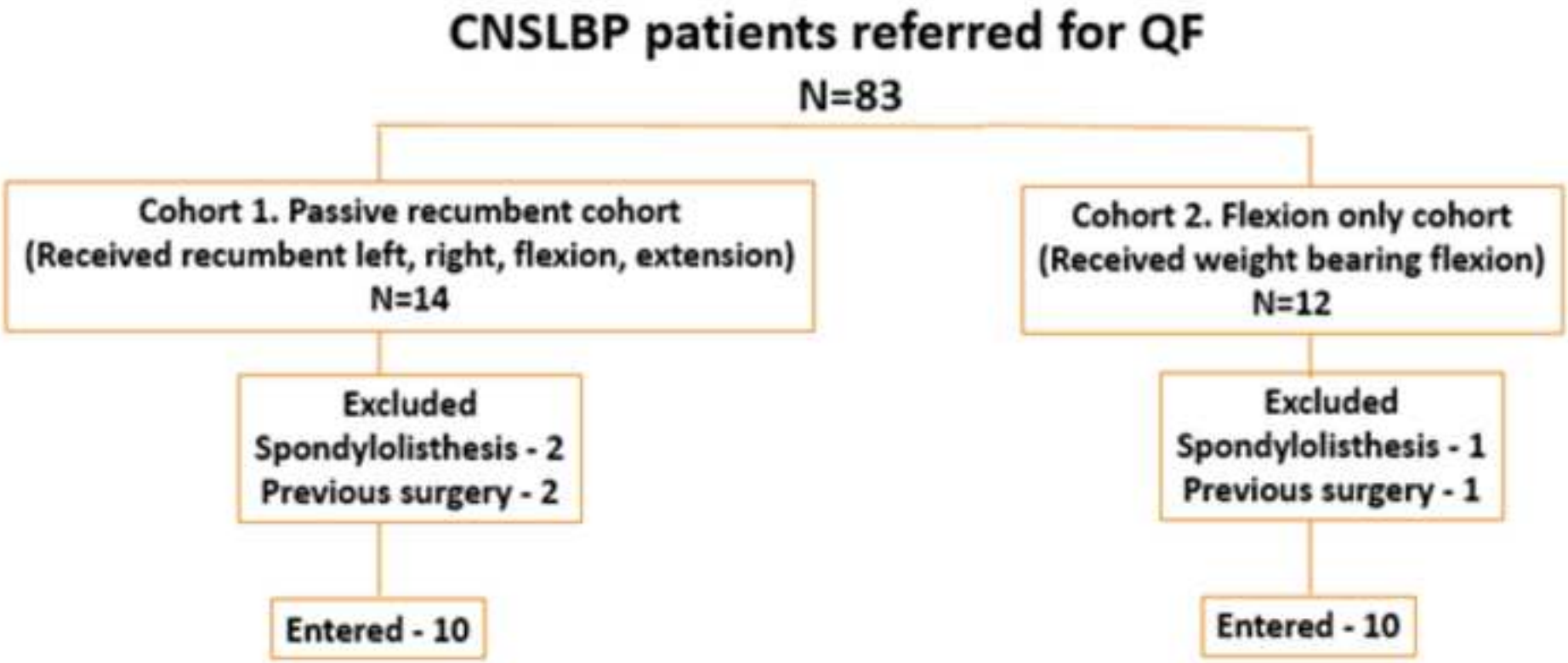

Figure

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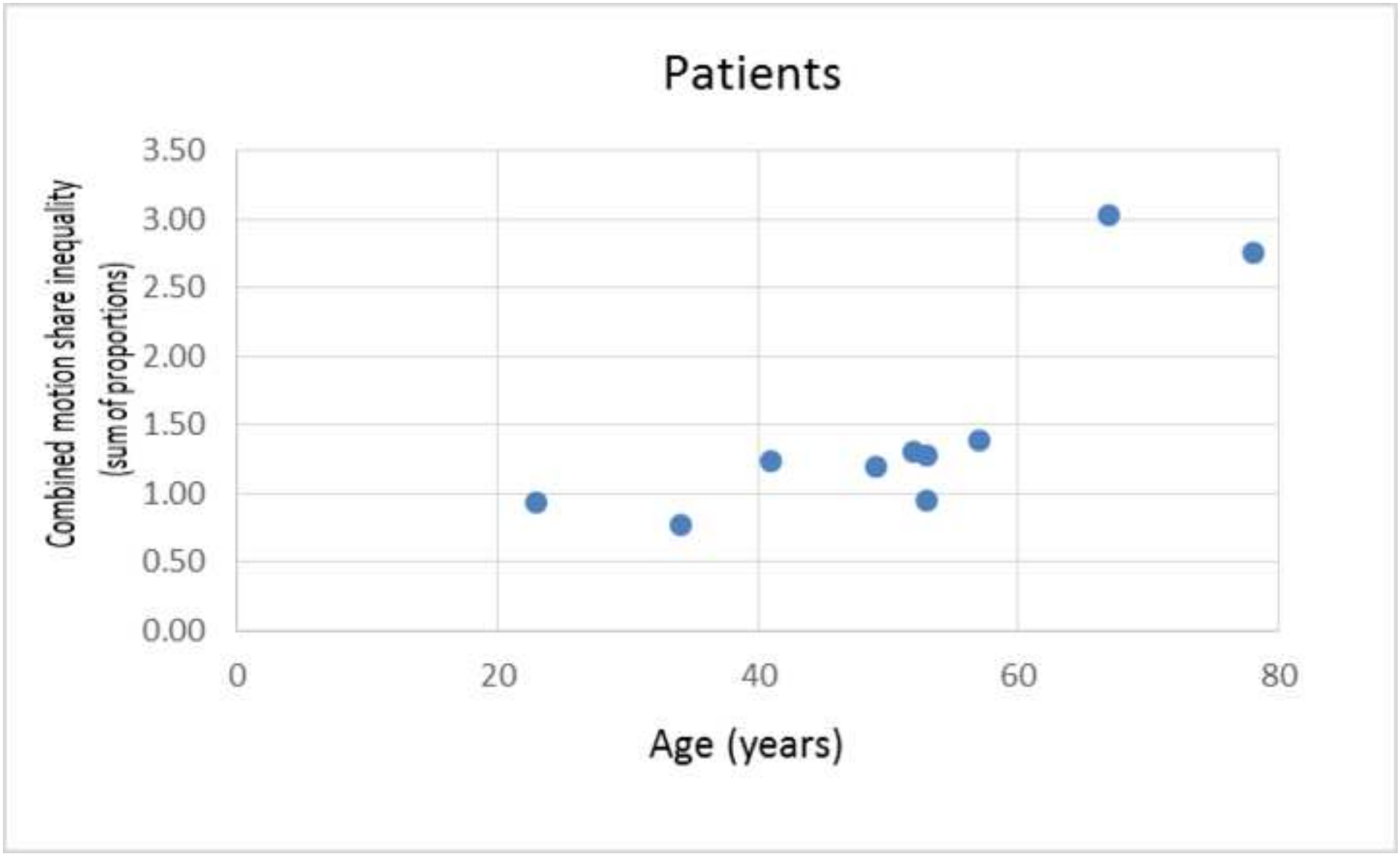

Figure

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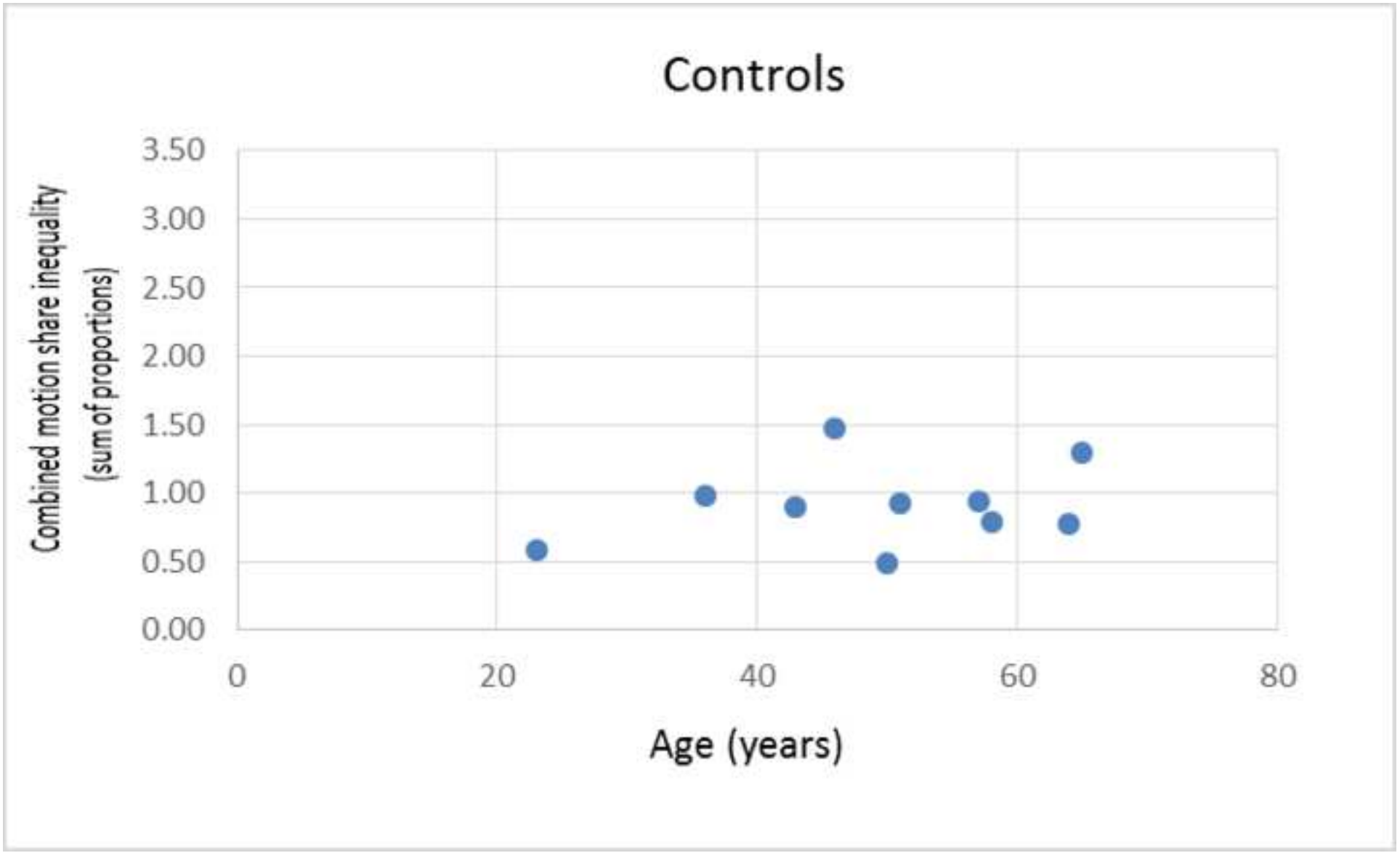

Figure

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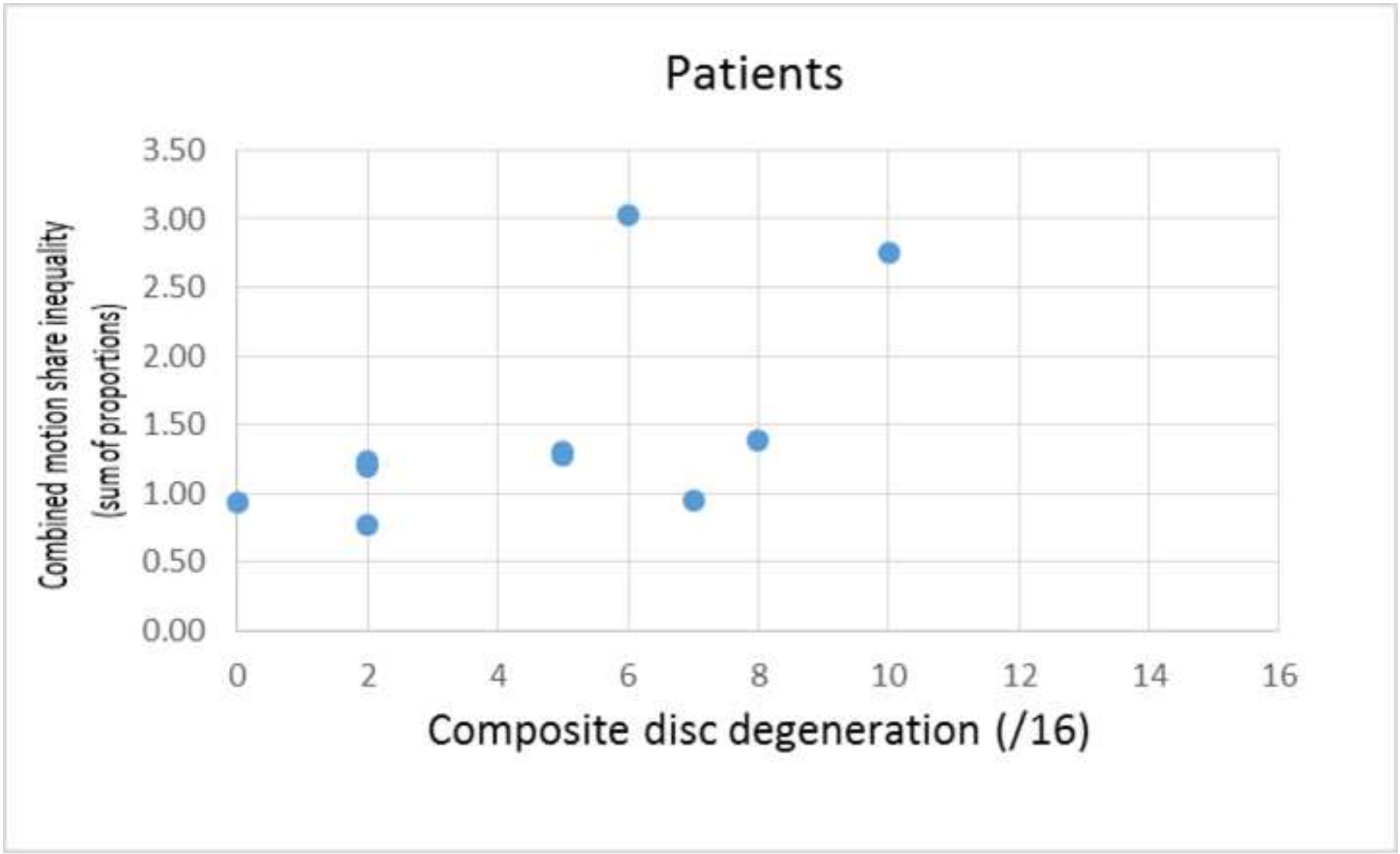

Figure

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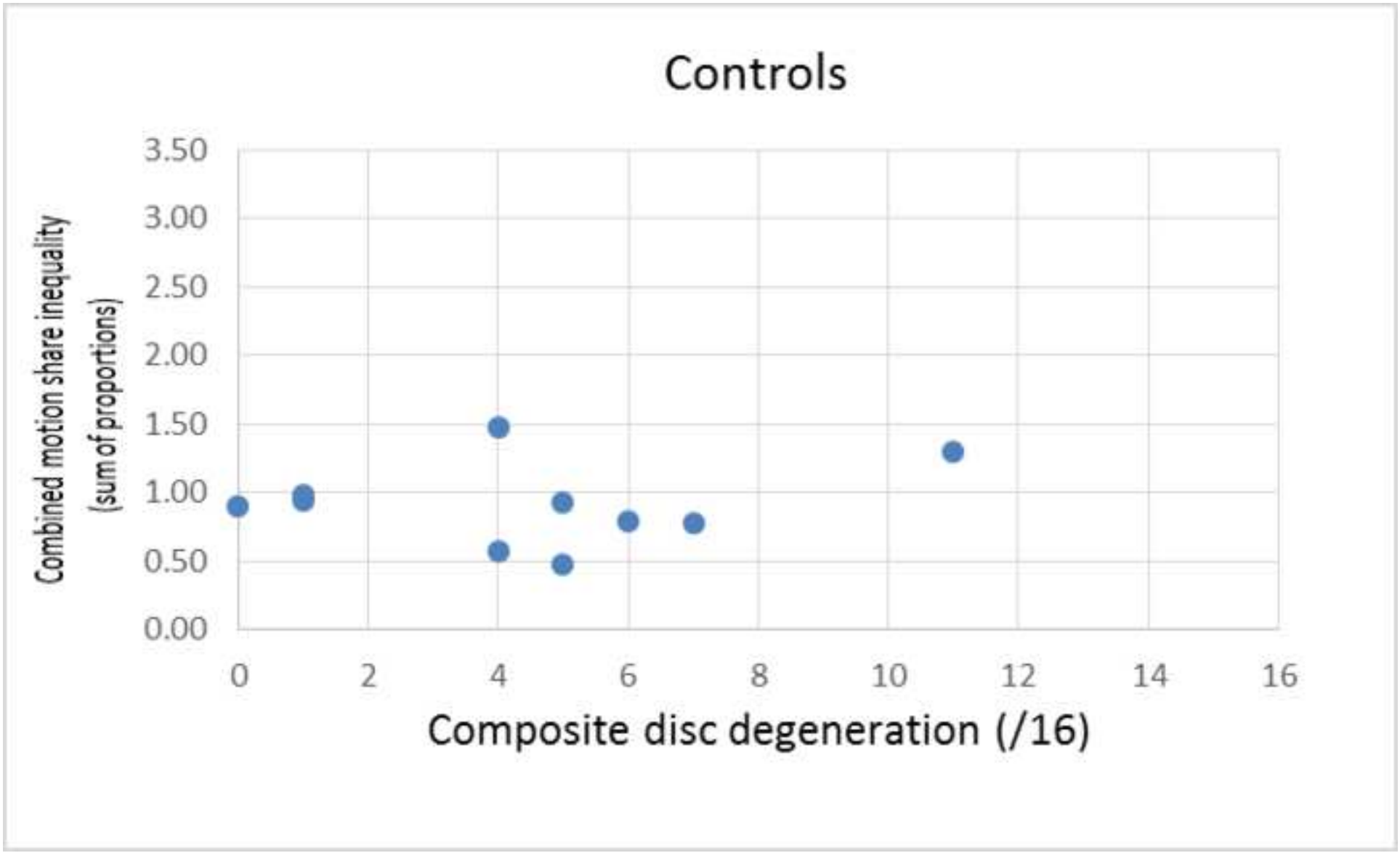

Figure

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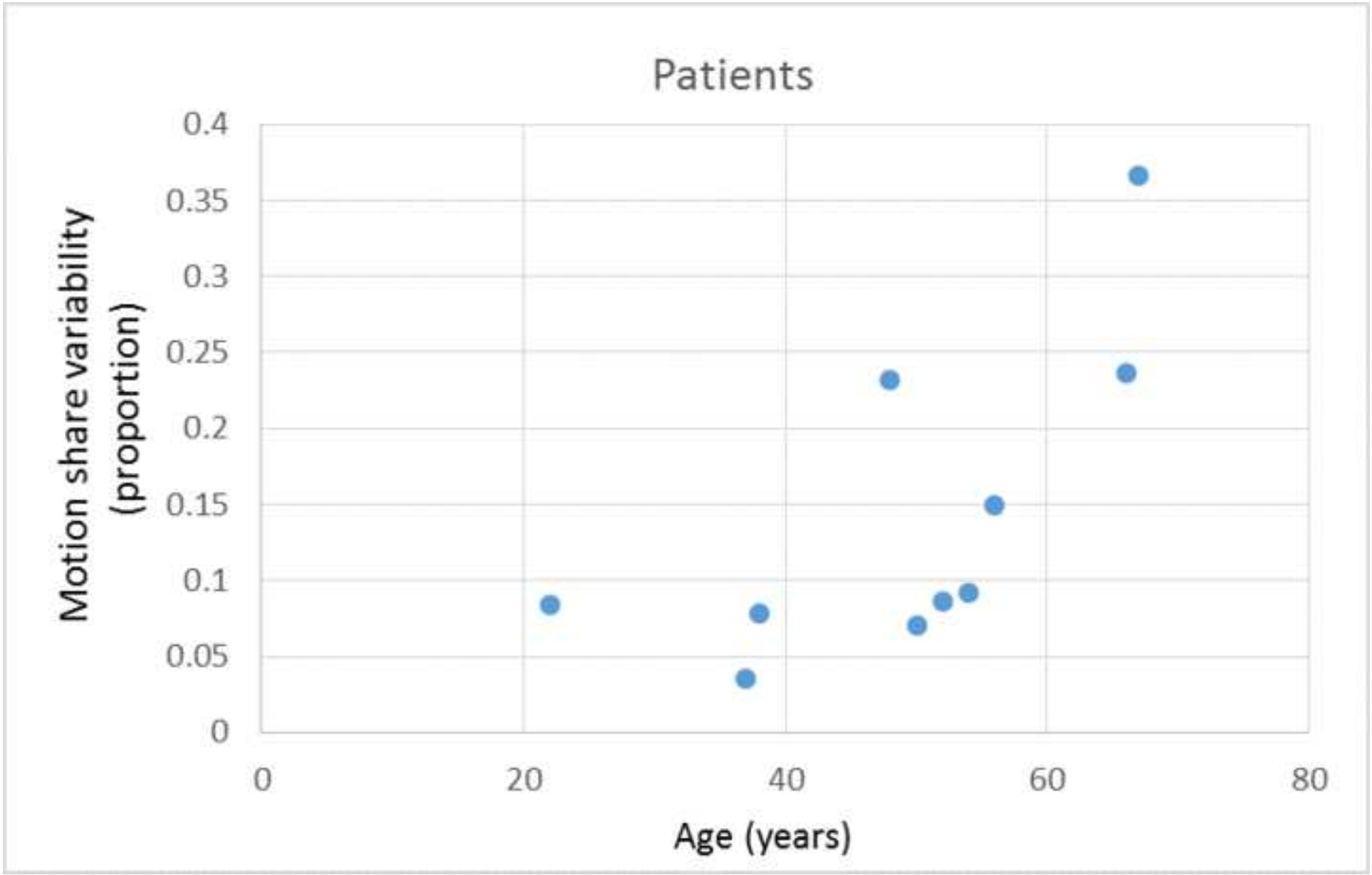

Figure

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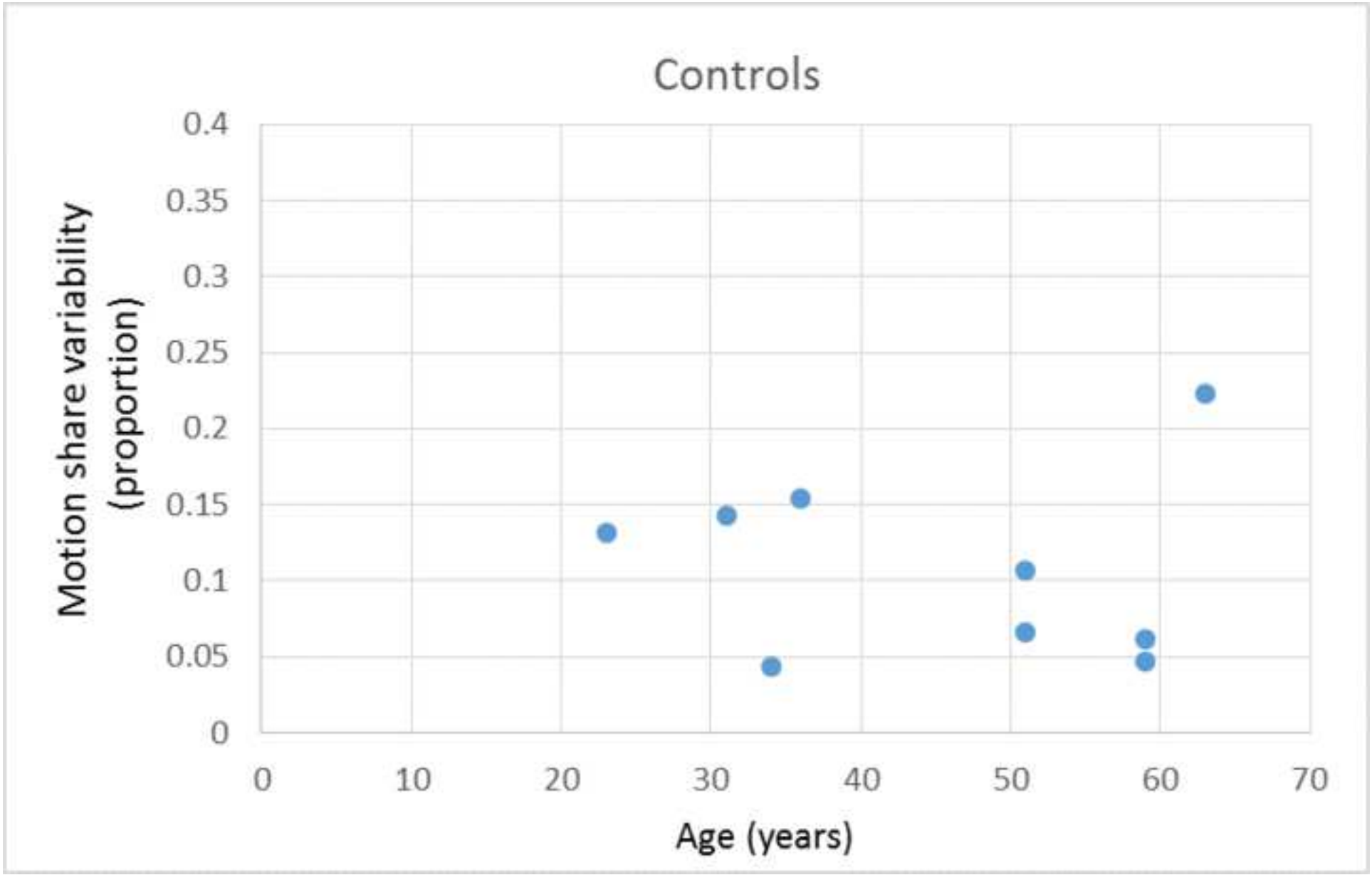

Figure

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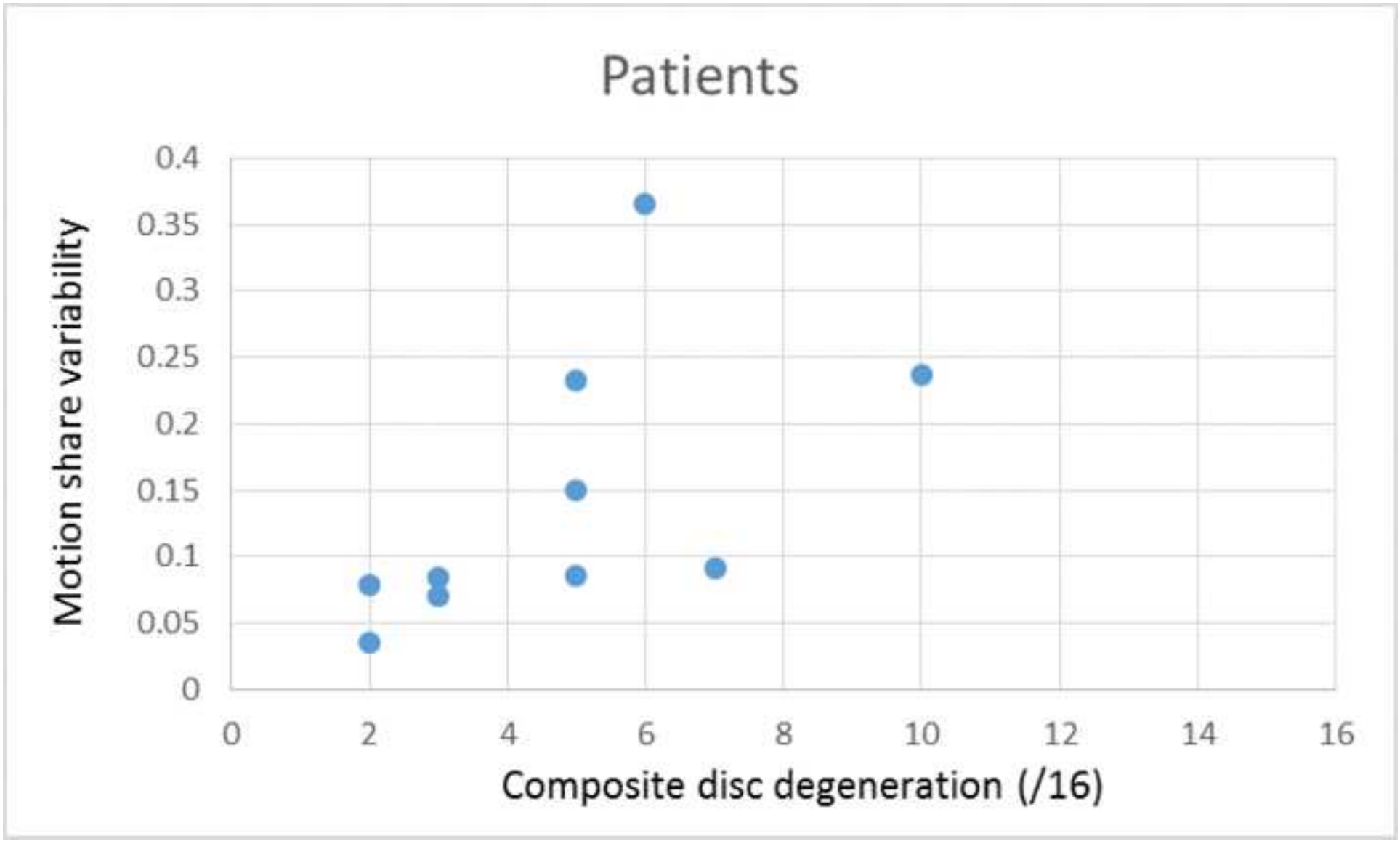

Figure

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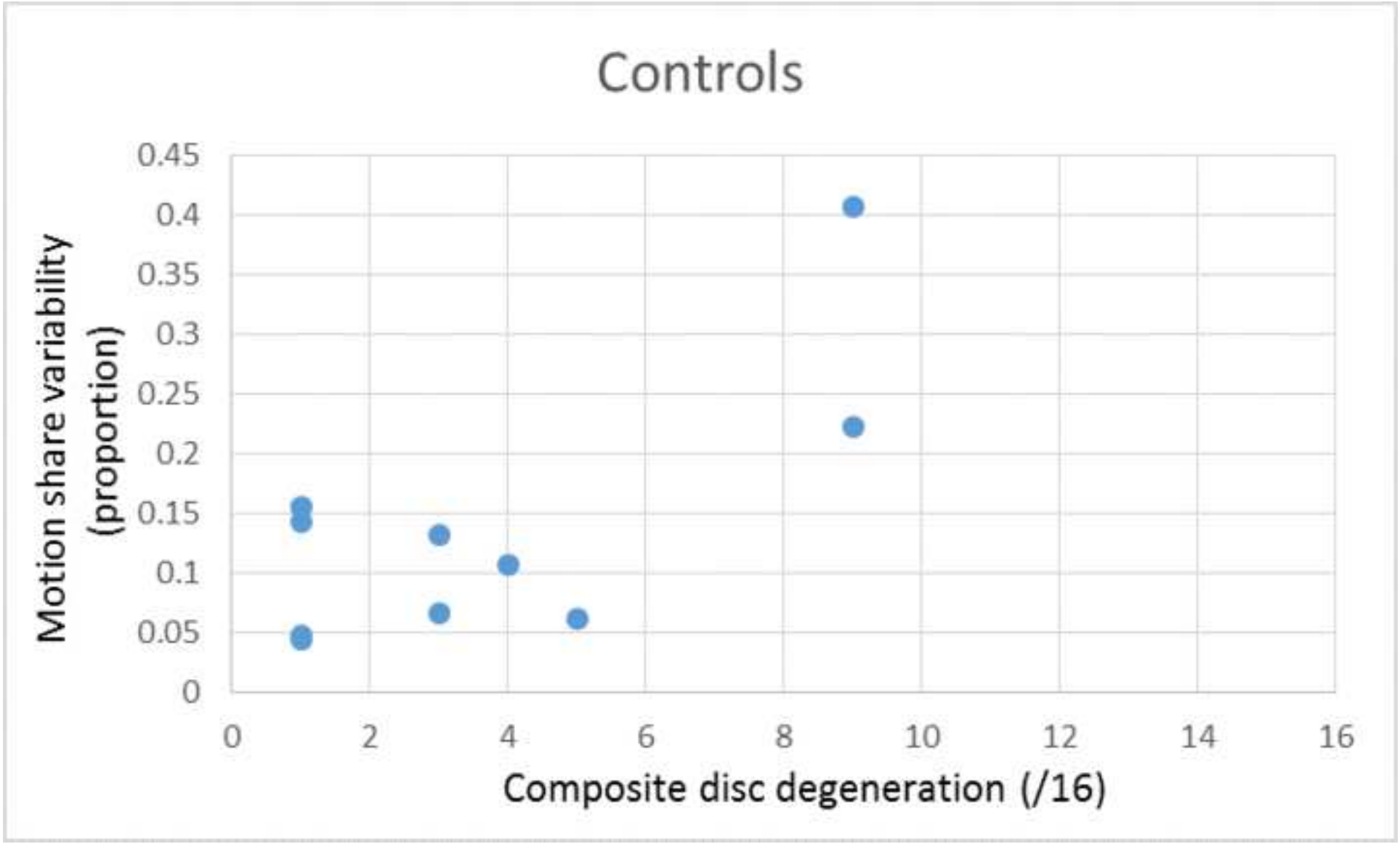

Table

Tables

Table 1

Characteristics of patients and controls (n=40)

		Patients	Controls	p
Cohort 1	N	10	10	
	Age (mean, SD)	51 (14.9)	49 (12.3)	0.83
	Sex	M7,F3	M8,F2	0.65
	DD/16 (mean, SD)	4.7 (3.0)	4.4 (3.1)	0.22
Cohort 2	N	10	10	
	Age (mean, SD)	49 (13.0)	47 (14.2)	0.78
	Sex	M6,F4	M6,F4	0.99
	DD/16 (median, range)	5.0 (2-10)	3.0 (1-10)	0.22

Table 2

Comparison of patients and controls by MSI and MSV

		Patients (n=10)	Controls (n=10)	p
Cohort 1 (lying flexion + extension + left + right)	MSI (mean)	1.48	0.92	**0.04**
	MSV (median)	0.19	0.15	0.25
Cohort 1 (lying flexion)	MSI (median)	0.29	0.22	**0.02**
	MSV (mean)	0.08	0.08	0.63
Cohort 2 (standing flexion)	MSI (mean)	0.39	0.33	0.25
	MSV (median)	0.08	0.14	0.97

Table 3

Correlations between age, combined disc degeneration, motion share inequality and motion share variability in patients and controls (n=40)

		Patients						Controls					
		CDD		MSI		MSV		CDD		MSI		MSV	
		R	p	R	p	R	p	R	p	R	p	R	p
Cohort 1 Recumbent	Age	**0.94**	**0.0003**	**0.85**	**0.004**	0.11	0.68	**0.76**	**0.015**	0.12	0.76	0.2	0.58
Flx + Ext + Left + Right	CDD			**0.70**	**0.03**	-0.21	0.54			-0.15	0.67	0.07	0.85
L2-5 (Spearman)	MSI					0.01	0.97					**0.77**	**0.01**
Cohort 1 Recumbent	Age	**0.94**	**0.0003**	0.27	0.58	-0.19	0.58	**0.76**	**0.015**	0.33	0.34	0.27	0.48
Flexion only	CDD			0.58	0.28	0.01	0.99			0.13	0.73	0.09	0.81
L2-5 (Spearman)	MSI					0.44	0.2					0.27	0.45
Cohort 2 Weight bearing	Age	**0.83**	**0.005**	0.54	0.11	**0.77**	**0.01**	**0.68**	**0.039**	0.26	0.24	0.25	0.49
Flexion only	CDD			0.43	0.23	**0.85**	**0.004**			0.39	0.26	0.47	0.18
L2-S1 (Spearman)	MSI					0.62	0.06					**0.67**	**0.01**