Articular Degeneration and Remodeling in Human Temporomandibular Joints With Normal and Abnormal Disc Position

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Temporomandibular joints with normal and abnormal disc positions, obtained from 15 females and 38 males (most between 15 and 50 years of age), were examined under a light microscope. Histologic changes attributed to degeneration and remodeling of the articular tissues were graded according to their prominence and extension along the articular surfaces. These changes seemed little affected by internal derangement in the condyle and the temporal component. In contrast, degeneration and regressive remodeling of the disc and, although to a much lesser degree, its attachments were more severe and increased more steeply with age in internally deranged as compared to normal joints. The present findings suggest that the disc indeed suffers from an abnormal position. However, this conclusion seems tenable only for adults, as rather prominent progressive remodeling changes have been observed in joints from adolescents, but no significant degenerative variations were noted.

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In a previous study[1] assessing synovial cavity dimensions of temporomandibular joints (TMJs) with normal and anterior disc position, it was hypothesized that, in addition to constitutional factors, articular remodeling related to growth or osteoarthritis (OA) could possibly account for discrepancies that were associated with internal derangement. The present investigation was undertaken to further evaluate this hypothesis.

Osteoarthritis is a noninflammatory disease of synovial joints that is characterized by destruction of cartilage and deterioration of the load-bearing surfaces and associated with extensive remodeling.[2,3] Based on examination of human limb joints obtained at autopsy or from surgical interventions, it is commonly, although not universally, believed that OA is preceded by less advanced aging and degenerative alterations.[2,3] Consequently, the severity of such alterations has frequently been graded when examining articular cartilage specimens macroscopically[4,5] and histologically.[2,6] Commonly used criteria for grading included various degrees of cartilage disintegration; the mildest form, referred to as fibrillation, is a focal roughening of the articular surface[2,3] and was considered a normal aging change when it was superficial and associated with an intact articular surface.[2] However, fibrillation with rising age may spread tangentially along the articular surface. In addition, it may also progress to more severe forms of tissue disintegration that affect deeper regions of the articular cartilage and, ultimately, lead to complete cartilage destruction and exposure of subchondral

bone.[4,5] Hence, increasing grades of severity have been assigned to morphologic features such as fissures, splits, or clefts extending to increasingly deeper parts, ie, the transitional, radial, or even calcified zone, of the articular cartilage.[6]

Johnson[7] divides articular remodeling into progressive, regressive, and circumferential or peripheral types. In relation to fibrillation and OA, progressive changes in cartilage, such as formation of chondrocyte clusters and production of proteoglycans resulting in enhanced histologic staining of the matrix, have been described.[6,8] Furthermore, bone may be added to the subchondral plate through apposition in existing osteons as well as through vascular invasion of cartilage and endochondral ossification.[6-8] Regressive changes would include chondrolytic removal of cartilage proteoglycans and loss of chondrocytes due to cell degeneration and necrosis, resorption of subchondral bone, and metaplastic conversion of soft connective tissue into cartilage.[6-8] Finally, circumferential remodeling can be regarded as a peripheral progressive remodeling consisting of cartilagenization and subsequent ossification of capsule or ligament insertions, which eventually lead to osteophytic lipping of the joint margins.[3,7]

In synovial joints whose articular surfaces are formed by fibrocartilage, eg, the sternoclavicular[9,10] and temporomandibular[11,12] joints, most of the histologic features attributed to degeneration and remodeling in hyaline articular cartilage have also been identified. Regarding the association between internal derangement and OA of the TMJ, there is almost universal consensus that the two conditions often occur concomitantly.[13-20] However, opinions differ as to the cause-and-effect relationship between the two disorders. The prevailing view seems to be that joint degeneration proceeds faster and to a more severe degree in cases of persisting internal derangement.[14-17,19] De Bont et al[20] state that internal derangement was one of the accompanying signs of OA, but Stegenga et al[21] consider OA the basic disorder. Several problems arise when attempting to evaluate these opinions. Autopsy studies[17,20,22] usually involve joints from aged individuals who, based on probability alone, might have suffered from degenerative diseases more often than young people. Also, some autopsy studies[23] fail to include a histologic examination to identify early signs of degeneration. Results from inspection of surgical specimens[16,24-29] may be biased by the fact that joint surgery commonly is performed only in severe cases of painful internal derangement refractory to conservative treatment. Finally, radiographic examination[14,16,19] often may not disclose mild osteoarthritic changes[11] and, therefore, will hardly allow a clear distinction of alterations due to degeneration and normal articular remodeling.

In the present investigation, TMJs (obtained postmortem from predominately adolescent, young adult, and middle-aged individuals, albeit with a few elderly subjects) with normal and abnormal disc position have been examined. On the basis of histologic criteria applied in studies of hyaline articular cartilage, an attempt was made to distinguish and grade alterations attributed to degeneration and remodeling.

Materials and Methods

Subjects and Specimens

This study examined the same left TMJs as used in a previous study,[1] obtained at autopsy from 15 female and 38 male subjects (Table 1; age range 15 to 92 years), whose medical, but not dental medical, histories were known. Seven joints from females and 13 from males exhibited internal derangement, and all except one from a male subject exhibited anterior disc position. Each specimen was first divided into five to eight sagittal slices about 2 mm thick. These slices were then processed for light-microscopic examination using either celloidin, 2-hydroxypropyl methacrylate (HPMA), or Technovit 7200 VLC embedding procedures.[1] For the present study, only 5-µm-thick sections made from decalcified tissue blocks embedded in HPMA and stained with toluidine blue O could be used, as only these preparations allowed identification of the morphologic details listed below. As a result, sagittal sections through

Table 1 Joint Characteristics of Male and Female Subjects

	Female (n = 15)		Male (n = 38)	
Age (y)	Normal disc position	Internal derangement	Normal disc position	Internal derangement
15–19	0	0	2	2
20–29	1	1	7	0
30–39	3	2	3	2
40–49	3	2	8	3
50–59	1	1	3	2
60–69	0	0	1	1
70–79	0	0	0	2
80–89	0	0	1	1
90–99	0	1	0	0

either the lateral and central or central and medial region of 16 joints, as well as preparations of the lateral, central, and medial region of 37 joints, were available for analysis.

Histologic Analysis

Without knowledge of the age and gender of subjects from which the joints came and independent of joint position, one observer examined sections using microscopic magnifications of × 100 and × 250. Joint regions evaluated separately were the anterior and posterior component slopes of the mandibular condyle; the articular eminence and mandibular fossa of the temporal component; the upper and lower part of the articular disc; and the posteroinferior, posterosuperior, anteroinferior, and anterosuperior disc attachments. In all these regions, histologic signs suggesting disintegration of articular tissues; deviation in form; and ongoing progressive, regressive, and circumferential remodeling were recorded (Table 2). For semiquantitative analysis, they were graded with respect to both prominence (Table 2) and extension along the surfaces against the synovial cavity.

Classification of prominence in tissue disintegration as well as in progressive and regressive remodeling largely relied on that used in earlier studies of hyaline articular cartilage.[2-6] This classification emanates from the assumption that the severity of disintegration rises with the increasing depth of fissures, splits, or clefts in the cartilage, whereas progressive and regressive remodeling would result in, respectively, an increase or decrease in the number of cells or the amount of some matrix constituents. It should be noted that the term remodeling as used here refers to alterations in tissue composition at the microscopic and possibly even molecular level. In contrast, reshaping processes that were referred to as remodeling in most previous studies of the TMJ[30-33] have been assessed with the parameter "deviation in form" (Table 2).

The extension of a histologic sign was estimated by dividing the total tissue/synovial cavity interface of a particular joint region, as seen in a section, into 10 arbitrary length units. This estimate was included because studies of limb joints[4,5] indicate that the severity of fibrillation increases not only by progressing to deeper zones of the cartilage, but also by spreading tangentially and affecting increasingly large portions of the articular surface. Therefore, the final score assigned in the present investigation to a particular tissue, joint region, and section was calculated as the product of prominence and extension. The scores assigned to

Table 2 Grading of Histologic Signs

Histologic signs	Prominence	Score
Articular tissue disintegration		
Fibrocartilage		
Normal appearance	absent	0
Superficial fibrillation associated with intact articular surface	mild	1
Fibrillation involving the articular surface	moderate	10
Deep vertical clefts and/or splitting in the depth of the cartilage or along the tidemark	severe	100
Complete destruction of fibrocartilage	overt OA	1000
Disc attachments		
Normal appearance	absent	0
Loosening of collagen fiber arrangement	mild	1
Superficial splits	moderate	10
Deep clefts	severe	100
Deviation in form		
Normal, smooth articular surface	absent	0
Microscopically irregular articular surface	moderate	10
Grossly deformed articular surface	severe	100
Progressive remodeling		
Fibrocartilage		
Diffusely increased cellularity or clusters of chondrocytes and/or increased metachromatic staining	absent moderate marked	0 10 100
Subchondral bone		
Vascular invasion of cartilage and endochondral ossification and/or osteoid seams in subchondral osteons	absent moderate marked	0 10 100
Disc attachments		
Synovial hyperplasia and/or increased collagen fiber density and/or metachromatic staining and/or adhesions	absent moderate marked	0 10 100
Regressive remodeling		
Fibrocartilage		
Decreased cellularity and/or decreased metachromatic staining	absent moderate marked	0 10 100
Subchondral bone		
Osteoclastic resorption and replacement by soft tissue	absent moderate marked	0 10 100
Disc attachments		
Synovial thinning or atrophy and/or decreased collagen fiber density	absent moderate marked	0 10 100
Circumferential remodeling		
Metaplastic conversion to fibrocartilage and/or ossification of insertions of disc attachments	absent moderate	0 10
Osteophytic lipping	marked	100

grades in prominence (Table 2), rather than denoting actual relationships between the severities of histologic signs, were chosen so that inferences could be made as to the particular morphologic appearance based on the final score. Thus, the observation of "moderate" disintegration of fibrocartilage along one tenth of the articular surface combined with "mild" alterations underneath three tenths of the articular surface would have resulted in a score of 1.3 ([10 × 0.1]+[1 × 0.3]) (Table 2). Conversely, these numbers provide the ability to trace back exactly to the corresponding description as long as an alteration does not extend over an entire joint region, which was never observed in the present material. (However, a score of 13 should not be taken to indicate that the respective morphologic change was 10 times more severe than one resulting in a score of 1.3.)

Statistical Analysis

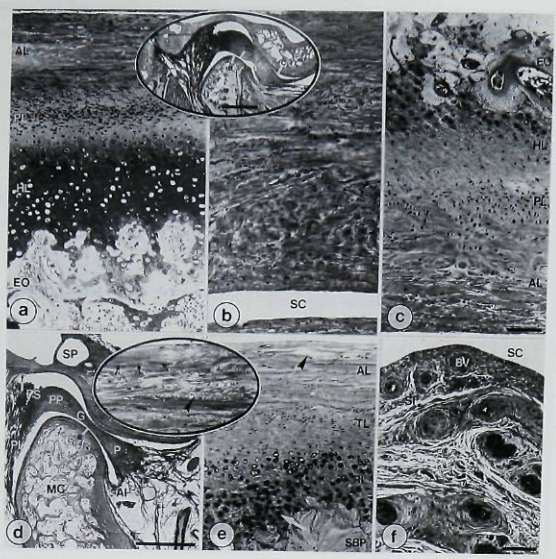
The maximal values obtained for each parameter were taken to characterize a particular region of a particular joint. This selection seemed justified because *(1)* for reasons of sample size, no distinction was made between various degrees of internal derangement, and *(2)* articular degeneration and associated remodeling seem to be focal processes[2] and, hence, may be underestimated when examining histologic sections that inevitably cover only a very limited portion of a specimen. Means and ranges were determined for specimens from female and male subjects with normal disc position and internal derangement from the data per joint. Differences between groups were statistically evaluated using Wilcoxon's rank test for two samples. As the ages of males whose joints exhibited normal as opposed to abnormal disc position differed considerably (Table 3), separate parameter means and ranges were also computed for each age decade. This, however, reduced sample sizes to an extent that statistical comparisons did not appear meaningful, and data were compared by visual inspection of graphical plots only. Likewise, findings from a joint of a 92-year-old female were not considered for statistical analysis, as the age of this subject deviated extremely from that of all other females. These findings will instead be described qualitatively.

Results

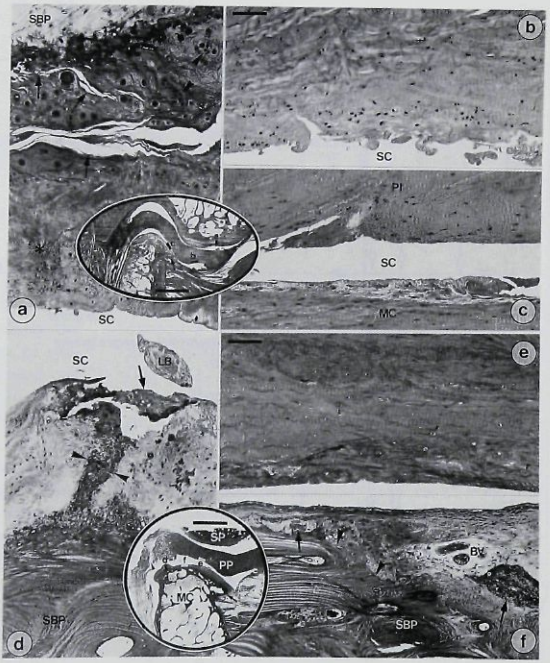
The joints of the subject under 20 years of age, irrespective of disc positions, appeared normal and showed no signs of tissue disintegration; the first signs of tissue disintegration were seen in joints from subjects in the third age decade (Figs 1a to 1c, 2a to 2c, 3a, 4a and 4b, 5a and 5b). The mandibular condyles exhibited signs of marked progressive remodeling characterized by chondrocyte hypertrophy, intense metachromatic matrix staining, and ongoing endochondral ossification (Figs 1a, 3b). These signs of progressive remodeling were much less prominent in the temporal component (Fig 1c), and they were weak to negligible in the disc (Fig 1b) and attachments (Figs 5c and 5d). All joint components largely lacked regressive alterations such as reduced cellularity or metachromatic matrix staining (Figs 3c, 4c and 4d, 5e and 5f), and circumferential remodeling did not seem to occur at all (Fig 3d).

Tissue disintegration usually was characterized by superficial fibrillation of fibrocartilage associated with an apparently intact articular surface (Fig 1d and 1e, inset) and, therefore, was classified as mild. The prominence and extension of alterations rose both on the average and in range (Figs 3a, 4a and 4b, 5a and 5b) in the fourth and fifth decades. Splits in deep cartilage layers and along the uncalcified-calcified cartilage interface, ie, tidemark (Fig 2a), as well as clefts of variable depth (Fig 2b) and fraying (Fig 2c) of articular surfaces were not infrequently observed. Although scores for tissue disintegration were considerably higher in internally deranged than normal joints in some isolated age groups, this was not generally true in the condyle (Fig 3a), the temporal component, and the disc attachments (Figs 5a and 5b). Thus, while fibrillation of fibrocartilage in the condyle (Fig 3a) and deterioration of the posterior inferior disc attachment (Fig 5a) differed highly between normal and abnormal specimens in the group aged 30 to 40 years, disintegration scores for both sites were similar in the fifth decade. In contrast to all other joint components, however, discs in an abnormal position consistently exhibited more severe and more extensive degenerative changes than did normally positioned discs (Figs 4a and 4b).

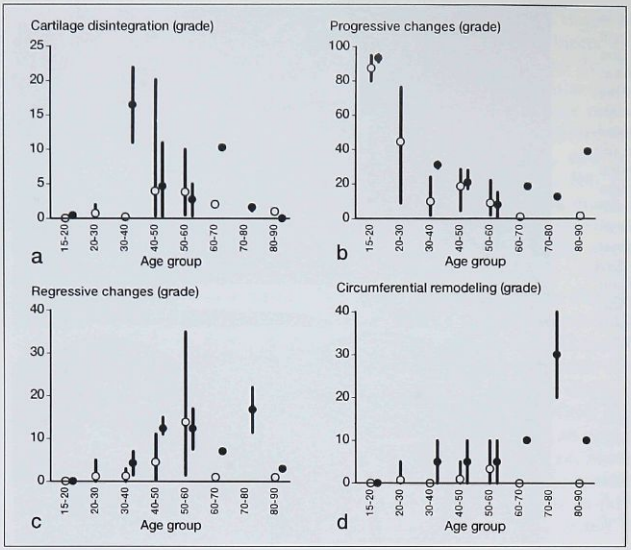
In specimens from 20 to 50 years of age, progressive remodeling seemed to gradually level off in the condyle (Fig 3b) and temporal component. Focally marked chondrocyte hypertrophy associated with intense metachromatic staining of cartilage matrix and signs of endochondral ossification (Fig 2e) were, however, regularly observed in both normal and internally deranged specimens. Furthermore, when fibrocartilage was disintegrated, chondrocytes often were arranged in clusters embedded in intensely stained pericellular matrix (Figs 2a and 2c). Histologic features suggesting progressive



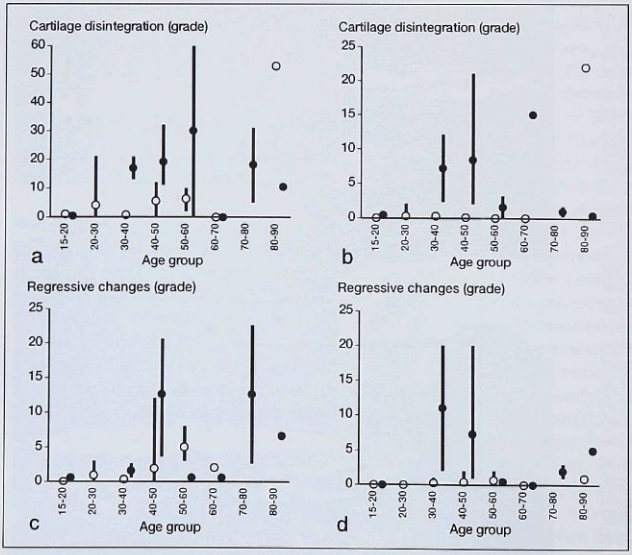
Figs 1a to 1f Normal articular tissues in condyle *(a)*, disc *(b)*, and temporal component *(c)* in a joint with anterior disc position obtained from an 18.5-year-old man. Approximate locations are indicated in the inset. Mild fibrillation subjacent to an intact condylar articular surface *(arrows in e and inset)* in a joint with normal disc position *(d)* obtained from a 40-year-old man. Fibrillation is associated with mild regressive changes *(small arrows in inset)* and progressive remodeling of the condylar cartilage *(large arrow in e)* and of the posterosuperior disc attachment *(f)*. (AI/AS = anteroinferior/superior disc attachment, AL = articular layer, BV = blood vessel, EO = zone of endochondral ossification, I = synovial intima, HL = hypertrophic layer, MC = mandibular condyle, P/G/PP = pes/par gracilis/pars posterior of the disc, PL = proliferative layer, PI/PS = posteroinferior/superior disc attachment, RL = radial layer, SBP = subchondral bone plate, SC = synovial cavity, SI = subintimal connective tissue, SP = squamous part of temporal bone, TL = transitional layer. Original magnifications = × 2.1 *(top inset*; bar = 5 mm); × 3.6 (*d*; bar = 5 mm); and × 100 *(a to c, e, f, bottom inset*; bars = 100 μm).



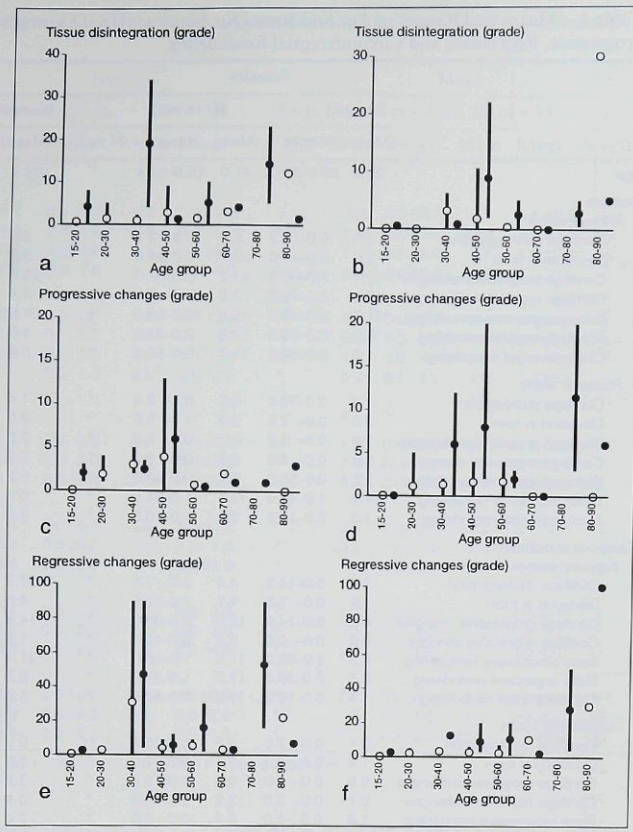
Figs 2a to 2f Tissue disintegration in the depth and along the tidemark of the temporal fibrocartilage *(a; large arrows)*, fraying of the articular surface of the disc *(b)*, fissuring of the surface of the stretched posteroinferior disc attachment *(c)*, and moderate osteophytic lipping of the condyle *(top inset; small arrow)* in a joint with anterior disc position obtained from a 60-year-old man. Disintegration of fibrocartilage is associated with reduction in cellularity *(*)* and clustering of chondrocytes *(arrows)*; approximate locations of the details *(a, b, c)* are indicated in the inset. Overt osteoarthritis in a joint with anterior disc position *(bottom inset)* of a 92-year-old woman: Bizarre spur *(d, arrows)* and exposure of mineralized tissue *(arrow)* at the condylar articular surface close to a loose body (LB); destruction of fibrocartilage *(arrows)* and subchondral bone *(arrows)* and their replacement by vascularized connective tissue. Note osteophyte at the anterior rim of the condyle *(small arrow in bottom inset)* as well as perforation of the posterior *(arrow in bottom inset)* and metaplastic conversion into fibrocartilage of the anterior portion of the posterior disc attachment *(e; arrows point at chondrocytelike cells)*. (Unless otherwise stated, labeling corresponds to Fig 1; original magnifications × 2.1 (*insets*; bars = 5 mm) and × 100 (*a to f*; bars in *b, e* = 100 μm).



Figs 3a to 3d Means (dots and circles) and ranges (vertical bars) of scores for cartilage disintegration *(a)*, as well as progressive *(b)*, regressive *(c)*, and circumferential remodeling *(d)* in the condyles of joints with normal (circles) and abnormal (dots) disc position from men in various age groups. (Numbers of individuals in age groups are shown in Table 1; definitions as to the scores are summarized in Table 2.)



Figs 4a to 4d Means (dots and circles) and ranges (vertical bars) of scores for cartilage disintegration *(a, b)* and regressive remodeling *(c, d)* in the inferior *(a, c)* and superior *(b, d)* part of normally (circles) and abnormally (dots) positioned discs from men in various age groups. (Numbers of individuals in age groups can be seen in Table 1; definitions as to the scores are summarized in Table 2).



Figs 5a to 5f Means (dots and circles) and ranges (vertical bars) of scores for tissue disintegration *(a, b)* as well as progressive *(c, d)* and regressive remodeling *(e, f)* in the posteroinferior *(a, c, e)* and posterosuperior *(b, d, f)* attachments of normally (circles) and abnormally (dots) positioned discs from men in various age groups. (Numbers of individuals in age groups can be seen in Table 1; definitions as to the scores are summarized in Table 2.)

remodeling were inconspicuous in the disc (Fig 2b), but in the disc attachments, particularly the posterosuperior one (Fig 5b), some degree of synovial hyperplasia, characterized by increased cell numbers in the synovial intima as well as by thickening of synovial folds and villi without any apparent round cell infiltrate, was seen occasionally (Fig 1f). Regressive changes rather than progressive remodeling seemed to be closely associated with tissue disintegration (Figs 3c, 4c and 4d).

Regressive changes included reduced cellularity as well as decreased staining of the matrix (Fig 1d, inset) and were often seen immediately adjacent to areas exhibiting increased cellularity (Fig 2c). Finally, signs of circumferential remodeling, such as conversion into cartilage of insertions of disc attachments, although present sometimes, did not exceed a moderate degree and were similar in joints with normal and abnormal disc positions (Fig 3d).

Samples were too small to draw valid conclusions as to the degree of articular tissue degeneration and remodeling in specimens from subjects in age groups above 60 years. The impression gained from the graphic plots (Figs 3 to 5) was that parameter scores in general did not change substantially above the ages of 50 to 60 years. On the other hand, the only joint affected by the most severe degenerative changes, which were classified as overt OA, was obtained from a 92-year-old female. The disc of this joint was positioned anteriorly and severely deformed, while the posterior disc

Table 3 Means and Ranges of Age and Scores for Fibrocartilage Disintegration, Deviation in Form, and Progressive, Regressive, and Circumferential Remodeling

	Females					Males				
	Normal (n = 8)		ID (n = 6)			Normal (n = 25)		ID (n = 13)		
	Mean	Range	Mean	Range	N vs ID	Mean	Range	Mean	Range	N vs ID
Age	38.2	28.6–52.0	41.0	26.8–57.4	*	38.4	18.7–81.2	49.6	14.9–84.2	ns
Condyle										
Anterior slope										
Cartilage disintegration	3.0	0.0–12.2	6.4	1.3–12.1	*	2.0	0.0–20.2	5.1	0.0–22.0	*
Deviation in form	3.0	0.0–12.0	22.7	1.0–99.0	*	5.2	0.0–64.0	12.8	0.0–70.0	ns
Cartilage progressive changes	21.1	1.0–60.0	41.2	5.0–82.0	*	25.8	0.0–99.0	39.5	0.0–91.0	*
Cartilage regressive changes	3.2	0.0–12.0	2.0	1.0– 3.0	*	1.4	0.0–11.0	7.5	0.0–31.0	†
Bone progressive remodeling	11.7	0.0–46.0	19.8	10.0–23.0	†	31.5	0.0–99.0	24.9	1.0–99.0	*
Bone regressive remodeling	5.8	0.0–25.0	19.8	2.0–81.0	*	5.9	0.0–70.0	12.8	0.0–43.0	†
Circumferential remodeling	1.5	0.0–10.0	11.7	0.0–50.0	*	0.9	0.0–10.0	8.8	0.0–40.0	††
Posterior slope										
Cartilage disintegration	1.3	0.0–10.0	0.7	0.0– 2.0	*	1.4	0.0–20.0	4.3	0.0–20.0	ns
Deviation in form	3.0	0.0– 7.0	2.8	1.0– 5.0	*	2.0	0.0– 6.0	8.9	0.0–43.0	*
Cartilage progressive changes	1.4	0.0– 5.0	0.7	0.0– 2.0	*	2.5	0.0–24.0	9.2	0.0–30.0	†
Cartilage regressive changes	0.8	0.0– 4.0	0.5	0.0– 3.0	*	0.2	0.0– 4.0	4.4	0.0–40.0	*
Bone progressive remodeling	2.6	0.0–10.0	5.7	0.0–10.0	*	5.2	0.0–50.0	6.1	0.0–42.0	*
Bone regressive remodelng	11.9	1.0–40.0	12.3	1.0–34.0	*	9.1	0.0–70.0	11.2	2.0–33.0	*
Circumferential remodeling	1.3	0.0–10.0	3.5	0.0–10.0	*	3.0	0.0–50.0	8.5	0.0–60.0	*
Temporal component										
Articular eminence										
Cartilage disintegration	2.8	0.0–13.2	4.4	2.0– 7.1	*	7.2	0.0–60.0	5.0	0.0–24.2	*
Deviation in form	1.6	0.0– 7.0	4.7	1.0–12.0	*	4.6	0.0–60.0	4.2	0.0–23.0	*
Cartilage progressive changes	4.6	0.0–14.0	12.0	2.0–30.0	*	14.6	0.0–90.0	19.0	4.0–42.0	†
Cartilage regressive changes	0.0	0.0– 0.0	2.3	0.0–10.0	*	1.9	0.0–20.0	1.8	0.0–14.0	*
Bone progressive remodeling	8.3	2.0–82.0	11.0	1.0–26.0	*	21.4	3.0–90.0	20.8	3.0–64.0	*
Bone regressive remodeling	8.7	0.0–30.0	11.3	1.0–25.0	*	8.0	0.0–30.0	10.8	0.0–31.0	*
Circumferential remodeling	1.4	0.0–10.0	12.2	0.0–50.0	*	5.8	0.0–99.0	5.8	0.0–30.0	*
Mandibular fossa										
Cartilage disintegration	0.4	0.0– 2.0	5.5	1.0–15.0	††	0.9	0.0– 5.0	5.5	0.0–33.4	*
Deviation in form	1.4	0.0– 6.0	3.3	0.0–12.0	*	1.5	0.0–10.0	2.2	0.0– 6.0	*
Cartilage progressive changes	0.9	0.0– 5.0	0.8	0.0– 2.0	*	3.2	0.0–50.0	2.5	0.0–10.0	*
Cartilage regressive changes	0.4	0.0– 2.0	2.2	0.0–10.0	*	0.5	0.0– 5.0	1.2	0.0– 7.0	*
Bone progressive remodeling	1.8	0.0– 4.0	2.2	0.0– 3.0	*	2.4	0.0–10.0	4.4	0.0–21.0	*
Bone regressive remodeling	5.5	1.0–22.0	7.0	2.0–15.0	*	3.7	0.0–12.0	9.4	2.0–30.0	††
Circumferential remodeling	1.3	0.0–10.0	0.0	0.0– 0.0	*	3.0	0.0–50.0	15.8	0.0–60.0	ns

Levels of significance: * = $P > .1$; ns = $.1 \geq P > .05$; † = $.05 \geq P > .01$; †† = $.01 > P \geq .001$.

attachment was perforated (Figs 2d and 2f, inset). A spicule of mineralized tissue was exposed to the synovial cavity (Fig 2d) at the zenith of the condyle, and the original fibrocartilage apparently had been destroyed and replaced by vascularized connective tissue along the entire anterior condylar slope (Fig 2f). There were signs of extensive bone resorption at the interface of this tissue and the subjacent bone, as well as along the posterior condylar slope, while osteophytic lipping was apparent at the insertion of the anteroinferior disc attachment (Figs 2d and 2f, inset). The portion of the elongated posterior disc attachment, which was interposed between the condyle and the articular eminence, lacked a synovial membrane, appeared cartilaginous, and exhibited numerous cells resembling chondrocytes embedded in intensely metachromatic matrix (Fig 2e). Such conversion into fibrocartilage of the posterior disc attachment was the only feature that was also observed in nearly all other specimens of younger individuals, except for one internally deranged specimen.

Means and ranges of most parameter scores varied little when the samples of joints with normal and abnormal disc position from women and men were compared as a whole (Tables 3 and 4). In particular, age as a possible contributing factor was similar in women and did not differ signifi-

Table 4 Means and Ranges of Scores for Tissue Disintegration, Deviation in Form, and Progressive and Regressive Changes

	Females					Males				
	Normal (n = 8)		ID (n = 6)			Normal (n = 25)		ID (n = 13)		
	Mean	Range	Mean	Range	N vs ID	Mean	Range	Mean	Range	N vs ID
Disc										
Inferior part										
Cartilage disintegration	1.7	0.0-11.0	6.2	1.2-12.0	†	6.0	0.0-53.0	15.3	0.0-60.0	ns
Deviation in form	1.6	0.0-11.0	16.0	3.0-40.0	††	2.9	0.0-11.0	17.9	0.0-60.0	ns
Cartilage progressive changes	0.5	0.0- 2.0	5.5	0.0-22.0	†	1.4	0.0- 5.0	3.2	0.0-10.0	ns
Cartilage regressive changes	0.9	0.0- 2.0	1.0	0.0- 4.0	*	3.6	0.0-50.0	5.2	0.0-22.0	*
Superior part										
Cartilage disintegration	2.0	0.0-12.0	2.3	0.0-10.0	*	1.0	0.0-22.0	4.7	0.0-21.1	§
Deviation in form	1.9	0.0- 5.0	1.5	0.0- 4.0	*	0.3	0.0- 3.0	4.5	0.0-23.0	§
Cartilage progressive changes	0.8	0.0- 3.0	3.5	1.0- 6.0	††	1.1	0.0- 4.0	1.3	0.0- 4.0	*
Cartilage regressive changes	0.1	0.0- 1.0	0.3	0.0- 1.0	*	0.3	0.0- 2.0	4.2	0.0-20.0	††
Disc attachments										
Inferior anterior										
Tissue disintegration	1.2	0.0- 4.6	8.9	2.3-15.0	††	4.2	0.0-42.0	8.6	0.0-52.0	ns
Progressive changes	1.8	0.0- 3.0	3.8	0.0-12.0	*	2.8	0.0-14.0	7.2	0.0-21.0	*
Regressive changes	1.1	0.0-70.0	10.0	3.0-30.0	*	5.1	0.0-60.0	8.5	0.0-42.0	*
Inferior posterior										
Tissue disintegration	1.4	0.0- 4.0	5.9	1.0-24.0	*	2.2	0.0-12.0	7.2	0.2-34.0	†
Progressive changes	3.8	0.0-20.0	1.0	1.0- 1.0	*	2.3	0.0-13.0	2.6	0.0-11.0	*
Regressive changes	4.6	0.0-12.0	8.0	2.0-22.0	*	7.4	0.0-90.0	20.3	1.0-90.0	ns
Superior anterior										
Tissue disintegration	0.4	0.0- 2.0	0.5	0.0- 2.2	*	1.9	0.0-30.0	5.5	0.0-40.0	†
Progressive changes	0.3	0.0- 2.0	1.7	0.0- 3.0	†	1.4	0.0- 6.0	1.6	0.0- 7.0	*
Regressive changes	0.9	0.0- 2.0	1.3	0.0- 4.0	*	2.0	0.0- 8.0	4.7	0.0-40.0	*
Superior posterior										
Tissue disintegration	2.0	0.0-10.0	8.3	0.0-24.0	*	2.2	0.0-30.0	3.4	0.0-22.0	†
Progressive changes	1.3	0.0- 4.0	0.8	0.0- 3.0	*	1.2	0.0- 5.0	5.3	0.0-20.0	ns
Regressive changes	2.5	0.0- 6.0	3.2	0.0-11.0	*	3.7	0.0-30.0	18.2	1.0-99.0	†

Levels of significance: * = $P > .1$; ns = $.1 \geq P > .05$; † = $.05 \geq P > .01$; †† = $.01 \geq P > .001$; § = $.001 \geq P$.

cantly in men (Table 3). Likewise, the scores of cartilage disintegration, deviation in form, and cartilage and bone remodeling were comparable in the condyle and temporal component (Table 3). Significant differences between normal and internally deranged joints were seen only occasionally and did not reveal a consistent pattern in females and males. In contrast, cartilage disintegration and deformation of the disc were significantly more severe in association with internal derangement as compared to normal disc position, although the lower side of the disc was affected in females and the upper side was affected in males (Table 4). Similarly, progressive remodeling was more prominent in abnormally positioned discs from women, while regressive changes were significantly more severe in those from men (Table 4). With respect to the disc attachments, significant differences between normal and internally deranged joints again were not seen uniformly in both genders (Table 4). It is worth noting, however, that all mean scores for tissue disintegration and regressive changes in the disc attachments were higher in association with an abnormal as opposed to normal disc position, and that a larger proportion of parameters than in the condyle and temporal component was significantly affected by internal derangement (Table 4).

Discussion

The findings suggest that degeneration and remodeling of all TMJ components except the disc complex itself are little affected by internal derangement. Discs positioned anteriorly, however, do appear to degenerate more severely and more extensively than those positioned normally. In addition, the disc attachments may also be affected

by internal derangement, although to a lesser degree than are the discs.

When identifying histologic alterations of the various articular tissues, care was taken to distinguish as far as possible changes due to either degeneration or remodeling. In accordance with studies of hyaline articular cartilage,[2,3] degeneration was conceived strictly as disintegration in the normal structural arrangement of a tissue. This definition included mild fibrillation associated with an intact articular surface, which sometimes cannot be completely ruled out as having resulted from histologic processing. In previous studies,[2,5] mild fibrillation has been considered a normal aging change. Such a concept, however, inevitably would have faced semantic difficulties in distinguishing aging and degeneration.[1] Similarly, some mild regressive changes, such as focal reductions in cartilage cellularity or in matrix metachromasia, which have been classified as aging changes elsewhere,[2] have been attributed to regressive remodeling in the present study to avoid such problems. Finally, osteophyte formation in accordance with the definition given by Johnson[7] was taken as a sign of circumferential remodeling, rather than as one of OA as in some earlier investigations.[11,15,34,35]

Scores assigned to the various grades of a particular morphologic sign were chosen arbitrarily. The primary goal of this choice was to allow inferences as to the appearance and extension of a particular change, rather than as to its clinical severity, which could not be estimated in the absence of a dental medical history. Thus, it can be concluded from the ranges in scores displayed in Table 3 that, for instance, the most prominent disintegration of condylar fibrocartilage in internally deranged joints from males was "severe" (Table 2) along two tenths and "moderate" along another two tenths of the anterior slope. However, it should not be inferred that this was about 20 times more severe than the minimal changes detected. Similarly, from equal scores regarding progressive and regressive remodeling, it cannot be derived that anabolic and catabolic processes were balanced. No attempt was made either to determine the error in judgment of these scores, as conclusions were drawn only from comparisons of groups of joints examined in the same way.

With respect to articular remodeling associated with internal derangement of the TMJ, there is wide consensus that anterior disc position elicits an adaptive response, particularly in the tissues of the disc and posterior disc attachments.[16,25-29,36,37] Observed histologic signs that suggest remodeling of the disc include rearrangement of collagen fibers,[24] decrease in cellularity,[27] and the appearance of a proliferative surface layer.[29] In the posterior disc attachment, fibrosis,[16,24,36] thickening of arterial walls[25] and other vascular changes,[27] hyperplastic tissue formation,[26,27,36] hyaline degeneration,[16,26] enhanced staining reaction for sulfated glycosaminoglycans,[28] splitting,[29] and perforation[22,26] have been described. Most of these morphologic changes have been considered typical for internal derangement and some, eg, synovial hyperplasia and enhanced staining for glycosaminoglycans, have also been recorded in the present study. However, these changes might well be related to the altered loading pattern of an abnormally positioned disc and posterior disc attachment,[24,27,28] rather than being associated with an attempt at repair of tissue damage due to degeneration.

With respect to articular degeneration, the microscopic findings of the present study compare well with results from macroscopic inspection of whole articular surfaces, although histologic examination of comparatively few sections is likely to underestimate the occurrence of degenerative alterations. In particular, mild fibrillation as early as the third or second age decade and a subsequent increase in severity and extension of tissue disintegration with age have also been observed in the hip and shoulder,[4,5] the sternoclavicular joint,[10,38] and the TMJ.[23,30] When considering the association between degeneration and sex or a particular site of the TMJ, however, results vary. The finding in this study of similar scores in males and females agrees with the observations of Kopp et al[38] and Axelsson et al,[32] but contrasts with other reports of a higher proportion of OA in women as compared to men.[11,30,35,39] Furthermore, similar scores of degeneration in all joint components as obtained in the present analysis agree with the results of Westesson and Rohlin,[17] but disagree with observations of more severe and more frequent changes in the temporal component and disc as compared to the condyle[30,32] as well as in the condyle as compared to the other joint components.[11]

Rather than confirming such a distinctive susceptibility to degeneration in general, the current analysis suggests that the various joint components react in distinctive ways to internal derangement. In fact, the disc seemed to be the only part of the joint whose degeneration and remodeling are significantly affected by anterior disc position. Respective parameter scores, in addition to being higher, also increased more steeply with age in internally deranged than in normal joints. As the degree of gross deformation in anteriorly posi-

tioned discs, which had been recorded previously,[1] suggested that abnormal disc position in middle-aged and elderly individuals of the current sample had existed for considerably longer time than in adolescents and young adults, this steeper rise with age could indicate that degeneration of the disc was accelerated as an effect of persisting internal derangement. In contrast, scores for degenerative and remodeling changes of the condyle, the temporal component, and the disc attachments exhibited associations with age that were not apparently affected by disc position. Thus, the present data do not support previous conclusions that uncorrected internal derangement constitutes a particular risk for development of severe structural hard tissue changes or overt OA in general.[15-17,19] Rather, degeneration of the joint components other than the disc may indeed be only an accompanying feature of abnormal disc position, as assumed by de Bont et al,[20] but could as well be considered its cause per Stegenga et al.[21]

Irrespective of such considerations as to possible cause-and-effect relationships, it should be emphasized that no significant signs of degeneration or regressive remodeling could be detected in internally deranged joints obtained from subjects under 20 years of age. Hence, early degenerative alterations are unlikely to account for abnormal disc position in adolescents. Rather, internal derangement in this age category conceivably could be related to progressive remodeling associated with late growth, which was found to be particularly prominent in the condyle. This is in agreement with previous observations that the condyle undergoes considerable changes in shape during the period of transition from growth to adulthood.[23,30] Progressive remodeling of the articular tissues, therefore, conceivably could account for dimensional joint alterations that, in turn, may predispose to the development of internal derangement.[1] However, the findings of this study neither rule out the possibility that abnormal disc position in adolescents is within the range of normal anatomic variation[18] nor that it is related to systemic factors[40] that need not necessarily result in morphologic alterations.

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Resumen

Degeneración Articular y Remodelación en Articulaciones Temporomandibulares Humanas con Discos en Posición Normal y Anormal

Se examinaron las articulaciones temporomandibulares con discos en posición normal y anormal, pertenecientes a 15 mujeres y 38 hombres (la mayoría entre los 15 y los 50 años), por medio del microscopio de luz. Se clasificaron los cambios histológicos atribuídos a la degeneración y a la remodelación de los tejidos articulares de acuerdo a su prominencia y extensión a lo largo de las superficies articulares. En el cóndilo y el componente temporal estos cambios parecieron ser poco afectados por el malfuncionamiento interno. En contraste, la degeneración y el remodelamiento regresivo del disco y, a un nivel más bajo sus inserciones, fueron más severos y aumentaron más severamente con la edad en las articulaciones afectadas por el malfuncionamiento interno, en comparación con las normales. Estos hallazgos indican que el disco indudablemente presenta posiciones anormales. Sin embargo, esta conclusión parece ser válida sólo para los adultos, ya que en las articulaciones de adolescentes se han observado cambios de remodelación progresiva bastante prominentes, pero no se han observado cambios degenerativos significativos.

Zusammenfassung

Artikuläre Degeneration und Remodellierung in menschlichen Kiefergelenken mit normaler und abnormaler Diskusposition

Kiefergelenke mit normaler und abnormaler Diskusposition von 15 Frauen und 38 Männern, die meisten von ihnen zwischen 15 und 50 Jahre alt, wurden lichtmikroskopisch untersucht. Histologische Zeichen, die auf Degeneration und Remodellierung der artikulären Gewebe schliessen liessen, wurden entsprechend ihrer Ausprägung und Ausdehnung entlang der Gelenkflächen in Schweregrade eingeteilt. Entsprechende Veränderungen des Kondylus und der temporalen Gelenkkomponente schienen durch eine abnormale Diskusposition wenig beeinflusst. Hingegen waren Degeneration und regressiver Gewebeumbau von Diskus und, allerdings in geringerem Umfang, von Diskusbefestigungen schwerwiegender in Gelenken mit abnormaler Diskusposition. Zudem nahm in diesen der Schweregrad der histologischen Diskus-Veränderungen mit dem Alter stärker zu als in normalen Gelenken. Die Befunde weisen damit darauf hin, dass ein Diskus in einer abnormalen Position tatsächlich Schaden nimmt. Allerdings scheint diese Schlussfolgerung nur für Erwachsene zuzutreffen, da in Gelenken von Adoleszenten keine signifikanten degenerativen Veränderungen, sondern lediglich Zeichen von ausgeprägtem progressivem Gewebeumbau beobachtet wurden.