

Intra-articular Disc Displacement Part II: Its Significant Role in Temporomandibular Joint Pathology

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Most painful temporomandibular joints (TMJ) have a displaced disc, which leads one to suspect that disc displacement plays a role in joint disease. Many surgeons have apparently reached the same conclusion, because treatment for the painful joint with an internal derangement includes repositioning or removal of the disc. As disc imaging began to be used postoperatively to document disc position, however, it became evident that most attempts to reposition the disc were unsuccessful, although the treatment seemed to relieve the pain. The more recent surgical modalities of arthroscopic lysis and lavage and arthrocentesis also seem to relieve pain and dysfunction, yet repositioning the disc is neither a goal nor a result of these procedures. This has led to confusion about the role of the disc and caused some surgeons to question the importance of disc position and function. But, as Merrill¹ has suggested, it seems premature to promote the idea that disc position is unimportant, because "it may well be that there is a long-term deleterious effect of not treating disc position." There also may be short-term deleterious effects.

The disc is a product of evolution and, therefore, one can reasonably assume that it serves an useful purpose. But, relatively little has been written about the role of the disc. Some functions of the disc, such as its contribution to joint stability, have been deduced from anatomic and functional observations.² The apparent role of the disc in promoting joint stability is based, in part, on the observation that it tends to be centered over the load-bearing part of the condyle when a load is applied and joint space decreases. The disc is thus able to move passively with the condyle during

sliding movements. There is also an active component attributed to disc movement during condylar excursion. During opening, the elastic fibers of the posterior attachment limit the anterior motion of the disc by maintaining the anterior band against the condyle between the articulating surfaces. During closure, the superior head of the lateral pterygoid muscle contracts and produces anterior and medial traction on the disc. This traction on the disc causes the posterior band to fill the space between the condyle and glenoid fossa and seems to be an especially important action that helps to stabilize the joint. The biconcave shape of the disc also seems to distribute loading over a greater area, and the disc may serve to cushion joint loads, as well as to facilitate lubrication and nourishment of the joint surfaces. Although these commonly accepted functions seem reasonable, and probably occur in the temporomandibular joint, there are little direct supporting data.

There are also three other apparent functions of the disc, for which there are more supporting data. These functions are based on correlations of disc position and specific outcomes. In this article the data will be reviewed that suggest normal disc position: 1) assists in alleviating pain, 2) prevents the gross degenerative changes of osteoarthritis, and 3) promotes growth of the mandible. These data constitute a strong argument for including disc reduction as an important goal of any treatment for the painful joint with a displaced disc.

Definitions

Although disc displacement is a central issue in this article, internal derangement (ID) is only one component of osteoarthritis (OA). The general pathologic process involving the TMJ is OA, a degenerative disease affecting cartilage, subchondral bone, and the soft tissue components of the joint.³ There also may be secondary inflammatory components. Whether disc displacement is a consequence or a cause of OA is not clear. A common feature of OA and ID is regressive change in the morphology of the joint, especially the

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FIGURE 1. Panoramic radiograph of mandibular condyles in a 37-year-old woman. A. The deformed right condyle. B. The deformed and dislocated left condyle. Both joints are typical of late-stage internal derangements and osteoarthritis. Disc imaging showed the discs to be displaced, deformed, and nonreducing. The woman has mandibular deficiency and Class II malocclusion. The skeletal deformity and malocclusion have worsened in the last few years, but there has been no joint pain.

condyle. If the disc becomes displaced during growth, the condyle sometimes becomes small and round. In late-stage OA and ID, the condyle usually goes through a poorly understood process that results in loss of mass, deformation and flattening (Fig 1). Avascular necrosis may be a variant or component of the process that leads to the deformed condyle. This regressive process is often simply referred to as degenerative joint disease (DJD). Thus, when disc displacement, disc dysfunction, ID, or DJD is used in this article, each should be viewed in the larger context of the pathologic process of OA.

Disc Displacement and Pain

Although it is possible that a displaced disc plays no role in the pain associated with OA and ID, it seems more reasonable that reversal of the ID to establish normal disc position has a positive effect on pain relief. Perhaps the best surgical data relating to the role of normal disc position in relief of pain comes from 64 patients on whom arthrograms were performed before and after modified condylotomy.⁴ All had a painful reducing disc displacement (Wilkes stage II, III)⁵ and all had the same operation performed by one surgeon. Twenty-one of the patients continued to complain of symptoms, including pain, after operation. The other

43 patients were from a larger group that became asymptomatic after operation. Among the asymptomatic patients, 72% had the disc in a load-bearing or reduced position, whereas the disc was reduced in only 43% of patients experiencing symptoms. For all instances in which the disc was reduced, it also functioned normally throughout excursive movements. These data support the proposition that normal disc position and function make pain relief more likely. But, they also show that disc reduction is neither necessary for pain relief nor does it always eliminate pain.

The data from attempts to reposition the disc with arthroscopy are more difficult to interpret. Kaplan et al⁶ imaged the disc in 20 symptomatic joints an average of 16 months after arthroscopy for disc repositioning. They found that none of the joints had normal arthrograms and 90% had decreased condylar mobility. Twenty-five percent of the discs were displaced. Because all of the joints were symptomatic, and none was normal by arthrography, no conclusions can be made regarding the relationship of joint pain to disc position, except that pain can occur even when the disc is reduced. Conway et al⁷ reported on the magnetic resonance imaging (MRI) evaluation of 25 joints after discoplasty. Only 12% of the joints had a normal disc position an average of 6 months after operation; another 28% of joints had improved disc position. Disc

mobility was markedly diminished in 90% of the joints and the disc failed to move forward appreciably on opening. All patients who had either normal or improved disc position were determined to have either excellent or good clinical results. Conversely, all patients with either fair or poor clinical results, had a disc that showed either little or no improvement in position after surgery. Because joints with a reduced disc were not separated from others in the group that were still displaced (improved disc position), it is difficult to draw conclusions about the relationship of disc reduction and pain. Lastly, Montgomery et al⁸ evaluated 74 joints an average of 2 years after surgical repositioning of the disc. Disc position was unchanged or worsened in 92% of the joints. The other 8% of discs were improved, but apparently none was reduced. Again, no conclusions can be drawn regarding the extent to which a normally positioned disc affected pain.

Nonsurgical reduction of a displaced disc by mandibular repositioning, like the condylotomy data, also suggests that normal disc position lessens pain. Lund et al⁹ randomly divided 63 patients with painful joints and a reducing disc into an onlay-repositioning group, a flat-plane splint group, and an untreated control group. Arthrograms were used to verify that the disc was reduced when the mandible was anteriorly repositioned. The new mandibular position was maintained by occlusal onlays. After 6 months of treatment, the disc-repositioning group had better function and less pain than the other two groups. When the onlays were removed, clicking and pain recurred in the 85% of patients who had obtained good relief. Westesson et al¹⁰ reported on the disc position in 18 of these 20 patients before the onlays were removed at 6 months. In 12 of the 18 patients, the disc was still in a load-bearing position, but in 6 it had displaced anteriorly, 2 of which had progressed to a nonreducing status. All 12 patients with the disc in a load-bearing position were free of subjective symptoms. On the other hand, the 6 patients with a displaced disc had subjective symptoms and these were especially marked in 3 of the patients who had developed clicking or locking. Thus, the recurrence of symptoms was associated with recurrence of disc displacement, and the severity of symptoms increased in parallel with the degree of mechanical disc dysfunction. Fifteen patients were examined by Lund and Westesson¹¹ 3 years after occlusal treatment to reposition the mandible and reduce the disc. Eleven of the 15 patients had arthrograms performed 3 years after occlusal treatment and disc position was compared with the intensity of subjective symptoms. They found only 2 patients with displacement of the disc, but could find no appreciable differences in the clinical status of these 2 patients compared with the other 9 who had a normal disc/condyle relationship.

These data clearly show that pain can be less after procedures that do not reduce the disc and, conversely, can be present in joints with normal disc position. But, the data also suggest that pain relief is more likely with disc reduction, especially when the mechanical signs and symptoms associated with a displaced disc are more severe. Normal disc position seems to be a factor in pain relief, but not the only factor, and probably not the most important factor. Nickerson's view¹² seems to fit the facts best; a reduced disc increases the threshold for pain from joint use.

Disc Displacement, DJD, and Skeletal Deformity

Boering's great contribution to the understanding of TMJ disease began with his 1966 thesis^{13,14} in which he presented his findings in 400 patients he observed for 4 to 8 years. All patients had signs and symptoms of OA and ID. Although disc imaging was not used, subsequent studies by Boering and others strongly suggest that the vast majority of these 400 patients did have OA and ID. Nickerson and Møystad¹⁵ later showed a high correlation of proven disc displacement (typically nonreducing) with the condylar and skeletal changes observed by Boering. Nickerson and Boering¹⁶ subsequently confirmed the association of a displaced disc and facial skeletal changes. Furthermore, disc imaging in 46 of Boering's original patients 30 years later¹⁷ confirmed their original diagnosis of OA and ID in 91% of symptomatic joints. All of Boering's patients were treated nonsurgically using therapies that were supportive and not intended to re-establish normal disc position.^{13,14,16} Thus, by careful observation of Boering's patients, the natural history of OA and ID has been largely delineated.

Boering noted that OA was progressive and resulted in gross radiographic changes in the condyles as seen in transpharyngeal radiographs (Figs 1 and 2). The number of joints exhibiting these changes increased by 23% between 2 and 5 years after his initial examination. The changes he described for the condyle are typical of the deformity in the terminal stage of OA. They consist of flattening of the articular surface, a decrease in the size of the condyle and sometimes dorsal bending of the condyle, and sclerosis of the articular cortical bone (DJD). In rare cases, a subcortical radiolucency or "cyst" was noted, as well as an occasional osteophyte or "lipping."

Boering also observed a high correlation of the condylar deformity (DJD) with a shortening of the condylar neck and ascending ramus. In 70% of the unilateral cases, there was a 4 mm or greater difference in the vertical ramus height, with a positive correlation between the deformed joint and the shorter ramus. More severe instances of this deformity resulted in vertical

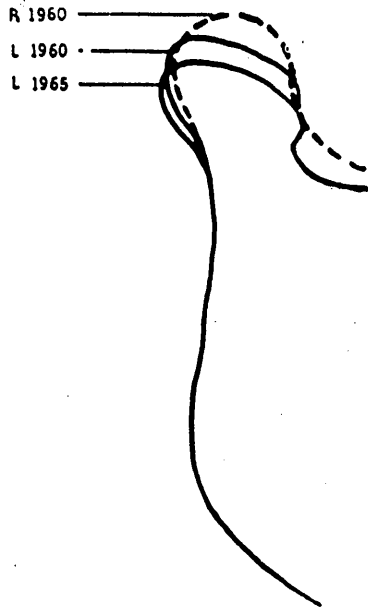


FIGURE 2. Tracings of transpharyngeal radiographs of 19.5-year-old woman. The tracings of the left side show a deformed condyle that demonstrates additional bone loss during the 5-year interval between radiographs. (Reprinted with permission.¹⁴)

ramus asymmetry in 13% of the patients. There also was noticeable chin point deviation to the affected side in 15% of patients. Boering presented a case showing the progression of radiographic changes in the condyle, and a worsening of mandibular retrusion and occlusion. The skeletal and occlusal deformities stabilized parallel with radiographic stabilization of the condylar deformity.

Ricketts¹⁸ independently observed an association between condylar deformity, shortening of the vertical ramus, and retrognathia. There also was no disc imaging of his patients, but the condylar deformity he illustrated was the same as Boering repeatedly documented and is typical for OA and ID. As disc imaging became more common, Nickerson and Møystad¹⁵ used arthrography to document ID in 91% of condyles shown to be deformed by transpharyngeal radiographic views. They also found a high association of condylar deformity and ID with mandibular deficiency. The condylar deformity described was typical for the terminal stage of OA and ID and, in fact, a nonreducing disc was highly associated with these deformities.

As MRI of the disc became available, large numbers of patients were imaged. Several reports¹⁹⁻²² now confirm a high degree of correlation of disc displacement (especially nonreducing), condylar deformity, and the skeletal deformities of mandibular deficiency, open-bite, and asymmetry. Link and Nickerson,²¹ confirmed disc status in 39 of 40 consecutive orthognathic surgical patients with Class II malocclusion, asymmetry, or open-bite. They found that 97% of these 39 patients

had disc displacement and 85% had Class II malocclusion. Schellhas et al²² retrospectively examined MRIs of the TMJs of 100 orthognathic surgical candidates with mandibular deficiency, some of whom also had an open-bite or mandibular asymmetry. Eighty-eight percent of patients had disc displacement and most discs were nonreducing. Condylar deformity was common. Eleven of the 12 patients with normal disc position had only mild chin retrusion and no asymmetry. Finally, in a series of 243 consecutive joints in which disc imaging and lateral radiographic views of the condyle were available, there was not a single occurrence of normal disc position associated with any type of condylar deformity.^{16,23} Thus, there seems to be a high degree of association between ID (especially nonreducing), DJD, skeletal deformity, and malocclusion.

The apparent long-term effects of a displaced disc can be determined from evaluation of Boering's patients 30 years after nonsurgical treatment.^{13,14,17}

Twenty-one percent of 33 joints with a clinical diagnosis of reducing disc displacement on initial examination had radiologic evidence of progression of DJD 2 to 4 years later. When the same 33 joints were examined 26 to 28 years later, 60% showed progression of radiographically apparent DJD and the changes were more severe.

Conversely, reestablishment of normal disc position seems to decrease progression of OA to gross DJD. Nickerson²⁴ reported 3 of about 150 patients develop a condylar deformity typical of late-stage OA and ID 1 to 8 years after being treated by modified condylotomy. This 2% incidence is only one tenth of the expected rate of a new occurrence of regressive changes (DJD), based on findings in Boering's patients during the first 2 to 5 years of his observations. McKenna²⁵ recently evaluated 15 of Nickerson's patients 10 years after modified condylotomy. The disc was in a reduced or load-bearing position in 61% of the 18 operated joints. None of the joints had the typical MRI changes seen in the TMJ with late-stage OA and ID (DJD). Furthermore, only 12% of the joints had progressed to the nonreducing stage in 10 years. McKenna's data are in sharp contrast to the accelerated rate of progression in Boering's patients. Based on the disappearance of clicking, de Leeuw¹⁷ estimated that 34% of Boering's original patients progressed from reducing disc displacement to nonreducing disc displacement in only 2 to 4 years. The data from Nickerson²⁴ and McKenna²⁵ suggest that re-establishing normal disc position does protect the joint from DJD. Their data also suggest that when normal disc position is established by modified condylotomy, there is a low relapse rate, a two thirds reduction in the rate of progression from a reducing to a nonreducing disc displacement, and protection against gross DJD.

In summary, there seems to be a strong association

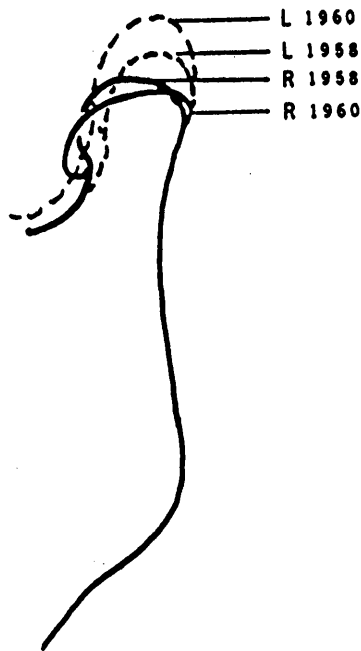


FIGURE 3. Tracings of transpharyngeal radiographs of 13.5-year-old girl. The tracing of the right side shows a deformed condyle with no evidence of growth during the 2-year interval between radiographs. The tracing of the left condyle shows normal condylar morphology and growth during the same 2-year interval. (Reprinted with permission.¹⁴)

of condylar deformity and the nonreducing disc displacement of late-stage OA with facial skeletal and occlusal deformity. These skeletal deformities and their accompanying malocclusions are predominantly mandibular deficiency; open-bite and mandibular asymmetry are much less common. Conversely, normal disc position is uncommonly associated with these facial skeletal deformities.¹⁹⁻²²

Disc Displacement and Mandibular Growth

DJD should subtract condylar bone mass and shorten ramus height to a similar degree in both adults and children. If OA and ID also affect growth, however, the total effect on ramus height would be more severe when it occurs in children than when it occurs in adults. In fact, this is what Boering^{13,14} found. He appears to be the first to call attention to the juvenile form of OA and ID and to point out the greater severity of the skeletal deformity. Boering compared serial lateral radiographs for ramus height and condylar deformity in 103 of his patients who were 20 years of age or younger. He found 31 (30%) showed little or no increase in ramus height, and all had condylar deformity. He concluded that a growth disturbance had occurred in these 31 patients because diminution of the size of the condyle alone could not account for the magnitude of the difference in ramus height (Fig 3). Katzberg et

al¹⁹ later noted the high incidence of ID in a group of pediatric-age patients and observed one boy who had an ID and progressive shortening of the condyle on one side sufficient to cause facial asymmetry. They suggested that a displaced disc may diminish growth of the mandible. Dibbets et al²⁶ also studied growing patients. They found that condylar deformity typical of late-stage OA and ID and a short mandibular corpus had a high statistical correlation with more severe skeletal Class II malocclusion. Nickerson and Boering¹⁶ presented an additional pediatric patient whose case demonstrated that significant mandibular deficiency and malocclusion develops in conjunction with condylar deformity and a displaced disc. The skeletal and occlusal changes stabilized with the development of a deformed condyle, a nonreducing disc, and skeletal maturity. These reports are all consistent with the hypothesis that the skeletal and occlusal deformities are the result of condylar bone loss and diminished mandibular growth secondary to a displaced disc.

Most instances of significant mandibular deficiency appear to be the result of a growth deficit related to disc displacement, rather than to genetic programming for less mandibular growth. Link and Nickerson²¹ found that 88% of 33 patients who had orthognathic surgery for a Class II malocclusion had bilateral disc displacement. They also suggested that a growth deficit alone had possibly caused the mandibular deficiency in the 59% who did not have DJD and its associated loss of condylar bone. More recently, Schellhas et al²⁰ used MRI to document the association of disc displacement, condylar deformity, and significant skeletal deformity in a pediatric population. Ninety-three percent of 60 patients with "obvious mandibular deficiency" had a displaced disc, usually the nonreducing disc observed in the late stages of OA and ID. They also noted an association of a displaced disc with mandibular asymmetry (deviation toward the ID), as have others,^{19,21} and anterior open-bite. They concluded that ID may contribute to the development of mandibular deficiency by retarding growth as well as loss of condylar bone. Conversely, it is uncommon to find instances of mandibular deficiency, the most common facial skeletal deformity, and normal disc position. Link and Nickerson²¹ found that only one of 33 patients with Class II malocclusion had normal disc position. Thus, it appears that mandibular deficiency may be largely acquired as the result of disc displacement in as many as 90% of affected patients and principally the result of genetic programming in the other 10%. It is also possible, however, that those with an acquired deficiency may have a genetic predisposition for OA and ID, and thus also have an indirect genetic component.

If a displaced disc is the major cause of significant mandibular deficiency, then ID of the TMJ is a significant public health problem in the United States. Using

published data, one can estimate the number of persons with significant mandibular deficiency and Class II malocclusion attributable to OA and ID. Ten percent of white youths from 12 to 17 years of age were found to have a Class II malocclusion with an overjet of 7 mm or greater.²⁷ Class II malocclusion correlates highly with mandibular deficiency.²⁸ Thus, if the US population is 250 million, and this sample of white youths is representative of the population, 25 million persons would have significant mandibular deficiency. If the proportion caused by OA and ID is about 90%, as suggested by three studies,²⁰⁻²² then 22.5 million persons may well have an acquired mandibular deficiency as a result of a displaced disc.

Implications

If subsequent studies confirm that the normally positioned disc plays a role in pain relief, prevention of DJD, and promotion of mandibular growth, as well as in other more commonly suggested functions, it would be useful to develop strategies to prevent the widespread deleterious consequences of untreated or inadequately treated OA and ID. But, there are several barriers that would have to be overcome. The majority of patients with OA and ID are asymptomatic and do not seek treatment. The best time to treat OA and ID probably is early in the disease process before significant disc, skeletal, and occlusal changes occur, and while the individual retains optimal capacity for tissue repair and growth. Most people with OA and ID who become symptomatic, however, do so after growth is complete. Furthermore, many symptomatic joints have already progressed to a nonreducing disc and DJD when the patient is first seen and no current procedure has been shown to achieve disc reduction reliably in late-stage disease.

Possible strategies to minimize the deleterious effects of a displaced disc must begin with an inexpensive, effective, and convenient means of screening children for OA and ID. None currently exists. There would also need to be an affordable and effective non-surgical means of disc reduction for the asymptomatic joint that would prevent development of mandibular deficiency; activator appliances or onlays that advance the mandible to reduce the disc may prove useful for this purpose. The use of activator appliances to treat developing mandibular deficiency when the disc is displaced has been previously discussed.²³ Finally, for the people who become symptomatic and require surgery, current procedures will need to be refined to achieve normal disc position and function as well as to relieve pain.

Conclusions

None of the studies cited proves the importance of disc position, because an association between variables

does not define a cause and effect relationship. However, the consistency of the observations showing a high correlation between disc displacement, DJD, facial skeletal deformity (mandibular deficiency, open-bite, and asymmetry), and malocclusion that is more severe when the ID occurs during growth, the virtual absence of these effects when the disc is normally positioned, and evidence that re-establishing normal disc position and function protects against DJD, constitute strong evidence in support of the importance of normal disc position for optimal jaw growth and protection against DJD. A reduced and functional disc also seems to have a role in alleviating pain associated with OA and ID. Finally, it is difficult to escape the intuitive notion that a normally positioned and functioning disc is a necessary component of an optimally healthy TMJ.

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