Effect of Excessive Occlusal Forces upon the Pathway of Gingival Inflammation in Humans*, **

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IN an earlier report¹ we indicated that in monkeys artificially created excessive occlusal pressure changed the alignment of the transeptal fibers and deeper periodontal ligament fibers so as to alter the pathway of gingival inflammation into the supporting periodontal tissues in periodontal disease. Interdentally, inflammation from the gingiva extended directly into the periodontal space rather than into the interdental septum. It was suggested that by altering the periodontal tissues and the pathway of gingival inflammation, excessive occlusal forces, particularly pressure, could be a factor in the etiology of infrabony pockets and angular bone resorption in periodontal disease. Other investigators² reported that excessive tension produced changes in the periodontal ligament of experimental animals which altered the pathway of gingival inflammation into the supporting periodontal tissues.

This is a report of microscopic observations regarding the relationship of occlusal forces and the spread of gingival inflammation into the supporting periodontal tissues in human jaws. The jaws were removed at autopsy, fixed in 10% neutral buffered formalin, decalcified in an equal mixture of 20% sodium citrate and 45% formic acid, embedded in celloidin and cut in serial section.

OBSERVATIONS

Area 1. Maxillary canine, premolar and molar area of a 59 year old male patient. Radiographs indicate wedge-shaped thick-

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Fig. 1. Radiograph of maxillary premolar area. Note wedge-shaped thickening of periodontal space mesial and distal to the second premolar.

Fig. 2. Survey mesio-distal section of premolar area shown in Fig. 1. Calculus and periodontal pockets are present on first and second premolar and molar roots. Hematoxylin and eosin. Orig. mag. x 6.
Fig. 3. Higher power of the interdental space distal to the second premolar (center) shown in Fig. 2. The transeptal fibers extend horizontally across the crest of the bone and perpendicular to the premolar and molar tooth surfaces. The alignment of the periodontal ligament fibers is normal. Hematoxylin and eosin. Orig. mag. x 40.

Fig. 4. Higher power of the interdental space mesial to the second premolar (center) shown in Fig. 2. The transeptal and the deeper periodontal ligament fibers on the mesial surface of the second premolar are oblique and angulated in an apical direction suggesting a compressive mesial force. Inflammation extends from gingiva directly into the periodontal ligament. Hematoxylin and eosin. Orig. mag. x 40.

Fig. 5. Higher power of alveolar crest area shown in Fig. 5. Note lacunar resorption along the angular bone surface. Hematoxylin and eosin. Orig. mag. x 100.

Fig. 6. Higher power of the interdental space distal to the second premolar (center) shown in Fig. 2. The transeptal fibers extend horizontally across the crest of the bone, perpendicular to the approximating tooth surfaces (Fig. 3). The alignment of the periodontal ligament fibers between the interdental bone and the roots is normal. Inflammation in the gingiva extends to and between the transeptal fibers.

Fig. 5. Section deeper in the block in area shown in Fig. 4. The transeptal fibers are perpendicular to first premolar root but angulated with respect to the second premolar. Inflammation extends along altered transeptal fibers directly into periodontal ligament. Hematoxylin and eosin. Orig. mag. x 50.

In the interdental space distal to the second premolar the transeptal fibers extend horizontally across the crest of the bone, perpendicular to the approximating tooth surfaces. The alignment of the periodontal ligament fibers between the interdental bone and the roots is normal. Inflammation in the gingiva extends to and between the transeptal fibers.
In the interdental space between the first and second premolars the direction of the transeptal fibers is oblique instead of horizontal (Fig. 4). The deeper periodontal ligament fibers on the mesial surface of the second premolar are also oblique and angulated in the direction of the root apex, suggesting a compressive mesial force on the second premolar. Distal to the first premolar the periodontal ligament fibers are distended and perpendicular to the root and interdental septum.

On the mesial surface of the second premolar in the coronal third the periodontal ligament is widened and wedge-shaped, compared to the periodontal space distal to the first premolar. The epithelial attachment at the base of the pocket on the second premolar is located further apically than the base of the pocket on the first
premolar. The interdental bone is reduced in height and presents an angular crestal defect mesial to the second premolar. Inflammation extends from the gingiva to the transeptal fibers and along the fibers into the periodontal ligament.

Sections deeper in the block (Fig. 5) show the transeptal fibers perpendicular to the root of the first premolar, but angulated and parallel to fibers deeper in the periodontal ligament mesial to the second premolar. The inflammation extends along the altered transeptal fibers directly into the periodontal ligament, and there is lacunar resorption along the angular bone surface (Fig. 6).

The first and second premolars are erupted to a comparable occlusal level. The angular defect on the mesial aspect of the second premolar is therefore not attributable to differences in the levels of the approximating cemento-enamel junctions.

Area 2. Mandibular canine and premolar area in a 54 year old male. Radiographically there is extensive angular bone destruction distal to the first premolar with slight angular destruction of the interdental septum between the first premolar and canine (Fig. 7).

Microscopic examination reveals suprabony pockets on the mesial and distal surfaces of the canine (Figs. 8 and 9). The
periodontal ligament around the first premolar is wider than on the canine.

Between the canine and first premolar the transeptal fibers are oblique instead of horizontal and extend toward the apex of the premolar (Fig. 10). The underlying periodontal ligament fibers are compressed suggesting excessive mesio-occlusal pressure. There is angular destruction of the interdental bone. The periodontal pocket on the mesial surface of the premolar is located further apically than the pocket on the distal surface of the canine. It extends beyond the level of the crest of the interdental bone and represents an early stage of infrabony pocket formation.

On the distal surface of the premolar there is an infrabony pocket with wide angular destruction of the bone (Fig. 11). Dense collagenous fibers cover the angular bone surface. The underlying periodontal ligament fibers are extended and the periodontal ligament is widened consistent with excessive tension. Inflammation from the periodontal pocket wall extends laterally between the fibers on the angular bone surface and apically into the periodontal ligament.

Area 3. Mandibular molar area in a 59 year old male. There are facets at the facio-occlusal angle of the molars and premolar (Fig. 12). Microscopic examination of the second mandibular molar reveals periodontal pocket formation on the facial and lingual surfaces (Fig. 13). On the facial surface, well formed periodontal ligament
fibers extend from the crest of the bone to
the cementum. Inflammation from the
marginal gingiva extends lateral to the
crestal fibers and external to the facial bony
plate (Fig. 14) into the attached gingiva.

On the lingual surface, instead of spread­
ing external to the lingual bony plate, the
inflammation extends into the periodontal
space. The periodontal space at the crest
of the bone is funnel-shaped and widened.
(Fig. 15). The normal arrangement of gin­
gival and alveolar crest fibers is destroyed.
The periodontal ligament fibers apical to
the widened area are compressed and oblique,
suggesting excessive occlusal pressure. There
is an angular defect in the bone.

Discussion

The findings in human autopsy material
corroborate the relationship between exces­
sive occlusal forces and the pathway of
gingival inflammation demonstrated under
experimental conditions in animals. The
periodontal changes in humans were com­
parable to those produced by excessive
pressure artificially induced in animals, and
can be attributed to similar etiology. Inter­
dentally, excessive occlusal pressure altered
the alignment of the transeptal fibers as
well as the deep periodontal fibers. Extent­
sion of gingival inflammation directly into
the periodontal ligament space, angular res­
sorption of alveolar bone and extension of
periodontal pockets between the tooth and
bone accompanied the alteration in the
periodontium induced by the excessive oc­
cclusal forces.

On the lingual surface, the gingival and
alveolar crest fibers and underlying fibers
of the periodontal ligament were altered by
excessive pressure and the inflammation
spread directly into the widened periodontal
ligament space rather than external to
the lingual bony plate.

Excessive occlusal forces do not change
the vascular and cellular features of ging­
gival inflammation, but they do change
the environment around the inflammation
and consequently lead to changes in its
direction. Angular and vertical patterns of
bone destruction and the development of
infrabony pockets result from the altered
pathway of the gingival inflammation as
well as from changes in the periodontal
ligament and bone created by the occlusal
forces themselves. The effect of excessive
occlusal forces was observed associated with
tension as well as pressure, and with forces
which were not severe enough to produce
necrosis of the periodontal ligament.

It is generally recognized that trauma
from occlusion and gingival inflammation
represent different types of tissue changes
which initially occur in different areas of
the periodontium. Gingival inflammation
starts in the gingival margin and is caused
by local irritation. Trauma from occlusion
involves the supporting periodontal tissues rather than the gingiva. It is the consensus of research findings that trauma from occlusion does not cause gingival inflammation or periodontal pockets. Orban noted that gingival inflammation and trauma from occlusion commonly occur together, possibly in 90% of all people. He described them as separate and different diseases of the periodontium, which could affect each other adversely.

Our findings demonstrate that when gingival inflammation and trauma from occlusion occur together they produce specific types of periodontal pathology, such as angular bone destruction and infrabony pockets. They are different pathological processes responsible for the tissue destruction in the disease, periodontitis, rather than two different diseases.

As long as the inflammation remains in the marginal gingiva, within the confines of the gingival and transeptal fibers it is unaffected by the presence of trauma from occlusion. However, when the inflammation spreads beyond the marginal gingiva, it and trauma from occlusion combine to become interrelated co-destructive factors in periodontitis.

The implications of our findings in terms of the nature of periodontitis, and the morphogenesis of angular and crater-like bone destruction and infrabony pockets are self-evident. The frequency with which gingival inflammation and trauma from occlusion combine to produce specific periodontal destructive patterns and the severity of irritational and occlusal factors required to bring about the combined destructive effect remain to be determined.

CONCLUSION

Gingival inflammation and trauma from occlusion are different types of pathologic processes which participate in a single disease, periodontitis. Together they exert a combined co-destructive effect which produces angular bone defects and infrabony pockets.

BIBLIOGRAPHY

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