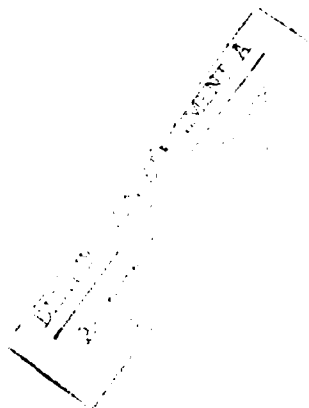LEVEL 20

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A COMPREHENSIVE REVIEW OF
NEW ATTACHMENT THERAPY

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A Comprehensive Review of
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Table of Contents

Table of Contents - 2

New attachment therapy is that which regenerates a more coronal attachment of the periodontal tissues to tooth, after the natural attachment has been destroyed by trauma or disease-- most often the chronic inflammatory periodontal disease, periodontitis. This is distinguished herein from reattachment, or healing by scar,[1] subsequent to surgical detachment. The terms new attachment and reattachment have been used interchangeably by some, and with specific intent by others. "True reattachment" of new cementum, periodontal ligament, and alveolar bone has been distinguished from "soft tissue reattachment" of epithelium and connective tissue coronal to the bony crest,[2] for instance.

In many cases no goal of new attachment or reattachment has been stated prospectively, at all; and various surgical procedures have developed empirically, based on subjective observations from clinical practice and conceptual speculations about pocket elimination.[3] There is a lack of a generally accepted philosophy and no agreement on a goal.[4] Rather than debate the merits of various "named" surgical procedures it appears time to examine the potential of periodontal tissues, considering the biological processes which bring about healing, and the application of knowledge to clinical benefit.[5]

Following his review of the world literature on periodontal therapy,[6] Ratcliff[1] proposed an analysis of repair systems in periodontal therapy. Ratcliff[1] stated, "There are four basic forms of periodontal therapy used for elimination of pockets. These are: (1) shrinkage, (2) excision, (3) healing by scar, and (4) new attachment." These were, essentially, accepting the existing dentogingival junction, or cutting it away and growing a new one. Shrinkage was considered the elimination of the edema developed as a part of the inflammatory reaction to irritants, and with a constant position of the epithelial attachment, shrinkage after hygienic procedures would reduce the pocket depth. Excision, such as gingivectomy, and healing by scar, such as with apically repositioned flaps, produce little to no regeneration of a new attachment. For purposes of analysis of new attachment, the tooth side of the periodontal lesion (Figure 1) was considered to have four zones. After the dissection and removal of the pocket lining (Figure 2), the contributions of the zones to new attachment were discussed.[1] Acknowledging Ratcliff's contribution[1] to our understanding, this review will consider other's points of view and later research findings in different zones on the tooth side and the periodontal tissues side, and their applications to new attachment therapy.

SUBGINGIVAL PLAQUE

There is little doubt that the removal of bacterial plaque is essential for the successful healing of a new dentogingival junction. The level of attachment is generally parallel to and congruent with the apical extent of bacterial plaque on the root surface.[7] The distances from the advancing "front" of the plaque to the nearest periodontal fibers on extracted teeth were measured and found to be 0.3 to 1.5 mm near the cementoenamel junction but only 0.2 to 1.0 mm nearer the apices.[8,9] In many cases after new attachment procedures, plaque is inadvertently left on the roots of teeth despite attempts at meticulous instrumentation with overlapping strokes. The removal of plaque is especially difficult if it is hidden in resorption bays in the cementum,[9,10] at the depth of the pocket, and in the furca.[9] After meticulous instrumentation of the roots and flushing with sterile saline, bacteria are dispersed on the tooth and soft tissue sides of the pocket, and it may be necessary to use a chemical disinfectant.[11]

Antimicrobial Chemicals

Pockets have been instilled, after scaling, with emetin,[12] "hydrogen ammonium fluoride," iodine,[13] "pyrozone" and "hydronaphthol",[14] acriflavin, metaphen, weak iodine or hypertonic salt solution,[15] Dakin's solution,[16] Dunlop oxygen[17] and zinc chloride solution dispersed with electrodes,[18] but without experimental evidence of effectiveness. These treatments were considered to be passing "fads" and not as important as basic scaling, curettage, and oral hygiene.[19] More recently, penicillin has been advocated, because infection was considered the enemy of reinsertion of connective tissue fibers in new cementum.[20] The users of strong salt solutions[21,22] and local delivery of tetracycline by hollow fiber devices[23,24] have claimed a marked reduction of spirochetes and clinical improvement. There is no support in the literature that local in tasche antimicrobial agents will effect, or assist in, healing by new attachment.

CALCULUS

There is unanimous agreement that calculus must be removed.[25-27] Despite the report that junctional epithelium may present a normal attachment to disinfected calculus,[28] sterilized calculus retains irritants.[29] Inadequate removal of calculus will reactivate periodontal disease even with good

supragingival plaque control.[7,9,10] The removal of calculus is difficult
due to its location, hardness, and modes of attachment which may be by cuticles,
surface irregularities, penetration of bacteria, undercuts in resorption bays,[30-32]
or penetration between separations of cementum.[33] Root planing enhances the
chances of the complete removal of calculus,[34] but smooth planed roots may still
have deposits.[35-37] The bacterial flora in a pocket are reduced after subgingival
calculus removal;[24,38,39] and good practice would seem to dictate the removal of
calculus, even if a surgical approach was required for access.[4,40] Past attempts
to use acid in pockets to dissolve calculus[41] were thought adverse because
experience showed that it made healing slow.[42]

ZONE OF ENAMEL

Since Ratcliff's analysis,[1] research has shown that epithelium not only
readapts to enamel after detachment,[43] but that it reforms an attachment which
seems identical to that before treatment.[44,45] The requirements for epithelial
attachment to enamel are most likely the absence of accumulated bacteria and
their toxins,[43] and regenerating junctional epithelium--not oral sulcular or
pocket epithelium.[46] The early strength of the new dentogingival junction in
resistance to rupture forces appears to be related to regeneration of epithelial
attachment to enamel.[47]

Connective tissue new attachment to enamel has not been reported, but a
cementum is found over cervical enamel[48] which Listgarten[49] has termed afibrillar
cementum. Epithelial reattachment can occur to afibrillar cementum,[50] and there
is one single instance reported of a short connective tissue attachment to
cementum on cervical enamel.[51]

Enamel projections protrude into furcation areas of multi-rooted teeth.[52-58]
Some investigators have found a relationship of enamel projections to pockets;[57,53]
and it has been recommended that enamel projections be ground away in order to
provide opportunity for connective tissue new attachment,[59] even though histology
shows they may be covered with cementum.[55,60]

ZONE OF PATHOLOGICALLY EXPOSED ROOT SURFACE

The Cementum Matrix

The cementum which can become involved in new attachment therapy may be of
the acellular, cellular, or intermediate type, depending on the depth of the
loss of attachment and the amount of previous instrumentation. Most often, the

acellular cementum in the cervical area is exposed. Even the dentin could be exposed. The cementum has matrix fibers that parallel the root surface[61,62] and finer fibrils in irregular arrangement[62,63] with the crossbanding of collagen. The matrix is mineralized and presents alternating dark and light bands on microradiography. The lines are thicker and more irregular in cellular than in acellular cementum.[62,64] The mineral crystals parallel the collagen fibers,[62] and mask the collagen matrix.[65] Principal fibers of the periodontal ligament penetrate the cementum at right angles to the surface and their cores are unmineralized.[62] In cellular cementum these Sharpey's fibers are not as densely packed as in acellular cementum.[62] Channels of cementocyte lacunae and their canaliculi are found in cellular cementum.[62] Intermediate cementum, found near the dentin surface, starts about one-third down the root and has a "collagen hiatus" thought to be protoplasmic inclusions of cells.[66,67] Between the Sharpey's fibers on the surface are found cell processes, finely granular material, and a three to five micron wide zone of collagen fibers and fine size mineral crystals, the precementum.[62,68] Repair of cementum may be by acellular or cellular new cementum.[48,60,61] Dentin intertubular matrix has an irregular arrangement of collagen fibers which are masked by mineral. The dentinal tubules are surrounded by a mineralized peritubular matrix which has no collagen fibers.[69]

Changes in the Matrix

The amino acid composition of the organic matrix of human cementum is that of collagen[70-72] with the $\alpha1(I)_2\alpha2$ composition typical of other Type 1 collagens which normally calcify.[72] No statistically significant differences in collagen composition occur in cementum which is pathologically exposed, except that if affected by dental caries there is loss of the hydroxyproline, proline and glycine typically found in collagen and a gain of amino acids thought to be of bacterial origin.[73] The bacterial cell-wall material, alpha, epsilon-diaminopimelic acid is found in carious cementum.[73] The amino acid composition of the outer dentin of periodontally diseased teeth may be altered.[74]

Changes in the Mineral

The mineral content of normal cementum changes only slightly from cervical to midroot or apical areas,[75,76] but the surface of exposed cementum is hypermineralized as revealed by microradiography.[32,77-83] This hypermineralized zone is usually about 10 microns wide, but is sometimes up to 50 microns deep.[78,80]

Electron micrographs show crystals on exposed surfaces that are disarranged, larger, or denser.[81,82,84,85] The denser surface was found to stop at the beginning of the epithelial attachment.[81] When specimens are demineralized, the surface zone often does not present collagen crossbanding in the cementum matrix.[81-83,85] Analyses of diseased root surfaces show significant increases of calcium,[78,83,86] magnesium,[78,86] phosphorus,[78,83,86] fluoride,[83,86,87] copper and zinc,[88] and decreased citrate.[87]

The research indicates a remineralization of the surface of cementum by plaque, once the junctional epithelium separates from the tooth. Scaled exposed root surfaces, whether of cementum or dentin, begin to show microradiopacity by four weeks and it is frequently found at eight weeks.[81] Both the serum in the inflammatory exudate and the saliva could be sources of this mineralization.[89] Armitage[90] suggested that if the fluoride were available for release, it may act as an inhibitor to adjacent cells, but that it is likely that fluoride is bound in ne crystals. Ratcliff[1] speculated that the hypermineralization made exposed cementum much like enamel, and so was a deterrent to connective tissue new attachment. It is possible for junctional epithelium to attach to hypermineralized root surface.[91]

Changes in the Physical State

Removal of the diseased root surface by root planing to produce a hard, smooth surface and to eliminate the necrotic surface layers of cementum has been universally recommended. The microhardness of cementum determined by diamond scratches has been scored 85 to 90, compared to 145 to 165 for dentin and 1600 to 1750 for enamel surface,[92] but there has been found no statistical difference in the hardness of exposed and unexposed cementum in periodontally-diseased teeth.[93-95]

The thickness of cervical cementum has been reported to be as little as 10 microns[48] to 16 microns.[96] Cementum normally increases in thickness with age,[48,97] but periodontally-diseased teeth do not show the same amount of apposition as normal teeth.[98,99]

The microtopography of normal cementum is finely serrated as mineral crystals project at the insertions of collagen fibrils.[62] Numerous small, knobby projections are seen as remnants of Sharpey's fiber insertions on the surfaces of extracted teeth.[100-105] Root surfaces exposed by periodontal disease were found significantly rougher than healthy areas.[106] The roots of diseased teeth have more resorptions than the healthy, but these defects are

5

primarily in the apical area.[107] At the cementoenamel junction there may be
a gap in the cementum 5 to 10% of the time.[65]

Studies with the diffusion of dyes and radioactive materials indicate
that there is a "circulation" of tissue fluids into cementum, but that it
may decrease in the cervical cementum in older teeth and in periodontally
involved human teeth.[108-115] Thus "vitality" remains a question of definition,
and the same fluid exchange which might contribute to healing might also allow
the penetration of bacterial toxins.

Histologic Changes

The cementum and sometimes the surface dentin in pathologically exposed
roots of teeth fixed in formalin, decalcified, and cut on a cryostat, present
fine granules and clumps of granules which are refractile and colored varying
shades of brown when viewed by transmitted light, are white with incident
light, and irreversibly cleared in alcohol.[116] They are not seen in ground
sections, except after decalcification.[117] On electron micrographs they
appear as vacuole-like formations, singly or in clusters.[118] These granules
may be related to the amount of calculus[119] or in some fashion to bacteria,
for pulpless teeth with open, infected chambers present granules in cementum
which was still attached to bone by the periodontal ligament.[120] Human teeth
removed because of unmanageable periodontosis-like bone destruction have none,
or very limited distribution, of these granules.[121]

Bacteria can penetrate into cementum in root caries lesions[122] and surfaces
exposed to plaque.[123] Even without bacterial invasion there is thought to be
an inhibitory factor in diseased roots, the quantity of which inhibitor may be
related to destructive effects and the resistance to destruction, and thus to
susceptibility to periodontal disease.[124] Some such inhibitory factor could
also prevent successful new attachment therapy.

Adsorption of Toxins

For many years it has been known that very toxic materials were to be
found in periodontal pockets,[125] and these may adsorb to diseased roots.
Bacterial cell wall muramic acid[126] and other antigens[127,128] are adsorbed on
diseased root surfaces. Periodontally affected human roots leach out a material
toxic to epithelial cells in culture.[129] A phenol-extracted material from
cementum of diseased roots was found toxic for fibroblasts in culture.[130]
Aleo, et al.,[131] demonstrated a reduced fibroblast adherence to periodontally

diseased root surfaces. Blocks of human diseased roots inhibit osteogenesis in an animal model,[132,133] and stimulate inflammation when implanted in human submucosal pouches.[134] Phenol[36,130,135,136] and acid[136] extracts of diseased root surface shavings have demonstrated the presence of bacterial lipopolysaccharide endotoxins.

Detoxification

Phenol extraction can increase the adherence of fibroblasts _in vitro_ to diseased root surfaces.[131] Hand scaling[36] and ultrasonic instrumentation[135] can markedly reduce the amount of extractable endotoxin from diseased tooth surfaces, while root planing can reduce the endotoxin to the level of healthy teeth[36,135] and restore fibroblast adherence _in vitro_.[131] Root planed blocks of diseased human roots seem to generate somewhat less inflammation than scaled blocks, when observed as submucosal implants.[134]

The depth of endotoxin absorption in diseased roots and the concentration level compatible with new attachment are not known. It may be possible to extract toxic bacterial materials with buffers of some ionic strength[137] or low pH-salt buffers[127,128] which are kinder to tissues than phenol and strong acids. Buffers and salts could be used after scaling, and thereby avoid deep and invasive root planing. Extracted human teeth with diseased roots treated with phosphate-buffered physiologic saline after scaling were used as controls for a series of experiments with biological agents, selected from a number of chemicals capable of dissociating or degrading endotoxins.[138] Sodium deoxycholate followed by human plasma fraction Cohn IV_1 produced a statistically significant increase in adherent fibroblasts, _in vitro_.[138] There remains much work to be done to perfect the biological preparation of diseased roots by chemicals and to test them in wound healing experiments for their ability to detoxify roots and abet new attachment therapy.

Rationale for Root Planing

Numerous opinions have been expressed advocating root planing. These have included the points of view that the root surface is pitted and a habitat for bacteria,[139] that the root surface is dead[140] or necrotic,[141] that the pericementum is necrotic,[142] or that remnant Sharpey's fiber ends are putrescent.[143] A fresh, clean surface has been recommended,[144,145] which some state should be only in the cementum[146,147] and others into the dentin.[148-150] Elimination of the biologically unacceptable surface cementum contaminated with substances that can elicit inflammation seems to be a current consensus.[151-153]

Root planing to a smooth surface in order to remove calculus will probably

remove most of the cementum.[34] The consensus of reports on the results of instruments suggests that curettes leave the smoothest root surface.[35,37,154-168] However, what feels like a smooth surface to the touch of a sharp explorer may be a grossly rough surface for the first adherent cells of a new attachment. Root planing will not prevent the later accumulation of plaque or calculus, which is related more to the patient's hygiene performance;[31,166,169,170] but it has been reported that rough tooth surfaces facilitate the retention of bacteria.[43,171]

As opposed to the point of view of smooth planed roots is the view that healing with new cementum occurs in nicks in the root[172] and that flat surfaces are inhibitory.[173] The recommendation for a microirregular surface[174] seems supported by only one abstract, a report of an experimental, intentional scarification of cementum after a surgical detachment.[175]

Demineralization

Nearly one hundred years ago "aromatic sulfuric acid" was used in deep pockets with claims of remarkable results of new attachment.[176] Claims of new attachment after use of hydrochloric acid[177] lactic acid,[178,179] salicyclic acid[180] and citric acid (used to "neutralize" antiformin)[181,182] have been made. Black[183] dismissed the use of acids as "radical" and pointed out that no proponents had demonstrated a connective tissue new attachment by histologic evidence.

Descriptions of histologic repair of surgically-created defects in animals have led to the selection of citric acid as a method for "conditioning" roots and promoting accelerated reattachment.[184-187] Acid etching of normal human cementum exposed the broad bundles and interlacing fibrils of collagen in the cementum matrix.[188] Citric acid applied to diseased root surfaces revealed no collagen fibers, unless the cementum was first removed by root planing.[189] The acid treatment also enlarges the dentinal tubules,[186-189] into which the healing connective tissue enters.[186,187] The report of Frank, et al.,[190] is used as a justification for artificial demineralization. In that study, electron micrographs depict scattered, fine inorganic crystals on the dentin surface among collagen fibers in an area of connective tissue new attachment, one month following a surgical procedure. What seems overlooked is that those investigators, in addition to suggesting that this was a crystal dissolution, also stated that the findings were compatible with beginning of mineralization,[190] or a precementum.

Decalcified normal roots implanted into subcutaneous tissues of rats did not seem to promote connective tissue attachment,[191] and when implanted into submucosa of humans led to resorption or exfoliation.[192] The replantation of endodontically-prepared monkey teeth which had been treated with acid caused exfoliation or accentuated resorption and ankylosis, compared to undemineralized controls.[193]

The citric acid conditioning of natural and artificial furcation defects in dogs has demonstrated some remarkable success in new attachment and closure of the defects.[194,195] The closure of deep pockets in humans has been reported.[196] Histologic study suggests this is mostly by a new, long attachment of junctional epithelium,[197] although some connective tissue adherence and a slight amount of new cementum at the apical extent of the pocket has been demonstrated.[198] In a comparison trial, citric acid conditioned teeth gained 2.1 mm clinical attachment and controls 1.5 mm, six months after modified Widman surgery.[199] Citric acid conditioning has not improved procedures for coverage of denuded roots in humans,[200,201] but has shown significant new connective tissue attachment in baboons.[202]

ZONE OF THE EPITHELIAL ATTACHMENT

The attachment of epithelium to the surface of the tooth provides a biological seal, which, with the attachment of connective tissue elements to the tooth, comprise the dentogingival junction.[203] The occluso-apical length of the junctional epithelium ranges from 0.1 mm to 2.7 mm in the periodontal pocket,[204-207] and it is quite variable. On extracted teeth it is seen as a "plaque free zone".[207-211] It is important to new attachment therapy to know that the junctional epithelium is continually renewed.[212] Although it has generally been thought that the strength of the dentogingival junction was due to the contribution of connective tissue, the epithelial attachment has surprising strength.[47,213,214] At the base of the pocket, bacteria are in contact with the junctional epithelium and "pioneer" organisms are found to have advanced into the interstices of deeper cells.[215] As a result, the junctional epithelium presents characteristics of epithelium over disturbed connective tissue.[216]

Probing the Epithelial Attachment

Most authors agree that the epithelial attachment should be removed to obtain a connective tissue new attachment.[1,207,217] Thin steel shims inserted in the gingival crevice appear to penetrate to the depth of the epithelial

9

attachment.[207] However, periodontal probes may wedge to a halt at variable positions related to the health of the tissues.[207,211,218-229] If pre-surgical "initial" therapy is performed to reduce gingival inflammation and dissection then carried out at the bottom of the probable pocket, the junctional epithelial cells may remain on the tooth side as depicted in Figure 2.

Operative dentistry procedures[230] and simple scaling[231] may tear or remove the epithelial attachment unintentionally, while intentional procedures to remove it may not completely eliminate it.[207,232,233] However, the presence of some patches of junctional epithelium may not prevent connective tissue new attachment more coronally.[234,235]

The Dental Cuticle

Between the junctional epithelium and the tooth surface there is a dental cuticle.[49,236-243] The dental cuticle may remain on exposed cementum.[30,81,237,244] The dental cuticle should be removed in attempts at connective tissue new attachment because new cementum may not deposit on it, according to Box[237] and Waerhaug.[207]

The exact nature of the dental cuticle is not known. Because it appeared hyaline in histological sections it was once thought to be keratin.[245] More recently it has been thought to arise as a gradual thickening and condensation of basement laminar material.[246] On the basis of stains it has been said to contain hemoglobin,[247] protein,[248-250] and carbohydrates, probably neutral mucopolysaccharides.[251,252] For others, there is thought to be carbohydrate which remains unidentified.[249,253,254] Sodium deoxycholate can remove epithelial cells _in vitro_, and leaves behind this cuticle and attachment substance.[255]

Dental cuticle was not seen under a new junctional epithelial attachment at two to three weeks after scaling,[44] but it has been seen at later intervals after reattachment,[256] on enamel and calculus after scaling,[28] four months after gingivectomy,[45] and 35 days to a year after flap operations.[50,257,258]

Forming A New Junctional Epithelium

The epithelial attachment is renewed by the coronal movement of cells,[259] and, after detachment, the artificially deepened crevice closes from the bottom, up, like closing a zipper.[260] With little to no inflammation present, the junctional epithelium has a low mitotic activity, but it rises dramatically

with inflammation.[232],[261] If some of the junctional epithelium is left on the tooth after a wound of minimal trauma, it participates only slightly in the epithelial regeneration, which is derived primarily from the oral epithelium at the gingival margin.[47],[262]

The Cementum Surface

Underneath the junctional epithelium, its attachment lamina, and the dental cuticle, the cementum surface appears to be varied.[68] Selvig[68] found it had a decreased density of the mineral crystals ranging from 0.2 to 18 microns deep, but generally to a depth of less than four microns in four of nine specimens. In other sections the mineral crystals were densely packed at the surface, while subjacent layers were almost devoid of crystals. The loss of mineral crystals revealed the crossbanding of the collagen, except in some regions where the surface decalcified zone failed to exhibit typical collagen structure.[68] It is most probable that even a light root planing in this zone which would scrape away the epithelial cells and their cuticle would also remove the thin altered surface. Would the exposed collagen fibers in the cementum which still retained their natural crossbanding unite with new collagen fibers of the healing wound? The first fine fibrils of collagen formed on dentin surfaces were generally oriented parallel to the root surface.[50],[187] Whether any "splicing" occurs has not been demonstrated.

ZONE OF THE CONNECTIVE TISSUE ATTACHMENT

The connective tissue attachment is where collagen fibers normally originate in the cementum and pass outward to insert in the gingiva, periosteum, alveolar bone proper, or across the interdental septum to adjacent teeth as transseptal fibers.[263] The distance from the crest of the alveolar bone to the epithelial attachment varies, but is about one millimeter on the average.[205],[206],[264]

In this zone, immediately apical to the epithelium, there is an area where the collagenous fibers are found in varying stages of degeneration in the presence of inflammation.[68],[263],[265-267] The width of this destruction ranges about 0.5 to 1 mm.[68],[265],[266] The fiber dissolution starts with decreased density of fibril packing and longitudinal splitting, progresses to loss of crossbanding, change of orientation, and then fibers appear to be cut off at the cementum surface; but they may retain their crossbanding under the surface.[68],[265] There is a demineralization of the surface of the cementum to a depth of about 0.2 microns in the area of complete fiber dissolution. In the area are found leukocytes, granular debris, and occasional microorganisms.[68] The fiber dissolution is thought to be enzymatic, as no

11

multinucleated cells are seen.[68,267]

The remainder of this zone is of densely packed collagenous fiber bundles and the finely fibrillar indifferent fibers,[68,268] cells and their processes, and precementum.[68] The precementum serves to maintain the attachment in health.[269-273] Sometimes part of the epithelial network of the former root sheath may be seen.[274] Resorptions of the root surface occur as a result of injury, necrosis, or perhaps inflammation, and are normally repaired by cementum to even out the root surface.[275-279] Sometimes resorption may go deep into the tooth and present an osseous metaplasia repair.[280]

Repair of the Connective Tissue Attachment

When an incision is made through the gingival crevice or gingival margin to the crest of the alveolar process, the collagen fibers in the zone of connective tissue attachment are severed. When a flap is raised and the root and bone exposed, the fibers are not readily apparent to the eye. Clinicians are repeatedly warned not to scrape them away.[1,281-283] These fibers have the potential to reattach to the fibers in the repositioned flap. If the root is undamaged, healing by scar does occur.[47,51,207,282,284-287] If the root in this zone is injured, even with a surgical detachment in normal gingiva, apical migration of the epithelium occurs.[50,185,186,207,286,238-294] Thus, careless instrumentation may result in an increased loss of connective tissue attachment.

Routine scaling may injure the connective tissue fibers as well as the epithelial attachment.[231] The cementoblasts in the area of the connective tissue attachment seem to be very sensitive to trauma, and they "disappear" for a short while after a wound.[261,295-299] The new cementoblasts may differentiate by some inductive property of the root surface acting on undifferentiated cells,[300] but studies seem to indicate a proliferation from the "native cells" on the cementum side of adjacent periodontal ligament.[287,296,297,301-303] There may also be a migration laterally from adjacent teeth along the transseptal fibers.[304] Obtaining a connective tissue reattachment to the root does not ensure bone regeneration.[294] The cells, which repopulate the wound, control the type of healing.[305] Periodontal ligament cells can produce new cementum and fibers on the root, but bone marrow cells produce resorption and ankylosis of the root.[296,304-308]

Cementogenesis

When new cementum forms following surgical detachment, it can form on "old" cementum[50,289,310] or dentin.[50,289,309-311] New cementum formed in surgical

12

detachment wounds often follows shallow surface resorption,[186,285,289,291,292] and is most often found at the apical extent of the wound.[50,186,285,289,309,310] The earliest material formed is a metachromatic band, probably of mucopolysaccharides,[50,312] which is later calcified and the collagen-polysaccharide matrix of cementum formed and calcified.[50,313] The dearth of collagen fibers in that first material would allow artifactitious splits in decalcified sections,[50] but it is not known if this makes the connective tissue reattachment "weaker", in vivo. The earliest collagen fibers of new cementum matrix parallel the root surface.[50,187,313]

There has been concern that connective tissue healing was impaired on non-vital teeth.[309,314] Root canal treatment can induce necrosis, resorption and repair of the cementum.[315] Following surgical detachment,[316,317] periapical curettage,[318] or replantation[319] normal healing was observed histologically on non-vital teeth; while case reports suggest good treatment results for combined endodontic-periodontic lesions.[320-323] Case reports claim acid treatment is effective in reattachment on non-vital teeth which do not respond to the usual therapy.[324]

THE PERIODONTAL SIDE

THE ZONE OF THE MARGINAL EPITHELIUM

With the objective of connective tissue new attachment therapy, the sulcular or pocket epithelium is removed.[1] Scaling with hand instruments and ultrasonic devices may injure the sulcular epithelium by tearing or even removing it, and with tears sometimes extending into the connective tissue.[231,325-327] With good hygienic measures this simple debridement has been demonstrated histologically to result in reformation of a normal epithelial attachment,[325,327,328] which may extend almost to the margin of the gingiva.[328]

Subgingival Curettage -- Histologic Studies

In subgingival curettage performed as a surgical procedure with the goal of connective tissue new attachment, one attempts, but does not always remove the pocket epithelium.[207,232,329-335] Remnants may be left at the gingival margin or near the epithelial attachment. The long epithelial rete extensions into the connective tissue may also be left,[232] but if thorough scaling and hygienic measures are used some time prior to curettage, these rete extensions will likely have disappeared.[328] Complete epithelium removal requires many strokes of the curette.[336-338]

Following curettage the basal germinative cells of the stratified squamous epithelium at the edge of the wound elongate and migrate as a sheet under the fibrin of the clot, and present evidence of phagocytosis.[339] Depending on the size of the wound and degree of inflammation, complete regeneration may occur in three to 18 days,[207,232,237,329,331,334,340-343] usually being complete at five to seven days. Excision of the marginal gingiva has been proposed with the rationale of delaying epithelial downgrowth into the wound, thus allowing more time for connective tissue healing, but no experimental evidence confirms that it is more effective.[336,344-349]

It is conceivable that connective tissue new attachment could result after a well-executed subgingival curettage. Waerhaug reported new cementum in four of 40 human teeth, but thought it was due to injury apical to the base of the pocket.[207] Box presented histology of one tooth which showed connective tissue against planed cementum, and an area of new cementum.[237,350] Schaffer and Zander[351] presented histologic material demonstrating new connective tissue and epithelial attachment in four cases, in a series where five of eight subjects had clinical pocket reduction after subgingival curettage. Cross[352] presented histologic sections of

14

three teeth in one human with epithelial and connective tissue new attachment. In a study with careful control of hygiene in two monkeys, a long new junctional epithelium was the predominant method of regeneration after root planing and curettage every three months. Occasional "windows" of resorption and connective tissue repair in the epithelium were seen at nine months.[235]

Subgingival Curettage -- Clinical Studies

An occasional case report claims considerable reduction of a pocket depth after treatment by subgingival curettage.[354] For six patients, one month after subgingival curettage, an average recession of 0.26 mm and 0.9 mm decreased pocket depth were noted.[355] In another practice, three months to two years after treatment of 20 patients, one to 7 mm gain of clinical attachments was recorded.[345] Significant improvement in 73 humans was recorded in a 36-month study.[356] Only five failures were reported in the periodontal maintenance of 103 patients over a six-year period.[20] For 157 patients, curettage resulted in a mean 3.0 mm attachment gain.[357] Another study, of four-month's duration, showed that subgingival curettage significantly reduced pocket depth and resulted in a gain of attachment for 40 subjects.[358] In an experiment on seven dogs treated with curettage six months after surgically creating pockets, 56.7% of the pockets were decreased in depth, with a mean overall gain of attachment of 0.83 mm, 10 months postoperative.[359]

Short-term surveillance of 44 patients treated with subgingival curettage by the Ramfjord group showed that 62% of the pockets initially deeper than 4 mm were improved, with a mean of 0.2 mm to 0.3 mm gain of attachment level.[360] At eight years, data for 78 patients in their long-term study were analyzed on the basis of initial pocket depth. For shallow (1-3 mm) pockets there was about one mm loss of attachment; but in the moderate (4-6 mm) and deep (7-12 mm) pockets there was a sustained gain of attachment and significant reduction of pocket depth.[361]

Chemicals in Curettage

Adjuncts to subgingival curettage have been used empirically until investigations or some years of experience demonstrated if any value existed in continued use. Calcium or sodium sulfide solutions or sodium sulfide and sodium carbonate pastes were once used to "dissolve" epithelium;[362-364] but several investigations showed that this very alkaline material was not a specific epithelial solvent, that pockets treated with it healed by epithelial proliferation to former level, and that there was

also connective tissue injury.[288,365,366] Sodium sulfide was found no more effective than sodium hydroxide.[365] Forty percent potassium hydroxide was once used, "neutralized" by hydrochloric acid.[41] Antiformin, a mixture of sodium hypochlorite, sodium carbonate, and sodium hydroxide, was also extremely alkaline, and was "neutralized" by applying citric acid.[181,182] Likewise, it was found not to dissolve all epithelium and to damage the connective tissue.[367,368] The use of highly alkaline agents was like an uncontrollable, chemical gingivectomy.[369]

Phenol, in strengths from 25% to 100%, has been used for an aid in removing epithelium, cuticle, and "infected granulation tissue."[234,370-373] Interestingly, both phenol and alkali can extract or degrade bacterial endotoxins; but phenol can cause damage to connective tissue[374] which, like strong alkalis, would impair new attachment.

The idea of complete removal of epithelium was questioned by Linghorne and O'Connell.[375,376] In a number of experiments where they interpreted the histology of healing wounds in dogs, they reported that there was a "creeping reattachment" of root resorption, cementum repair and bone regeneration which proliferated coronally at a slow rate, despite the use of procedures which did not attempt to remove pocket epithelium. These results could not be repeated by independent investigators.[382] However, one report, where the flap was reflected, the tooth side of the flap coated with gum copal and collodion varnish, autogenous bone from a nearby tooth socket implanted, and the flap closed, showed that connective tissue had formed a new attachment on the tooth, with pocket epithelium nearby in the flap instead of on the tooth surface.[376]

Excising the Pocket Epithelium

In variations, clinicians have curetted the epithelium before elevating a gingival or mucoperiosteal flap, have tried to curette the epithelium from the flap after elevation or have dissected the epithelium from the gingival flap by internal bevel incision. The ability of these variations to remove the pocket epithelium and epithelial attachment have not been evaluated to the same extent as has subgingival curettage. A "self-limiting epithelial scalpel" was found to reliably remove epithelium on 14% of proximal tissues and about 80% of labial or lingual surfaces.[?] Epithelial rete projections in the proximal pocket soft tissue walls between mesial and distal tooth surfaces often come close to touching each other, and so the epithelium can be eliminated completely only by removal of the papilla.[?]

16

The early postoperative histology of one study shows that epithelium removal by incision was almost perfect.[341] It is thought that epithelial removal by incision is less traumatic than curettage and permits better postoperative adaptation to the tooth.[380]

After dissection, the new epithelium migrates from the cut edge of the marginal epithelium under the clot, and any residual junctional epithelium does not appear to participate in the regeneration.[262] The new epithelial attachment has normal ultrastructural appearance on dentin and cementum.[19,18] The epithelial new attachment is reformed in about seven days,[214,299,340,341] at which time it has formed a strong new attachment.[214] In some areas a 40 nm to 120 nm thick space which may contain amorphous electron dense material is present at four months between epithelium and the root.[254]

THE ZONE OF THE GINGIVAL CONNECTIVE TISSUE

The residual gingival flap after incision (Figure 1) and the dissection and removal of the lining of the pocket (Figure 2) would ideally leave a consistent wound surface between the epithelium and the alveolar crest.[1] The idealized condition as carried out would appear as in Figure 3. Note that the gingival fibers and the vascular plexus approximately parallel the tooth surface. The thermal effects of ultrasonic curettage may cauterize this surface facing the tooth,[335,381-383] or it may be no different than after incision.[384] Thermal or chemical coagulation might impede healing.

Hygienic measures and scaling to resolve the gingival inflammation should reduce the possibility of residual ends of pocket epithelium rete in this zone. Those inadvertently left may not constitute a hazard.[385] Treatment prior to surgery provides for a regeneration of the free gingival and circular connective tissue fibers.[386] Scaling prior to surgery should also reduce hemorrhage, lessen the likelihood of excess granulation tissue in healing, lessen the chance of infection, promote quicker healing, make the tissue firmer and technically easier to incise, and reduce the time required at operation.[387-390] However, these views are not supported by direct evidence.

Gingival Flaps -- Histologic Studies

A blood clot of fibrin and red blood cells fills the space between the connective tissue and tooth, and is infiltrated with polymorphonuclear leukocytes to form a

17

"poly band."[214,299] Granulation tissue proliferates from the periodontal ligament and the flap.[214,299] The new fibroblasts may come from activation of local fibrocytes, dormant mesenchymal cells, or perhaps from cells of the blood stream.[391] They migrate into the wound on fibrin strands, followed by capillary buds.[392] Interdentally, granulation tissue may also proliferate from marrow spaces. The fibroblasts have a random orientation of polarity on the collagen fibers of the normal gingival connective tissue.[393] With the very close adaptation of a flap having fibers parallel to the tooth, and successful replacement of a thin clot by new fibrous tissue, it is most likely that the result would appear to be what has been called "collagen adhesion."[394] There is no evidence that cells of the gingivae have an osteogenic potential and the origin of cementoblasts is uncertain.[301] It has even been questioned whether gingival connective tissue is able to form anything more than "contact" to a dentin surface.[307] Formation of a "collagen adhesion" may prevent or retard the coronal proliferation of periodontal ligament cells which could secure the connective tissue attachment by new cementum. There is no information on the relative strengths of wounds healed by a new attachment with cementum, collagen adhesion, or a long junctional epithelium. It is reasoned that a connective tissue new attachment would give support to the tooth and that a long epithelial attachment could detach and reform a pocket.[395]

As was found with subgingival curettage, the predominant mode of healing by gingival flaps is a long junctional epithelium new attachment.[256,299,340,341,345,396-398] Only slight amounts of connective tissue attachment and cementum occur, usually in grooves cut into teeth at the base of the wound.[130,258,398]

Gingival Flaps -- Clinical Studies

Case reports attest to the clinical success of periodontal disease management by gingival flap procedures.[16,344,399-402] Clinical studies have followed the results of gingival surgery over short and long-term periods. On 12 patients a postoperative reduction of pocket depth and gingival fluid flow was reported up to four weeks.[403] Significant pocket reduction and a slight loss of attachment four months after surgery was reported in a study of 40 patients.[154] Over a one-year period, 15 patients were found to have reduction of pocket depth and over one mm gain of attachment.[404] Another report of 153 pockets in 95 humans demonstrated an average of 3 mm regeneration in 127 pockets, and only seven pockets showed no improvement.[405] In nine patients, over 2 mm new attachment was found at 12 months,

with a mean of 1.9 mm at three years and 1.5 mm persisting at five years.[406-408] Gingival flaps, even without an attempt to return to original preoperative level at the completion of surgery, were found to have a 0.9 mm gain of attachment at six months.[409]

After eight years of clinical trials, the Ramfjord group has found that shallow pockets (1-3 mm) had about one mm loss of attachment, as did those treated by subgingival curettage; but in the moderate (4-6 mm) and deep (7-12 mm) pockets there was a sustained gain of attachment and a significant reduction of pocket depth.[361]

Gingival Grafts

Gingival grafts constitute a use of the zone of connective tissue. In the coverage of denuded roots, no case report or study has documented a new attachment with a free gingival graft.[410-413] Pedicle grafts moved laterally to cover denuded roots are claimed to be successful in almost every instance.[414-418] A clinical study of pedicle grafts in 15 humans found a significant gain in root coverage.[419] In another study, a significant 3 mm mean gain in attachment was found at six months postoperative in 14 procedures,[420] which was sustained at a three-year followup.[421] Twenty-eight days after creating a denuded root surface surgically, pedicle grafts were performed on 11 dogs in order to study the histology for up to 180 days postoperative. The average result was a 2 mm attachment of epithelium and a 2.1 mm new attachment of connective tissue.[422] Two human teeth treated with mucoperiosteal pedicles were seen to have varying amounts of epithelium and connective tissue new attachment.[423] In another report of two cases, the one treated with a split thickness pedicle healed with long junctional epithelium, while one treated with full thickness pedicle had connective tissue and epithelium new attachment.[424] Full thickness pedicle grafts have been claimed successful in cases for management of two-walled bony defects,[425] furcation problems,[426] and adjacent to an edentulous ridge.[427]

The Periosteum

The success of connective tissue new attachment with mucoperiosteal pedicle grafts moved laterally, as contrasted with replaced gingival flaps, is thought to be associated with the periosteum and its participation in the formation of a union.[428]

Small fragments of bone often remain on the periosteum elevated from alveolar process.[429] New bone formed on the top and bottom of bone spicules which remained attached to periosteum elevated from alveolar process, but little new bone formed where separation had been by elevation through the cambium layer of the periosteum.[430] Combination osteoperiosteal flaps produced more new bone when transposed as extraoral pedicle grafts into muscle than did periosteal flaps.[431] Osteomucoperiosteal pedicle flaps were not successful in forming new attachment to denuded roots in miniature swine.[432] Where these flaps were placed over recipient area bone, a new periosteum formed between recipient and donor bone.[432]

With the idea that the cambium layer is thin and dormant in the adult, and may be injured when elevated from bone, histologic examination was made of periosteum traumatized by needles jabbed through the gingiva. In three weeks the inflammatory response was followed by proliferation and osteogenesis on the surface of the bone.[433] Case reports of pedicle grafts with "stimulated" periosteum have demonstrated the technique but have not documented the results.[434,435]

Free grafts of periosteum wrapped around roots during bone implant procedures have not produced a connective tissue new attachment.[308,436] A descriptive histologic study of free periosteal grafts packed interdentally reported new cementum and connective tissue attachment in the notches marking the depth of the pockets.[437] On the basis of case reports, attachment success for free grafts of osteoperiosteal tissues from the tibia has been claimed.[438] There has been little work done with pedicle grafts of periosteum.[439]

Coronal Positioning

Beginning with a gingival flap, then elevating a mucoperiosteal flap, the clinician has the options of replacing the flap or moving it laterally, apically, or coronally. Coronal positioning could bring periosteum onto the root surface with prospects for new attachment and perhaps even increased bone support. Success has been claimed in case reports of coronally positioned mucoperiosteal flaps where the procedure has been started with a gingivectomy[440] as well as the internal bevel incision.[441,442] Successful cases of coronally positioned pedicle grafts for coverage of denuded root surfaces of individual teeth have been reported.[443-445] One report has demonstrated 1.43 mm of new attachment 12 months after surgery.[446] Citric acid treatment of diseased roots in conjunction with coronally positioned pedicles has not shown an

advantage in two human clinical studies.[200,201] Coronally placed pedicles were
found to have no significantly different results from laterally placed pedicles,
except that donor teeth for lateral pedicles had slight recession.[447]

Pedicles of mucoperiosteal flaps, elevated after gingivectomy, with their
bases at the cuspids and the free ends at midline have been proposed for new
attachment,[448] but others could not obtain the same results.[449] Limiting such
"bridge" pedicles to single teeth, a case report has claimed new attachment,
sustained at 18-month follow-up.[450]

Adaptation

An important requirement of new attachment therapy is close adaptation of the
gingival tissues to the tooth at the completion of the surgical procedure.[451]
When the gingiva is separated by a great amount of clot or debris the healing is
delayed.[262,299] It is a generally held point of view that intimate contact favors
a shorter epithelial attachment.[290,452,453] Close adaptation is thought to result
in early formation of an epithelial seal, protecting the connective tissue from
bacterial irritants which could interfere with cementogenesis.[299,454]

Cell Adherence

An area of study with possibly great implications for new attachment therapy
is the application of fibronectin in promoting cell adherence to the root surface.
Fibronectin is a cell surface glycoprotein which is found on human gingival
fibroblasts and provides adherence to culture vessels.[455] The attachment of
gingival fibroblasts to collagen is via the fibronectin, and the attachment
probably accounts for the axial orientation of cells to fibers and perhaps even
to orientation of new fibrils into bundles.[456] The attachment of fibroblasts to
diseased root surfaces could theoretically be improved by demineralization,
thereby exposing collagen for a collagen-fibronectin-cell attachment.[457] Experiments
with non-diseased root surfaces showed that demineralization could double the
number of attached fibroblasts in vitro.[457] The amount of fibronectin in implanted
demineralized collagenous bone matrix was found to increase prior to cell
proliferation, differentiation, and calcification.[458] Fibronectin has an affinity
for fibrinogen, too, and this may be involved in the locomotion of cells[459]
which migrate into blood clots. There are many exciting new studies which can
apply this biological process to new attachment therapy.

THE ZONE OF THE BONY DEFECT

It is thought by some that only when the pocket is within the alveolar process that a new attachment can occur.[460] Pockets apical to the alveolar crest are classified according to the number of bony walls.[461] The number of bony walls serves as a rational basis for therapy with the three-wall bony defect thought to have the most favorable chance for new attachment therapy.[461]

Decortication

In a study of human cadavers, most bony walls observed were of cancellous bone, but sometimes a more sclerotic wall was noted.[461] Cells of osteogenic potential are found in periosteum, endosteum and marrow.[462] It is thought that perforation of sclerotic bony walls should be made to open into marrow spaces to establish a good blood clot, ensure its rapid revascularization, and facilitate ingress of osteogenic marrow cells.[463,464] The major repair tissue is from the bone marrow,[465] and bone resorption would have to open a sclerotic wall before the marrow cells could enter the wound. The arguments for perforation are rational, but there is no experimental evidence of its value.

Transseptal Fibers

Transseptal fibers follow the bony wall surface in interdental angular bony defects.[466,467] It has been recommended that these fibers be removed, for if left they may be a barrier to proliferation from the marrow.[461,466,468] Again, this seems rational, but it is done without the support of direct investigation. The granulation tissue which fills the remainder of the defect could have formed from perivascular cells of the gingiva, periosteum, bone marrow, or periodontal ligament. It contains chronic inflammatory cells and the elements of repair; but it is also riddled with elongated epithelial rete,[469] therefore it is removed.

The number of bony walls is related to the amount of periodontal ligament that is exposed. A three-wall bony defect might have a V or U-shaped exposure of periodontal ligament.[454] If the usual maximum proliferation outward of the periodontal ligament is about 2 mm, then a complete new attachment is possible in narrow, or in shallow, bony defects.[454]

Curettage -- Clinical Studies

Case reports have indicated a profound response of pocket reduction and apparent bone "fill" on X-ray after treatment of acute periodontal abscesses.[470-478] One

point of view holds that the healing is due to a favorable biological state of the root surface following rapid destruction, as opposed to all the changes which occur in chronic pockets with a longer history.[476] Another view is that there is a radiolucent bony matrix in these lesions, which can be rapidly remineralized as healing occurs.[470,473] There is really very little evidence in this matter.

Other case reports have claimed "reattachment" and bone fill of chronic defects treated with curettage, as shown by X-ray.[479-487] However, X-rays may not accurately depict bony defects or the form of the final results.[395,488-491] Photography of re-entry operations has demonstrated bone fill.[482]

Clinical studies with careful documentation have demonstrated gain of attachment upon probing and bone fill in 3-wall, 2-wall, and combination bony defects treated by curettage.[492-498]

Curettage -- Histologic Studies

Histologic evaluation of a bony defect in a monkey treated by flap and curettage, and having a 4 mm gain of attachment on probing and bone fill on X-ray, revealed at one year that bone had filled the defect, but that junctional epithelium had migrated apical to the level of planing on the root surface.[499] In two other animals at one-year postoperative, very little connective tissue new attachment had formed at the base of the defect, and again junctional epithelium was seen on the root in areas of bone fill.[500] A complete histologic new attachment was observed in 56.3% of 20 monkey bony defects treated by curettage, but about 10% of all specimens had alternating zones of connective tissue attachment and epithelium on the root surface.[501]

One human case treated by curettage, which had a shallow crevice on probing and where the X-ray showed bone fill at six months postoperative, disclosed on histology new bone and cementum and a disorganized periodontal ligament.[502] New bone and cementum were seen in only seven of 21 human block sections in another practice, however two of 21 with no bone were removed at only 14 days postoperative.[503] In another study, no bone fill was found in three human blocks removed one year after curettage, and only one specimen had 0.1 mm new cementum, the junctional epithelium having migrated apical to the alveolar crest in all three.[504]

It seems that following curettage, even with probing, X-ray, and re-entry evidence of bone fill, that there may not be a concomitant connective tissue new

attachment. Histology must be done to confirm that epithelium has not migrated to the depth of the former pocket.

Retarding Epithelization

In experiments to determine what could happen if that "bugaboo", epithelium, were excluded, teeth with bony defects were amputated, the defects curetted, and the roots buried by mobilizing flaps.[397,505,506] Histology at six to eight months usually showed connective tissue new attachments, although the fibers of the new periodontal ligament were parallel to the roots.

Since the epithelium of free gingival grafts usually completely dies and sloughs, it was conceived that bony defects could be covered with free grafts, delaying the migration of epithelium and allowing more time for connective tissue new attachment.[507] Complete regeneration as determined clinically was found in 60% of 88 human bony defects using this technique, as compared to 40% treated with conventional flaps.[507] In a histologic study on 20 monkeys, reepithelization was determined to have been retarded by 10 to 12 days by free grafts, and they generally had shorter epithelial attachments.[508]

Implants of Oral Bone -- Clinical Studies

The healing tissue which fills a bony defect could form a dense, fibrous scar, or new bone. It is thought that the matrix of bone conatains some inducing or organizing substance and so implanted bone could by its resorption release the stimulus for the formation of new bone in the recipient site.[509] A number of cases have been reported of successful fill and attachment after the use of autogenous bone implant material obtained from oral donor sites.[464,510-522]

Clinical studies have quantitated the bone fill seen on X-rays after autogenous bone implants.[523,524] An increase of over 3 mm mean fill was reported in 44 patients.[525] With conventional flap approach, 40% of bony defects obtained complete regeneration in another study.[507] In eight carefully documented cases, autogenous transplants from oral sites did not appear to markedly influence the outcome, compared to curettage, in 2 and 3-wall defects.[494] In a study of 91 humans, the same conclusion was made.[493] In 25 cases, involving one-wall and furca defects as well as 2 and 3-wall, about 2.9 mm mean bone fill was found with autogenous oral bone implants.[525] In a larger group of subjects, it was found that, regardless of the form of the bony defect, there was a statistically significant increase of fill with implants compared to curettage.[527] The 2-wall were better when implanted but not the one-wall in another study.[528]

Implants of Oral Bone -- Histologic Studies

Histologic study has demonstrated that the implantation of autogenous bone chips from an oral site caused bone to regenerate further from the bony defect walls after surgical detachment, than when no implant was used.[300] Chips smaller than one mm seem to be most effective.[529] In epithelized, surgically-created defects the result of autogenous bone implants was "reattachment" in dogs.[270] Another study in dogs found histologic evidence of new bone, liagment and cementum in defects implanted, but long epithelial attachment in those not implanted.[530] New cementum was seen in grooves at the bottom of defects implanted with fresh autogenous bone from an oral site in another study in dogs.[531] In 12 monkeys, new cementum, ligament and bone formed in implanted and non-implanted defects.[501,508] In another study on monkeys both implanted and non-implanted bony defects eventually filled with bone.[532]

New cementum formed on old cementum and on dentin, along with new bone formation in one human case at eight months after implanting autogenous cortical and cancellous bone chips.[533] However, the ligament fibers were parallel to the tooth.[533] Another single case report, at 57 months after implanting autogenous bone chips, showed bone fill, but the ligament had considerable artifactual tearing away from the tooth in root planed areas.[534] One block section from a clinical study showed new bone, ligament and cementum on a root planed surface which was not denuded of cementum.[525] Another instance showed new cementum on old cementum which had been planed, 28 months postoperatively.[535] Histologic study of three human teeth at six to 13 weeks after implanting autogenous oral bone showed new bone, a cellular ligament with some functional organization, and new cementum formed on old cementum and on dentin.[536] Autogenous bone from oral sites implanted in six human cases when observed at one year showed new bone and cementum in three root planed specimens, but, without root planing, only one of three specimens had new cementum.[504] There was junctional epithelium on the root apical to the regenerated bone in two of three specimens not root planed, and one of three which were root planed.[504] A specimen examined 28 weeks after autogenous bone implant was found to have bone regeneration, but with epithelium on the root nearly to the base of the original defect. New cementum was limited to the base of the wound.[537]

Implants of Autogenous Red Marrow and Bone -- Clinical Studies

Case reports have been presented of the successful use of autogenous cancellous bone and marrow from extraoral sites with the thought that red marrow was the most

osteogenic implant.[538-543] However, there have also been reports of fresh
marrow causing root resorptions of clinical significance.[544-547] When using
fresh, refrigerated, and frozen red marrow, a clinical study of 52 humans has
reported dramatic bone fill in all classes of bony defects.[548] For others,
comparison studies of autogenous red marrow with autogenous cancellous bone
from oral sites have demonstrated no significant clinical difference in bony
defects.[494,526] It is clinically significant, however, that autogenous red
marrow has resulted in new bone coronal to the alveolar crest, in "overfilled"
implant use.[2,548]

Implants of Autogenous Red Marrow and Bone -- Histologic Studies

Histologic investigation of autogenous red marrow in bony defects in monkeys
showed that fresh and frozen marrow from the hip both produced comparable amounts
of new bone. The frozen marrow was much better, though, for new cementum and
ligament regeneration free of ankylosis.[501] Red marrow implants have resulted
in more bone regeneration than non-implanted sites in dogs, but resorption and
ankylosis were more common.[303] Fluoride treatment of the donor tissue and recipient
site has not affected cementum deposition with red marrow implants in bony defects
in dogs.[549] When composite implants of oral site cancellous bone chips on the
root surface and fresh red marrow in the remainder of 2-wall bony defects of
monkeys were compared to implants of fresh marrow only, the composite grafts were
found to have less resorption and ankylosis. While they often had new cementum
on the roots, there was also epithelium apical to the alveolar crest.[550]

New cementum was seen on one extracted tooth from a human clinical trial of
marrow implants in 52 patients.[548] New bone, cementum, and a functionally-
oriented ligament were observed on four human teeth in a study of 13 humans with
documented pocket reduction and bone fill.[2] This new cementum formed upon
dentin or old cementum, and upon resorbed and unresorbed root surfaces.[2] The
implanted marrow was replaced by new connective tissue at 14 days, and osteogenesis
was seen at 24 days, in a series of 15 autogenous bone implants in humans.[503] New
cementum formed at the level of bone regeneration in narrow or shallow defects;
in the deep and wide defects the new bone was more coronal to the new cementum.[503]
New cementum formed over old cementum or denuded dentin.[503] After nine months the
implanted bone had been incorporated into new bone or was replaced.[503]

Implants of Allogeneic Marrow and Bone -- Clinical Studies

Allogeneic bone, preserved in several ways, has been used with success according to case reports.[436,510,551-554] A clinical study of 20 patients treated with frozen allogeneic bone showed at five to 26 months a mean fill of over 3 mm in all sites.[555] This was comparable to results with autogenous bone and marrow, including "crestal" apposition.[555] In another report of eight patients, about 1.5 mm mean bone fill was seen at re-entry one year postoperative.[556] Freeze-dried allogeneic bone implants in 350 patients were reported to have complete or greater than 50% fill in 60% of the implant sites re-entered.[557,558] Another report of nine patients also found greater than 50% fill in 60% of sites implanted with freeze-dried bone, but this was not different than the results in control defects treated by curettage.[559] Decalcified, freeze-dried allogeneic bone implants were found in three cases to have 4 mm to 10 mm new bone produced in defects re-entered at 32 to 104 weeks.[560]

Implants of Allogeneic Marrow and Bone -- Histologic Studies

Histologic observation of fresh allogeneic red marrow implants in surgical defects in 12 dogs showed new bone and cementum at four and eight weeks after implantation.[561] Resorption and repair at the base of bony defects were seen in four dogs, whether the sites received merthiolate-preserved allogeneic bone chips or not.[562] In six dogs, new bone and cementum attachment were observed in histologic sections of merthiolate-preserved allogeneic bone implanted into surgically-created bony defects.[436] One human case of an implant from a bone bank into an intrabony pocket, but which became infected and was removed, showed new bone forming on and between the implanted chips at $3\frac{1}{2}$ months.[563] New bone and connective tissue but no cementum were seen on histology of a 2-month postoperative specimen implanted with a frozen allograft of red marrow.[564] In a companion report, new cementum was seen on old cementum at the base of the bony defect of a one-month specimen.[555] Fresh and frozen allogeneic red marrow implants in four monkeys were found both to result in new bone, cementum and functionally-oriented ligament fibers.[565] New cementum and connective tissue attachment were seen in five of six human frozen allogeneic red marrow implants at six months or one year, but junctional epithelium was also seen on the tooth apical to the regenerated bone in four of the six specimens.[504] Freeze-dried bone implants were seen on histology to have formed new bone at six weeks postoperative, but the results in four dogs were not different than defects treated by curettage.[566] Decalcified, allogeneic bone matrix was replaced by new

bone in 27 dogs, but at 13 weeks there was ankylosis of new bone to the roots.[567] In a human case, a biopsy at 17 weeks showed new bone forming on decalcified freeze-dried bone, but no root was available to determine if there was cementogenesis.[560]

Implants from Another Species

Case reports of xenografts, or implants of non-human bone, have claimed good results in a number of instances.[510,538,568-572] Clinical studies have shown some pocket reduction, attachment gain and bone fill,[492,573,574] but in comparison to curettage there was essentially no difference.[492]

Histologic examination showed that bone powder from sheep implanted in six dogs resulted in new bone, ligament and cementum after surgical detachment.[575] Boiled cow bone powder in dogs appeared to hasten reattachment in surgically-created defects.[576] A human block section removed 15 years after the implant of boiled cow bone into an 8 mm bony defect showed new cementum and connective tissue on an old, irregular cementum surface presumed to be the former exposed root.[577] Commercially-produced cow bone was found to be not completely replaced after six months in surgically created defects in six dogs.[578] Decalcified, despeciated cow bone was found to be incorporated in host bone faster than undemineralized implants when placed under periosteum on alveolar process of dogs.[579] Anorganic bone has been found to be quite inert.[580,581] Defatted, deproteinated ox bone was found to be no different than oral autogenous bone chips in 12 monkeys.[582]

Implants of Non-Bony Tissues

Implants of collagen have not shown significant differences from controls in regeneration.[583-586] Using dentin and cementum chips from human teeth as implants in surgically-created defects in six dogs, epithelium was found on the tooth apical to the new bone in two specimens.[587] The implantation of cementum and dentin shavings into surgical defects of animals has resulted in osteoid formation on the smaller chips and new cementum,[588,589] but at four months some areas of ankylosis had occurred.[589] Cartilage allografts have been claimed to result in new attachment in human cases,[590,591] and the histology of cartilage implants showed they were replaced by bone in four monkeys.[592] However, a comparison in dogs showed no difference in repair whether implanted with cartilage or not.[562] Implanted human sclera has been reported to reduce pockets in human cases.[593,594] Histologic study of sclera implants in human bony defects shows they are incorporated into host fibrous connective

28

tissue but are not replaced by bone,[595],[596] and with no cementogenesis at one year.[596]

Implants of Alloplastic Materials

Plaster of Paris implants into surgically created bony defects in 10 dogs were resorbed by six weeks, but showed no more bone than unimplanted defects.[597] No bone was found at six months in 10 defects in five humans when plaster was mixed and inserted in bony defects as a paste;[598] but when implanted as set plaster pellets 79% of the defects in 35 cases demonstrated "fill" on X-rays.[599] Tricalcium phosphate ceramic materials implanted into surgically-created bony defects in dogs were slowly resorbed by multinucleated giant cells, which may have retarded its replacement with bone, but defects were repaired at 22 to 24 weeks.[600-602] In six humans, one ceramic implant was exfoliated at three weeks, but in the other five bone fill was seen on X-ray at two to 16 months.[603] Compared to allogeneic bone implants, ceramic was found to be no different.[556] Zirconium oxide ceramic cloth implanted in surgical defects had bone form on its surface in monkeys and dogs.[604] In five human bony defects, four pyrolytic graphite implants were exfoliated and there was no regeneration in the other.[605] No new attachment has been found with these materials.

THE INTERRADICULAR ZONE

Implants of Autogenous Oral Bone

The furcae within multi-rooted teeth have always been a difficult management problem in periodontics. The implantation of autogenous bone from oral donor sites into surgically-created furcal defects in dogs resulted in a variable amount of bone regeneration and new cementum in the root nicks marking the base of the defects, while unimplanted controls had bone loss and epithelium grew down into the furcae.[531] Oral cancellous bone implants in 17 monkeys developed total closure of the furcae in 44% of sites, with new cementum in 34%. Controls with no implants had only four of 27 sites (15%) with total closure and new cementum.[606] Devitalized oral cancellous bone resulted in no apparent difference than fresh oral cancellous bone in 19 monkeys.[607] Oral cancellous bone implanted in six dogs did not result in complete regeneration. Epithelium migrated into the furcae of 33 out of 36 sites, despite the flaps having been sutured up on the crowns of the teeth.[608] No difference was found between use of oral cancellous bone and defatted, deproteinated ox bone. Few sites developed new cementum on the "roof" of the furca.[582]

29

Implants of Autogenous Marrow and Bone

Autogenous red marrow implanted into the furcae of two human molars was found to have resulted in complete fill on re-entry at five to six months.[539] Fresh red marrow implants in monkeys formed total closure in 12 of 24 sites (50%) and new cementum in 17%, while frozen red marrow had complete closure in 30% and new cementum in 28%. However, 75% of the fresh marrow sites had resorption and ankylosis.[606]

Demineralization

Significantly more bone fill was found in citric acid-treated furcae in eight dogs, compared to the controls, for 13 of 23 sites had complete connective tissue new attachment with cementum and eight of 23 sites had connective tissue attachment but no new cementum, at six weeks.[194] In another study, 27 of 35 sites had complete cementum regeneration and five partial regeneration in furcae of dogs at 12 weeks.[609] Comparing coronal flap positioning with closure to the preoperative level in conjunction with citric acid treatment of furcae in dogs showed no difference in a third study.[610] The overall results of the three studies was a complete closure in 71% of sites.[610] A preliminary report has indicated new attachment in natural defects as well as surgical defects in dogs.[195] Comparable investigations in humans have not been reported.

As was found with treatment by curettage, bony defects treated with implants of various materials may have probing, X-ray, and re-entry evidence of bone fill, and still may not have a connective tissue new attachment. Only histologic evidence can confirm that epithelium has not migrated apically to its former depth. It is generally thought that bone implants are not needed in "narrow" defects, but there is no study which has established "narrow" limits. Implants have not really demonstrated an inductive effect, but the results with many cases suggest that some particulate implants may be conductive. That is, they may facilitate the migration of osteogenic tissue into the defect. In a similar fashion, do implants of small bone chips provide space for coronal migration of periodontal ligament cells? What is the limit of the migration, and how predictable is it? These questions have not been answered by investigative evidence. Implants of bone do facilitate the regeneration of host bone in sites with low potential for innate regeneration, such as one-wall bony defects, supracrestal and furcae.[611,612] However, there is still no conclusive evidence that bone implants increase the predictability of connective tissue new attachment.[613,614]

THE KEY TO SUCCESS

It is a prevalent point of view, eloquently stated by Zander, et al.,[615] that
surgical pocket elimination is an essential part of therapy; that to maintain
hygiene and the integrity of the attachment there must be re-established "normal"
sulcular dimensions. Pocket elimination surgery does not necessarily improve
plaque control,[616] however. Another view is that it really may be the therapist's
choice of the procedures to use in new attachment therapy; as long as good oral
hygiene and maintenance care are ensured, teeth can be maintained in health and
function even with a long junctional epithelium, or collagen adhesion, and
without new bone fill.[617-629] The question is, can a dentition survive in that
status?

Patients who receive routine periodontal maintenance every three months do
not exhibit a dramatic or prolonged improvement in the absence of personal oral
hygiene.[621] Personal oral hygiene can rapidly reverse gingival inflammation in
persons with gingivitis,[622] and significantly alter the flora of plaque.[623]
Those persons with periodontal pockets need both frequent professional care and
personal oral hygiene. Subgingival scaling can have a dramatic effect on the
flora of pockets.[24,39] Scaling and optimal plaque control can resolve inflammation
and reduce pocket depth.[624-628] Scaling, root planing and oral hygiene can
cause significant gains in clinical attachment as determined by periodontal
probing.[627,629-631] Scaling and hygiene can even perform as well on attachment
gain as subgingival curettage or other surgical procedures.[632-635]

Inflammation in the healing wound is a primary factor in determining the
resultant clinical attachment. Epithelium will most likely extend to the depth
of the inflammation.[234,530] Patients with poor personal hygiene performance
before new attachment therapy were found to not have a significant gain of
attachment clinically.[636] Those persons with poor hygiene postoperatively were
found to have a loss of attachment.[496,637,638] Even with careful experimental
control of hygiene, the junctional epithelium may migrate to the depth of root
planing, but the health of the junctional epithelium and connective tissue in
the new dentogingival junction will be resistant to probing.[499,500] With control
of hygiene and the prevention of postoperative inflammation, significant bone
regeneration occurs.[495,496,498,639,640] Even in the difficult to manage furcation,
postoperative hygiene can result in significant bone regeneration.[641,642]

Long-term case histories and clinical studies of patients treated for periodontal
disease attest to the importance of professional maintenance and personal hygiene

31

by the patient.[152,361,408,635,643-649] According to Feinstein,[650] clinicians constantly perform "experiments", the experimental material being a person, prepared by nature, who has much to say about who the investigator will be, and the time, place, and the goals of the experiment. The clinician observes data such as signs and symptoms of illnes, demographic, morphologic, and microbiologic evidence. His purpose is to repeat a success of the past with well established procedures. The enumeration of case successes is as scientific as the titration with a biuret or weighing with an analytical balance. Thus these long-term case histories support the value of periodontal therapy. A deepened sulcus or a long epithelial attachment can be considered an acceptable result as long as there is no observation of data which show disease recurrence.

One bit of evidence that the clinician cannot readily gather is the chemical state of the exposed root surface. The laboratory experiments in this area have not determined all the deleterious effects of root exposure nor the most beneficial methods with which to revitalize that surface. It is hoped that patients will not have to shed too much blood in clinical "experiments" before the answers are provided.[']

It may be that, before long, chemicals or biologic agents will be used as adjuncts to the steel of curettes and scalpels. An antibacterial agent to complete the "toilet of the cavity" after scraping away plaque and calculus would probably help prevent infection. An agent that would dissociate and bind up endotoxin, or a buffer solution that would elut adsorbed toxins and antigens, appear to be sound, innovative and rational. Application of surface binding agents may improve the "plating efficiency" of connective tissue cell adherence, and perhaps prevent the apical migration of epithelium. In these experimental developments, it seems well to remember, "First, do no harm," and to avoid harsh, caustic chemicals.

The current status of new attachment therapy, which seems to be supported by sound prior research, is that clinicians who employ it accomplish a new dentogingival junction of a long epithelial attachment, backed up by healthy collagenous connective tissue, which is functional and maintainable for a long time. The key to success is the attention to hygienic measures.

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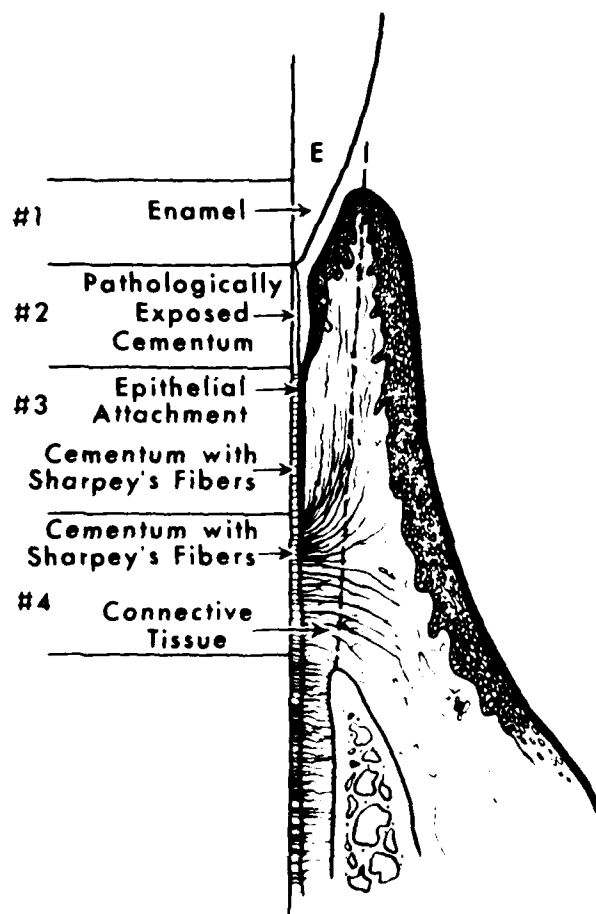650. Feinstein, A. R.: Clinical Judgement. Baltimore, Williams and Wilkins, 1967.

Fig. 1. Dotted line represents cut of internal gingival incision leaving relatively constant connective tissue gingival wound surface between epithelium and alveolar process.
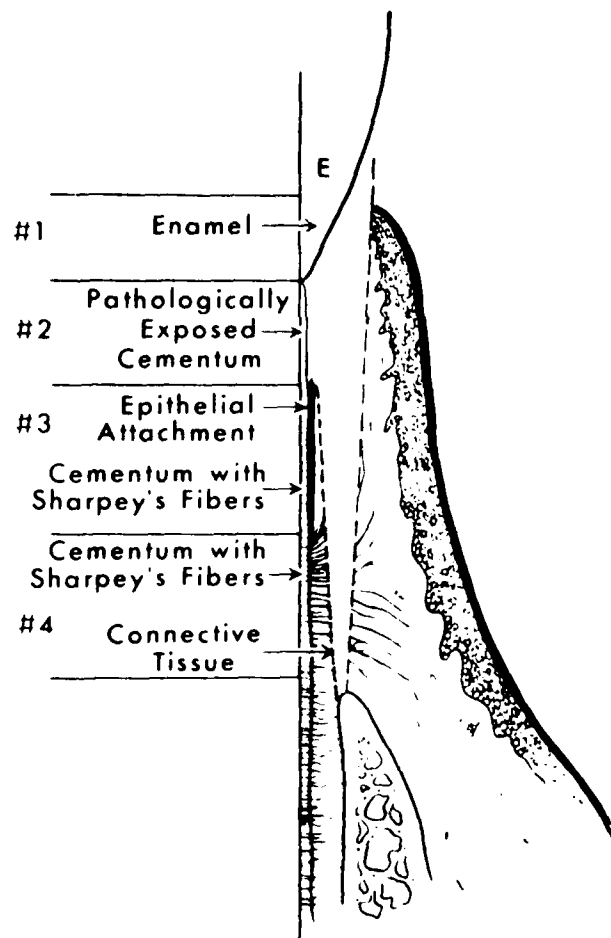
Fig. 2. After removal of a wedge of tissue note that tissue remnants remain in areas of the epithelial attachment and the connective tissues of zones 3 and 4.

Figure 3. Histologic section of tooth and gingiva, immediately after internal
bevel incision and removal of pocket lining to the depth of probing. (Rhesus
monkey, H and E stain, original magnification X10).

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Dental surgical procedures have developed over the years based on strong clinical impression and empirical practices. Important advances have been made in the field of clinical trials both in measurement of conditions and in analysis of clinical data. Many articles in the past have dwelled upon methods of handling soft tissues and bony defects, while recently there has been interest in the treatment of the diseased root surface. This report is a review of the literature in 1980, and highlights the biological nature of the healing dentogingival junction.
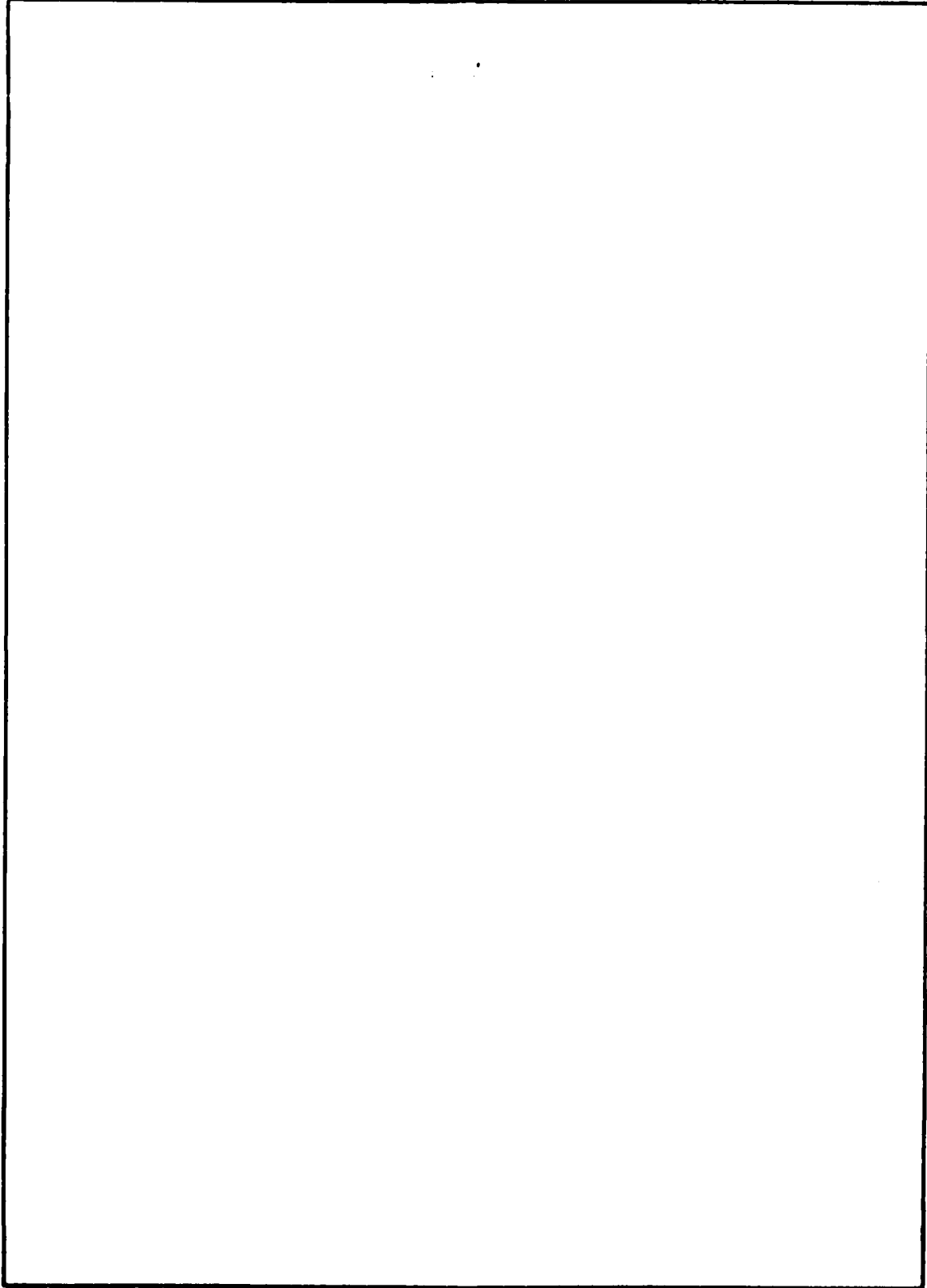
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