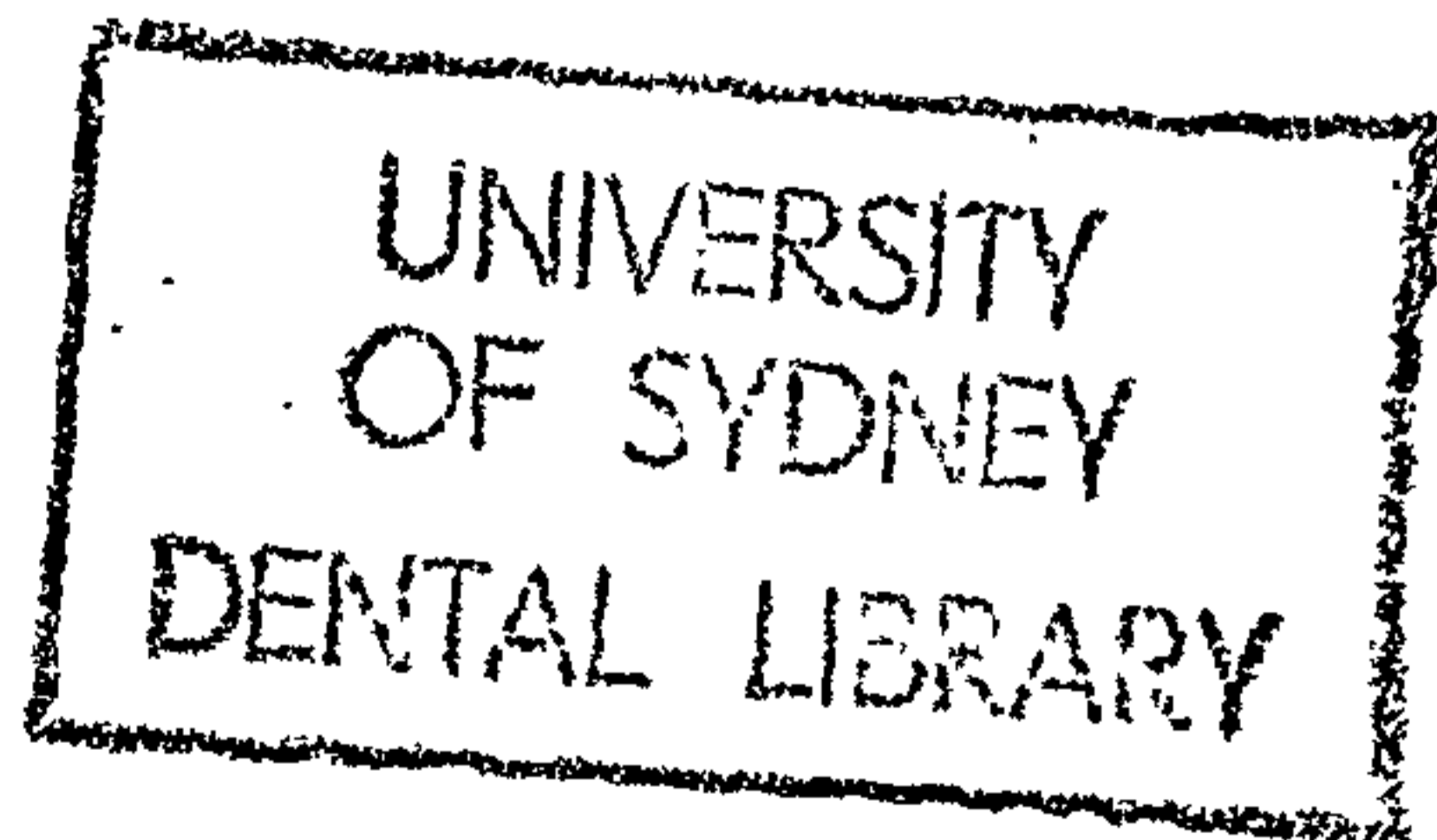


SCANNING ELECTRON MICROSCOPY OF ROOT SURFACES  
FOLLOWING PERIODONTAL INSTRUMENTATION

A.T. MCHUGH, B.D.S.

*A.T. McHugh*

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Department of Preventive Dentistry  
Faculty of Dentistry,  
University of Sydney.

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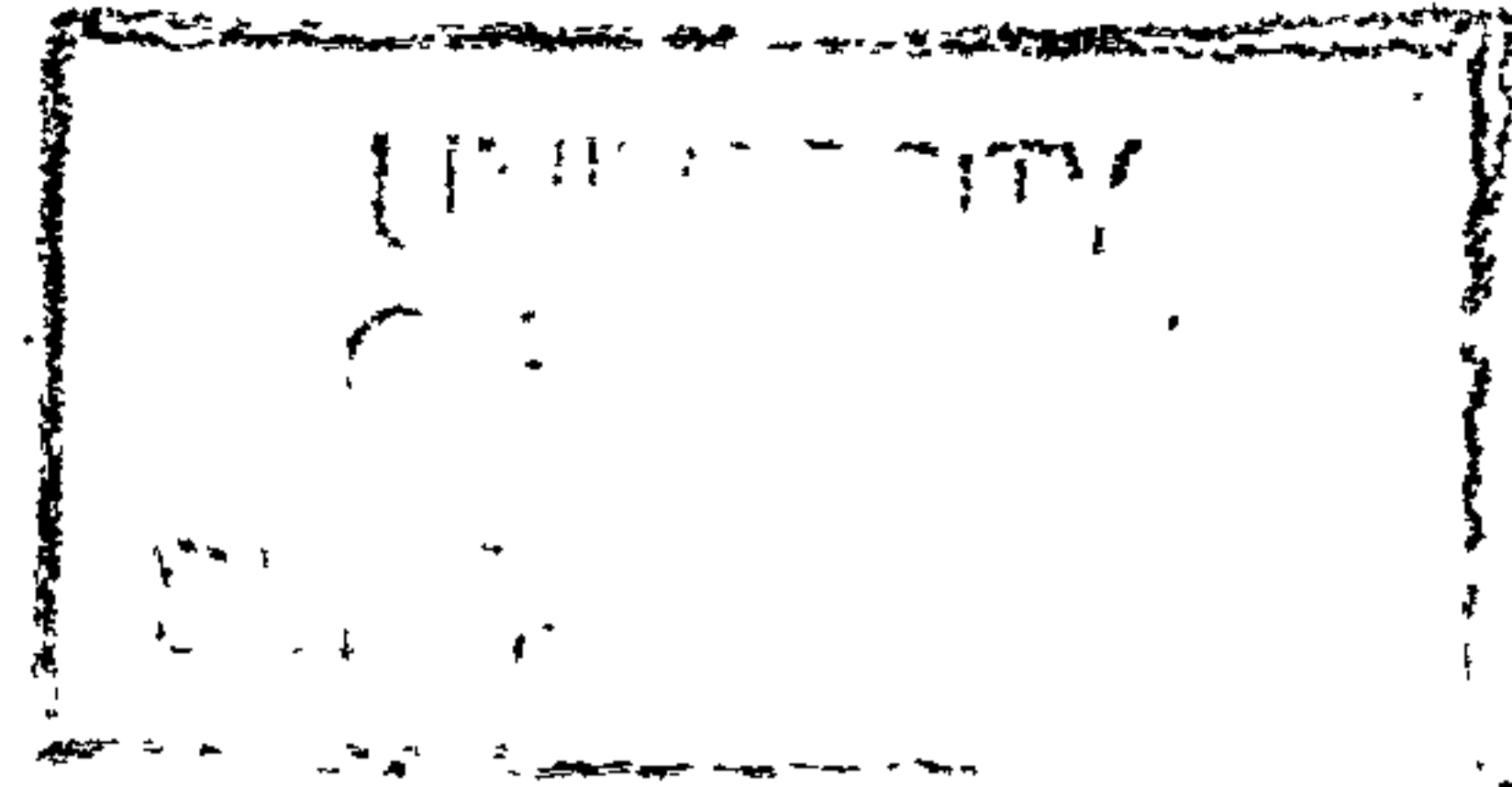
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INTRODUCTION

Scaling and root planing are considered important procedures in periodontal therapy.\* However, the problem of how best to treat the root surface to control periodontal disease and to potentiate new attachment, still remains. Three aspects have been referred to by various authors\*\* as being desirable objectives for root surface preparation. These are:

- (1) total removal of uncalcified and calcified deposits;
- (2) removal of pathologically affected cementum; and
- (3) production of a smooth, hard surface.

Root surface features, where preparation is made for adaptation of either a periodontal flap, or free gingival graft, may well differ from those considered desirable supragingivally.

Study of the root surface following instrumentation is important for two main reasons. Firstly, the formation and retention of plaque and calculus, and subsequent ease of removal of these acquired layers, may be related to surface features<sup>18,94</sup>. Secondly, such knowledge would assist the clinician to utilize biological factors to enhance repair of the tissues involved in new attachment<sup>236</sup>.

---

\* 9, 11, 15, 20, 25, 26, 66, 67, 100, 153, 175, 199, 240.

\*\* 16, 22, 23, 83, 101, 109, 179, 202, 246, 248.

Recent investigators evaluating root surface texture following instrumentation have used either the profilometer<sup>20, 66, 100, 192</sup>, the scanning electron microscope<sup>39, 93, 163, 271, 275</sup>, or a combination of the two<sup>147, 262</sup>.

The profilometer gives a numerical assessment of the average height of irregularities in surface texture<sup>66, 263</sup>. This method, however, is most suited to measuring planar surfaces, or surfaces having a regular curvature, such as a ball bearing; not irregular tooth surfaces<sup>263</sup>. The profilometer does not distinguish the changes produced by instrumentation from normal root surface features such as cemental undulations<sup>93</sup>. Without the use of higher magnifications, as obtained using the scanning electron microscope, to reveal the actual surface characteristics, profilometer roughness values may be misinterpreted<sup>163</sup>.

The scanning electron microscope is ideally suited to the direct examination of tooth surfaces<sup>18</sup>. It has a greater depth of focus than the transmission electron microscope, or the light microscope<sup>18</sup>, and can resolve about 150 Angstroms<sup>271</sup>. All parts of a rough surface can therefore be in focus. It is also possible to easily examine a wide range of magnifications, thus allowing rapid assessment of the fine detail of an area of interest<sup>80</sup>.

The aims of this study were:

- (1) to review the literature concerning healthy and diseased root surfaces; their histology and

pathology, and subsequent surface changes following periodontal instrumentation; and

- (2) to examine root surface changes following various scaling and planing procedures, using scanning electron microscopy.

Instruments used included hand curettes and scalers, and three commercially available ultrasonic dental scaling units, each with differing tip motion as claimed by the manufacturers: the Cavitron\* - elliptical motion<sup>6</sup>; the Amdent\*\* - linear motion<sup>219</sup>; and the Odontoson\*\*\* - rotary motion.<sup>56</sup>

Three test surfaces were evaluated:

- (1) root surfaces coronal to the epithelial attachment, ie, supra-attachment root surfaces;
- (2) root surfaces apical to the epithelial attachment, ie, infra-attachment root surfaces; and
- (3) a homogeneous model test surface of polished acrylic.

---

\* Dentsply Cavitron  
ultrasonic instrument,  
Amalgamated Dental, London, England.

\*\* Amdent 6 ultrasonic instrument,  
Siemens, Bensheim West Germany

\*\*\* Odontoson ultrasonic instrument,  
Goof, Herlev. Denmark.

PART 1

REVIEW OF LITERATURE

SECTION ONECEMENTUM

Cementum has been defined as "the thin calcified layer which covers the roots of teeth. It is a specialized calcified tissue of mesodermal origin; a modified type of bone covering the anatomic root of the teeth"<sup>169</sup>.

On a dry weight basis, cementum from fully formed permanent teeth contains about 45% to 50% inorganic substances, and 50% to 55% organic material and water<sup>7</sup>.

The inorganic portion consists mainly of calcium and phosphate in the form of hydroxyapatite,  $\text{Ca}_{10}(\text{PO}_4)_6(\text{OH})_2$ . Numerous trace elements are found in cementum in varying amounts. It is of interest that cementum has the highest fluoride content of all the mineralized tissues<sup>7</sup>.

The organic portion of cementum consists primarily of collagen and protein polysaccharides<sup>7</sup>.

The main function of cementum is to provide a structure for the attachment of collagen fibres that bind the tooth to surrounding tissues. Cementum serves as the major reparative material for root surfaces, as well as being the tissue that makes functional adaptation of teeth possible<sup>7</sup>.

Root cementum has been divided into two, and possibly three types on the basis of morphologic and physico-chemical differences; namely, acellular cementum, cellular cementum, and intermediate cementum.

It has recently been suggested that the conventional classification of cementum into cellular and acellular

types is inadequate in terms of development and structure. Osborn and Ten Cate<sup>162</sup> state that it may be more useful to think in terms of "predominant Sharpey fibre cementum" and "partial Sharpey fibre cementum". The first formed cementum is "predominant Sharpey fibre" type and does not contain cells. The later formed cementum is "partial Sharpey fibre" type and, although it usually contains cells, it can sometimes be acellular.

#### Acellular Cementum

Morphologically, acellular cementum has been seen in human teeth primarily at the coronal portion of the root. Its thickness varies from 20 to 50 microns (u) near the cervix, to 150 to 200 microns (u) near the apex<sup>130</sup>.

At the ultrastructural level, Selvig<sup>212</sup> describes acellular cementum as a finely lamellated tissue characterized by numerous incremental lines running parallel to the root surface. The cemental surface appears irregular, with collagen fibrils of the periodontal ligament inserting into cemental projections. In some areas, acellular cementum incorporates fibrils which are arranged irregularly or parallel to the root surface and, thus are not part of the tooth attachment apparatus. Furthermore, the cemental surface facing the periodontal ligament appears more mineralized than cementum near the dentinal interface. The crystals at the periodontal surface are smaller, thereby supporting the concept of continuous cementum apposition<sup>212</sup>.

Acellular cementum contains mostly radially oriented fibrils (attachment fibrils), thus being particularly well suited to serve as an anchoring medium for the periodontal attachment apparatus. This may represent a significant observation for the clinician, since acellular cementum is the root structure most frequently removed in treatment<sup>235</sup>.

Formation of acellular cementum seems slower than cellular cementum<sup>166, 212</sup>; although cemental apposition usually occurs throughout life<sup>278</sup>. The degree of apposition is probably in the range of a few hundred micra during a lifetime, which suggests that the embedded fibrils may have a slow turnover rate. This observation is of particular clinical interest to the periodontist, since Selvig<sup>212</sup> suggests that the turnover of these fibrils may be by molecular rearrangement rather than new fibril formation. If this mechanism is operative, it may be of significance in the "collagen reattachment potential" at the root surface<sup>120</sup>.

Finally, lining the surface, there is a layer of precementum (cementoid), which separates surface cells from cementum. This layer has been stated to vary in width between 0.25 and 5 microns ( $\mu$ ), and contains densely packed collagen fibrils<sup>43</sup>. Precementum, which is also seen along cellular cementum, has been considered a major factor in limiting cementum resorption.

Apposition and resorption of acellular cementum is usually seen in association with cell types which have been traditionally associated with this activity; namely cementoblasts and cementoclasts<sup>235</sup>. Some researchers<sup>118, 123, 124, 166, 224</sup> however, have indicated that the epithelial root sheath cells apparently secrete the initial acellular cementum.

### Cellular Cementum

Cellular cementum differs from acellular cementum in its location, (it is usually found in the more apical portion of the root), and by its cellular content. The thickness of cellular cementum varies from one to several millimeters (mm); its thickness increases with age<sup>130</sup>.

At an ultrastructural level, cellular cementum contains more irregularly arranged collagen fibrils than are seen in acellular cementum. According to Selvig<sup>212</sup>, most of these fibrils are not directly engaged in attachment. Many calcified fibres are also found to be orientated parallel to the root surface.

Under normal conditions, growth of cementum is a rhythmic process, and as a new layer of precementum (cementoid) is formed, the old one calcifies<sup>7</sup>.

Cementum which has had to be deposited rapidly, consists of wider lamellae, and contains many more trapped cells in the matrix. Cementum which has been elaborated slowly may have few, or no cells in its matrix<sup>170</sup>. Deposition of cellular cementum has been found to be more rapid than acellular cementum. In

fact, the highest rate of continuous cemental apposition may take place at the apex<sup>278</sup>.

In addition to surface apposition and resorption seen along the root surface, Jande and Belanger<sup>90</sup> also reported on cementolysis. This is resorption of pericellular cementum which resembles similar pericellular apposition and resorption patterns observed in bone<sup>10, 258</sup>.

A thin layer of precementum can usually be observed on the cemental surface. This precementum is lined by cementoblasts. Connective tissue fibres from the periodontal ligament pass between the cementoblasts into the cementum<sup>7</sup>.

#### Intermediate Cementum

Intermediate cementum is an ill-defined zone of the cementodentinal junction which contains cellular remnants of Hertwig's epithelial root sheaths embedded in calcified ground substance<sup>154</sup>. These cellular remnants may play a function in the formation of cementum<sup>28, 118, 154</sup>. This layer is predominantly seen in the apical two thirds of roots of molars and premolars, and is only rarely observed in incisors or deciduous teeth<sup>154</sup>.

Scanning Electron Microscope Appearance of Normal  
Cemental Surface

In extracted teeth which are otherwise untreated, the actual cementum is only rarely exposed to view. We generally find that it is obscured by a large amount of debris from the periodontal membrane. (Refer Plates 1 and 2)



PLATE 1 (100x)

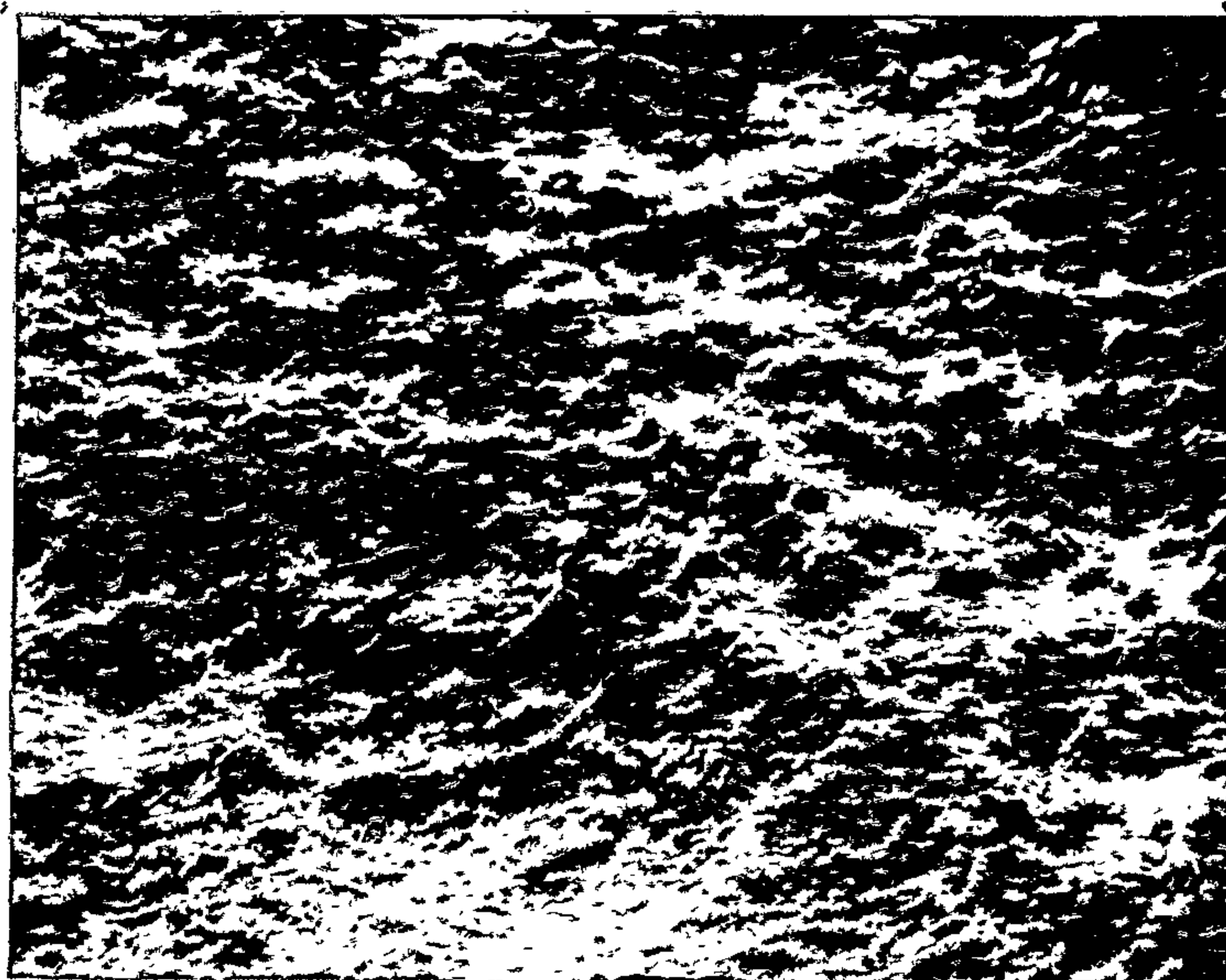


PLATE 2 (1000x)

It is possible to find small areas where the root surface is free from overlying material and we can observe a pattern of ruptured Sharpey's fibres (Refer Plate 3) and, in some places, randomly orientated fibrils lying parallel to the surface. (Refer Plate 4)

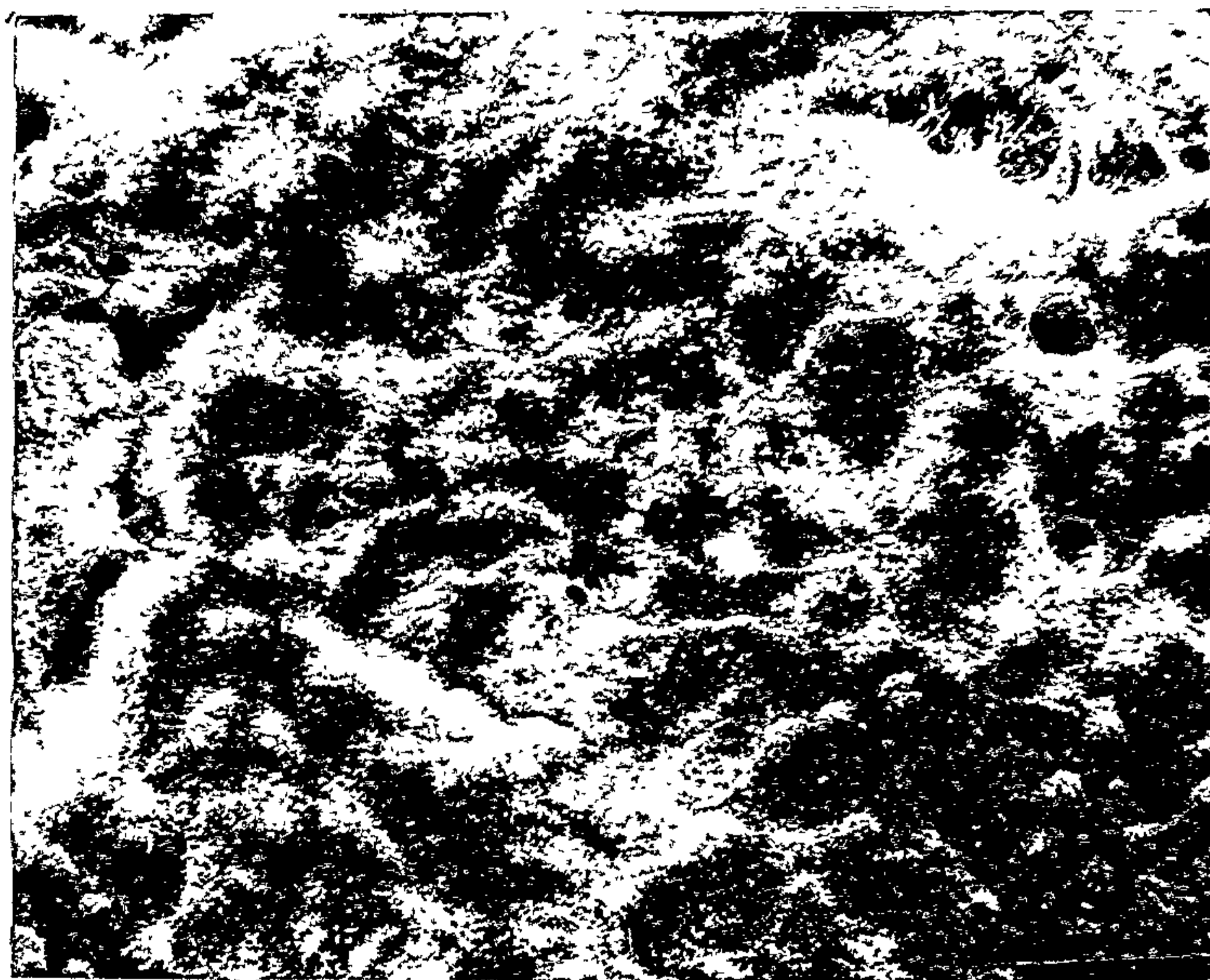


PLATE 3 (1000x)

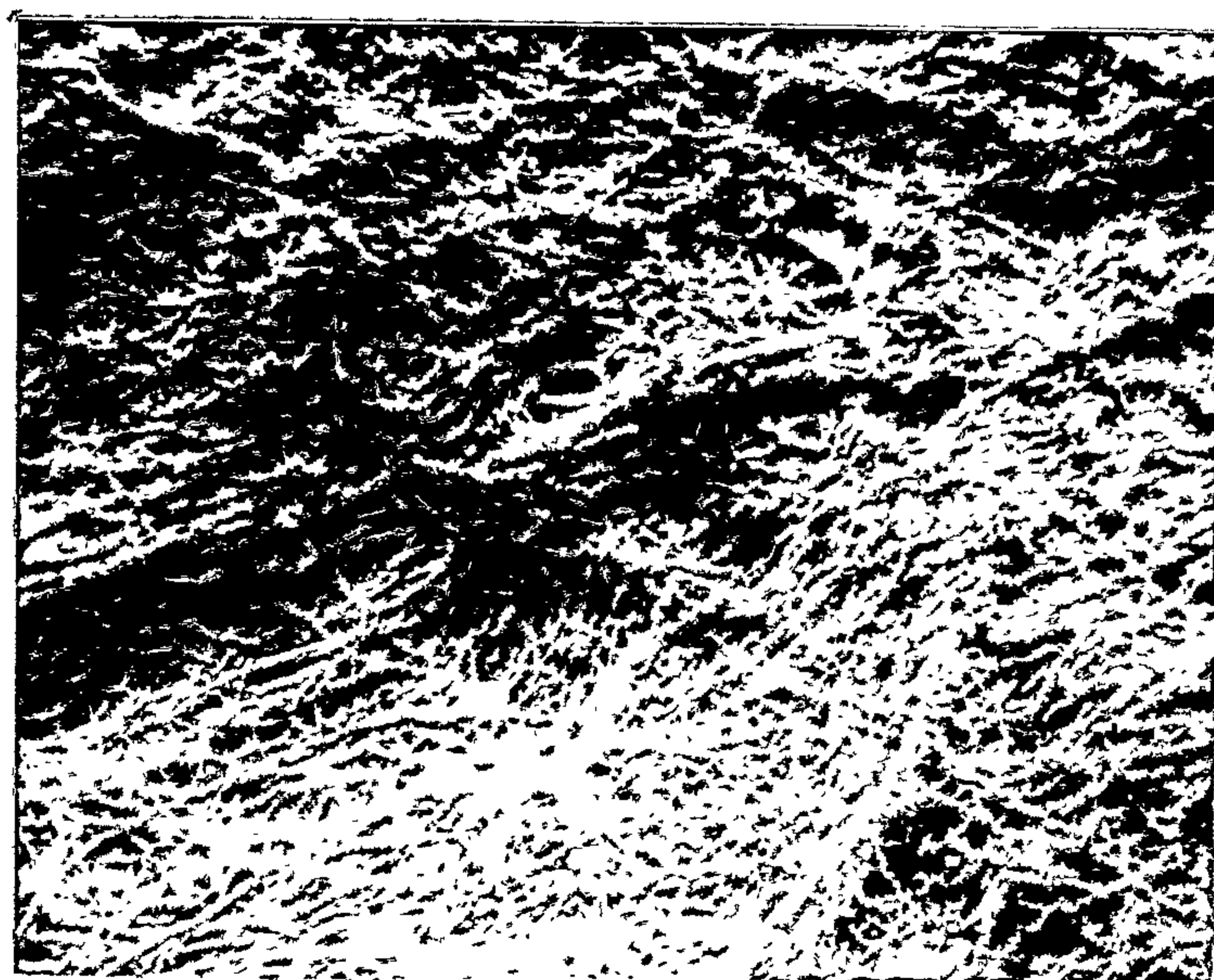


PLATE 4 (5000x)

Many other fine features of the cemental surface such as cementocyte lacunae, resorptive lacunae, lateral canal openings, and apical foramen morphology have been identified by Landay and coworkers<sup>113</sup>.

SECTION TWOCEMENTAL CHANGES IN PERIODONTAL DISEASEThe Role of Cementum in Periodontal Disease

Gottlieb, as early as 1922<sup>57</sup>, proposed the interaction between periodontal inflammation and cemental response as being a key factor in the pathogenesis of periodontal disease.

Since cementum is a relatively static tissue when compared to the biologic turnover capabilities of surrounding tissues, any change in its structure or chemical make-up could have long term effects; the ultimate effects depending upon host response<sup>2</sup>.

The cemental changes due to exposure to the oral environment, interfere with healing following periodontal therapy, and make the new attachment of connective tissue to the root surface very unpredictable. This fact has been a continuous subject of discussion for some fifty or more years\*.

The role of cementum in periodontal disease is still unresolved despite extensive clinical and experimental study. In particular, the question remains as to whether cementum has any significance in periodontitis other than merely as a vehicle for plaque and calculus attachment. Recent evidence<sup>1, 2</sup> indicates that cementum associated with periodontal disease undergoes morphological and biochemical changes that may contribute to the perpetuation of periodontitis.

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\* 12, 45, 57, 58, 60, 81, 121, 146, 161, 167, 178,  
185, 224, 235, 242.

Three important questions should be considered<sup>185</sup>:

- (1) What is the nature of the root changes?
- (2) Do these cemental changes merely represent an unimportant result of periodontal breakdown, or rather act as a stimulus to perpetuate periodontal disease?
- (3) Is the cementum able to cause cytotoxic changes on the surrounding gingiva directly, or does it also act via humoral mediators of inflammation?

Changes affecting cementum may be divided into those involving pathologically exposed cementum, ie. supra-attachment cementum, and those involving non-exposed cementum beneath or below the epithelial attachment, ie, infra-attachment cementum.

#### Changes in Supra-Attachment Cementum

Changes in exposed cementum and the underlying dentine, associated with periodontal disease, have been reported in a number of studies. These changes include: softer root surface<sup>180, 181</sup>; removal of collagen<sup>213, 255</sup>; diminution in the organic portion<sup>245</sup>; dissolution of the dentinocemental junction<sup>255</sup>; cemental splitting<sup>82</sup>; thinner layers<sup>86</sup>; more resorption<sup>79, 210</sup>; demineralization<sup>213</sup>; hypermineralization<sup>211, 214, 245</sup>; and bacterial penetration<sup>44</sup>.

In a scanning electron microscope study of the root surface, Young<sup>276</sup> demonstrated either unmineralized Sharpey's fibres, or completely static mineralized insertions. He suggested that the presence of mineralized fibres running parallel to the root surface

may create difficulty in gaining new attachment at these sites.

Landay<sup>114</sup> showed how areas of cementum exposed in periodontal disease, may show different changes depending on their location. At the base of the pocket, the most recently exposed cementum showed a partial filling in of the spaces between projections. Cementum which had undergone longer exposure showed complete covering of the normal projections with what appeared to be a flat sheet of calculus. There were no holes or spaces where Sharpey's fibres had once been. Wilkinson and Maybury<sup>271</sup>, while studying the results of planing, described a similar layer of calculus.

Plate 5 shows a supra-attachment root surface where spaces between Sharpey's fibre projections have been partially or completely filled in by a mineralized layer.

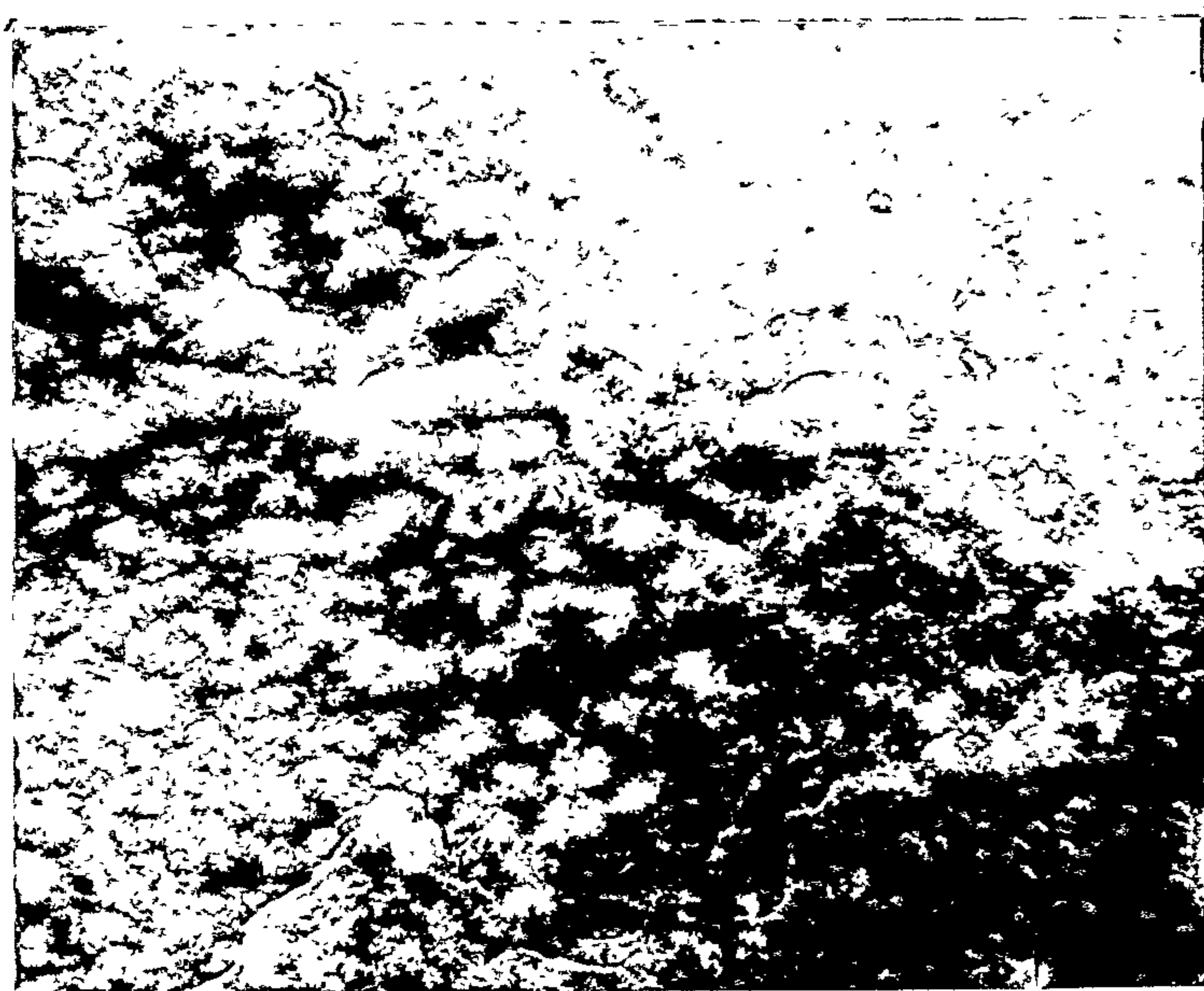


PLATE 5 (1000x)

Jones<sup>94</sup> described the same surface projections as did Landay<sup>114</sup>. She also observed an exposed cementum surface which was covered by thin mineralized layers. These were sometimes continuous with calculus (Refer Plate 6), and thought to be mineralized pellicle. She thought, however, that it was possible that mineralized cuticle contributed to the layer.

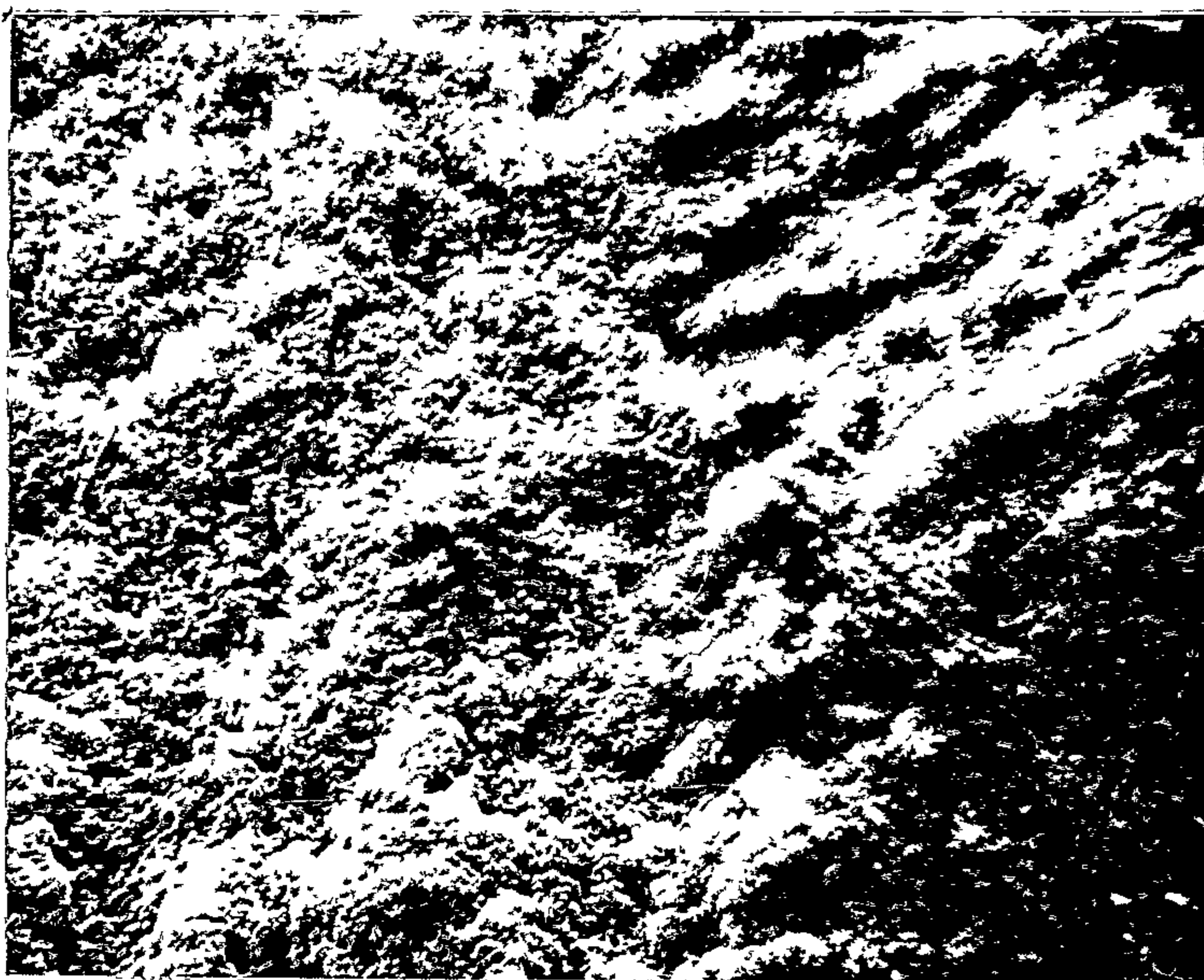


PLATE 6 (1000x)

Cytotoxic Capacity of Diseased Cementum

Robinson<sup>185</sup> has summarized several hypotheses regarding the cytotoxic capacity of diseased cementum.

These relate to the:

- (1) direct toxic effect by means of bacterial products incorporated within diseased roots;
- (2) indirect effect of incorporated bacterial products by means of initiating a cytotoxic immune response;

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- (3) morphological and biochemical changes in the cementum, interfering with the repair of normal connective tissue attachment; and
- (4) biochemical changes in the collagenous component of cementum initiating an autoimmune response.

The research of Aleo and co-workers<sup>1</sup> has stimulated the concept that the incorporation of bacterial products within cementum, as well as cemental structural changes, may contribute to the pathogenesis of periodontitis. Aleo and co-workers<sup>1</sup> extracted endotoxin (lipopolysaccharide), a component of the cell wall of Gram-negative bacteria<sup>104</sup>, from exposed roots with 45% phenol in water. They used the limulus lysate test, as performed by Rojas-Corona and colleagues<sup>187</sup>, to determine the presence of endotoxin. These toxins were then introduced into a tissue culture of mouse fibroblasts. They found a decreased cell growth, and an increase in non-viable cells as the concentration of endotoxin increased. Controls of non-exposed roots produced no response to the limulus lysate assay, and no response in tissue culture. The authors concluded that their data has shown that a substance, toxic in tissue culture of mouse fibroblasts could be extracted from exposed cementum. This toxic substance had the characteristics of endotoxin.

The possible role of endotoxin in the aetiology and progression of periodontal disease is well established<sup>225</sup>. Other investigators have implicated endotoxin in bone inhibition<sup>158</sup>, bone resorption<sup>77</sup>, and collagenase

production by endotoxin-activated macrophages<sup>77, 267</sup>. Additionally, endotoxin has been demonstrated to reduce the rate of oxygen metabolism of cells and tissues that make up the periodontium, that is, bone cells and fibroblasts of the periodontal ligament<sup>184</sup>.

Endotoxin, it appears, either functions in a multipotential fashion, or its actions are the sequelae of its initial inflammatory response<sup>2</sup>.

The results of Aleo and co-workers<sup>1, 2</sup> show that extracted cementum-bound endotoxin is capable of both cell death and decreased cell proliferation; the results depending upon the concentration of endotoxin used. Neiders and Weiss<sup>156</sup>, on the other hand, have demonstrated that endotoxins from two different organisms enhanced the detachment of two different cell types from glass, and that the enhanced detachment was not demonstrably dependent on the toxicity of the endotoxin. Their studies, conducted in the absence of total complement activity, show that endotoxin, by itself, in sublethal doses, is locally disruptive and, or destructive to cell attachment.

Whether the phenol extract in Aleo's experiments<sup>1, 2</sup> contains heat stable toxic substances other than endotoxin has not been determined. Nor has the nature of the endotoxin binding been established. It is very possible that in spite of the careful cleaning of the root surfaces, they were merely dealing with dental plaque material which was trapped in the surface imperfections of diseased cementum. Acquired pellicle

may also contain such material. It is, however, possible that cementum free of acquired accretions, could perpetuate periodontitis by structural changes which would not allow normal attachment of connective tissue fibres to occur, and by bacterial products within the cementum matrix interfering with the normal repair mechanisms<sup>185</sup>.

At the present time, there is no conclusive clinical evidence that directly correlates cemental changes with the progression of periodontitis.

Hatfield and Baumhanners<sup>76</sup> described cytotoxic results on epithelium cultured in vitro with periodontally diseased roots. Root surfaces from unexposed, impacted third molar roots produced no change in a similar culture.

Additionally, Morris<sup>151</sup> implanted portions of human dental roots with bone subcutaneously in the dorsum of rats. Morris interpreted his findings as indicating that diseased roots inhibit bone growth, whereas healthy roots do not affect bone growth.

The results of both these groups of investigators are difficult to relate to clinical periodontitis. A more acceptable system would have to include methods that can detect changes that occur within cementum, and see if these changes reflect the degree or rate of the pathogenesis of periodontitis.

Wicken and Knox<sup>269</sup> reported on lipoteichoic acids, a class of bacterial antigen contained on the cell wall of almost all Gram positive bacteria. The presence of

extracellular lipoteichoic acid has a number of implications in regard to the pathogenic potential of Gram positive bacteria<sup>105</sup>. With respect to the tooth surface, an important property of lipoteichoic acids is that they have a high phosphate content, and so have a number of ionized groupings that could react with calcium of the hydroxyapatite in the tooth.

Knox<sup>105</sup> has shown that teichoic acids will bind very firmly to hydroxyapatite. The adherence of certain streptococci to the tooth has generally been equated with their ability to form dextran, but as it now seems likely that the dextran layer will be permeated with lipoteichoic acid, it may be that this lipoteichoic acid is a crucial factor in determining the ability of organisms to adhere to the tooth<sup>105</sup>.

Knox<sup>105</sup> goes on to say that although there are suggestions that immunological reactions involving immunoglobulin A, may be concerned in the prevention of adherence of bacteria to the tooth surface, the immunological properties of lipoteichoic acids are more likely to be of importance in periodontal disease. It is generally accepted that the tissue changes that occur in periodontal disease involve immunological reactions, and it would be of importance if extracellular lipoteichoic acid could diffuse into tissues, and either induce antibody formation, or react with antibodies already present.

Lipoteichoic acid, like the lipopolysaccharides of Gram negative bacteria, may also be implicated in the

resorption of bone in periodontal disease<sup>78</sup>.

Reports have also been made on the antigenic potential of cementum.

Slavkin<sup>222</sup> notes that preliminary data suggests that enamel proteins are autoantigens. Since enamel proteins and acellular cementum proteins are antigenically identical<sup>70, 204.</sup>; Slavkin<sup>223</sup> concludes, "It is quite fascinating to consider that demineralization of acellular cementum could 'unmask' the enamel protein-like antigenic determinants, and severely challenge the host's immune mechanisms, resulting in an 'amplification' of the aggressive and destructive phases of periodontitis".

Robinson and Rowlands<sup>183</sup>, have suggested, on the basis of tooth transplantation studies, that changes in the cementum of tooth grafts may account for the host's chronic inflammatory reaction, which eventually results in loss of tooth transplants. This chronic, non-specific response takes place even with genetically compatible teeth, and in the case of replants.

Since the organic phase of cementum has been characterized as essentially collagenous, with a scant cell population<sup>186</sup>, and there is morphologic evidence that cemental collagen changes are associated with periodontal disease<sup>213, 255</sup>, it appears that one reasonable approach to determine the role of diseased cementum is to characterize its collagen immunologically. This approach is especially appropriate in light of recent evidence of elevated levels of collagen -

specific antibodies in patients with other chronic inflammatory diseases<sup>239</sup>.

The possible role of damaged cemental collagen is given additional significance by the findings of Kirrane and Glynn<sup>103</sup>; and Steffin and co-workers<sup>238</sup>. They reported enhanced immunogenicity of denatured collagen. This finding lends support to Willoughby and DiRosa's hypothesis on the role of damaged tissue in chronic inflammation. Willoughby and DiRosa<sup>273</sup> have suggested that the immune response to diseased, damaged collagen can perpetuate the cycle of chronic inflammation in situ. These observations could account, in part, for the character of the lesion in periodontitis, as well as the great difficulty encountered in the therapeutic control of this chronic disease.

#### Changes In Infra-Attachment Cementum

Significant changes have been reported in cementum immediately below the most apical border of the junctional epithelium in periodontal disease. Gottlieb<sup>58, 59</sup> first suggested that the layer of noncalcified precementum covering the cemental surface may act as a barrier to prevent apical migration of the epithelium. Thus its destruction might lead to pocket formation.

Kerr<sup>98</sup> stated that continuous deposition of cementum is a protective biologic process without which the supporting structures could not be maintained.

He further noted that cemental resorption took place at sites of active inflammation just apical to the bottom of periodontal pockets. These reports should be coupled with histologic observations of a loss of cementoblasts and a reduction or loss of the precemental layer in areas immediately below the most apical position of the junctional epithelium. Such responses are most vividly seen after the infliction of a gingival injury and its accompanying inflammatory response<sup>235</sup>. In experimental animals, lysis of cementoblasts and alteration in cemental surface at the wound periphery have been observed within minutes after injury<sup>174, 229, 257</sup>.

At the ultrastructural level, Selvig<sup>213</sup> reported changes in the area immediately below the apical position of the junctional epithelium in moderate gingival inflammation. These changes consisted of partial destruction of collagen in a zone 0.5 to 1 mm below the apical border of the junctional epithelium, and complete destruction of collagen in a narrow zone immediately below the epithelium. Thus, the cemental surface immediately below the epithelial level was denuded of collagen. This space contained granular debris. The cemental surface showed decreased electron density due to a reduction in number and size of mineral crystals and loss of collagen in this area<sup>213</sup>. Such changes may be responsible for repeated findings that apical epithelial migration and gingival recession was greater at the site of gingival injury

than at non-injured sites in rats followed over a one-year postsurgical period<sup>230, 256</sup>. Furthermore, as reported by Hurzeler and Zander<sup>86</sup>, human teeth with periodontal disease showed less cemental apposition than teeth without periodontal pathology.

Stahl<sup>235</sup> suggests that gingival inflammation, caused by either local irritants or gingival surgery, may affect the integrity of acellular cementum immediately adjacent to the site of soft tissue insult, by lysis of cementoblasts and changes in the precementum and cemental surface. These responses may play an important part in the subsequent ability of the junctional epithelium to migrate apically.

In addition, injured epithelium, by phagocytic activity<sup>160</sup> might affect the integrity of the cementoblasts and cemental surface, and fibrocytes might be engaged in collagen breakdown, as well as formation during situations requiring rapid remodelling<sup>251</sup>. Thus, gingival inflammation acting either alone, or with other biologic mechanisms, may affect acellular cementum at the site of inflammation<sup>235</sup>.

This concept is further supported by Dragoo and Sullivan<sup>30</sup>, who reported on their histologic evaluations of iliac bone grafts placed in infrabony pockets. In their cases, external root resorption was associated with chronic inflammation in the adjacent gingiva.

In cellular cementum, as in bone, loss of cells within the tissue graphically depicts loss of vitality. In acellular cementum, different criteria for

"vitality" have to be applied. Pathologic changes in acellular cementum may be considered as:<sup>235</sup>

- (1) lysis of cementoblasts;
- (2) loss or change in the precementum - cementum interface;
- (3) decrease in surface mineralization; and
- (4) cemental resorption.

### SECTION THREE

#### ACQUIRED LAYERS ON THE ROOT SURFACE

Jones<sup>94</sup> reported three main acquired coatings on pathologically exposed root surfaces: the acquired pellicle, plaque, and calculus.

##### Acquired Pellicle

The acquired pellicle is a thin layer of salivary proteins, mainly glycoproteins<sup>188</sup>, which is formed within minutes on tooth surfaces or other firm surfaces in the mouth<sup>117</sup>. It is from 0.05 to 0.8 microns (u) thick, acellular and initially bacteria free<sup>141, 253</sup>. It is a smooth, colourless, translucent film which adheres firmly to the underlying tooth surface<sup>142</sup>.

Although it is said that the formation of dental plaque can proceed directly on tooth surfaces<sup>42</sup>, it usually appears to commence on the acquired pellicle<sup>115, 138</sup>. As the plaque matures, the underlying pellicle may persist, undergo bacterial degeneration, or become calcified<sup>115</sup>.

From chemical data<sup>144</sup>, it is likely that acquired pellicles naturally formed on root surfaces are not pure derivatives of salivary proteins, since they may also contain substantial fractions of bacterial protein. Armstrong<sup>8</sup> supports the view that acquired pellicle is a complex of a salivary mucoprotein matrix intimately associated with embedded bacterial cell wall elements and lysed bacterial matter. This may be significant in the periodontal disease process and subsequent therapy, when we

consider the antigenic nature of certain bacterial cell wall components, discussed by Knox<sup>106</sup>.

The precise mechanisms involved in the initial protein adsorption have not been fully described. If, however, hydroxyapatite can be regarded as an amphoteric material, it could bind negatively and positively charged groups on salivary proteins<sup>190</sup>. Negatively charged groups in macromolecules could be bound to calcium ions on the surfaces of the hydroxyapatite.

### Plaque

Genco<sup>47</sup> defines dental plaque as, "a product of microbial growth, tenaciously attached to the surfaces of teeth and adjacent gingiva, and exhibiting a definite microscopic architecture".

The principle significance of dental plaque in the aetiology of gingival and periodontal disease is its concentrations of bacteria and their products<sup>47</sup>.

Evidence for the aetiological role of dental plaque in periodontal disease comes mainly from:

1. individual clinical observation and epidemiological studies<sup>68, 196, 203</sup>.
2. animal experiments<sup>48, 87, 102, 195, 217, 226, 260</sup>.
3. short term clinical experiments in man<sup>91, 129, 131, 132, 254</sup>.

The inflammatory changes in periodontal disease are characteristic of an infection, and attention has generally been directed at two possible mechanisms<sup>46</sup>:

1. a direct effect on the tissues by bacterial enzymes and cytotoxic substances; and

2. immunopathological processes deriving a host response to bacterial reactions.

Plaque flora will vary from site to site within the same mouth<sup>17, 157, 261</sup>, as well as from individual to individual<sup>17, 134</sup>. Facultative and anaerobic, saccharolytic Gram-positive species, tend to inhabit supragingival plaque, whereas the more anaerobic nonsaccharolytic Gram-negative species are usually found in the subgingival plaque.

At least three broad types of plaque can be identified according to clinical conditions : a nondisease-associated plaque, a caries-associated plaque, and a periodontal disease-associated plaque<sup>135</sup>. These categories are not distinct bacterial entities, but rather, appear to be different proportions of the same basic flora.

A caries-associated plaque is adapted to convert sucrose quickly and efficiently to insoluble intra- and extracellular polymers<sup>149</sup>; and has elevated levels of *S. mutans* and lactobacilli, and low levels of *S. sanguis* and Gram-negative rods.

A periodontal disease-associated plaque has elevated levels of Gram-negative anaerobic rods and actinomyces species<sup>272</sup> and no *S. mutans*. In marginal gingivitis, *A. israeli* and *A. viscosus* increase proportionately<sup>134</sup>, whereas in periodontosis, the motile Gram-negative anaerobic rods dominate<sup>157</sup>.

The general microbial population shifts during plaque development from a predominantly coccal

facultative flora, to a more complex flora containing a high proportion of Gram-negative anaerobes<sup>74, 182</sup>.

Theilade and co-workers<sup>254</sup>, correlating gingival condition in specific areas with plaque composition, found that clinical inflammation could be diagnosed at about the time the complex flora was established. However, subclinical inflammation starts as a reaction to the first phases of plaque formation<sup>165, 254</sup>.

Possible interactions between the acquired pellicle and bacteria may include the following:

1. Interaction of the acquired pellicle proteins with extracellular, bacterial polysaccharides<sup>69</sup>.  
Examples of bacterial polysaccharides or polysaccharide-containing materials that may interact with the protein portion of the acquired pellicle are: dextran, levan, peptidoglycan (or murein, a structural component of bacterial cell walls), teichoic acids, and lipopolysaccharides.
2. Aggregation of bacteria by binding to the surfaces of other bacterial cells, initiated by a salivary macromolecule<sup>49</sup>.
3. A portion of the carbohydrate component of some salivary glycoproteins of the acquired pellicle may serve as substrate for extracellular, bacterial neuramidases<sup>116</sup>.
4. It is theoretically possible that bacterial cells may interact with the acquired pellicle through covalent bonds<sup>69</sup>. For example, a hydroxyl from the carbohydrate moiety of the acquired

pellicle may be linked covalently to the N-acetyl muramic acid, or the N-acetyl glucosamine portion of the bacterial cell wall.

Possible interactions between tooth substance and bacteria may include the following factors:

1. Bacteria may bind directly to tooth substance materials. This is suggested by the observation that bacterial cells are frequently in direct contact with the crystalline enamel surface<sup>207</sup>. The interactions occurring between the external aspect of bacterial cells, and the hydroxyapatite, are probably analogous to the use of hydroxyapatite in column chromatography as a protein and, or polysaccharide adsorbent<sup>189</sup>.
2. Another possibility is the interaction of external bacterial materials with components of the organic matrix of tooth substance<sup>69</sup>. This is especially applicable to cementum or dentine surfaces, where there is substantial organic matrix present. These interactions are probably similar to those of 1. and 2. in the previous section concerning acquired pellicle and bacteria.

### Calculus

"Calculus is an adherent, calcified or calcifying mass that forms on the surface of natural teeth and dental prostheses".<sup>50</sup> It may be classified according to its relation to the gingival margin as supragingival or subgingival. In both instances, the organic matrix

consists, to a large extent, of microorganisms which have become mineralized to varying degrees<sup>206</sup>. In the case of supragingival calculus, the mineral component is believed to originate primarily from saliva, whereas, in subgingival calculus, a substantial portion of the inorganic material is thought to come from gingival fluid.

It should, however, be noted that dental calculus may also occur in germfree animals<sup>252</sup>, an observation which suggests that under unusual circumstances, an organic matrix derived from sources other than microorganisms, may also serve as a nidus for calculus formation.

Attachment of calculus to tooth surfaces has been studied many times by conventional histological techniques. The relative frequency of various modes of attachment was first analysed by Zander<sup>277</sup>. According to this, and subsequent investigations<sup>109, 139, 148, 209, 264</sup> calculus, can be attached by means of:

1. an organic cuticle;
2. by direct attachment of microorganisms to the tooth surface;
3. by mechanical locking into irregularities of the tooth surface, such as resorption lacunae, and scaling grooves; and
4. by penetration into cracks, enamel lamellae, and carious defects.

More recently Selvig<sup>215</sup> used the electron microscope to study the ultrastructural nature of the interface between dental hard tissue, and soft and calcified deposits. He found that an intimate relationship existed in most instances between the intercellular matrix of calculus, and the organic matrix of the dental hard tissues, as well as between the mineral crystals of calculus, and those of the underlying tooth surfaces.

Selvig<sup>215</sup> further observed frequent mechanical locking of the deposits in cracks, resorptions, carious lesions, and other defects of the tooth surface. Moreover, calculus was closely adapted to the submicroscopic irregularities which were present even on so-called "smooth" tooth surfaces. At the ultrastructural level, the predominant mode of attachment appeared to be the adhesion of organic interbacterial substance to the tooth surface, in addition to intercrystalline forces of organic nature acting between crystals of calculus, and underlying tooth substance. The relative importance of the latter factor was presumed to increase as the concrement matured.

It is widely recognized that the presence of calculus is detrimental to periodontal health, and soft tissue new attachment to the tooth surface<sup>51, 53, 64, 168</sup>. Although bacterial plaque is considered to be of greater significance in the aetiology of periodontal disease than calculus per se<sup>127</sup>, the substantial contribution by bacteria to the matrix of calculus, and

the findings by Allen and Kerr<sup>3</sup> that calculus following autoclaving, remains irritating to tissues - suggest that, even in the mineralized state, calculus is able to exert a toxic influence on cells.

Hatfield and Baumhammers<sup>76</sup> have also demonstrated a toxic effect on cultured gingival epithelial cells by root surfaces from teeth with periodontal disease, possibly caused by residual plaque components.

Gingivitis has been shown to occur in the absence of calculus<sup>143</sup>, and the formation of plaque leads to gingivitis, which disappears when plaque is removed.<sup>203, 254.</sup> It is difficult to separate the effects of calculus and plaque upon the gingiva, because calculus is almost always covered with a nonmineralized layer of plaque<sup>205</sup>. There is a positive correlation between calculus and the prevalence of gingivitis<sup>173</sup>, but it is not as high as between plaque and gingivitis<sup>220</sup>.

O'Bannon<sup>159</sup> consistently found chronic inflammation associated with periodontal pockets clinically deeper than 3 mm, whether they had been scaled or not.

Cheraskin and Ringsdorf<sup>24</sup> reported a significant reduction in gingivitis after scaling, but pointed out that the response to scaling was not always what one might expect. After scaling, subgingival calculus accumulated steadily in both jaws over the 12-week period, until, by the end of the experimental period, the calculus surface index had reached its original level. There was a slight improvement in papillary and marginal inflammation over the first two weeks

after scaling, but this was not maintained, and by the fourth week, the incidence of papillary and marginal inflammation was back, almost to its original level. This would seem to coincide with maturation of pocket plaque.

Subgingival calculus may be the product, rather than the cause of periodontal pockets<sup>140, 243</sup>. Plaque initiates the gingival inflammation which starts pocket formation, and the pocket provides a sheltered area for plaque and bacterial accumulation. Increased flow of gingival fluid associated with gingival inflammation, provides the minerals which convert the continually accumulating plaque into subgingival calculus.

Mandel<sup>140</sup> concludes that calculus is important primarily because of its bacterial content. Allen and Kerr<sup>3</sup> agree that the non-mineralized plaque on the calculus surface seems to be the principal irritant, however, they also signify the importance of the underlying calcified portion as a contributing factor. They contend that, although it does not irritate the gingiva directly, it does provide a fixed nidus for the continued accumulation of irritating surface plaque, and holds the plaque against the gingiva.

SECTION FOUR

RATIONALE OF PERIODONTAL THERAPY

"Consistent success in treatment of periodontal lesions is dependent on an understanding of therapeutic objectives, and a knowledge of the processes involved"<sup>175</sup>

The ultimate goals of periodontal therapy are:

1. to halt the destruction of the tooth supporting apparatus; and
2. to establish an environment that the patient is able to maintain in health<sup>279</sup>.

The prevention of gingival inflammation is based primarily on removal of bacterial plaque and calculus which have formed on the root surface, or permeated the cemental surface subsequent to its exposure through pocket formation\*. Two principal pathways lead toward this goal:

1. local, chemical prevention of plaque and calculus accumulation, or dissolution and removal of already existing soft and hard deposits; and
2. mechanical removal of microbial plaque and concretions.

Despite partially successful experiments to chemically prevent the formation of deposits, or to dissolve already existing deposits, Donze and co-workers<sup>29</sup> conclude that these methods cannot yet be generally recommended for the dental practice, or for home care.

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\* 16, 22, 23, 29, 83, 101, 109, 119, 179, 202, 246, 248.

In addition, Flotra and coworkers<sup>40</sup>, using 0.1 to 0.2% chlorhexidine solutions, showed results which suggest that topical antimicrobial agents are not likely to enter the periodontal pocket greater than 3 mm, and reach the subgingival flora.

Mechanical removal of uncalcified and calcified accretions still seems to be the therapy of choice.

Establishment of an environment that facilitates removal of plaque by the patient is thought by many to be best achieved by total eradication of the periodontal pocket<sup>96, 111, 279</sup>. There are a number of forms of periodontal therapy used for elimination of pockets<sup>175</sup>. These comprise:

1. procedures to produce shrinkage. Where re-establishment of normal osmotic gradient is dependent on the removal of irritants, and elimination of the inflammatory reaction;
2. excisional procedures. Where tissues are removed that are not attached to the tooth or alveolar process, thus eliminating one wall of the pocket;
3. procedures which result in reattachment; and
4. procedures which result in new attachment.

Reattachment and new attachment are incorporated in a variety of therapeutic procedures including scaling, root planing, curettage, treatment of certain infrabony pockets, and various mucogingival surgical techniques.

Reattachment is the reuniting of gingival tissues and root surfaces following surgical, or traumatic separation<sup>96, 99</sup>. Segments of attached fibres left on the root surface may guide or join the newly formed fibres produced by the soft tissue segment of the wound<sup>175, 236</sup>. Under such circumstances, the periodontal attachment will be similar to the presurgical level<sup>107, 228</sup>. If accepted, this concept would demand clinical reorientation regarding the extent of root planing during the surgical procedure, since much greater attention would have to be paid to the level of attached fibres, and their clinical identification<sup>233</sup>. Reattachment may take place more often than the clinician realizes, since complete removal of all inserted collagen from all involved root surfaces is not easily accomplished<sup>236</sup>.

New attachment implies the reunion of soft and supporting tissues to root surfaces that were pathologically exposed in a periodontal lesion. It involves creation of new epithelial and, or connective tissue attachment elements<sup>96, 99</sup>. Rosenberg<sup>191</sup> states that new attachment may be influenced by: the morphology of the osseous deformity, contour of the root, chronicity of the defect, tooth mobility, definitive preparation of the root surface, flap design, thoroughness of enucleation of the granulo-matous tissue and connective tissue lying immediately over the osseous walls, decortication, presence of a blood clot within the defect, asepsis, prophylactic

antibiotic coverage, postoperative maintenance care, and effective plaque control at the surgical site. Certainly, a recent study by Rosling and coworkers<sup>193</sup> relates the success or failure of new attachment attempts to the presence or absence of plaque and inflammation in the healing areas.

## SECTION FIVE

### INSTRUMENTATION FOR SCALING AND ROOT PLANING

Scaling generally means removing hard deposits from visible surfaces of the teeth; while the term root planing, indicates more than removal of deposits, and includes the removal of tooth structure, with a view to smoothing the surface of the root<sup>72</sup>.

The skilful use of instruments is a basic requisite of adequate scaling and root planing. There are many sets of instruments of various types, designed to meet the needs of most situations encountered.

#### Hand Instruments

There are five basic types of hand instruments<sup>54</sup>,  
<sup>63</sup>: chisels, scalers, hoes, files and cures.

(Refer Figure 1) Each of the five types is designed for a specific use, and sometimes, for access to a specific tooth surface. The chisel, scaler and hoe are designed for the removal of heavy calculus, whereas, files and cures are intended for the finer and final planing of the root surface to the bottom of the pocket<sup>63</sup>.

#### Chisels

The chisel is designed for removal of extensive supragingival calcified deposits, especially those located in the mandibular anterior region. When calculus occupies the interproximal and lingual area, the chisel is used in a labiolingual direction with a push stroke to dislodge the gross mass<sup>63</sup>.

#### Hoes

Hoes are very efficient in removing calculus<sup>9</sup>.

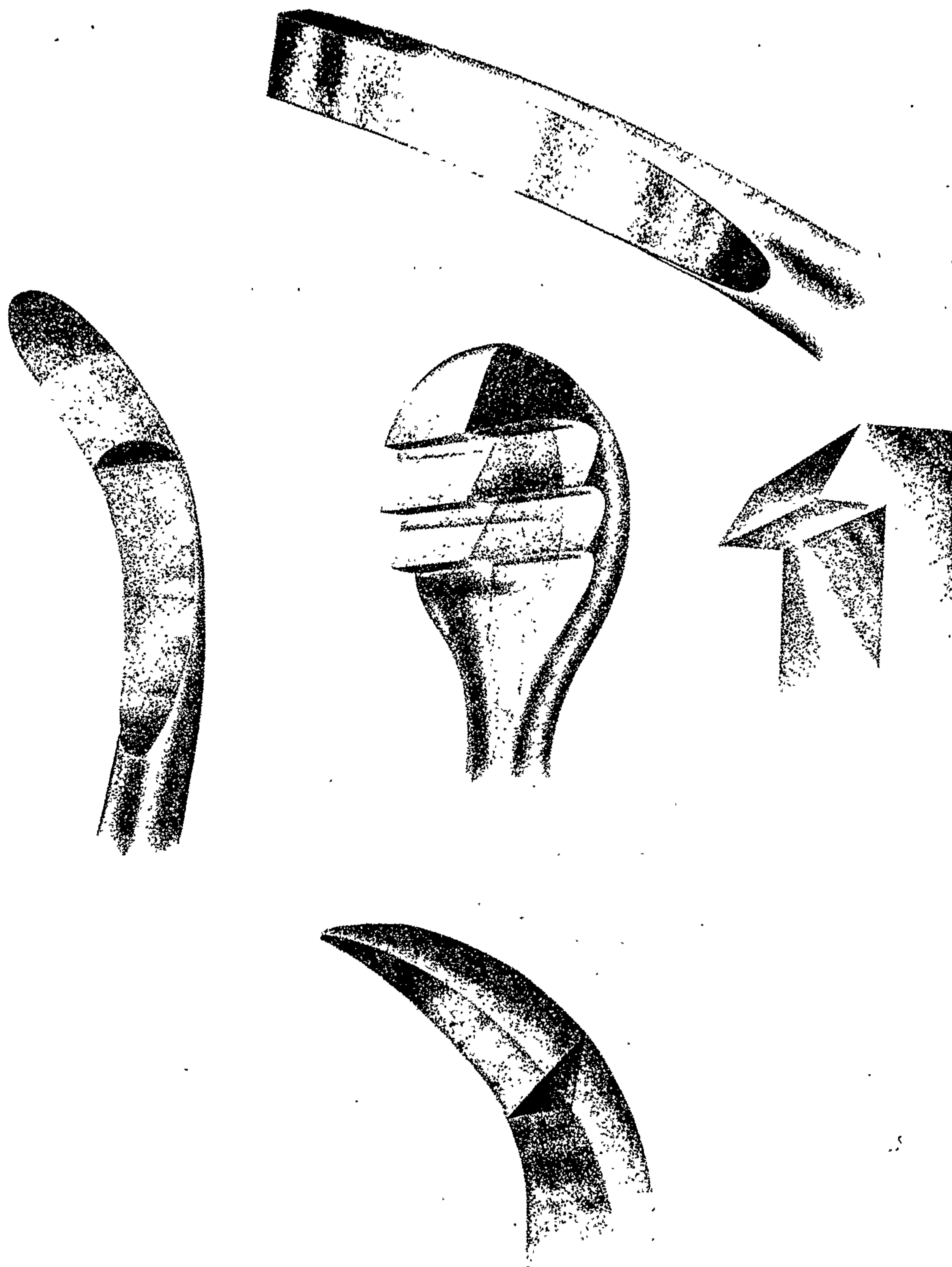


FIGURE 1<sup>55</sup>

shows:

- chisel (top)
- curette (middle left)
- file (centre)
- hoe (middle right)
- scaler (bottom)

They are not good for planing because they tend to groove or scratch the root surface<sup>66, 199</sup>. If hoes are used for debridement, the root should be planed with curettes<sup>200</sup>.

### Scalers

Although they are used primarily for supra-gingival deposits, scalers may be used sparingly beneath the gingiva<sup>200</sup>. Because of their design, sickle scalers are inefficient in removing subgingival deposits in narrow pockets, or ones of moderate depth; Morse scalers are slightly better suited. Scalers are not designed to plane the tooth<sup>200</sup>.

### Files

These instruments can be used to remove calculus, but they do so by fragmentation. They are less efficient in removal of calculus than hoes or curettes<sup>9</sup>. A file is a poor instrument for planing because there is a tendency to scratch and lacerate the root surface<sup>66, 199</sup>. If files are used for subgingival debridement, they should be followed by curettes to plane the root surface<sup>200</sup>.

### Curettes

These are the most efficient hand instruments to remove minute deposits<sup>9</sup>. Of the hand instruments, they produce the smoothest root surfaces in planing<sup>66, 199, 200</sup>.

Curettes can be used with either pull or push strokes. Many curette designs exist, however, they are basically the same cross-sectionally. Curettes

have two cutting edges. Some, (for example, Gracey's), are designed to use one cutting edge against the tooth for removing deposits, and the other against the soft tissues. Others, (for example, McCall's), are designed to use either cutting edge on the tooth, or on the soft tissues<sup>200</sup>.

## Ultrasonic Instruments

### Introduction

Ultrasonic instruments are used routinely in periodontics for removal of calculus and other accretions from root surfaces. Some authors<sup>25, 34, 153</sup> suggest that ultrasonics may be used for root planing, others disagree<sup>200, 247</sup>.

### Working Mechanism

The ultrasonic instruments used in periodontal treatment operate at frequencies between 25,000 and 42,000 Hz. The amplitudes in the longitudinal axis of the handpiece range from 0.006 to 0.1 mm<sup>247</sup>.

Ultrasonic vibrations for periodontal therapy are produced by inserting a stack of magnetostrictive material, (usually metal which can change its dimension in an electromagnetic field), into a shockproof, cylindrical handpiece. The stack is then subjected to the influence of both a standing, and an alternating electromagnetic field. Since a tip is attached to the end of the stack of magnetostrictive metal, ultrasonic vibrations will be transferred to the tip<sup>247</sup>. The pattern of vibrations of the tip appears to depend on its design<sup>34</sup>.

During the generation of ultrasonic vibrations, a large amount of heat is produced. The necessary cooling is effected by circulating water round the stack of magnetostrictive metal in the handpiece. The water is then expelled, and cools the vibrating tip. At one or two points of the working tip, where there are maximum vibrations, a spray is produced<sup>26</sup>. This phenomenon is called "cavitation" (bubbling effect), and has been discussed by Hueter and Bolt<sup>85</sup>. It was thought at one time that cavitation liberated some energy for removal of deposits<sup>33, 250</sup>. However, it is now generally accepted that the mechanical energy produced by the vibrating tip is responsible for deposit removal<sup>26, 92</sup>  
200

#### Handling of the Instrument

Information concerning the handling of ultrasonic instruments is available<sup>26, 35</sup>. In general, the lowest power setting consistent with effectiveness should be used. The handpiece and tip should be applied with a very light pressure, making a small angle of approximately  $15^{\circ}$  (degrees) with the tooth surface.

As the instrument is being used, the tip should be kept in motion at all times when the machine is on. In addition, it has been suggested that an ultrasonic tip should be applied in such a way that its pattern of vibrations is correctly orientated to the tooth surface, otherwise it may knock the tooth and result in pain and surface roughness.<sup>89, 97</sup>. It must be remembered,

however, that in clinical work, the irregular anatomy of the tooth makes it impossible to keep the angle between instrument motion and the tooth at its correct theoretical value at all positions<sup>89</sup>.

Periodontal exploratory instruments, for example a sharp No. 17 explorer used by Kerry<sup>100</sup>, are often recommended for use during ultrasonic instrumentation in pockets. Also, an ample amount of water spray should be provided, particularly in pocket areas, where the flow at the tip may be impaired<sup>89</sup>.

It is important not to use tips that have rough surfaces or spurs, which would scratch the tooth surface<sup>26</sup>. The more common brands of ultrasonic scalers have a universal tip, for example the Cavitron TFI 10 or P10, which may be used in most situations.

## SECTION SIX

### COMPARISON OF ULTRASONIC AND HAND INSTRUMENTATION

There have been many reports concerning different effects of hand instruments and Cavitron<sup>\*</sup> ultrasonic instruments. A short review of certain aspects investigated follows.

#### Calculus Removal

There seems to be little, or no difference in the ability of either ultrasonic or hand instruments to remove calculus, in most cases<sup>21, 92, 93, 137, 153, 240, 244</sup>.

However, where there is the existence of deep pockets, pronounced root irregularities, open bi- and tri-furcations, and crowding, the ultrasonic instruments seem to be more limited than conventional hand instrumentation. This is probably because of the ultrasonic's diminished tactile ability, and design of the standard tips<sup>29, 153</sup>.

In the study by Jones and co-workers<sup>93</sup>, ultrasonic instrumentation either left the calculus intact, or dislodged it piecemeal; while scalers and curettes fractured off part or all of the calculus, and burnished the surface.

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\* Dentsply Cavitron ultrasonic instrument,  
Amalgamated Dental, London. England.

### Stain Removal

Ultrasonic instruments can usually remove stains more efficiently, and with less discomfort to the patient, than hand scaling instruments<sup>21, 92, 200, 244</sup>. It has been noted, however, that conventional techniques, using the rubber polishing cup and paste, are superior to ultrasonics in removal of stains<sup>137, 274</sup>.

### Smoothing of the Root Surface

Reports on the effect of ultrasonic and hand instrumentation on root smoothness at first glance seem inconsistent\*. However, the contradictory reports can be explained by the materials used, and the methods of evaluation in each case. These will be more fully explained in the "Discussion of Results" section.

Generally speaking, the Cavitron ultrasonic scaler with TFI-10 insert, used as directed, produces a smoother surface than a sharp curette, when viewed at 1000 magnification. Where a sharp curette has been used in the instrumental sequence, the surface is more likely to be level, and uniformly planed<sup>163, 262</sup>.

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\* 39, 66, 93, 100, 147, 163, 192, 262.

### Planing the Root Surface

Although the removal of some surface structure is possible with the ultrasonic unit<sup>15, 25, 34, 153</sup>, true definitive root planing, where the operator wishes to remove tooth structure to a specific depth in a given area, is probably best performed with a sharp curette<sup>26, 240</sup>.

### Removal of Pocket Epithelium

Pocket epithelium has been shown to be partly removed during hand scaling<sup>152, 172</sup>. Similarly, Schaffer and co-workers<sup>201</sup> observed comparable partial removal of the pocket epithelium during ultrasonic, as well as during hand scaling. They furthermore found that the apical part of the pocket epithelium was more frequently removed than the coronal part. This is in agreement with the findings of Nadler<sup>155</sup>.

### Regeneration of Pocket Epithelium

The regeneration of pocket epithelium after ultrasonic instrumentation has been found to be faster than after hand instrumentation<sup>52, 197, 201</sup>. Bhaskar and co-workers<sup>13</sup> found that the healing of inflamed gingiva was faster after ultrasonic scaling than after hand scaling. Donze and co-workers<sup>29</sup> however, found that gingival inflammation decreased at the same rate in cases of pure gingivitis, whether they were treated ultrasonically or manually.

### Operator Physical Strain

With ultrasonics, scaling is performed with less pressure and decreased wrist motion than hand instrumentation<sup>29</sup>. This fact is likely to reduce fatigue where a great deal of periodontal scaling is performed.

### Time Saving

Many investigators found that ultrasonic instruments removed deposits faster than hand instruments<sup>13, 29, 41, 92, 153, 227</sup>. Burman and co-workers<sup>21</sup> however, reached the conclusion that both types of instruments required more or less the same time to remove deposits. Stewart and others<sup>244</sup> observed that ultrasonic scaling was faster than hand scaling in patients with heavy supragingival calculus. It has been reported that ultrasonic scaling can save as much as 20% to 50% of the time needed for hand scaling<sup>29, 41</sup>.

### Tactile Sense

Compared to hand instrumentation, the tactile sense during ultrasonic instrumentation has been reported to be poor<sup>11, 21, 71, 153, 200, 202</sup>. One's tactile sense may be increased by momentarily allowing the unit to be turned off while the tip is passed over the surface being covered<sup>38</sup>.

### Ease of Control

A number of authors have pointed out that a longer training period is needed to master the special ultrasonic scaling techniques<sup>26, 65, 108</sup>. However,

ordinary hand scaling also requires considerable technical skill if it is to provide the desired result<sup>88</sup>.

Ultrasonic instrumentation is subject to a larger number of variables than hand instrumentation. These are mostly related to energy factors<sup>26, 89</sup>.

#### Patient Comfort During Treatment

There is general agreement that most patients prefer ultrasonic scaling to hand scaling<sup>29, 41, 92, 137, 244, 274</sup>. Possibly the absence of tugging and scraping during ultrasonic scaling helps decrease anxiety in the patients<sup>92</sup>.

#### Incidence of Bacteraemia

Transient bacteraemia has been shown to be present after curettage, gingivectomy, and other periodontal procedures<sup>73</sup>. Brandt and co-workers<sup>19</sup> compared the incidence of bacteraemia produced by hand scaling and that produced by ultrasonic scaling, and found no significant difference.

#### Bacterial Counts in Air

According to a well controlled study carried out by Lorato and colleagues<sup>136</sup>, air organism counts increased 30 times during ultrasonic scaling. The authors concluded that the very light weight, organism-bearing droplets might contain pathogenic organisms; these could create a potential health hazard if inhaled.

#### Effects on Hearing

Temporary shifts in hearing threshold have been

reported to occur in some patients following ultrasonic  
instrumentation<sup>150</sup>.

PART II

ORIGINAL WORK

## MATERIALS AND METHODS

### Study 1

In vitro conditions were used in this study to provide optimal control of periodontal instrumentation.

Broad mesial and distal root surfaces from a total of twenty-five extracted anterior teeth were selected for investigation. The teeth represented random variations in age, sex, race, and periodontal status. Of the fifty available test surfaces, forty-seven were treated in an area on the coronal half of each root; the surrounding untreated portions acted as controls. Also, the remaining three surfaces were kept as controls.

Of the forty-seven treated surfaces, thirty-seven were instrumented in areas apical to the epithelial attachment, ie, infra-attachment regions, (Refer Figure 2, Zones 3 and 4) thus helping to eliminate variables involving disease or previous instrumentation damage. Ten specimens were instrumented in areas exposed by periodontal disease, ie, supra-attachment regions (Refer Figure 2, Zone 2). The supra-attachment regions were clinically unaffected by caries or previous instrument damage.\*

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\* After preliminary studies, it was decided that infra-attachment and supra-attachment root surfaces should be distinguished as separate types of surfaces when evaluating changes following instrumentation, especially where little alteration

was produced. Infra-attachment root surfaces show either periodontal fibre remnants obscuring the cementum. (Refer Plate 2), or Sharpey's fibre mounds. (Refer Plate 3). Supra-attachment root surfaces, on the other hand, show either calculus completely covering the cementum, or a mineralized pellicle "sheet", partially and, or completely filling in the spaces between the Sharpey's fibre mounds. (Refer Plate 5,) Where an instrument had little tendency to alter the surface such as with a Cavitron or Amdent ultrasonic at three-quarter (medium) setting, the resultant surface would exhibit features characteristic of its own anatomy, rather than resultant features characteristic of the instrument. However, where an instrument removed a significant amount of tooth substance such as with a sharp curette, the surface would be more likely to exhibit features characteristic of the changes caused by the instrument itself.

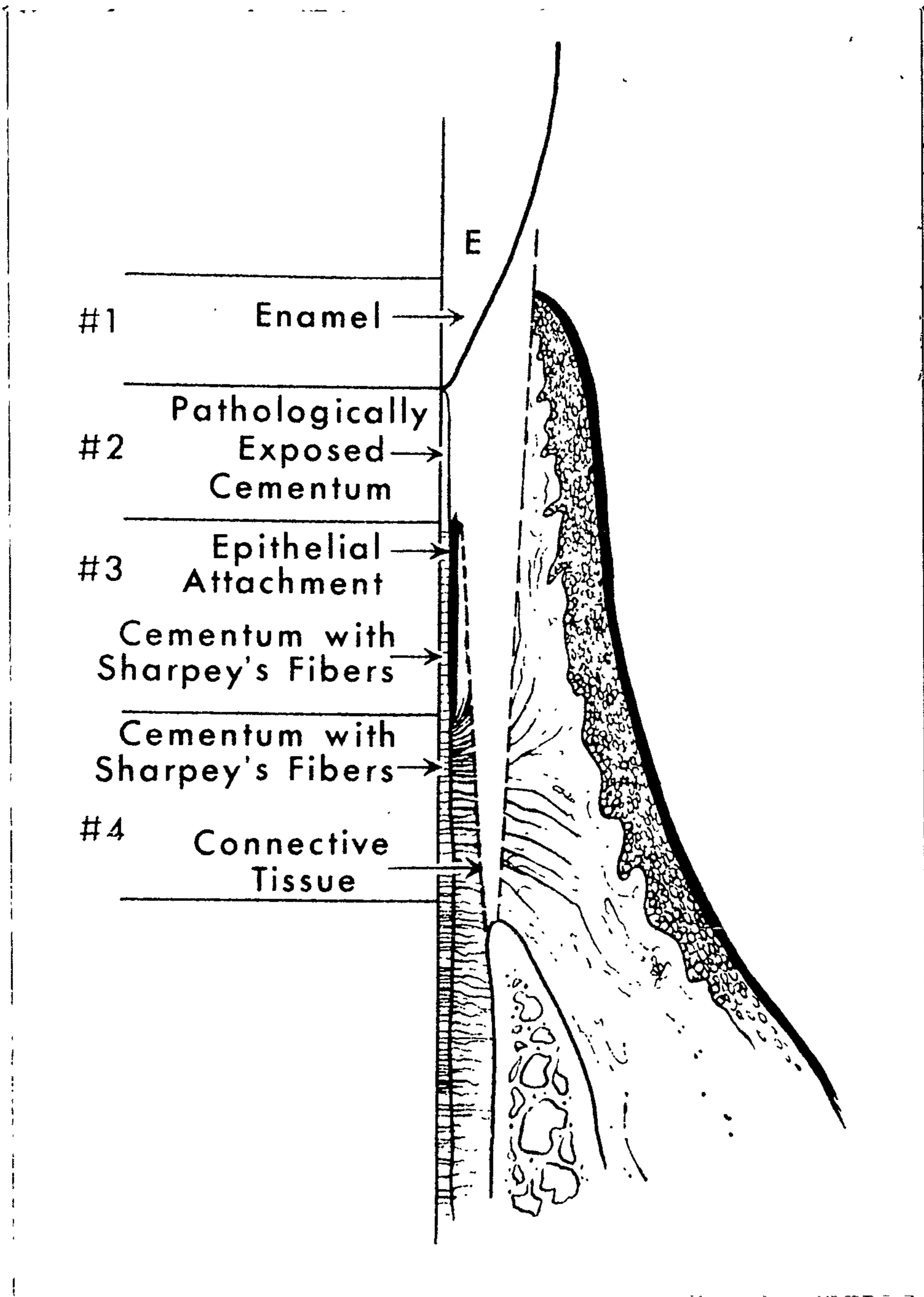


FIGURE 2<sup>176</sup>

Illustrates zones on root surface following removal of a wedge of tissue.

Allocation of instruments to tooth root surfaces is shown in Table 1.

TABLE 1

| Instruments used  | <u>Numbers and type of specimens</u> |      |
|---|--------------------------------------|------|
|   | (i) = infra-attachment specimen      |      |
|   | (s) = supra-attachment specimen      |      |
| Sharp curette   | 8(i)                                 |      |
| Dull curette  | 4(i)                                 |      |
| Sharp scaler  |                                      | 1(s) |
| Dull scaler   |                                      | 2(s) |
| Sharp hoe   | 1(i)                                 |      |
| Cavitron three-quarter (medium) setting                           | 5(i)                                 | 1(s) |
| Cavitron full (high) setting                                      | 3(i)                                 | 2(s) |
| Amdent three-quarter (medium) setting                             | 4(i)                                 | 2(s) |
| Amdent full (high) setting  | 3(i)                                 | 2(s) |
| Odontoson half setting  | 2(i)                                 |      |
| Odontoson three-quarter setting                                   | 2(i)                                 |      |
| Sharp curette followed by Cavitron three-quarter (medium) setting | 1(i)                                 |      |
| Sharp curette followed by Amdent three-quarter (medium) setting   | 1(i)                                 |      |
| Cavitron three-quarter (medium) setting followed by sharp curette | 2(i)                                 |      |
| Amdent three-quarter (medium) setting followed by sharp curette   | 1(i)                                 |      |
|   | Total = 37(i), 10(s).                |      |

Examples of hand instruments used in the study are shown in PLATES 6, 7, and 8.

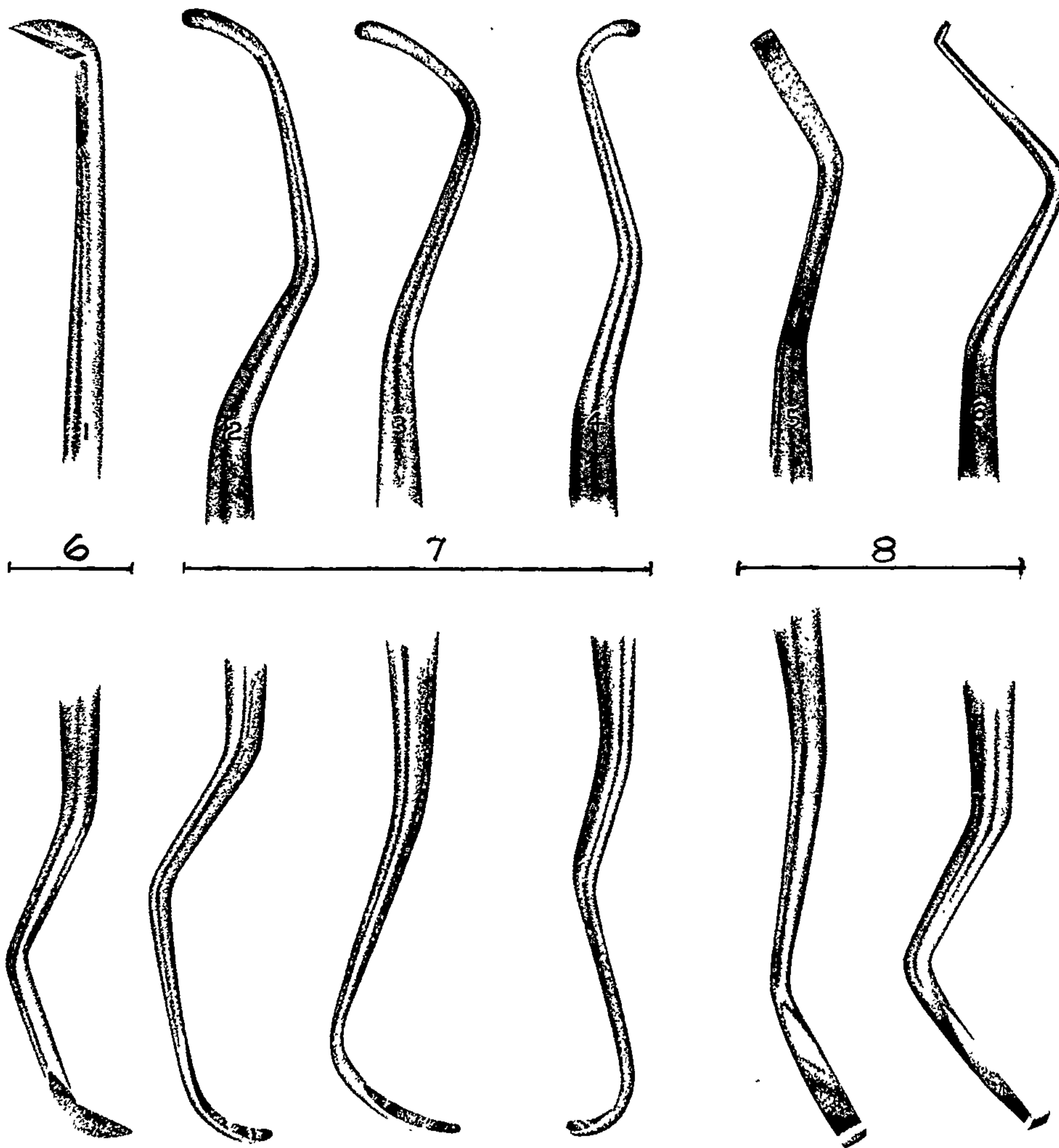


PLATE 6 - Periodontal scaler

PLATE 7 - Periodontal curette

PLATE 8 - Periodontal hoe

An example of ultrasonic instruments used in the study is shown in PLATE 9a and 9b.

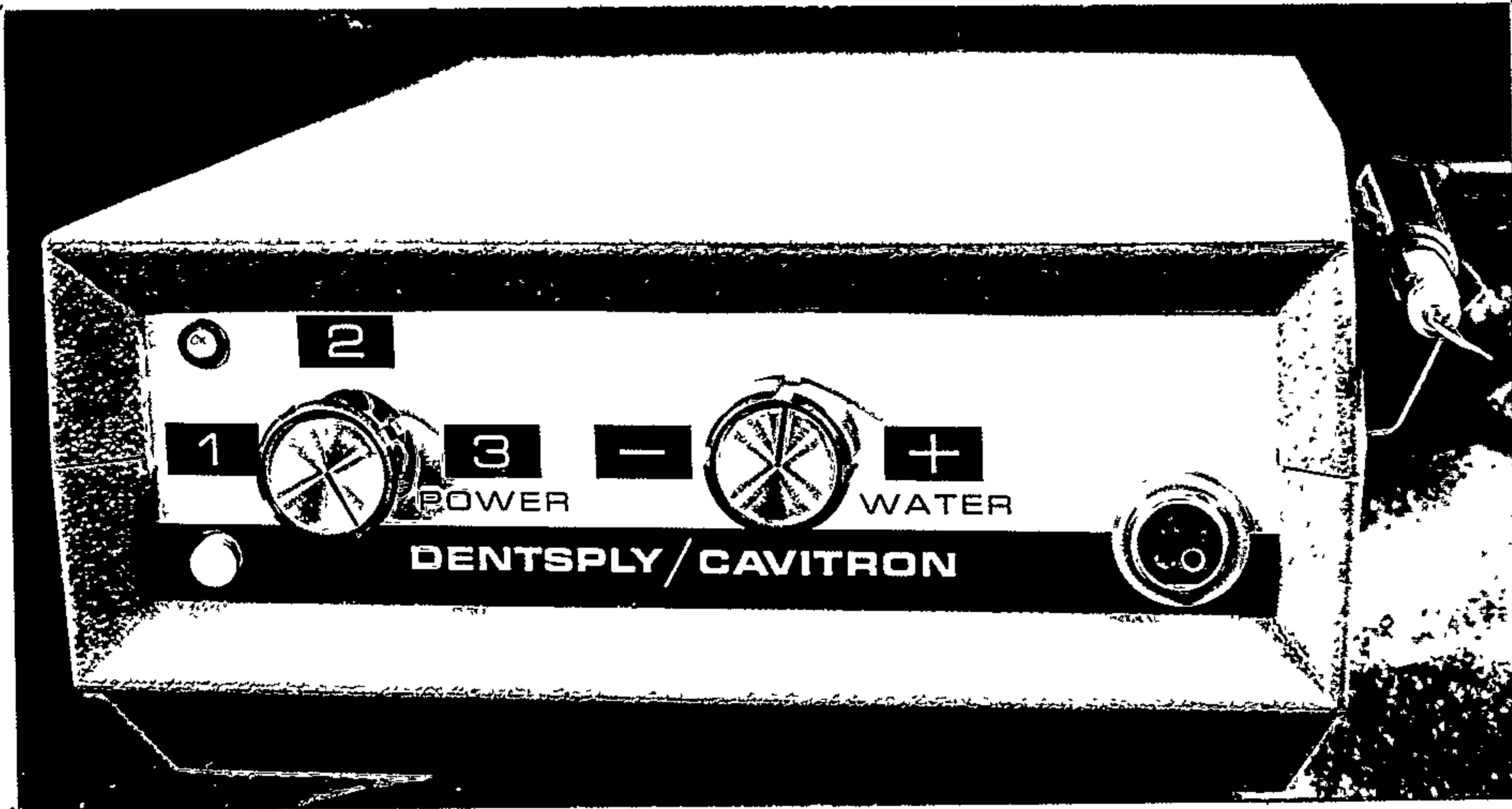


PLATE 9a - Cavitron 700II  
ultrasonic scaling unit

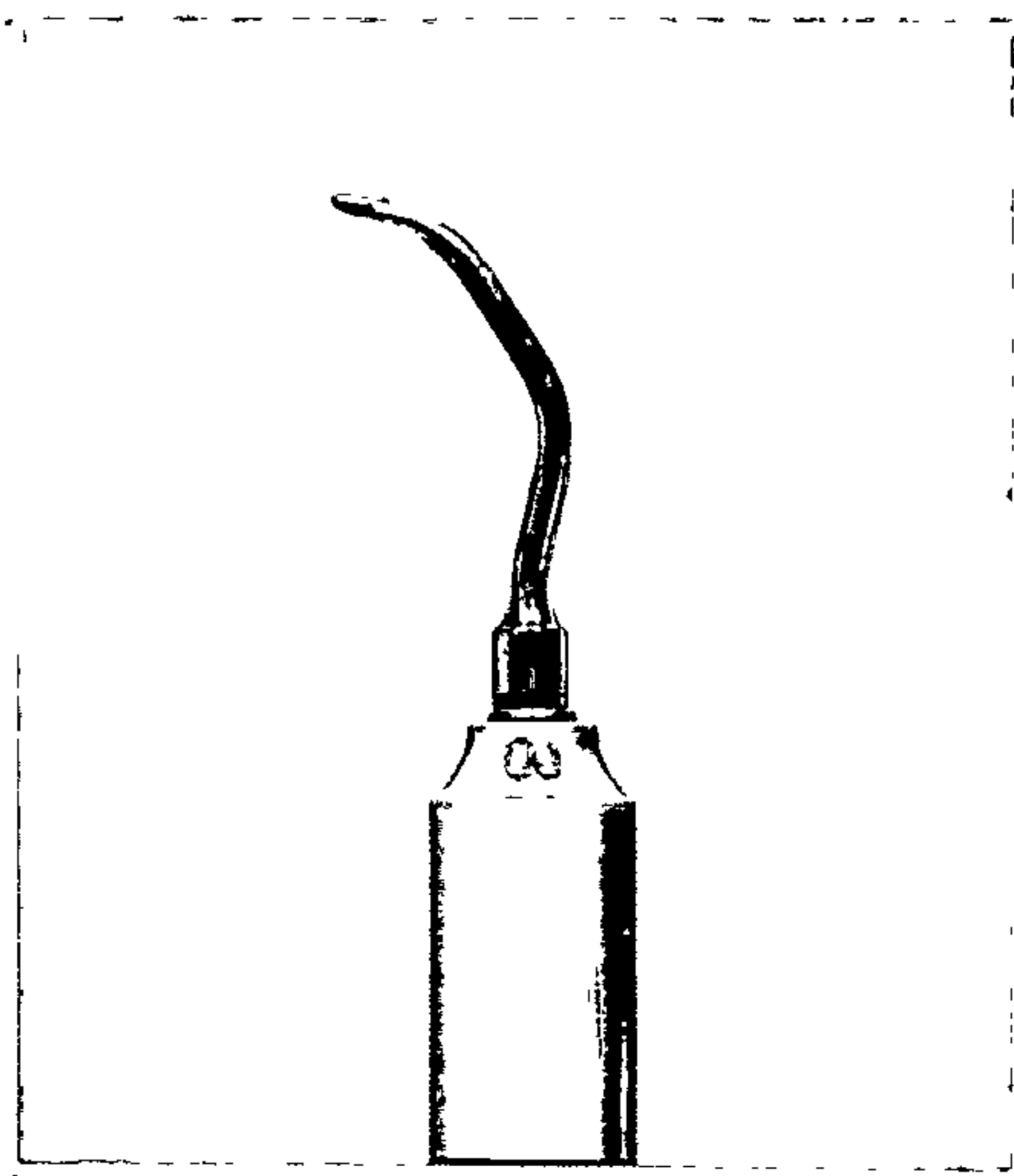


PLATE 9b - Cavitron TFI-10 tip

An example of ultrasonic instruments used in the study is shown in PLATES 10a and 10b.

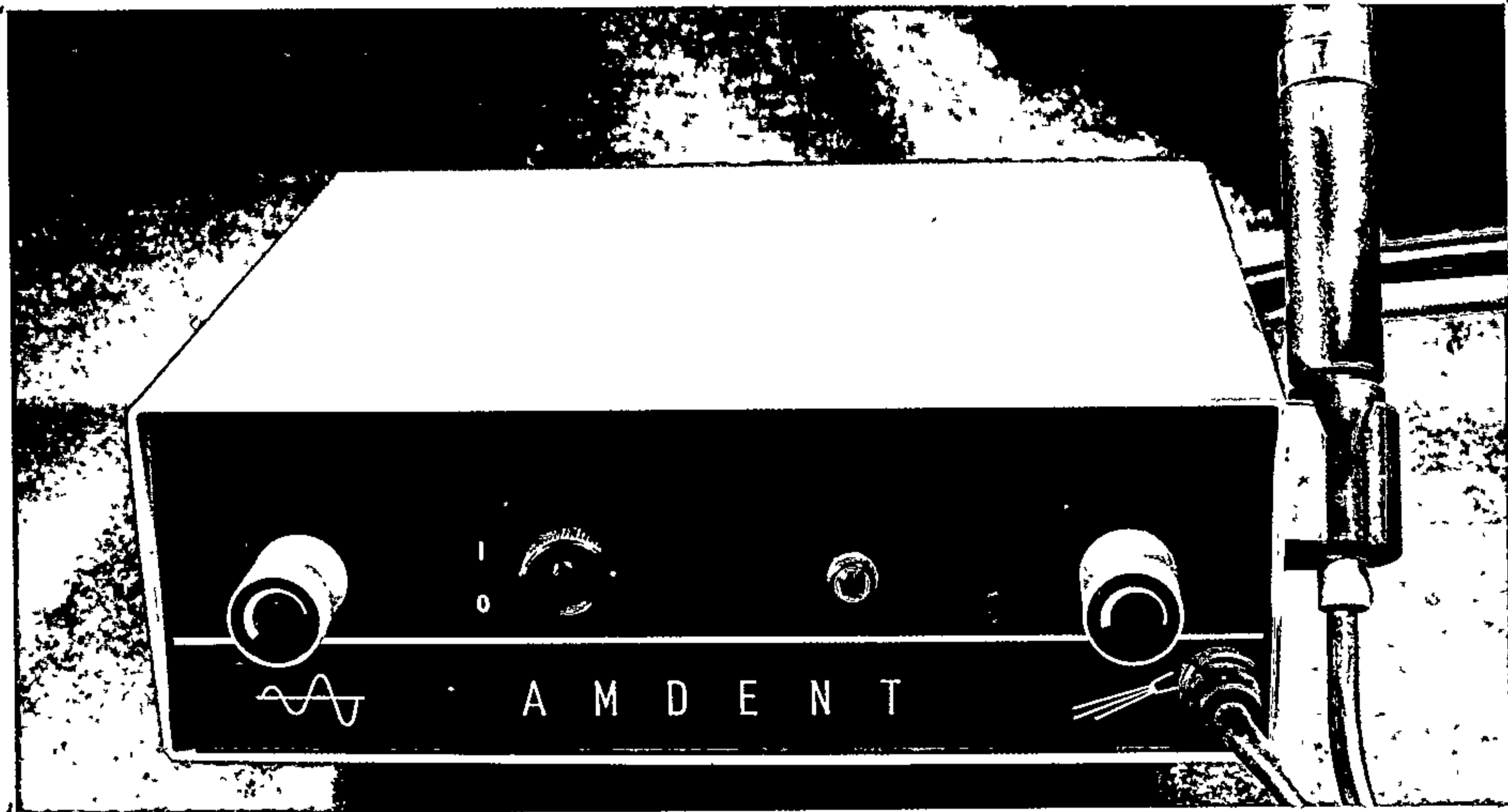


PLATE 10a - Amdent 6  
ultrasonic scaling unit



PLATE 10b - Amdent No. 18 tip.

An example of ultrasonic instruments used in the study is shown in PLATES 11a and 11b.

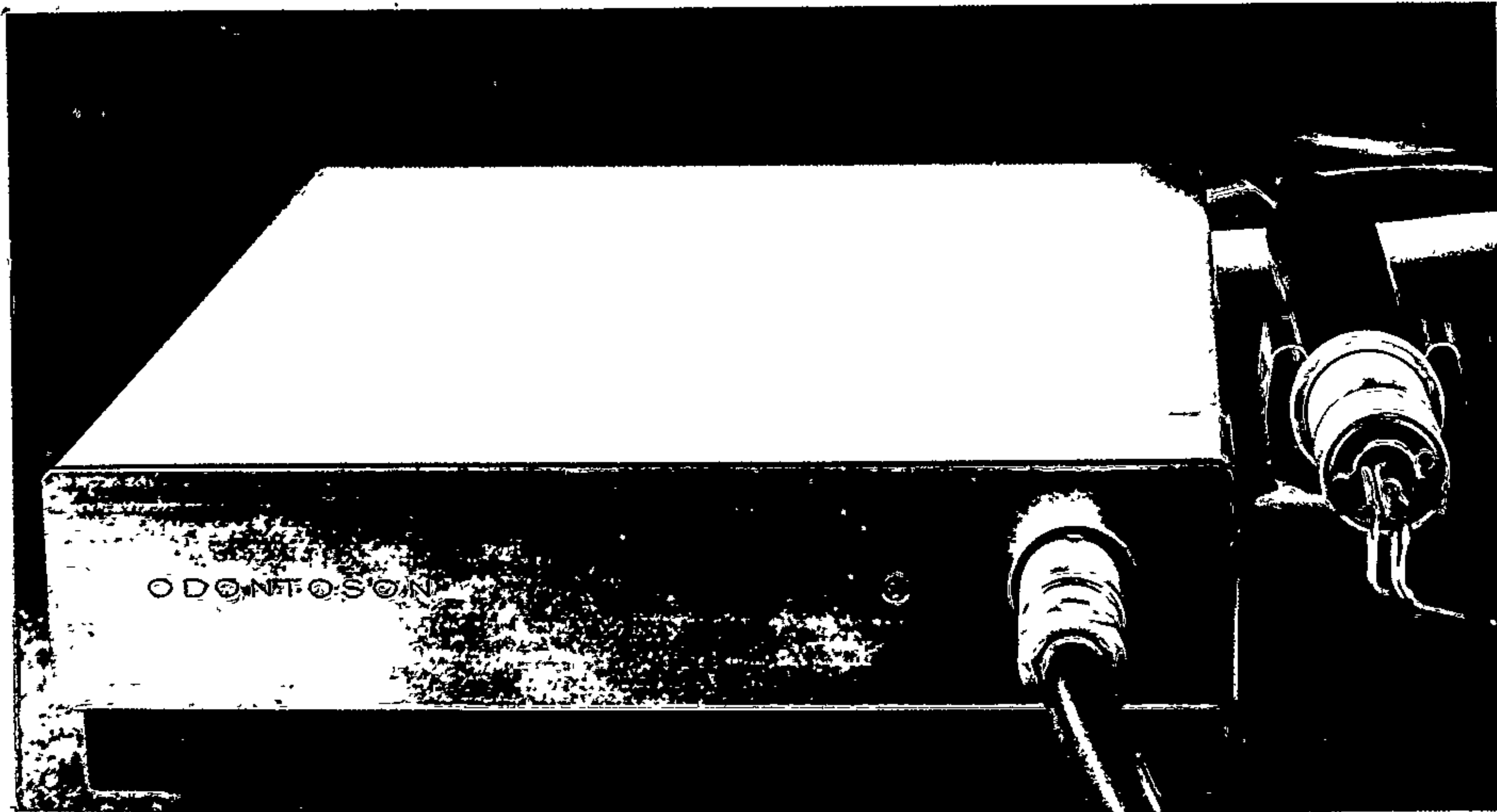


PLATE 11a - Odontoson  
ultrasonic scaling unit

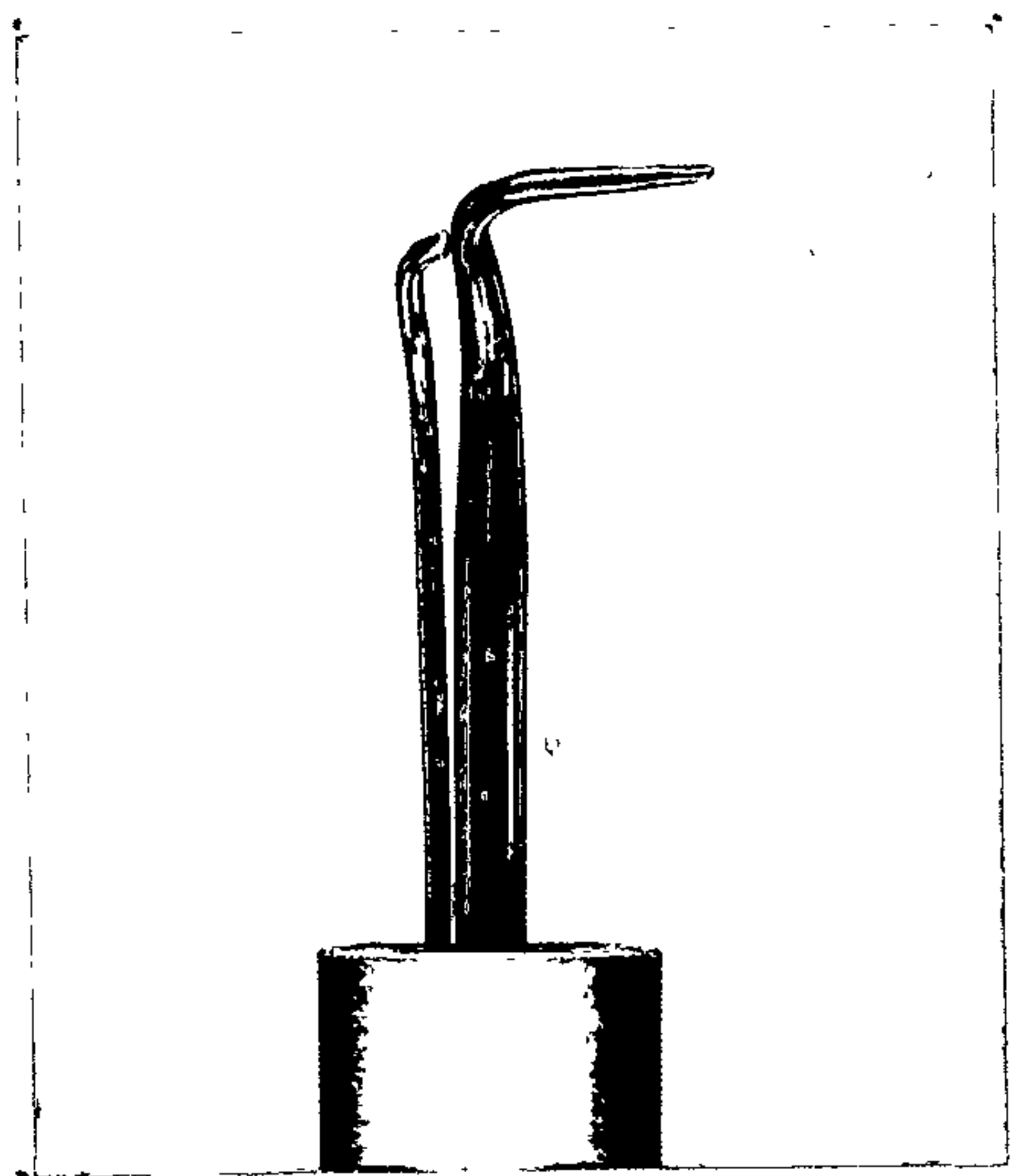


PLATE 11b - Odontoson  
contra-angled R tip

Technical data, related to the three types of ultrasonic scaling units used in these studies, is shown in Table 2.

|                                | Cavitron 700<br>II         | Amdent 6               | Odontoson                   |
|--------------------------------|----------------------------|------------------------|-----------------------------|
| Rated frequency                | 25 KHz                     | 25 KHz                 | 42 KHz                      |
| Frequency adjustment           | automatic                  | automatic              | automatic                   |
| Instrument transducer          | metal blades               | metal blades           | ferrite (ceramic) rod       |
| Specified tip motion           | elliptical                 | linear                 | rotary                      |
| Water supply to instrument tip | passage through instrument | separate tube          | separate tube               |
| Tips used*                     | "universal" TFI-10         | "universal" No. 18     | "universal" contra-angle R. |
| Amplitude Settings used**      |                            |                        |                             |
| - Lower setting                | three-quarter (medium)     | three-quarter (medium) | half                        |
| - Higher setting               | full (high)                | full (high)            | three-quarter               |

\* All tips used were kept dull

\*\* Two comparison settings for ultrasonic instruments were decided upon following preliminary studies. The Odontoson ultrasonic scaler at full setting was found clinically impractical because of patient discomfort due to vibration, and difficulty for operator manipulation. Readings of Cavitron full

(high), Amdent full (high), and Odontoson three-quarters were selected for the higher amplitude settings. Lower amplitude settings comprised: Cavitron three-quarter (medium), Amdent three quarter (medium), and Odontoson half.

In the present in vitro study, an attempt was made to achieve the smoothest surface possible, as judged clinically via unaided visual means and by tactile sense through the instrument. For the curette, an average of 10 to 15 "pull" strokes were performed in approximately the same direction. For the ultrasonic instrument, an average of 10 to 15 "back-and-forth" strokes were performed in approximately the same axis.

The portion of each instrumented test surface measured about 5mm x 5mm. Each instrumented area, and an additional 2 to 3mm either side, was excised and isolated using a water-cooled high-speed, tungsten-carbide bur; then prepared for scanning electron microscope examination. The average dimensions of each specimen for examination were 7mm (width) x 7mm (length) x 2mm (depth). (Refer Figure 3).

The preparation sequence for each tooth root specimen will now be detailed:

1. Teeth were extracted using bucco-lingual purchase to avoid damage to mesial and distal areas.
2. The teeth were immediately rinsed in running tap water to remove loose debris.
3. Teeth were fixed in 2% glutaraldehyde for a minimum period of seven days.
4. The teeth, were hand-held, instrumented in the selected areas. The teeth were then stored in 2% glutaraldehyde.

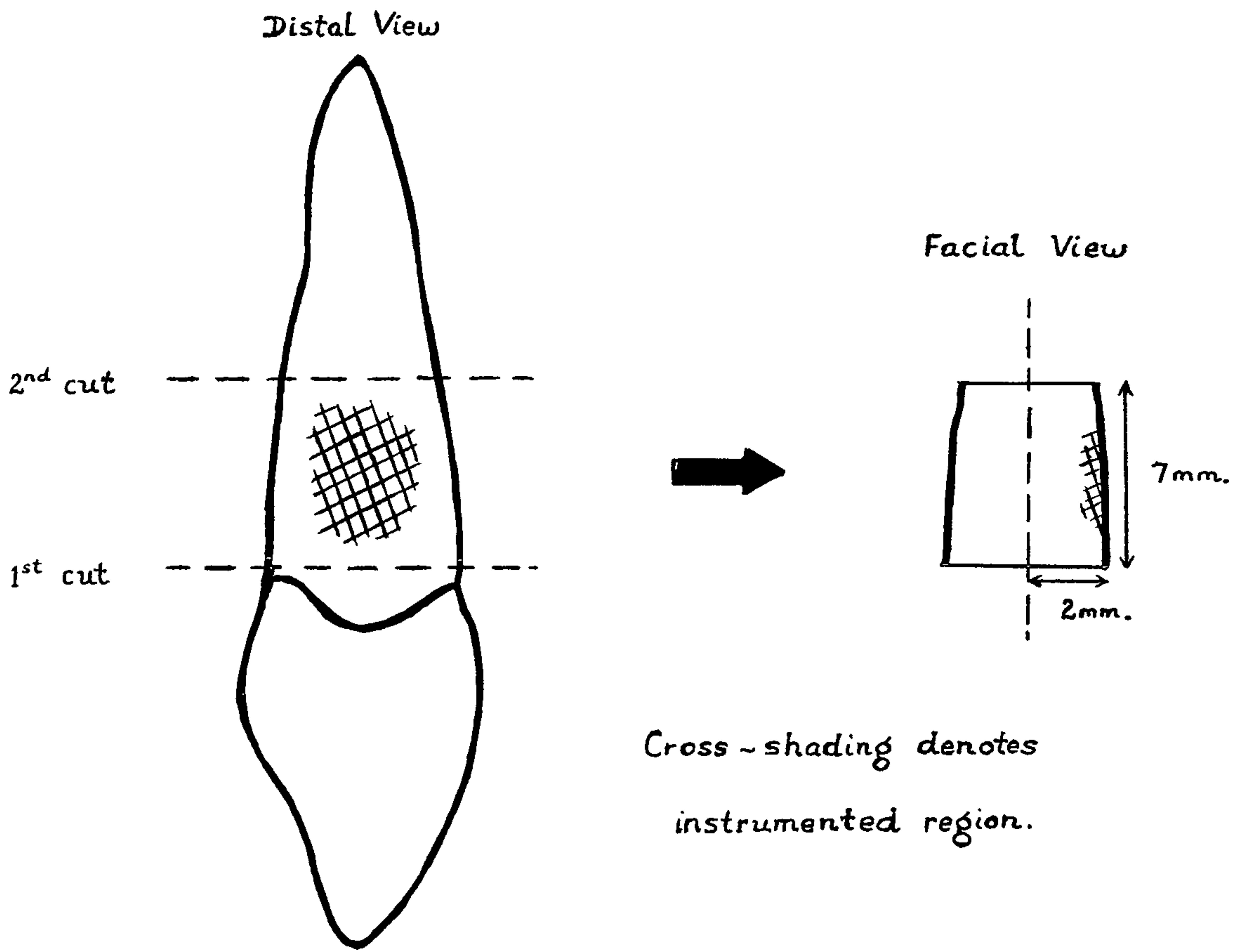


FIGURE 3 - Diagrammatic representation of sectioning of teeth to desired size for examination.

5. Fixed teeth were sectioned. (Refer Figure 3)  
Sectioned specimens were replaced in glutaraldehyde\*.
6. Specimens were then cleaned ultrasonically in distilled water for two separate five minute sessions and were transferred to trays lined with lint-free tissue paper.
7. The specimens were dried in a dessicator over a period of seven days at room temperature.\*\*
8. The dehydrated sections were mounted with silver conducting paint (colloidal silver) on a specimen stub. (Refer Plate 13).

---

\* Preliminary studies also used post-fixation of specimens in osmium tetroxide,  $\text{OsO}_4$ . The root surface following post-fixation seemed largely denuded of organic substance. The expected reduced tendency of "charging" of the specimen under scanning electron microscope examination did not eventuate. This was also reported by Kvam<sup>112</sup>.  
It was decided not to use  $\text{OsO}_4$ .

\*\* Preliminary studies used graded acetone solutions to facilitate dehydration. However total air drying of specimens over a longer period of time was found equally effective. In fact, the more gradual process may help decrease cracking of acellular cementum. Jones, Lozdan, and Boyde<sup>93</sup> state, however, that some crazing of acellular cementum surfaces appears to be an inevitable consequence of any drying technique because of stresses built up between tissues of different structural arrangements. (Refer Plate 12)



PLATE 12 - Illustrates cracking of acellular cementum (top right), as well as the splitting apart of the functional plane between cementum and dentine.

(200x)

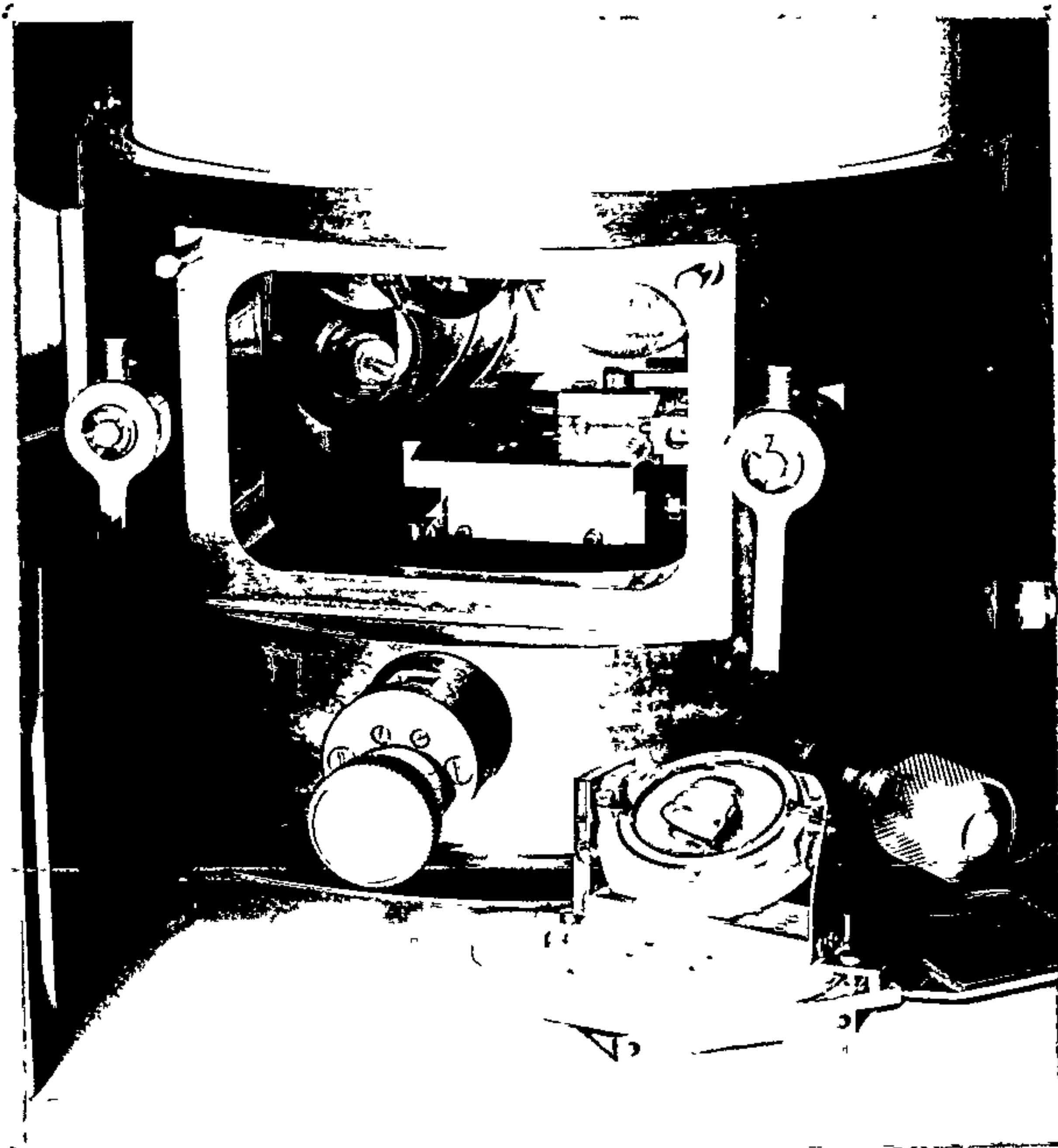


PLATE 13 - Specimen stub ready to be introduced into vacuum chamber of scanning electron microscope.

9. The specimen received a layer of carbon, followed by a coating of either gold, gold-palladium, or aluminium (approximately 200 to 400 Angstroms thickness). A uniform coating was achieved by rotating and tilting the specimen through a series of angles to the filament in the Dynavac coating machine.
10. The specimens were examined in the Cambridge Stereoscan 600 scanning electron microscope, (Refer Plate 14), operated between 7.5 KV and 25 KV of the beam voltage, using secondary electron images.
11. Secondary electron images were recorded on Kodak Panatomic - X (ASA 32) film.

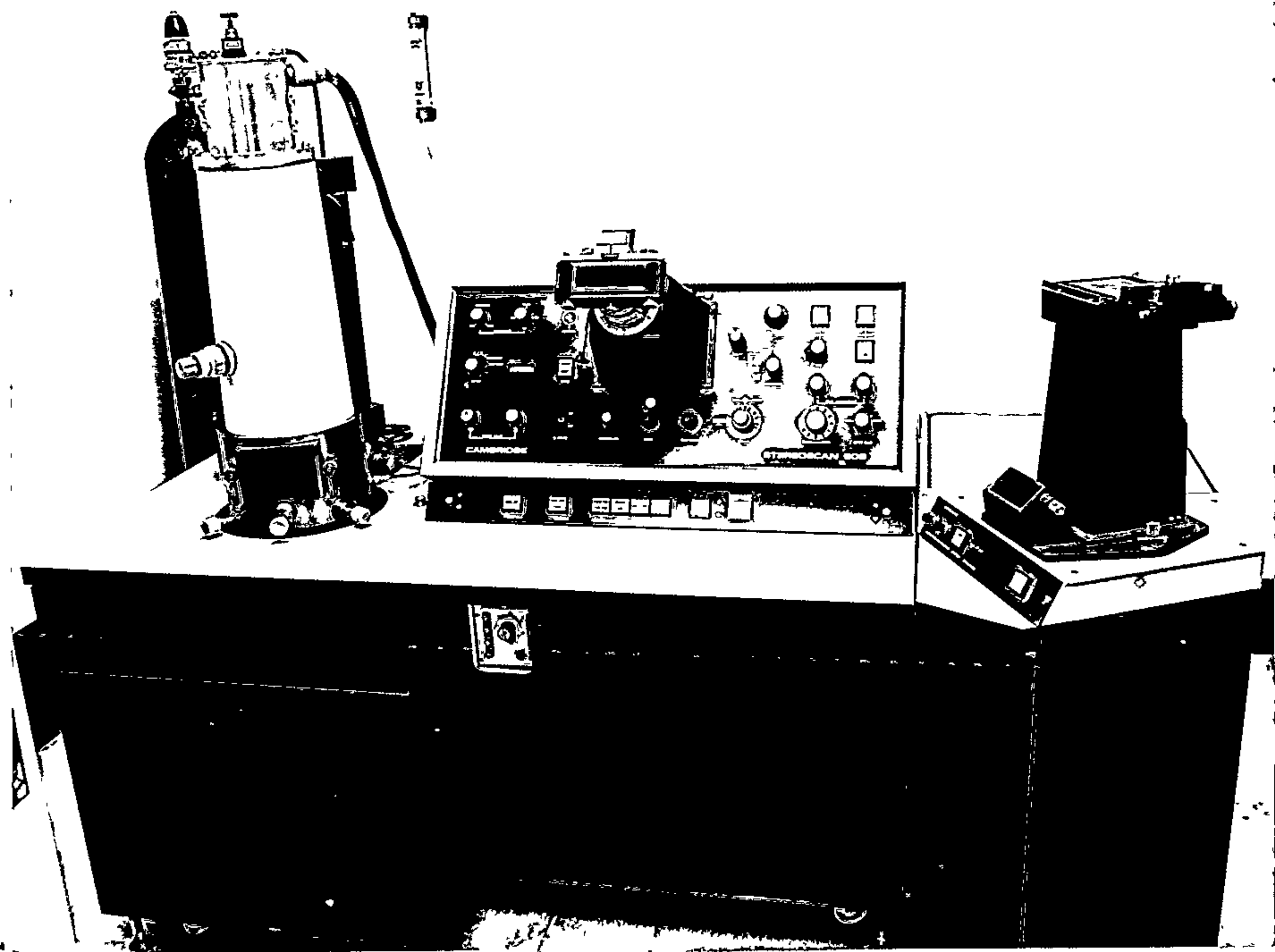


PLATE 14 - Cambridge Stereoscan 600 scanning  
electron microscope.

Study 2

Similar procedures to Study 1, using polished acrylic surfaces\*, were carried out in this study.

Sixteen highly polished labial faces of plastic Frasco teeth were used. Two of these prepared surfaces were used as controls.

- 
- \*. The desirability of a standardized model root surface came about because of the conflicting results which have been reported concerning the relative usefulness of hand versus ultrasonic instruments on tooth roots<sup>39, 66, 93, 100, 147, 163, 262, 271</sup>. Variability attributed to tooth root surfaces include the following items:
1. The type of tissue evaluated may vary. Tissue may be predominant Sharpey fibre cementum or partial Sharpey fibre cementum, or it may be dentine. Sharpey's fibre mounds may mask changes produced on the surface by the instrument. The root surface may have been exposed to the oral environment, or it may be covered with periodontal fibres. Damage due to previous instrumentation may or may not have existed.
  2. The physical properties of the tissue may vary. These include hardness, scratch resistance, abrasion resistance, and acoustic impedance. These properties may

vary with age, state, and duration of the disease, from person to person, from tooth to tooth in the same person, and from area to area in the same tooth.

Polished acrylic surfaces, it was hoped, could be used as an easily controlled, homogeneous, model surface to test the potential for periodontal instruments to damage tooth roots.

Allocation of instruments to acrylic surfaces is shown in Table 3.

TABLE 3

| <u>Instrument used</u>                    | <u>Number of specimens</u> |
|---|----------------------------|
| . Sharp curette                           | 2                          |
| . Cavitron three-quarter (medium) setting | 2                          |
| . Cavitron full (high) setting            | 2                          |
| . Amdent three-quarter (medium) setting   | 2                          |
| . Amdent full (high) setting              | 2                          |
| . Odontoson half setting                  | 2                          |
| . Odontoson three quarter setting         | 2                          |
| Total                                     | = 14 (plus 2 controls)     |

For the curette, 15 "pull" strokes were performed in the one direction. For the ultrasonic instrument, 15 "back-and-forth" strokes were performed in approximately the same axis of direction.

The plastic teeth were then prepared in blocks the same dimensions as in Study 1, and examined as in Preparation Steps (6) to (11). However, in the case of the acrylic specimens, reflected primary electron images were used instead of secondary electron images. This was found to give better three dimensional images of acrylic specimens which have comparatively homogeneous structures and features.

## Presentation of Results

It was decided to combine the results of Study 1 and Study 2 to aid continuity and comparison.

The entire surface of the mounted specimen could be viewed on the scanning electron microscope at a magnification of 20x. At this setting, gross surface topography of the specimen could be assessed. (Refer Plate 15)

Additional surface detail was evaluated at 1000x magnification.

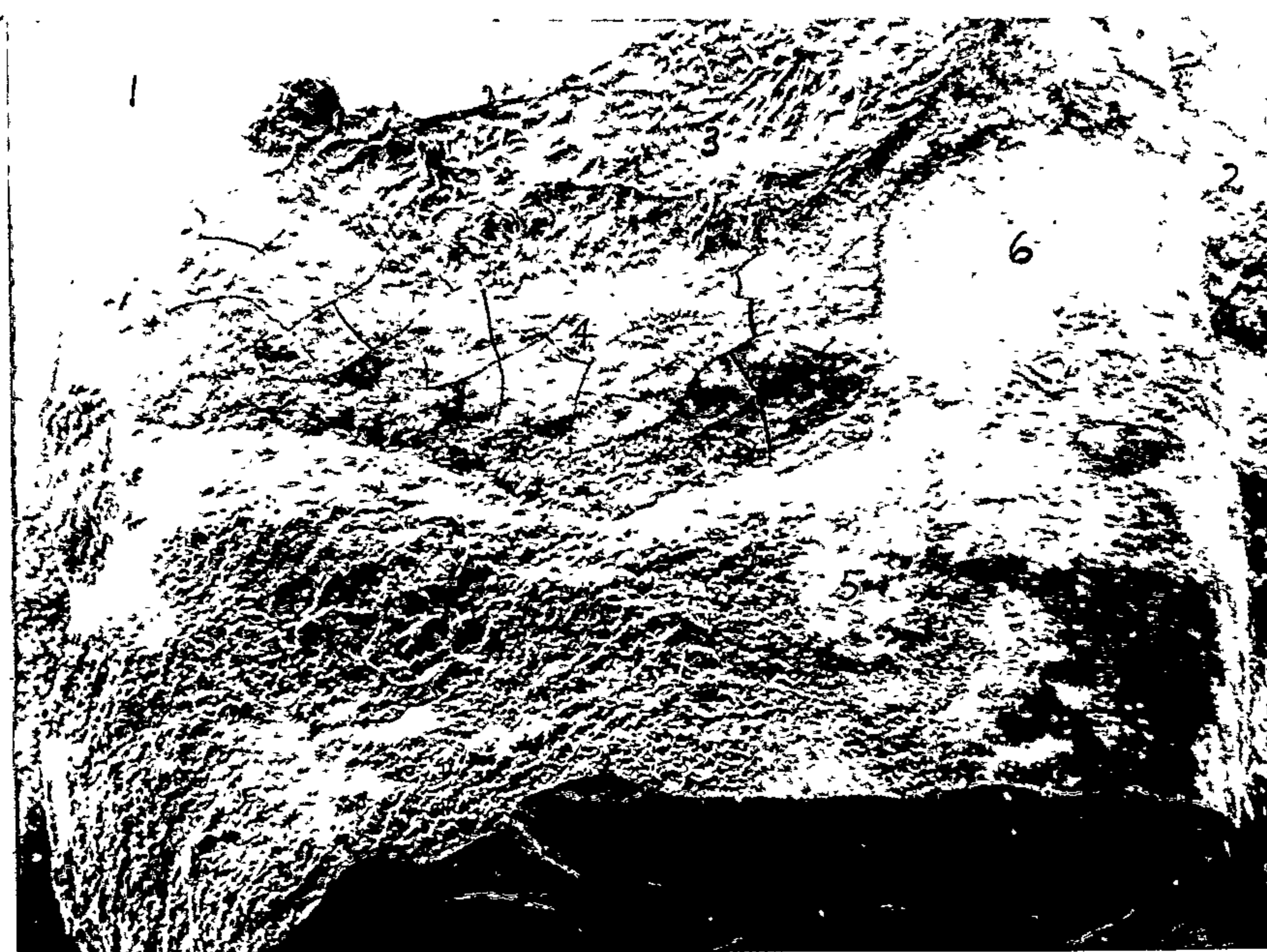


PLATE 15 - Sample of mounted root section (instrumented with curette). Shows typical appearance of: 1. metal specimen stub; 2. conducting paint used to secure specimen; 3. adherent periodontal fibres; 4. acellular cementum; 5. cellular cementum; and 6. dentine.

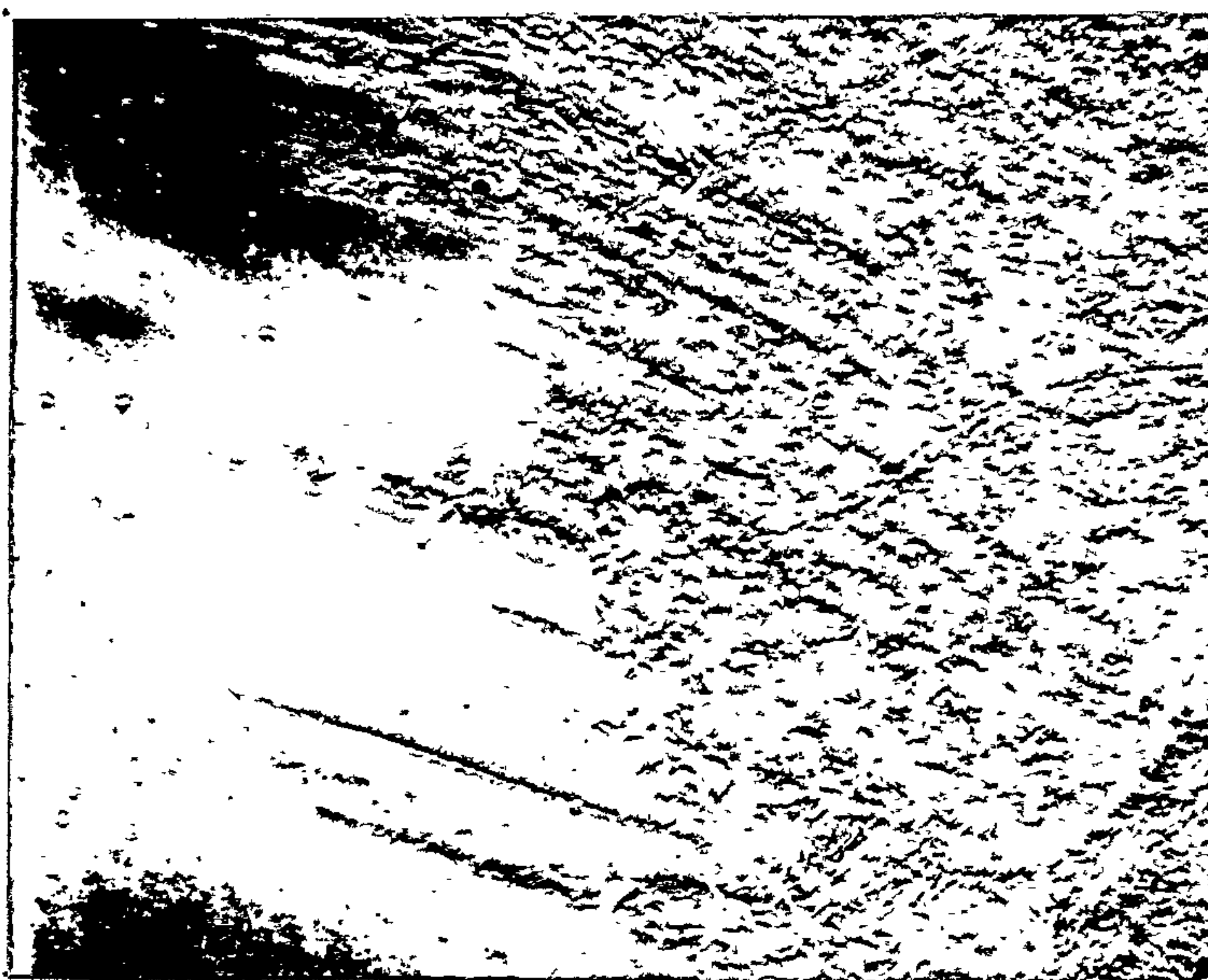
In the electron images to follow, the area shown at higher magnification (1000x), was selected in the vicinity of the red indicator dot shown in the lower power micrograph. (20x)

A range of magnification between 20x and 1000x were used to select and localize representative areas. An example of area localization is shown in Plate 16 (a) (b) (c) (d).

PLATE 16 - Acrylic surface following Cavitron at  
high setting.

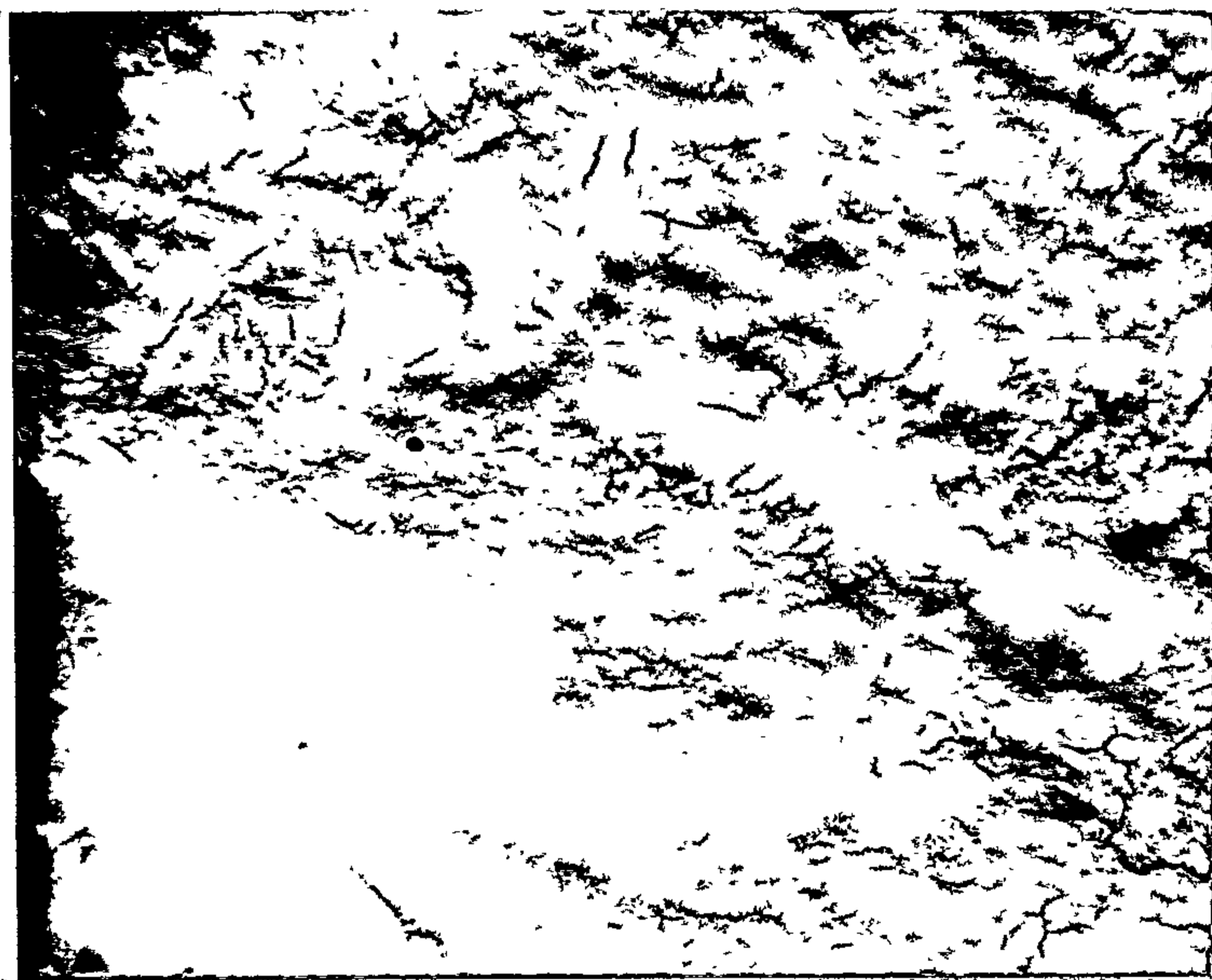


16 (a) 20x

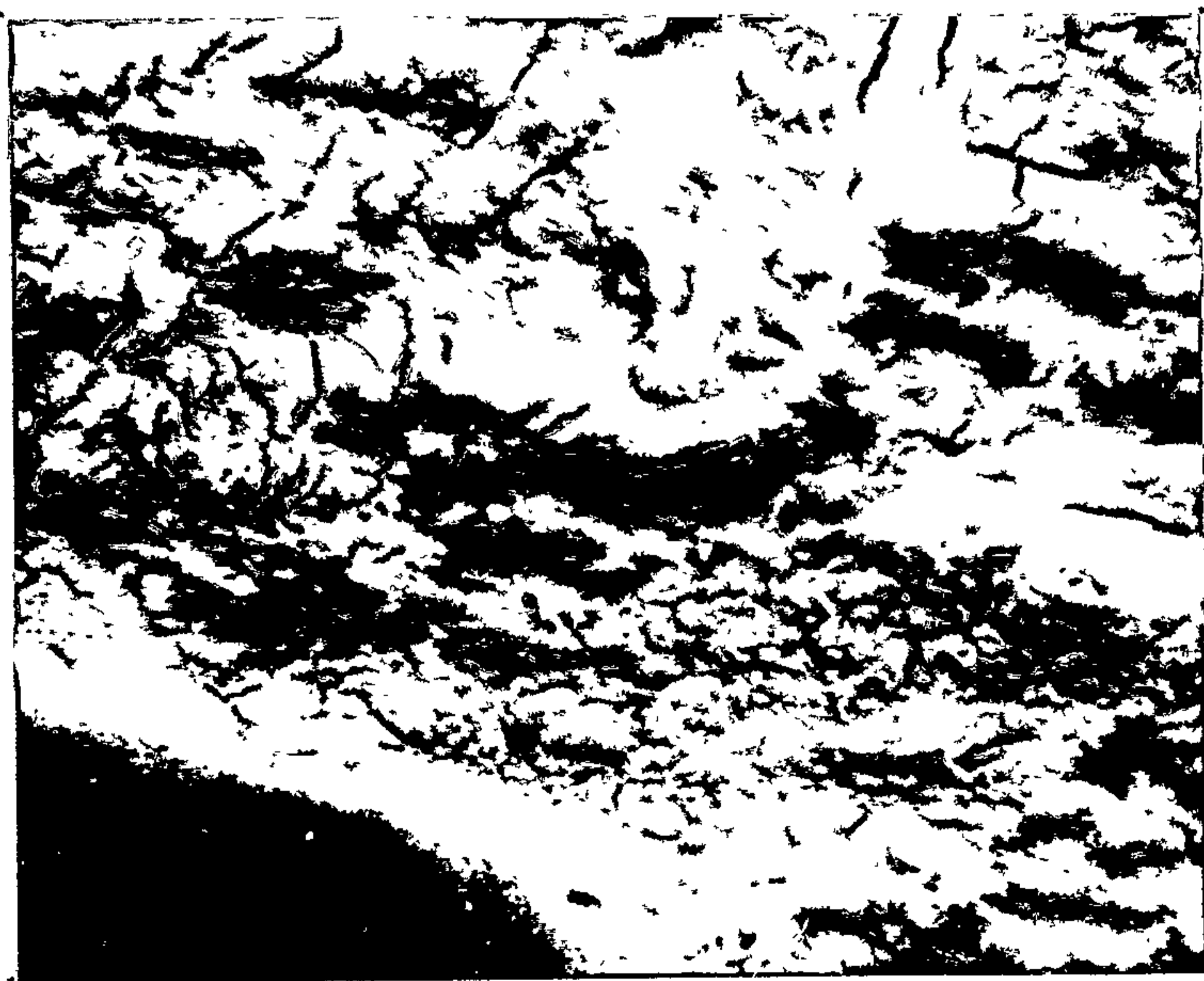


16 (b) 100x

PLATE 16 (continued) - Acrylic surface following  
Cavitron at high setting.



16 (c) 500x



16 (d) 1000x

RESULTS

In the following electron micrographs, the area shown at higher magnification on the bottom of the page (1000x), was selected in the vicinity of the red indicator dot shown in the lower power micrograph (20x) on the top .