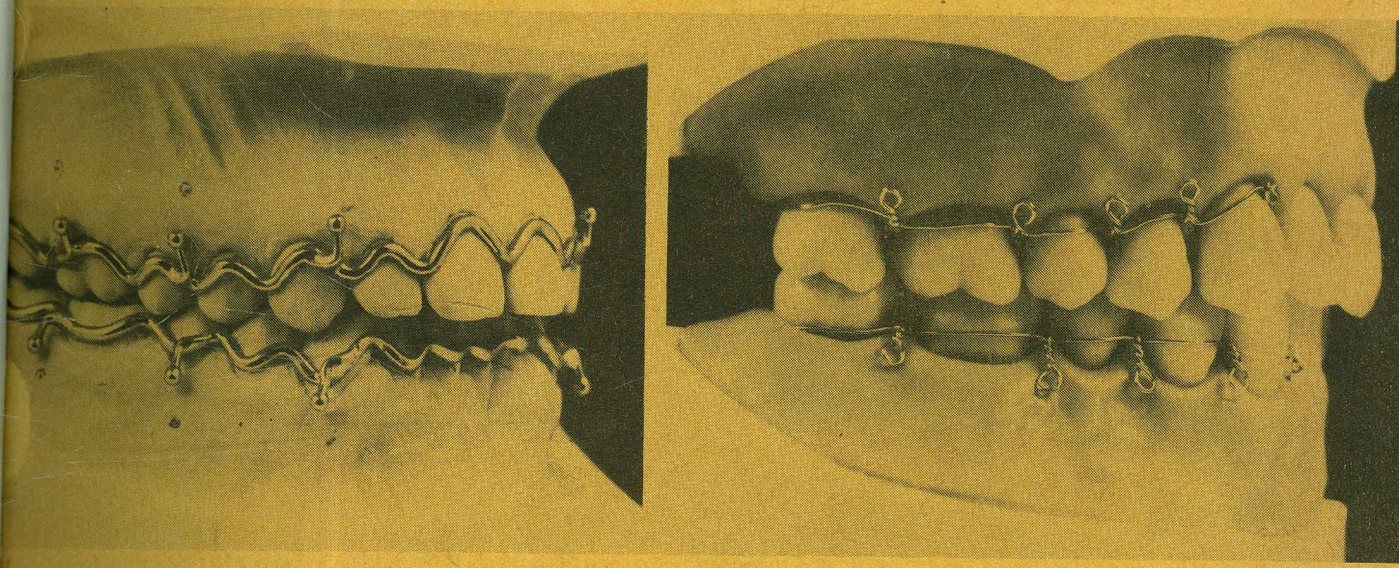
THE REACTION OF PERIODONTAL TISSUES TO SPLINTS USED IN MAXILLO-FACIAL SURGERY



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THE REACTION OF PERIODONTAL TISSUES TO SPLINTS USED IN MAXILLO-FACIAL SURGERY

by

Dr. David Nazareno Bernard Ngassapa, D.D.S. (Romania)

A dissertation submitted in partial fulfilment for the degree of Master of Science (Oral Morphology) in the University of Dar-Es-Salaam, 1983.

FOR REFERENCE ONLY

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I. INTRODUCTION

1. Definition and use of splints

The word splint is often carelessly used in the literature and is frequently taken to mean only those rigid appliances used in the fixation of fractured jaws.

In this dissertation the word will correctly be used as defined in the Heinemman Modern Dictionary for Dental students. Here a splint is defined as an appliance which may be rigid or flexible, used to immobilize the ends of fractured bones or to restrict the movement of joints. A dental splint is defined as any form of appliance or device used to fasten and immobilize the teeth. Therefore splints used in maxillo-facial surgery include all the appliances in the form of wire ligatures, arch bars (flexible or rigid) and acrylic devices used in the treatment of jaw fractures as well as fixation after jaw osteotomies. The splints are usually applied to teeth, the alveolar processess and other skeletal parts of the face. Their function is to immobilize the fractured or osteotomized jaws, maintain them in a reduced position, and facilitate intermaxillary fixation if so desired.

2. Types of splints

A short review of the most commonly used splints and some of their modifications will be given. It must be noted that it is beyond the scope of this study to give an exhaustive oversight of all splints, all modifications, their history and use.

a. Wire ligatures:

These can be used singly or as continuous wire ligatures. The following are the most commonly used wire ligatures:

i) Gilmer's direct interdental wiring (Gilmer, 1887).

It is the simplest form of ligature. In the maxilla and mandible, single wire ligatures are applied with 0.2mm. soft stainless steel wire. For intermaxillary fixation, the maxillary wires are wound together with the mandibular wires (Fig. 1 A and B).

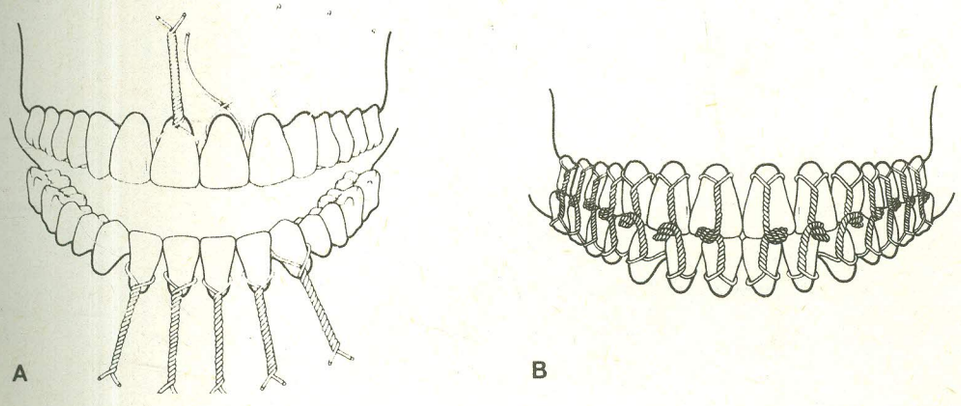


Fig. 1 A and B: Gilmer's direct interdental wiring.
(With kind permission of the Quintessenz Verlags-GmbH, Berlin, quoted from the book by Professor Krüger and Professor Schilli "Oral and Maxillofacial Traumatology, Vol. I".)

According to a recommendation by Hering (1960), the entire vestibular dental arch can be strengthened with cold-curing acrylic after placing the ligatures.

ii) Ernst's ligature (1972)

It is frequently used for emergency fracture immobilisation.

Two adjacent teeth are looped in a figure of 8 with a 0.4mm. diameter stainless steel soft wire. One end of the wire is drawn from the vestibule through the anterior interproximal space, the other through the posterior interproximal space. Together they are returned to the vestibule through the central interproximal space. The ends, twisted together over the vestibular wire loop, are cut to approximately 5mm. and adapted to the gingival margin so that a hook is formed to take up intermaxillary tie wires or rubber bands (Fig.2).

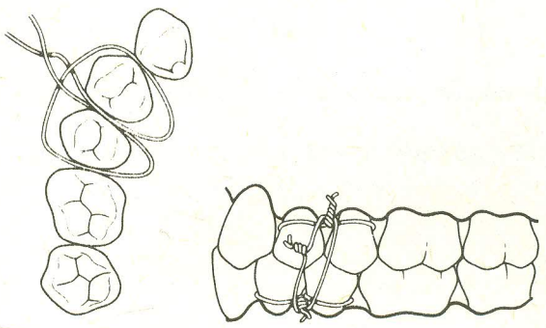


Fig. 2: Ernst ligature.
(With kind permission of the Quintessens Verlags-GmbH, Berlin, quoted from the book by Professor Krüger and Professor Schilli "Oral and Maxillofacial Traumatology, Vol I".)

The hook may be coated with cold-curing acrylic for better stabilization (Grimm, 1969). Also, not infrequently, the wire ends from opposing maxillary and mandibular ligatures are twisted as in Gilmer's method.

iii) The Kazanjian button (1933).

As a modification of the Ernst ligature is made from two individual

ligatures from adjacent teeth. The two twisted wires are retwisted together into a small button that serves so hold intermaxillary ligatures or rubber bands.

iv) The eyelet method (Eby, 1920, Ivy, 1922).
Also may be used for intermaxillary fixation in emergency situations. A wire is twisted about an instrument e.g. a bur, to establish a loop. Both ends of the wire are drawn through the interproximal space of two adjacent teeth so that the loop lies vestibularly. One end of the wire is then drawn around the anterior tooth, the other around the posterior tooth. The posterior wire end is passed through the loop or between the interdental wires and twisted together with the anterior wire. Tie wires are drawn through the loops of the opposing upper and lower eyelet wires for intermaxillary fixation (Fig. 3).

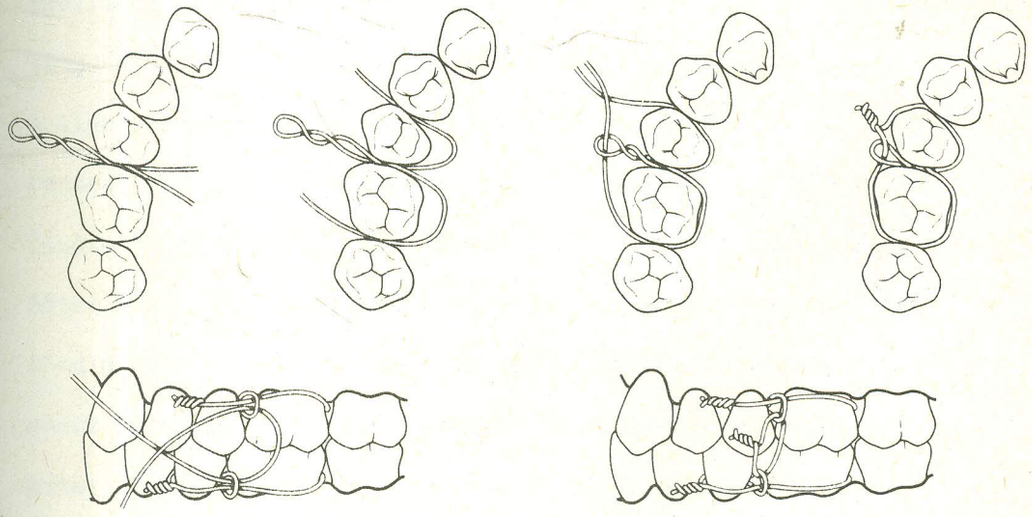


Fig. 3: The interdental eyelet wiring (Eby, 1920, Ivy, 1922).
(With kind permission of the Quintessenz Verlags- GmbH, Berlin, quoted from the book by Professor Krüger and Professor Schilli "Oral and Maxillofacial Traumatology, Vol I").

v) Continuous or multiple-loop wiring (Stout, 1943, Obwegeser, 1952). The wiring is undertaken with a 0.5mm. soft stainless steel wire. The buccal end is adapted to the dental arch and should only be long enough to reach the other side, proceeding somewhat beyond the midline. The lingual end, which must be significantly longer, is then brought through the adjacent mesial interdental space in a lingual direction. (Fig. 4 A). The end of a pliable round rod, approximately 3-4mm. thick, which has previously been adapted to the dental arch, then is inserted into the loop. The lingual wire is pulled tight and led in a buccal direction through the next interdental space (Fig. 4 B), brought around the rod and once again led through the same interdental space. In this manner all the teeth of a quadrant are encircled by the lingual end of the wire. At the midline, the lingual end is only led through to the labial side and not back again. It is loosely twisted with the buccal end. Then the round rod is pulled out with a rotating motion (Fig. 4 C). The individual uniform loops are now tightened with a ligature forceps by a few twists above the buccal wire, which caused this to be pressed into the interdental space and the lingual wire to be firmly adapted to the tooth cervix (Fig. 4 D). After tightening, a smaller loop should remain behind. This should be bent back in an occlusal direction to provide a means for receiving the tie wires which are to be applied after the fracture ends are reduced (Fig. 4 E). If a progressive reduction is desired, the loops are bent back toward the gingiva to hold elastic bands (Fig. 4 F).

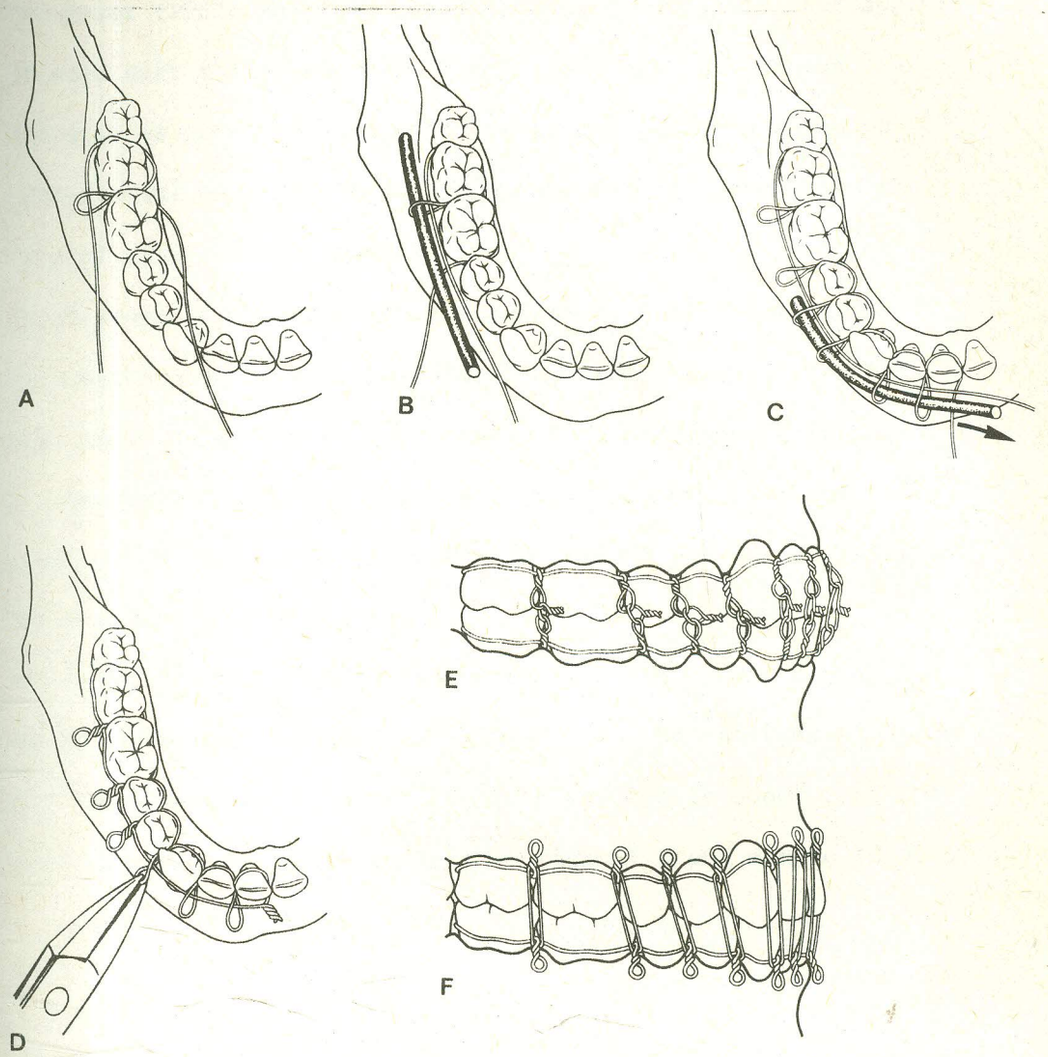


Fig. 4 A to F: Stout's continuous or multiple loop wiring

Fig. 4 A to B: The interdental loops are placed over a buccally adapted rod and around the buccal wire end.

Fig. 4 C: When the loops have been completed on one side, the rod is withdrawn.

Fig. 4 D: The loops are tightened with a ligature pliers.

Fig. 4 E: Intermaxillary fixation with tie wires.

Fig. 4 F: Intermaxillary fixation with elastic rings. The loops are bent gingivally for this purpose.

(With kind permission of the Quintessenz Verlags-GmbH, Berlin, quoted from the book from Professor Krüger and Professor Schilli "Oral and Maxillofacial Traumatology, Vol. 1".)

Obwegeser (1952) modified the technique of applying this splint. He used soft stainless steel wire 0.5 - 0.7mm. thickness. The wire is preformed, so that the interdental loops can be pulled simultaneously through the interdental spaces with the help of silk threads (Fig. 5A).

Round loops are formed from the two strands of the interdental wire which have been pressed together with a special forceps. One end of this forceps is fitted with a hole and the other with a spike which penetrates it. With this, the distal end of the wire is led forwards along the arch (Fig. 5 B) and twisted with the mesial end of the wire.

The loops are tightened with the special forceps on top of the buccal wire, whereby this is pressed into the interdental space, and the lingual part of the ligature is tightly adapted to the neck of the tooth, just as in the original Stout method (Fig. 5C).

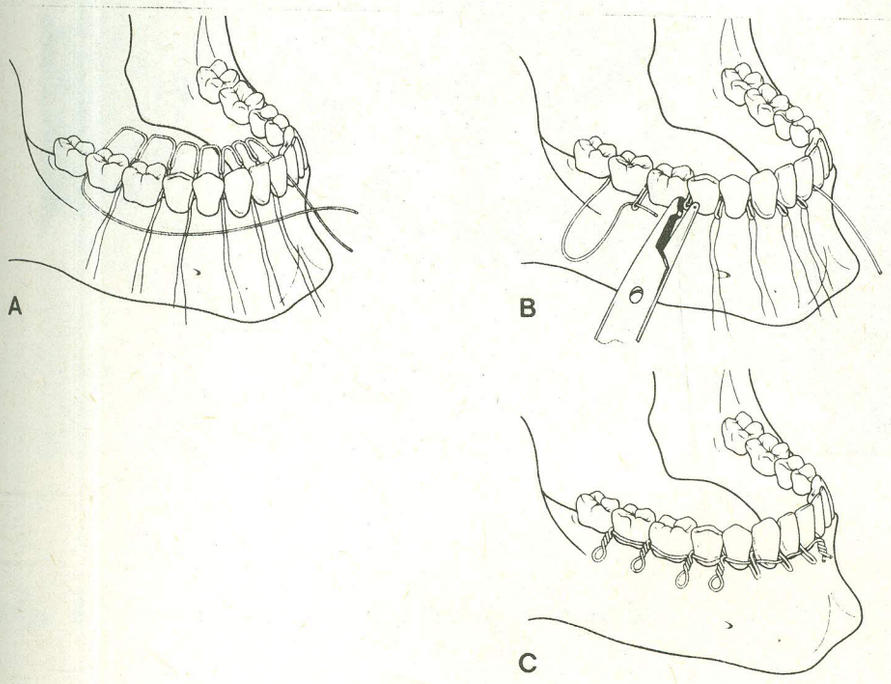


Fig. 5 A to C: Obwegeser's placement technique for continous loop wires

Fig. 5 A: Pulling the pre-shaped loops trough the interdental spaces with wilk threads.

Fig. 5 B: Pulling the buccal end of the wire through the loops.

Fig. 5 C: Tightening the loops.

(With kind permission of the Quintessenz Verlags-GmbH, Berlin, quoted from the book by Professor Krüger and Professor Schilli "Oral and Maxillofacial Traumatology, Vol 1".)

N/B Usually the loops are no longer placed with threads since 15 years ago. They are just pushed with the fingers. (Personal communication, Freihofer, 1983).

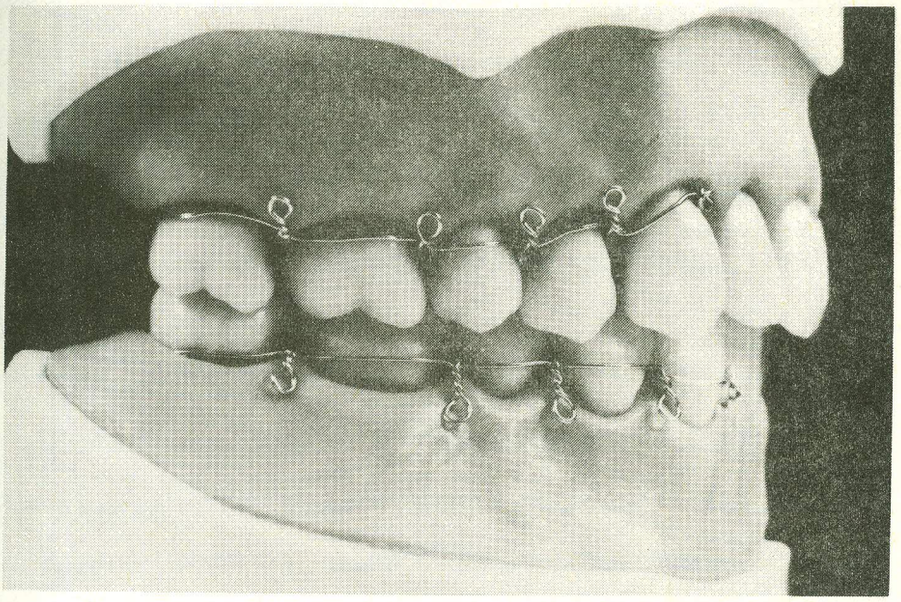


Fig. 5 D: Obwegeser splint fixed on a model.

b. Arch bars:

The most commonly used arch bars are:

i) Sauer's arch bar (1889)

This is a simple arch bar without hooks attached to it. A thick wire is pre-bent to fit the gingival contour (this is more easily done on a model) and attached to each tooth with two single ligatures. The twisted portions of the two single ligatures, which are adjacent to each other can remain about 5mm. long and can be bent apically. In this way small hooks are formed for the attachment of elastic bands for intermaxillary fixation.

ii) Hauptmeyer's arch bar (1924)

This is a modification of the Sauer's arch bar in the sense that the arch bar itself is contoured into "hooks" at every interdental papilla.

iii) Jelenko arch bar (Quoted by Krüger and Schilli, 1982).

iv) Winter arch bar (1943)

v) Erich arch bar (1941)

vi) Niro arch bar (Quoted by Krüger and Schilli, 1982).

These are prefabricated arch bars of malleable metal and have small hook attachments. These hooks serve to receive elastic bands or tie wires for intermaxillary fixation (Fig. 6).

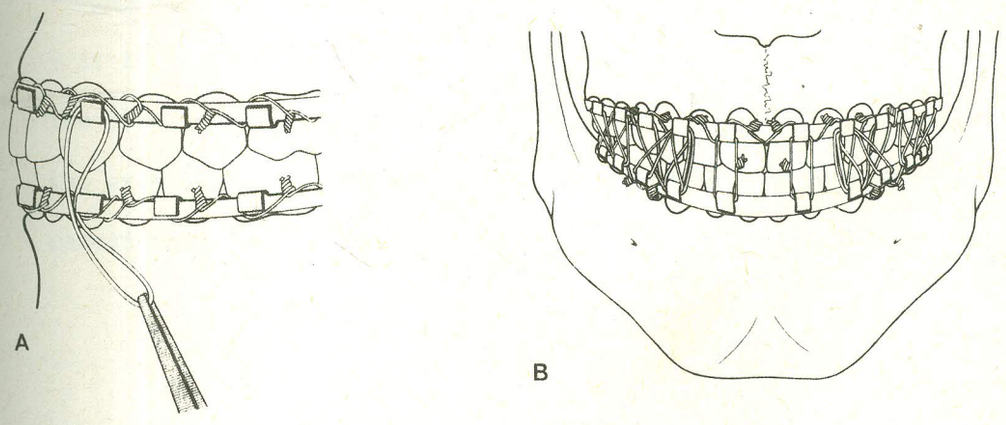


Fig. 6: Erich arch bars with intermaxillary fixation.
(With kind permission of the Quintessenz Verlag-GmbH, Berlin, quoted from the book by Professor Krüger and Professor Schilli "Oral and Maxillofacial Traumatology, Vol 1".)

Essentially these arch bars differ in their types of hooks attached to them.

vii) The Merkx individual prefabricated arch bar (unpublished). The arch bar was introduced in practice by Prof. C.A. Merkx of the Department of Oral and Maxillo-Facial Surgery of the University of Nijmegen in 1959.

Technique of making the arch bar:

- An impression is taken from the patient's mouth for both the upper and lower jaws and sent to the technican who casts models.
- A semi-roundish stainless arch bar of 1.5 mm diameter and as long as the jaw dental arches is softened by heating over a bunsen burner flame and left to cool.
- It is then contoured over the buccal surfaces while following the exact contour of the gingival margin.
- Cusps are welded at several places on the arch bar.
- It is then polished.

The most important feature particular to this splint is that the arch bar is contoured to follow precisely the gingival margin (fig. 7).

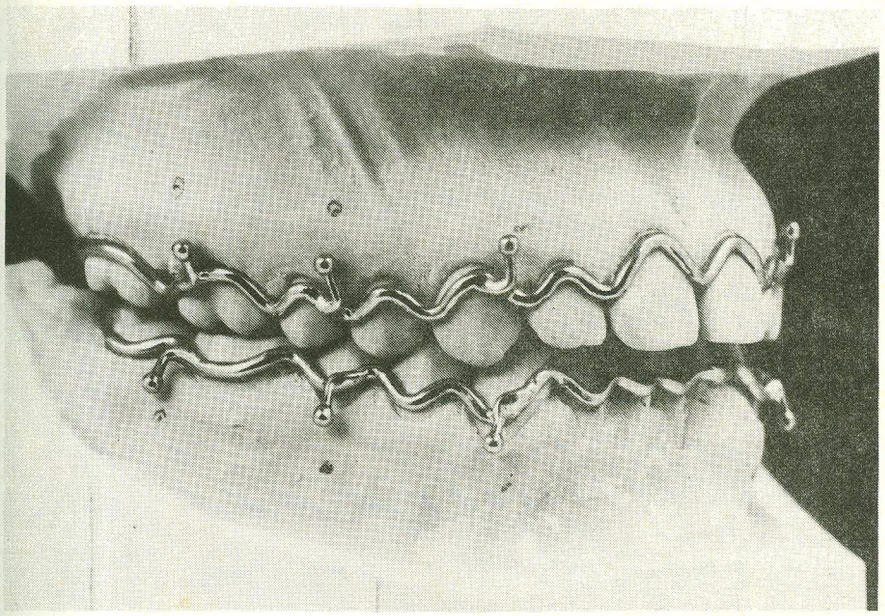


Fig. 7: The Merkx individual prefabricated arch bar.

viii) The wireacrylic arch bar (Schuchardt, 1956).
This is made from a half round soft stainless arch wire with cross wires soldened to it. The arch wire is adapted to the dental arch and the occlusal ends of the cross-wires are curved over the occlusal surface of the teeth. Thus, the bar is supported occlusally and cannot slip in a gingival direction. Cold-curing acrylic is then applied to its buccal surface.

c. Cap splints:

The following are most commonly used cap splints:

1. Cast acrylic splints

i) The mandibular lingual splint (Hofer, 1939)

This is the simplest kind of acrylic splint. It adjoins the alveolar process and the dental arch on the lingual side, but leaves the occlusal surfaces free. Fixation is ensured by ligature wires on the teeth. The splint is provided with holes, drilled at each interdental space through which wire ligatures are pulled.

ii) Sectional splint (Kersting, 1904)

consists of lingual and buccal portions which grip the alveolar process and the dental arch on both sides.

2. Cast metal splints

i) Cap splint

This is a splint which holds each individual tooth with a cap. It can be one piece or different segments which are reassembled by locking pieces.

ii) Open cap splint

This is a modified cap splint in the sence that it leaves the occlusal surfaces exposed.

d. Prosthetic and orthodontic splints

Although these types of splints are basically different from the ones so far discussed, they will not be presented here since they are normally used in very specific situations.

3. Advantages and disadvantages of different types of splints

a. Wire ligatures:

Advantages:

- Only steel wire and a few instruments are needed. There is no need of laboratory work and a technican; therefore these splints are cheap.
- Application always takes place directly in the patient's mouth and therefore the time required for preparation is minimal and the wire ligature can be used in emergency situations.
- They permit leeway in treatment when occlusion is not definitively determined.

Disadvantages:

- Because of their limited stability, they are unsuitable for monomaxillary fixation or fractures of the alveolar process unless they are reinforced with acrylic.
- In case of interdental wiring (e.g. Gilmer's direct interdental wiring (1887); Ernst ligature (1927) ; and the eyelet wiring) all the force is applied on certain teeth that bear most of the strain (Thoma, 1969).
- In case of the Stout (1943) - Obwegeser (1952) wiring, during its application, if the wire breaks, then the whole process has to be repeated (Harper, 1977).
- Periodontal damage may occur (Krüger and Schilli, 1982).
- They are difficult to apply in case of deciduous dentition.

b. Arch bars:

Advantages:

- Offer more stability to the immobilized fragments.
- In the case of prefabricated arch bars of malleable metal (Jelenko, Winter, (1943), Erich (1942), and Niro) there is no need of laboratory work as application always takes place directly in the patient's mouth.
- These splints can be used in emergency situations.
- They permit to a certain extent some leeway also.
- Can be used for mono- and bimaxillary fixation.
- The Merkx (1959) prefabricated individual arch bar is totally passive and in this way has no orthodontic effect on the teeth. Furthermore like any other prefabricated individual arch bar, it can be used as a guide in osteotomy operations and in the reduction of jaw fractures. In the case of osteotomies a mock-operation is done on plaster models to establish the desired dental relationship.
  The splint is then constructed on the models. During the real osteotomy, the desired correct osseous relationship is considered achieved when the splint fits correctly.

Disadvantages:

- Damage to enamel and periodontium can occur (Krüger and Schilli, 1982).
- In case of the Merkx (1959) prefabricated individual arch bar there is a need of laboratory equipment and a qualified technician; this makes the splint expensive. It is also time consuming to make and therefore cannot be used in emergency

situations.

- They permit little leeway compared to wire ligatures.
- Prefabricated splints which are moulded in the patient's mouth may have an orthodontic effect on teeth.

c. Cap splints

Advantages

- Cap splints afford a more satisfactory and stable means of effecting immobilization than any other type of splints. This is achieved by virtue of the absolute immobility of the properly constructed and affixed splints, and the fact that the whole crown of the tooth is enclosed within the capsplint. Bucco-lingual rotation about a vertical axis, is therefore less liable to occur.
- Can better be used in case of deciduous dentition, because they can additionally be stabilized with circumferential wires.
- Do not touch the gingiva.

Disadvantages

- Between the articulating surfaces of the teeth are interposed two thicknesses of splint metal and two layers of the cementing medium and these layers can bring about occlusal and articulating error. But this disadvantage can be overcome by the use of open cap splints.
- They are time consuming to make and therefore cannot be used in emergency situations.
- They are very expensive.
- They permit no leeway for adjustments in monomaxillary fixation.

II. STATEMENT OF THE PROBLEM AND AIM OF THE PRESENT INVESTIGATION

Modern technology has developed more splints for the use in the treatment of jaw fractures and osteotomies than ever before. One may review the literature only to find that it is not uncommon to come across a situation where one author advocates the use of a particular type of splint for a given case whereas another advocates a completely different splint for the same situation without any particular reason. It is possible that a surgeon uses a particular type of splint because of sheer habit, or for technical or financial reasons rather than serious considerations being taken to compare thoroughly all aspects of advantages and disadvantages of the different types of splints. Going through the list of advantages and disadvantages of different types of splints as presented in section 3 of the introduction, one finds that more often than not, splints are evaluated from technical points of view and the biological aspect is not given the weight it deserves. Mostly a splint would be considered advantageous if, it can be applied with ease (less work to the surgeon), it is less time consuming to make and less expensive. No note is taken considering its side effects on the patient.

Especially the effect of the splints on periodontal tissues has not been given sufficient consideration although some authors are aware of the problem (Härle et al. (1977), Bienegräber et al. (1973), Fröhlich and Gäbler, 1981, Krüger, (1982).

Artificial periodontitis has been induced in experimental animals by ligature placement around the necks of teeth (Rovin et al., 1966; Kennedy and Polson, 1973; Lindhe et al., 1975;

Schoeder and Lindhe, 1975 and Lindhe and Ericsson, 1978).

There is no doubt that in use, splints act as plaque retentive ligatures and therefore must have an effect on periodontal tissues.

Since splints vary not only in shape but also in size and method of application, consequently their contact relation with the periodontal tissues varies also and correspondingly their effect on the periodontium.

Unfortunately most of the splints have not been subjected to evaluation of such side effects.

In view this of this the following questions arise:

1. What is the effect on the periodontal tissues of applying a splint? (both at clinical and histological levels)
2. Does the duration of the splinting period influence the splint effect?
3. Is the effect on the periodontal tissues the same for whatever type of splint after putting and leaving it in place for a certain period of time?
4. Is the splint effect on the periodontium reversible?
5. How long does it take for the periodontal tissues to establish a definitive state, be it permanent damage or complete restoration after splint removal?
6. In case of permanent periodontal impairment, to what extent does it affect an individual?

The aim of the present investigation is to evaluate the reaction of periodontal tissues to different types of splints used in the department of Oral and Maxillo-Facial Surgery

(Head:Prof. Dr. H.P.M. Freihofer), University of Nijmegen, and compare them.

Due to the limited number of experimental animals and time available it is logical to make first an attempt to answer the first three questions in order to determine whether a long term effect might be expected.

For the same reasons only a limited number of splints can be tested.

Under normal circumstances the Merkx (1959) individual prefabricated splint and the Obwegeser (1952) splint are used in the department. Since the Merkx splint belongs to the group of arch bars, while the Obwegeser splint is a continuous wire ligature, it appeared to be an ideal choice to compare these two. Not only do they belong to basically different groups of splints but they also are examples of those two groups in which periodontal changes are most likely to occur.

In the cap splint group these changes are not expected to be seen.

III. MATERIALS AND METHODS

A. MATERIALS:

1. Experimental animals

1.1 Choice of animal model:

For such a clinically oriented experiment, ideally, a primate, which is very close to man, would have been an animal of choice. However, cost, housing conditions, risk of serious infections and availability made it impossible to utilise an animal such as the monkey.

Preferentially, the next animal of choice is the dog, and especially the beagle for the following reasons:

a. the beagle is small, short haired docile breed which makes it easy to handle. This is very important, when oral hygiene is to be taken into account (Andersen, 1970).

b. At gross anatomical as well as histological level, the beagles periodontium is quite similar to that in man (Attström and Graf-de Beer, 1975; Lindhe and Rylander, 1975) and is well documented.

c. the beagle has been used in many clinical and histopathological studies on periodontal disease as the following have been demonstrated:

- gingival inflammation concomitant with debris and calculus deposition, pocket formation, periodontal abcess and extreme tooth mobility occur in the beagle dog just as they do in man (Rosenberg et al., 1966; Gad, 1968; Hull et al.,1974; Hamp and Lindberg, 1977).
- plaque accumulation has been shown to produce gingival inflammation (Schroeder et al., 1975; Lindhe and Rylander,1975)

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changing into a periodontitis (Saxe et al., 1967, Lindhe et al., 1973, Schroeder and Lindhe, 1975) which deteriorates with time (Saxe et al., 1967, Lindhe et al., 1975).

The above findings imply that the beagles periodontium reacts quite similarly to human periodontium when insulted by external factors.

However it is worthwhile mentioning the drawbacks due to the beagles tooth morphology:

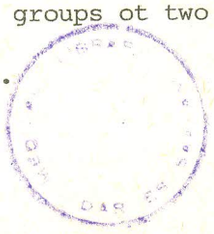
- compared to the human dentition the beagle root furcations are high;
- the bucco-lingual thickness of the premolars and molars is smaller and the mesio-distal thickness is bigger;
- there are no contact points in the premolar region;
- there is no occlusion in the premolar region.

Inspite of these still it is considered the animal of choice.

1.2 Source of animals:

The animals were born and raised in the central laboratory for animal experimentation of the University of Nijmegen (Head: Dr. J.L. v.d. Gulden). The age of the animals was between 19 and 20 months. At this age permanent dentition had already erupted and hence splinting was easier than it would have been the case with deciduous dentition.

The animals were caged in groups ot two in cages measuring approximately 2 x 1.5 mtr.



They were kept under normal laboratory conditions, fed with standardized pelleted food HOPE FARMS COMPLETE DOG FOOD D-B Woerden, The Netherlands, one meal per day.
Water was given ad libitum.

The 6 animals were divided into 2 groups of threes.
The first group was splinted for 48 hours while the second for 6 weeks.

2. The splints to be investigated

2.1 Merkx splint.

The procedural steps for constructing a prefabricated individual arch bar for the dog are essentially the same as those for the human being. However, the anatomical situation of the dog's oral cavity demands an additional step, and that is, the construction of an impression tray, followed by other steps similar to those done in human beings.

2.2 Obwegeser splint.

A stainless steel wire of 0.4mm. diameter was used.

B. METHODS

1. Clinical evaluation of periodontium and statistical analysis of the periodontal index scores:

Before actually splinting each dog several clinical procedures were done in order to establish the state of the periodontium at the beginning.
The same procedures were repeated after splint removal to

establish changes, if any, due to splinting. All these procedures were done while the animal was under general anaesthesia. For general anaesthesia, NARCOVET (Pentobarbitalum Natrium) was administered intravenously at a dose of 30 mg. per kg. body weight.

0.5 mg. of Atropine were also administered intravenously to keep the oral cavity dry.

The following is a detailed description of every clinical procedure done.

1.1 Radiographic procedures.

Standardized oblique-lateral radiographs were made for the right and left halves of the mandibles and maxillae of each dog.

The animals were given an intravenous general aneasthesia as described above, and radiographs made, using a specially constructed cephalostat (Maltha, 1982).

The cephalostat consisted of a wooden tray in which the dog could be placed.

On this tray adjustable ear pins were mounted, as well as a pin in the mid-sagittal plane, which was adjustable in a rostro-distal direction. The adjusting mechanism of the ear pins was constructed in such a way that the position of the two ear pins was always symmetrical towards the mid-sagittal plane (Fig. 8, Maltha, 1982).

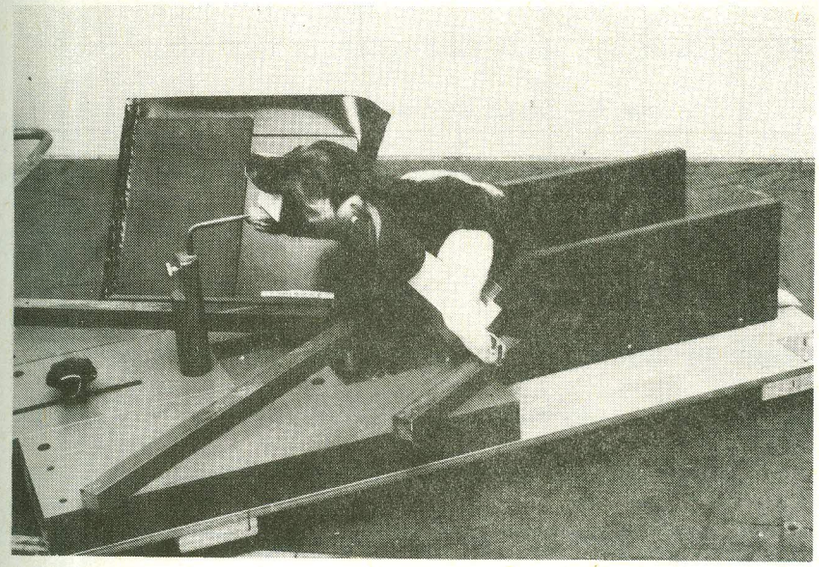


Fig. 8: Dog placed in a cephalostat.

The horizontal tray could be rotated around a vertical axis in the mid-sagittal plane between the ear pins and the adjustable pin in the mid-sagittal plane. It could be fixed in two positions. In both fixed positions the mid-sagittal plane made the same angle of approximately 70° with the central X-ray beam, so that radiographs were taken about three quarters profile.

The X-ray source was mounted on a wall at a distance of 2.6 m. away from the axis of rotation of the cephalostat. The vertical position of the X-ray source was 1.5 m. above the cephalostat, so that the distance between the focus and the rotation point of the cephalostat was 3.0 m. The angle between the central X-ray beam and the horizontal was 30°.

The aneasthetized animal was placed in the cephalostat, the earpins were adjusted in the external auditory canals and the adjustable pin in the mid-sagittal plane was placed between the mandibular central incisors at the gingival margin.

The mouth of the dog was then opened wide and held in that position by a cardboard sheet.

Then the X-ray film was placed on a board resembling a bookrest which could be placed in a standardised position perpendicular to the central X-ray beam at a distance of 5 - 10 cm. behind the part of the mandible and maxilla to be radiographed.

The film used was Cronex LO-Dose Mammography with an image intensifier screen (Dupont). Exhausted black Polyethylene bags served as a casette.

One half of the film was then covered by a 1mm. thick sheet of leadrubber, apparently dense enough to avoid the exposure of the covered part of the film.

A Philips Practic X-ray machine, adjusted to 20 mA and set at a voltage of 90 KV, was used.

The exposure time was 4 seconds.

After exposure the wooden tray with the dog in situ was rotated 140^{o} into the other fixed position. The X-ray film holder was then replaced at the other side of the cephalostat, the sheet of lead rubber shifted and a radiograph of the other half of the mandible and maxilla made.

After exposure the films were processed in a R.P.X. Processor (Kodak) for a duration of 90 seconds.

This set up of radiographic procedure has an advantage over normal lateral radiographs in the sense that structure superimposition is

completely avoided for one half of the mandible although it remains for the maxilla. Another advantage is that since the central X-ray beam is approximately perpendicular to vertical lines on the body of the mandible at the side farthest away from the X-ray source, distortions of the vertical dimensions of the mandible and the teeth on the radiographs are minimized, although this is again not valid for the maxilla.

The greatest disadvantage of this method is that for assessing gross qualitative changes of alveolar bone, the images are not clear enough especially in the maxilla. Therefore, in addition to this radiographic procedure standardized postmortem radiographs were taken.

This was done after sacrificing the animals and sawing off their jaws. A Philips Practic X-ray machine, adjusted to 15 mA and set at a voltage of 85 KV was used.

The exposure time was 3 seconds. The jaws were radiophotographed at a vertical distance of 80 cm.

1.2 Inspection of the oral cavity.

A general visual inspection of the oral cavity was done. General oral condition was assessed, and any abnormalities in the oral cavity noted.

1.3 Coloured clinical photographs.

Photographs were taken for both the experimental and control quadrant for each type of splint.

1.4 Clinical assessment of the periodontium.

The animal was placed with its side on the operation table and covered with surgical towels so that only the mouth was exposed. The procedures were carried out under moderate sterile conditions. Clinical assessment was done using the periodontial indices described below.

1.4.1 Plaque index system (Silness and Loë, 1964).

Score and criteria:

0 = no plaque

1 = A film of plaque adhering to the free gingival margin and adjacent area of the tooth. The plaque may be seen in situ only after application of disclosing solution or by using the probe on the tooth surface.

2 = Moderate accumulation of soft deposits within the gingival pocket or on the tooth and gingival margin which can be seen with the naked eye.

3 = Abundance of soft matter within the gingival pocket and/or on the tooth and gingival margin.

1.4.2 Calculus index system (Ramfjord, 1959).

Score and criteria:

0 = No calculus

1 = Supragingival calculus extending only slightly below the free gingival margin (not more than 1 mm.)

2 = Moderate amount of supra and subgingival calculus, or subgingival calculus only.

3 = abundance of supra and subgingival calculus.

1.4.3 Gingival index system (Loë and Silness, 1963)

Score and criteria:

0 = absence of inflammation.

1 = Mild inflammation - slight change in colour and little change in texture. No bleeding on probing.

2 = Moderate inflammation - moderate glazing, redness, oedema and hypertrophy. Bleeding on probing.

3 = Severe inflammation - marked redness, hypertrophy and oedema. Tendency to spontaneous bleeding.

1.4.4 Sulcus (pocket) depth:

With the help of a calibrated periodontal probe, the sulcus (pocket) depth on each of the four sides of each tooth was recorded.

Scoring was done on four teeth (P2, P3, M1, M2 for maxilla and P2, P3, P4, M1 for mandible) on each quadrant.

Each of the four surfaces of each tooth (buccal, mesial, lingual/palatal and distal) was given a score from 0 to 3 and recorded in mm. in case of pocket depth measurements. The recordings were then entered in a form designed for the purpose. (See appendix i).

Scoring was done always by the same person.

Statistical analysis:

The data obtained from periodontal index scores was transferred to an IBM 4341 computer and analysed as follows: statistical analysis was based on the assumption that the effect of splinting depends on 3 factors and their interactions.

The first factor represents the individual variation between the

dogs. This factor is taken to be random. The other two factors are fixed and represent the position in the mouth and the type of splint. We are interested in the splint factor.

In order to obtain periodontal index for one tooth, indices for each side (buccal, mesial, palatal/lingual and distal) were added together. For each tooth then the effect of the splint was calculated as follows:

(index at the end of splinting - index before splinting) - (control index at the end of splinting - control index before splinting) = Splint effect.

N/B The calculus index was omitted because it was nearly almost zero and hardly changed during the experiment.

For each group mean values and standard deviations were calculated for each index.

From these values graphical illustrations were drawn for ease of understanding.

This model was analysed with a mixed three-way analysis of variance and analysis was done in the same way for the first group as well as for the second group (after 3 weeks and 6 weeks of splinting of animals).

All the above clinical procedures were done in both groups of dogs.

2. Splinting

First group (splinted for 48 hours):

Dog no. 1		Dog no. 2		Dog no. 3	
MS	Control	Control	OS	Control	MS
Control	OS	MS	Control	OS	Control

Second group (splinted for 6 weeks):

Dog no. 4		Dog no. 5		Dog no. 6	
MS	Control	Control	OS	Control	MS
Control	OS	MS	Control	OS	Control

Fig. 9: Splinting plan
MS = Merkx splint
OS = Obwegeser splint

On one dog, each of the two types of splints was fixed on one quadrant leaving the other as a control for that particular splint.

With this method both types of splints could be tested on the same animal and thus eliminating the animal factor in the interpretation of the results. Also by using one side of each jaw as a control for a particular type of splint, the number of animals required for the experiment was reduced.

The possibility of the animal avoiding chewing on one side (splinted side) was greatly reduced as both sides were splinted.

And finally it was possible also to test the two types of splints on different sides of each jaw (left and right).

Splinting was done for both types of splints to cover the distance from the canine to the second molar.

The possibility of intermaxillary fixation was considered but later on abandoned due to the difficulties in feeding the dogs in such a condition.

In the course of splinting it was necessary to pierce the inter-dental papilla in order to secure a fixed position for the splints. This is due to the dog's dentition which differs morphologically from that of the human being.

After 48 hours, 3 and 6 weeks of splinting for the respective 2 groups of animals:

a) a general examination of the oral cavity was done again

b) coloured clinical photographs were taken

c) the splints were carefully removed to avoid further trauma to the gingiva (this was not done after 3 weeks for the 6 weeks group)

d) X-rays of the maxilla and mandibles were taken so as to assess gross qualitative changes (not detailed measurements) of alveolar bone. (This was not done after 3 weeks for the 6 weeks group).

3. Perfusion of the animals and histological procedures.

While still under general anaesthesia, the animal was given an intravenous injection of 5 ml. (1 mml = 5000 UI) of HEPARINUM NATRICUM (THROMBOLIQUE) and killed by an overdose of PENTOBARBITALUM NATRIUM (NARCOVET).

The abdomen was opened and blood from the head region cleared by Saline solution (NATRIUMCHLORIDE solution 0.9%) through the

abdominal aorta and the superior vena cava. The procedure was considered successful when the tongue and other oral tissues became completely pale.

3 litres of Saline solution were used.

The animal was then perfused with 2 litres of 4% Neutral Formaldehydesolution for fixation of tissues.

The jaws were dissected and sawed out of the skull and fixed in jars containing 10% buffered Formaldehydesolution for 2 weeks.

With the aid of an electric saw, smaller pieces were then sawed off for fixation and these were from the following parts for each jaw.

Mandible:

M1	buccal part - for frontal sections
P4 - M1	interdental (proximal) part - for mesio-distal sections
P3 - P4	interdental (proximal) part - for mesio-distal sections

Maxilla:

M1	buccal part - for frontal sections
P3 - M1	interdental (proximal) part - for mesio-distal sections
P2 - P3	interdental (procimal) part - for mesio-distal sections.

For each quadrant 3 pieces were sawed off i.e. 12 pieces from each dog and therefore 36 pieces for 3 dogs.

Each of these pieces was fixed in a bottle with 4% buffered Formaldehyde solution.

Histological procedures: (carried out in the Dept. of Oral Histology; Head: Dr. J. Maltha, University of Nijmegen).

After two weeks these segments were washed in tap water, and decalcified in a solution of 25% Formic acid and 10% Sodium citrate in water.

After embedding in paraffin wax, sections of 7mm. apart were picked up and stained with Haematoxylin and Eosin.

Each section was examined under a light microscope and the following were considered:

1. Epithelium:
   - integrity
   - presence/absence of rete pegs in sulcular epithelium
   - apical migration of juntional epithelium
   - loss of epithelial attachment.

2. Gingival connective tissue:
   - integrity
   - presence/absence of cellular infiltrate (inflammatory cells)
   - diameter of blood vessels
   - proliferation of small blood vessels
   - gingival fibre orientation (organisation).

3. Alveolar bone:
   - presence/absence of osteoclasts
   - presence/absence of inflammatory cells in the marrow spaces of the alveolar bone.

4. Periodontal membrane:

- integrity
- presence/absence of inflammatory cells
- quantity of blood vessels
- diameter of blood vessels.

The results were then entered in specially designed forms for scoring and quantification. (See appendix ii). The epithelium and connective tissue on each section were divided into 4 parts (1,2,3 and 4) and (5, 6, 7, and 8) respectively and each part was then carefully studied.

All the produres were done also in the second series of dogs in the same manner as in the first group except that in the latter clinical procedures were done also after three weeks of splinting.

IV. RESULTS

1. Clinical and radiological results

a. Inspection of the oral cavity

48 hour group:

All the dogs which showed a satisfactory oral hygiene before splinting, show a different picture after 48 hours.
Small amounts of food particles are seen especially around the splints and in the interproximal parts.
On the experimental sides the gingiva is inflammed, reddish in colour and bleeds at the sites of wire penetration. On the control sides, the situation remains more or less the same as it was before splinting.

6 week group:

All the dogs showed a satisfactory oral condition before splinting (Figs. 10 and 13). After 3 weeks of splinting, there is foetor oris from the oral cavity. In all the dogs there is an accumulation of food debri on the splinted sides (Figs. 11 and 14). There is more food debri accumulation on the side with Merkx splint than that with the Obwegeser splint. (compare Figs. 11 and 14).
The gingiva is reddish in colour, inflammed and bleeds easily on probing. Gingival ulceration was seen on the experimental side fixed with the Merkx splint in one of the dogs (Fig. 12).
After six weeks of splinting the situation seen after 3 weeks has worsened and gingival ulceration is seen in two of the dog's experimental sides.

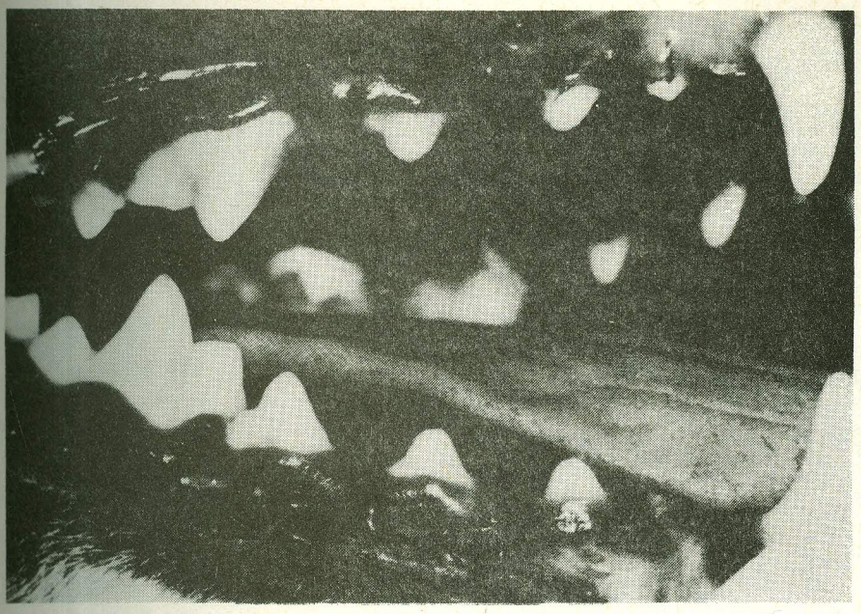


Fig. 10: Situation in the oral cavity before MERKX splint is fixed to the right quadrant of the dog's maxilla.

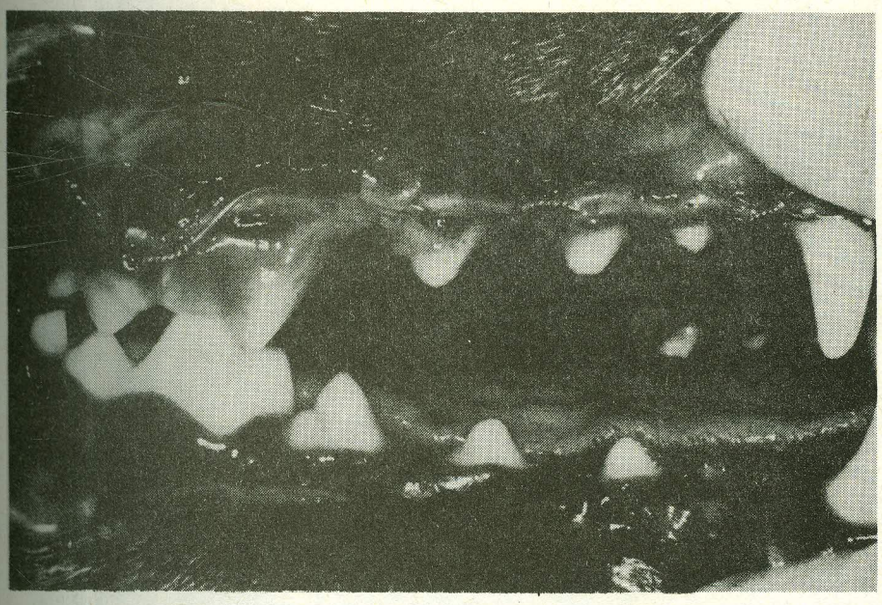


Fig. 11: Situation in the oral cavity of the same dog after 6 weeks of splinting with MERKX splint on the right quadrant of the maxilla.

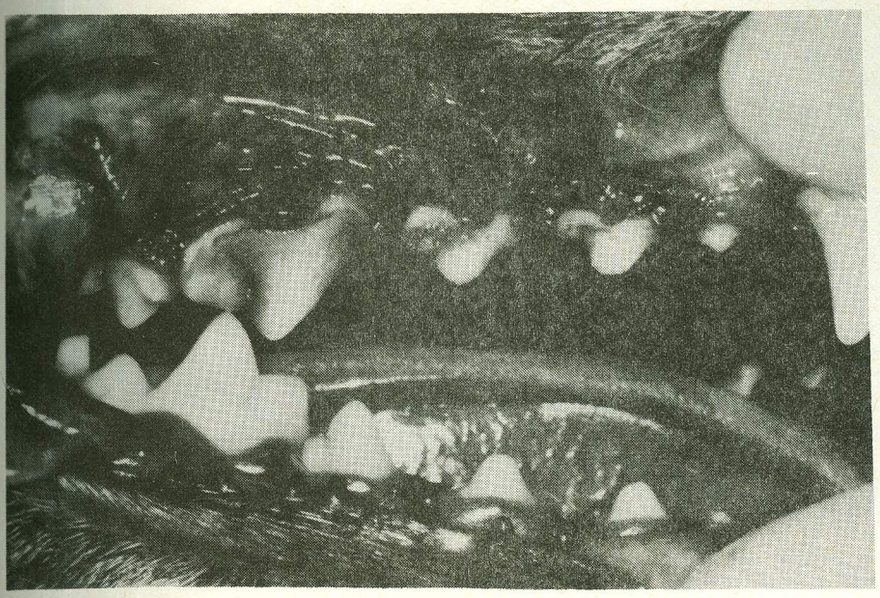


Fig. 12: Situation in the oral cavity of the same dog after 6 weeks of fixing with the MERKX splint on the right quadrant of the maxilla, immediately after splint removal and washing away food debri. Note the gingival ulceration and hypertrophy.

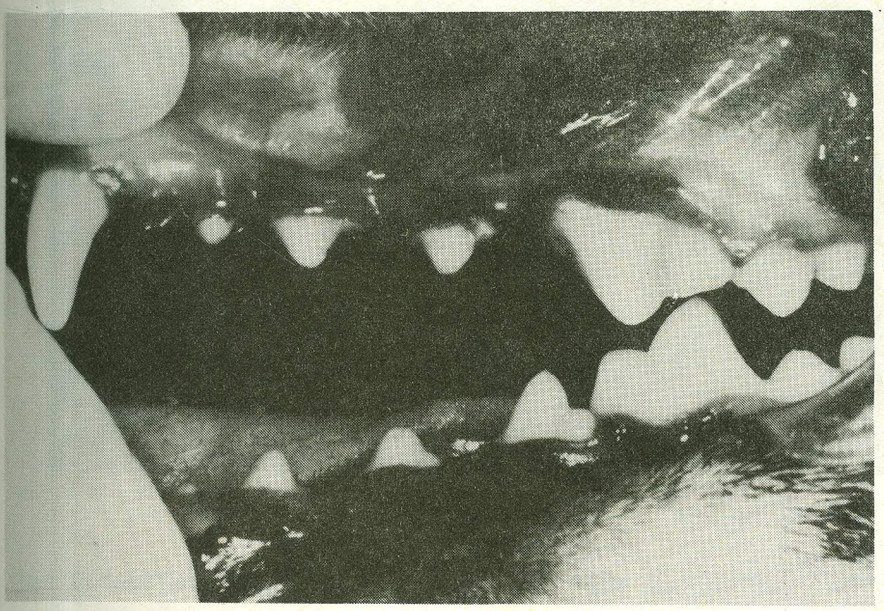


Fig. 13: Situation in the oral cavity before OBWEGESER splint is fixed on the left quadrant of the mandible.

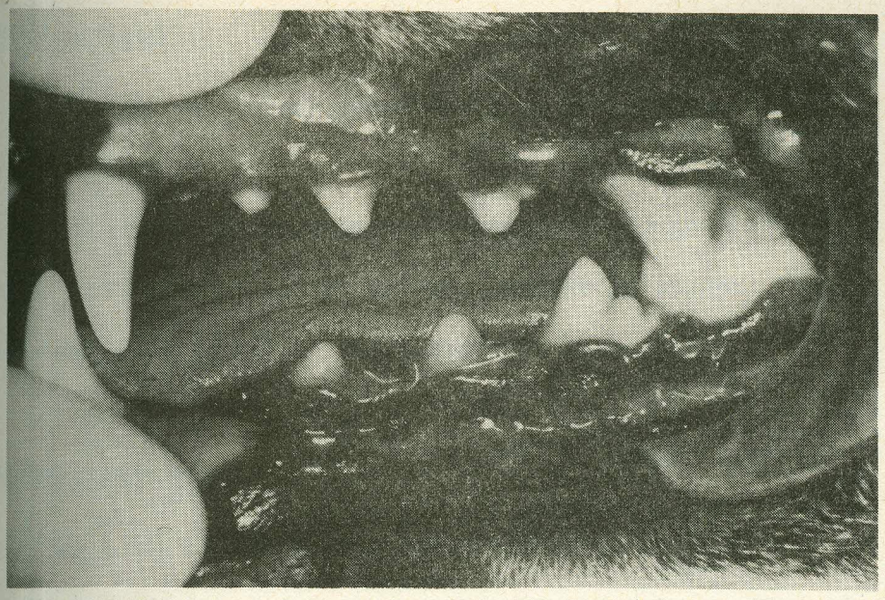


Fig. 14: Situation in the gingiva of the same dog after 6 weeks of fixation with OBWEGESER splint on the left quadrant of the mandible. Note that the splint is for the greater part covered by the inflammed and hypertrophic gingiva.

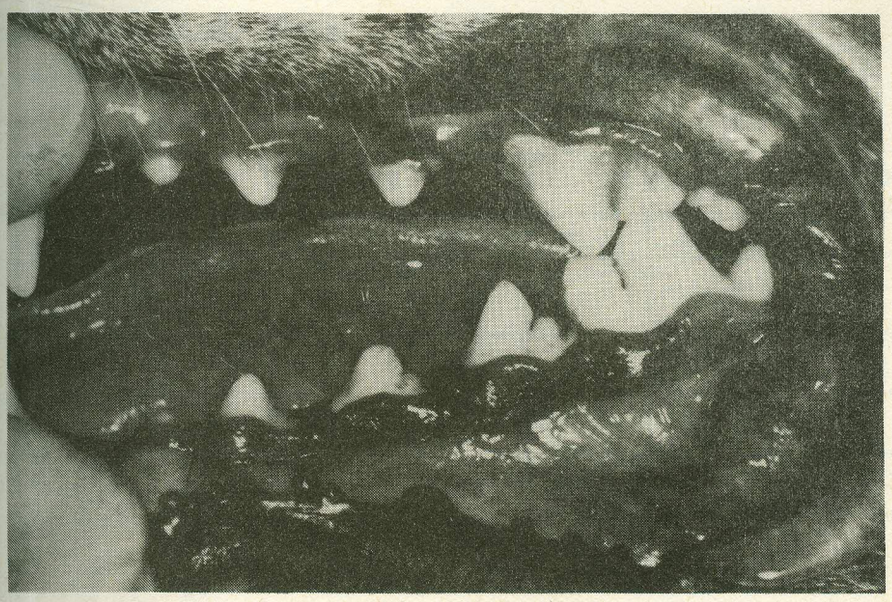


Fig. 15: Situation in the gingiva of the same dog after 6 weeks of fixation with OBWEGESER splint on the left quadrant of the mandible, immediately after splint removal and washing away food debri.

b. Radiographic results:

In the 48 hour group no bony changes can be seen in the radiographs.

In the 6 week group the interdental septae have changed in morphology from convex to flat or even concave in appearance. This is seen more clearly in the postmortem radiographs.

c. Periodontal index scores:

1) The first group of three dogs splinted for 48 hours.

All the periodontal index scores show higher values after 48 hours of splinting than before.

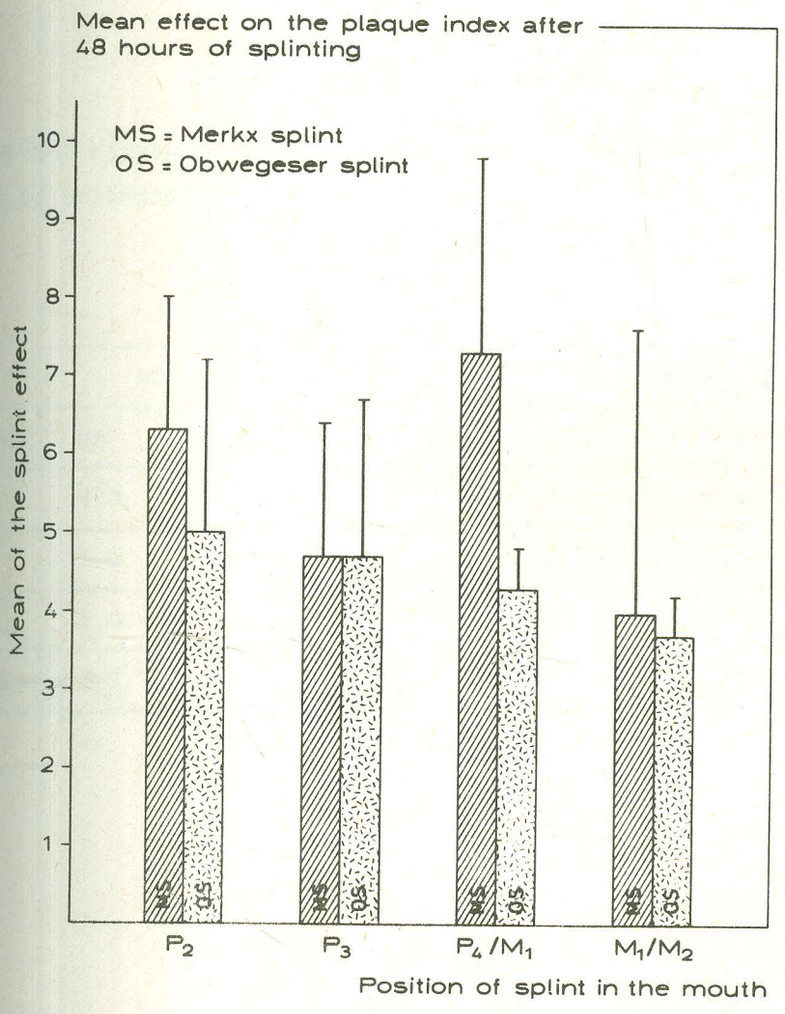
i) plaque index.

The results are presented in table 1 and graph 1.

TABLE 1

The splint effect on the plaque index of each tooth after 48 hours of splinting.

	P2		P3		P4/M1		M1/M2	
dog no.	MS	OS	MS	OS	MS	OS	MS	OS
1	7.0	2.0	3.0	2.0	4.0	4.0	2.0	4.0
2	4.0	6.0	4.0	7.0	8.0	5.0	1.0	4.0
3	8.0	7.0	7.0	5.0	10.0	4.0	9.0	3.0
$\bar{x} \pm$ s.d.	6.3±1.7	5.0±2.2	4.7±1.7	4.7±2.0	7.3±2.5	4.3±0.5	4.0±3.6	3.7±0.5


Mean effect on the plaque index after
48 hours of splinting
MS = Merkx splint
OS = Obwegeser splint
Mean of the splint effect
10
9
8
7
6
5
4
3
2
1
MS
OS
P2
P3
P4 /M1
M1/M2
Position of splint in the mouth

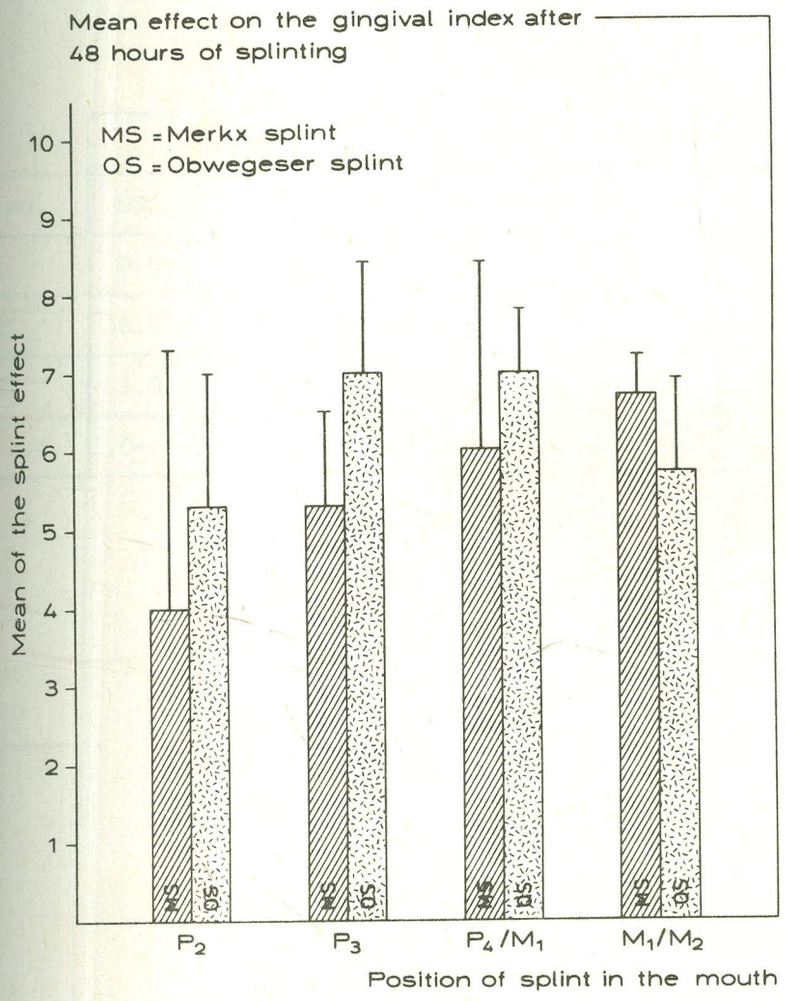
ii) Gingival index:

The results are presented in table 2 and graph 2.

TABLE 2

The splint effect on the gingival index of each tooth after 48 hours of splinting.

	P2		P3		P4/M1		M1/M2	
dog no.	MS	OS	MS	OS	MS	OS	MS	OS
1	-3.0	3.0	4.0	5.0	3.0	6.0	6.0	7.0
2	7.0	6.0	5.0	8.0	9.0	7.0	7.0	4.0
3	8.0	7.0	7.0	8.0	6.0	8.0	7.0	6.0
$\bar{x} \pm$ s.d.	4.0±3.3	5.3±1.2	5.3±1.2	7.0±1.4	6.0±2.4	7.0±0.8	6.7±0.5	5.7±1.2

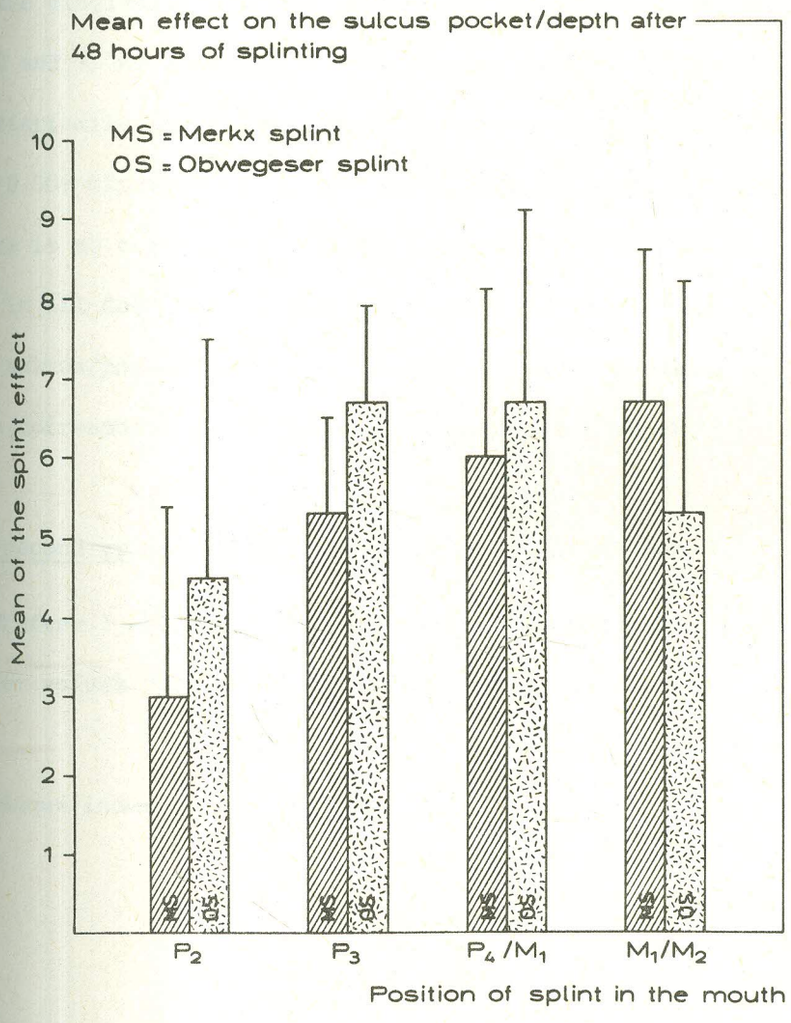

Mean effect on the gingival index after
48 hours of splinting
MS = Merkx splint
OS = Obwegeser splint
Mean of the splint effect
10
9
8
7
6
5
4
3
2
1
MS
OS
P2
P3
P4/M1
M1/M2
Position of splint in the mouth

iii) Sulcus/pocket depth.

TABLE 3

The splint effect on the sulcus/pocket depth of each tooth after 48 hours of splinting.

	P2		P3		P4/M1		M1/M2	
dog no.	MS	OS	MS	OS	MS	OS	MS	OS
1	0.0	3.0	4.0	5.0	3.0	5.0	4.0	2.0
2	6.0	2.0	7.0	7.0	7.0	5.0	8.0	5.0
3	3.0	9.0	5.0	8.0	8.0	10.0	8.0	9.0
$\bar{x} \pm$ s.d.	3.0±2.4	4.5±3.0	5.3±1.2	6.7±1.2	6.0±2.1	6.7±2.4	6.7±1.9	5.3±2.9


Mean effect on the sulcus pocket/depth after 48 hours of splinting
MS = Merkx splint
OS = Obwegeser splint
Mean of the splint effect
10
9
8
7
6
5
4
3
2
1
MS
OS
P2
P3
P4 /M1
M1/M2
Position of splint in the mouth

It is clear from the results in table 1, 2 and 3 and graph 1, 2 and 3 that all the periodontal indices are related to a significant level after 48 hours of splinting.

Although the Merkx splint brings about more splint effect on the plaque index, and the Obwegeser splint more splint effect on the gingival and sulcus/pocket depth indices (compare graphs 1, 2 and 3) no significant difference could be demonstrated statistically between the two types of splints (Plaque index $P = 0.50$; Gingival index $P = 0.55$; Pocket depth $P = 0.74$).

There is no correlation between plaque index and other indices. But in all dogs there is a correlation between gingival index and sulcus/pocket depth as a higher gingival index is accompanied by a corresponding increase in sulcus/pocket depth.

2. The second group of three dogs splinted for 6 weeks

Still after 3 and 6 weeks, all the periodontal index scores show higher values than those before splinting.

i) Plaque index

TABLE 4

The splint effect on the plaque index of each tooth after 3 and 6 weeks of splinting respectively.

	P2				P3				P4/M1				M1/M2			
	MS		OS		MS		OS		MS		OS		MS		OS	
dog no.	3wk	6wk	3wk	6wk	3wk	6wk	3wk	6wk	3wk	6wk	3wk	6wk	3wk	6wk	3wk	6wk
1	7.0	6.0	2.0	2.0	7.0	5.0	2.0	2.0	8.0	9.0	1.0	5.0	4.0	8.0	1.0	3.0
2	10.0	5.0	1.0	1.0	7.0	8.0	2.0	0.0	8.0	5.0	2.0	-3.0	8.0	5.0	-2.0	1.0
3	5.0	5.0	8.0	5.0	4.0	4.0	3.0	3.0	5.0	5.0	-1.0	-3.0	3.0	1.0	-1.0	-3.0
$\bar{x} \pm$ s.d.	7.3±2.1	5.3±0.5	3.7±3.1	2.7±1.7	6.0±1.4	5.7±1.7	2.3±0.5	1.7±1.2	7.0±1.4	6.3±1.9	0.7±1.2	-0.3±3.7	5.0±2.2	4.7±2.9	-0.7±1.2	0.3±2.5

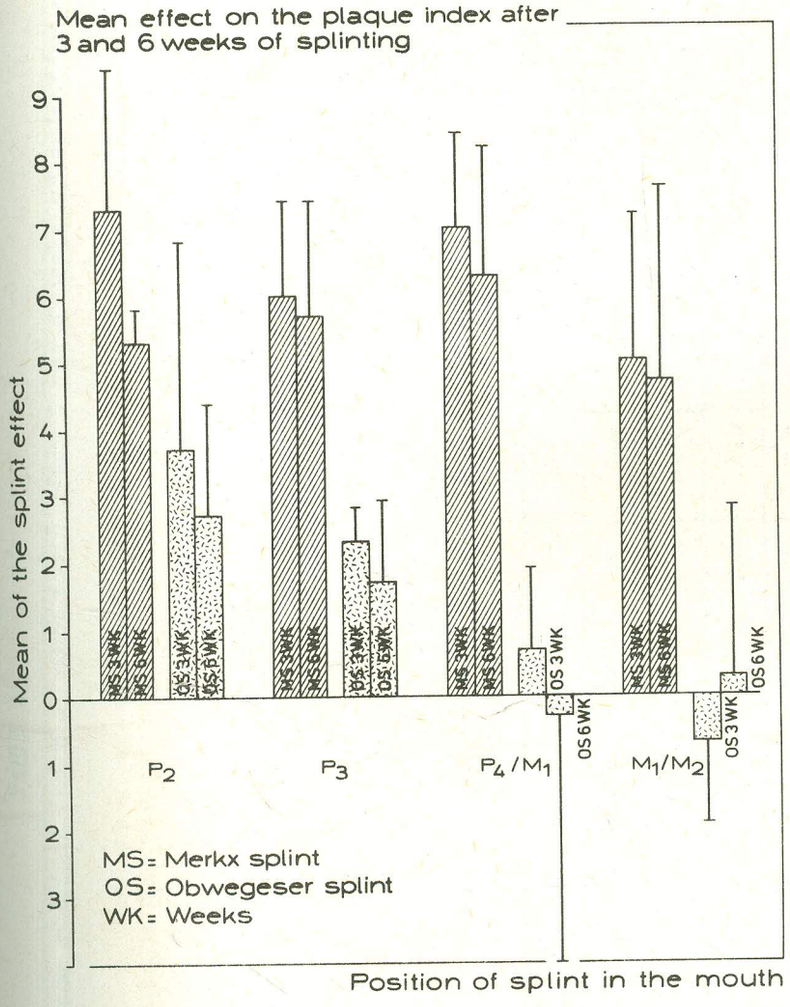

Mean effect on the plaque index after
3 and 6 weeks of splinting
Mean of the splint effect
9
8
7
6
5
4
3
2
1
0
1
2
3
MS 3WK
MS 6WK
OS 3WK
OS 6WK
P2
P3
P4/M1
M1/M2
MS= Merkx splint
OS= Obwegeser splint
WK= Weeks
Position of splint in the mouth

TABLE 5

The splint effect on the gingival index of each tooth after 3 and 6 weeks of splinting respectively.

	P2				P3				P4/M1				M1/M2			
	MS		OS		MS		OS		MS		OS		MS		OS	
dog no.	3wk	6wk	3wk	6wk	3wk	6wk	3wk	6wk	3wk	6wk	3wk	6wk	3wk	6wk	3wk	6wk
4	-2.0	2.0	0.0	0.0	-1.0	2.0	1.0	2.0	-1.0	4.0	1.0	3.0	1.0	2.0	1.0	1.0
5	4.0	3.0	2.0	6.0	3.0	3.0	1.0	5.0	2.0	1.0	-1.0	3.0	2.0	1.0	-3.0	3.0
6	4.0	3.0	3.0	2.0	9.0	2.0	2.0	5.0	8.0	-2.0	2.0	4.0	3.0	1.0	0.0	2.0
$\bar{x} \pm$ s.d.	2.0±2.8	2.7±0.5	1.7±1.2	2.7±2.5	3.7±4.1	2.3±0.5	1.3±0.5	4.0±1.4	3.0±3.7	1.0±2.4	0.7±1.2	3.3±0.5	2.0±0.8	1.3±0.5	-0.7±1.7	2.0±0.8

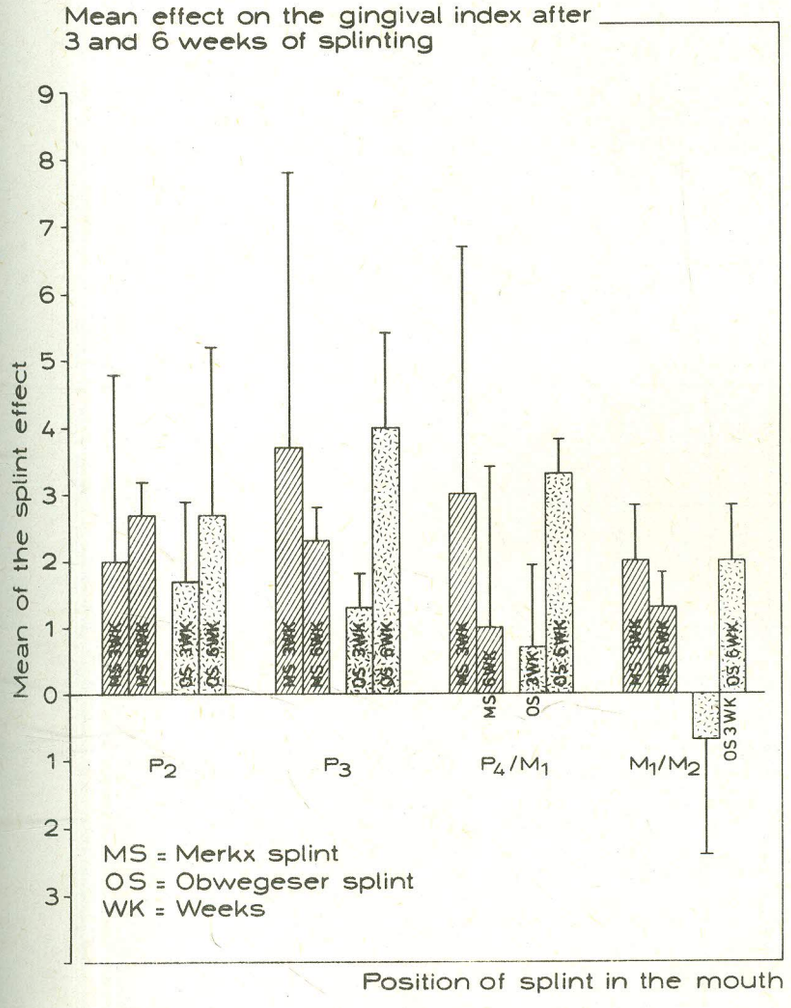
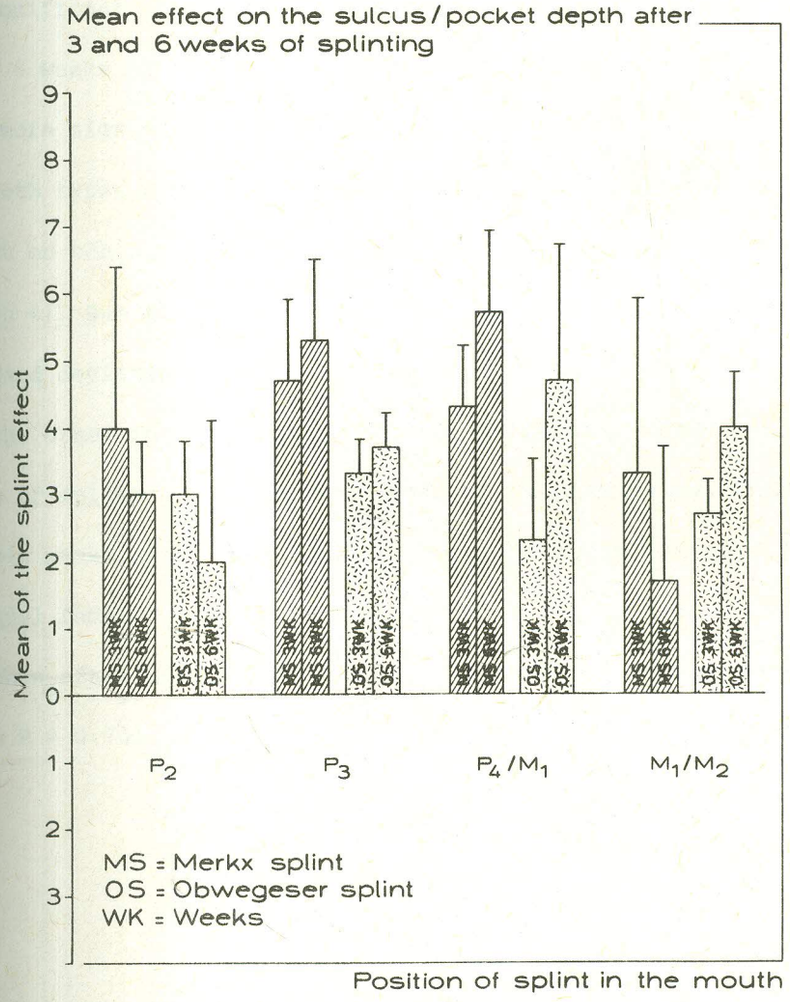

Mean effect on the gingival index after
3 and 6 weeks of splinting
Mean of the splint effect
9
8
7
6
5
4
3
2
1
0
1
2
3
MS 3WK
MS 6WK
OS 3WK
OS 6WK
P2
P3
P4/M1
M1/M2
MS = Merkx splint
OS = Obwegeser splint
WK = Weeks
Position of splint in the mouth

TABLE 6

The splint effect on the sulcus/pocket depth of each tooth after 3 and 6 weeks of splinting respectively.

	P2				P3				P4/M1				M1/M2			
	MS		OS		MS		OS		MS		OS		MS		OS	
dog no.	3wk	6wk	3wk	6wk	3wk	6wk	3wk	6wk	3wk	6wk	3wk	6wk	3wk	6wk	3wk	6wk
4	1.0	2.0	2.0	-1.0	4.0	4.0	3.0	3.0	3.0	4.0	2.0	2.0	2.0	4.0	2.0	3.0
5	4.0	3.0	4.0	4.0	3.0	5.0	4.0	4.0	5.0	7.0	1.0	5.0	1.0	-1.0	3.0	4.0
6	7.0	4.0	3.0	3.0	7.0	7.0	3.0	4.0	5.0	6.0	4.0	7.0	7.0	2.0	3.0	5.0
$\bar{x} \pm$ s.d.	4.0±2.4	3.0±0.8	3.0±0.8	2.0±2.1	4.7±1.7	5.3±1.2	3.3±0.5	3.7±0.5	4.3±0.9	5.7±1.2	2.3±1.2	4.7±2.0	3.3±2.6	1.7±2.0	2.7±0.5	4.0±0.8


Mean effect on the sulcus / pocket depth after 3 and 6 weeks of splinting
Mean of the splint effect
9
8
7
6
5
4
3
2
1
0
1
2
3
MS 3WK
MS 6WK
OS 3WK
OS 6WK
P2
P3
P4 /M1
M1/M2
MS = Merkx splint
OS = Obwegeser splint
WK = Weeks
Position of splint in the mouth

In the 6 week group we find that still the periodontal indices are raised up to a significant level for both types of splints (Tables 4, 5 and 6 and graph 4, 5 and 6).

3 weeks after splinting the Merkx splint has a greater effect on the plaque index than the Obwegeser splint, and the difference is significant ($P = 0.009$).

After 6 weeks of splinting the difference increases to become even more significant ($P = 0.002$).

For both types of splints there seems to be a fall in the splint effect on the plaque index between 3 and 6 weeks of splinting (Graph 4). But the fall is not significant as shown by the large standard deviations in the graph.

For the other indices no significant difference between the two types of splints could be demonstrated statistically, three and six weeks after splinting (3 weeks after splinting: gingival index $P = 0.39$ and pocket depth $P = 0.34$).

(6 weeks after splinting: gingival index $P = 0.39$ and pocket depth $P = 0.70$).

2. Histological results

2.1 MS fixed for 48 hours.

(a) Frontal sections of M1 part:

2.1.1 Epithelium

Continuity of the epithelium is generally maintained.

Epithelial destruction tends to be highly localized, in the sence that it is observed in those areas where wire ligatures of the MS were located (Fig. 16).

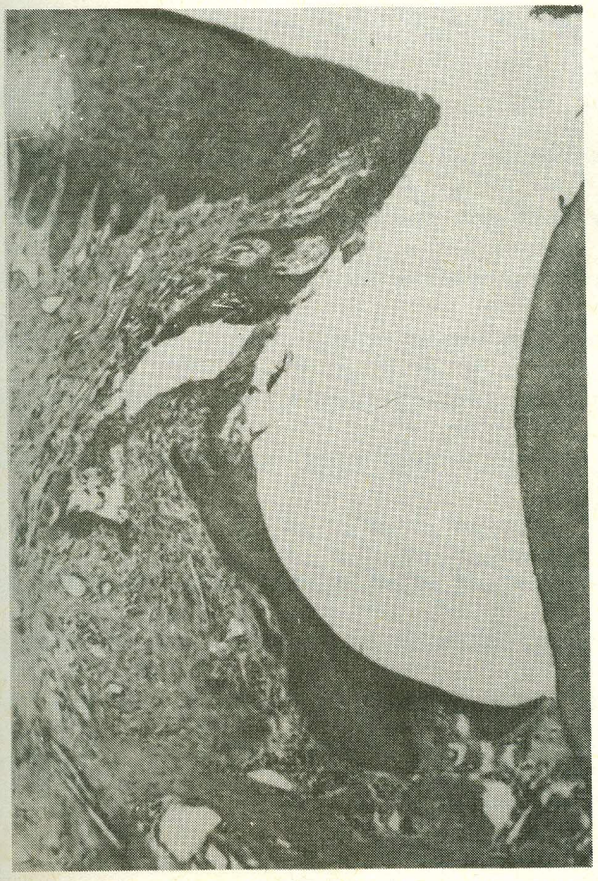


Fig. 16: Mesio-distal section of a maxillary P3-M1 part showing the localized epithelial destruction at the place of splint location. H.E. 120 x (Merkx splint).

In all sections, except one, the epithelium in both the buccal and lingual/palatal aspects is equally affected. In this one exceptional section, in the buccal aspect, the epithelium is totally destroyed.

In 80% of the cases the epithelial keratin layer is intact.

Sulcular epithelium is intact in 90% of the sections, and does not show any epithelial proliferations (rete pegs). Again the lesions here are strictly localized.

In all the sections neither apical migration of junctional epithelium nor loss of epithelial attachment are observed.

No plaque accumulation is seen in the sulcus.

2.1.2 Gingival connective tissue

The integrity of the connective tissue maintained except in those areas where wire ligatures passed through the gingiva. These are seen as holes in the gingival connective tissue.

In all the sections, the buccal aspect shows only scattered cellular infiltrate while the lingual/palatal aspect shows a moderate cellular infiltrate (more severe than in the buccal aspect).

Generally the cellular infiltrate affects the whole connective tissue evenly without any tendency of localization. However, it is worthmentioning that more inflammatory cells are seen around lesions (Fig. 17).



Fig. 17: The same section as before showing in more detail the localized epithelial destruction. Note that cellular infiltrate is concentrated around the lesion. H.E. 300 x (Merkx splint).

45% of the sections show a moderate proliferation of small blood vessels which, on average, are equally distributed in the lingual/palatal and buccal aspects.

50% of the blood vessels are dilatated and 93% of these are seen in the lingual/palatal aspect.

Gingival fibres are found to be disorientated in 41% of the sections, and 92% of these are seen in the lingual/palatal

aspects.

2.1.3 Alveolar bone

In all the sections examined, there are no osteoclasts seen on the alveolar bone.

No inflammatory cells are seen in the marrow spaces of the alveolar bone.

2.1.4 Periodontal membrane

The integrity of the periodontal membrane is maintained as the orientation of the periodontal fibres is undisturbed.

There are no inflammatory cells seen in the periodontium.

The number and diameter of the blood vessels are within the normal limits.

(b) M-D-sections of P3 - M1 (maxilla) P4-M1 (mandible) Inter-dental part:

2.1.5 Epithelium

Continuity of the epithelium is disrupted in most parts of the cases and complete epithelial destruction is seen in 67% of the sections.

In 91% of all the sites examined in the sections the epithelium has been either partially or totally destroyed (Fig. 18).

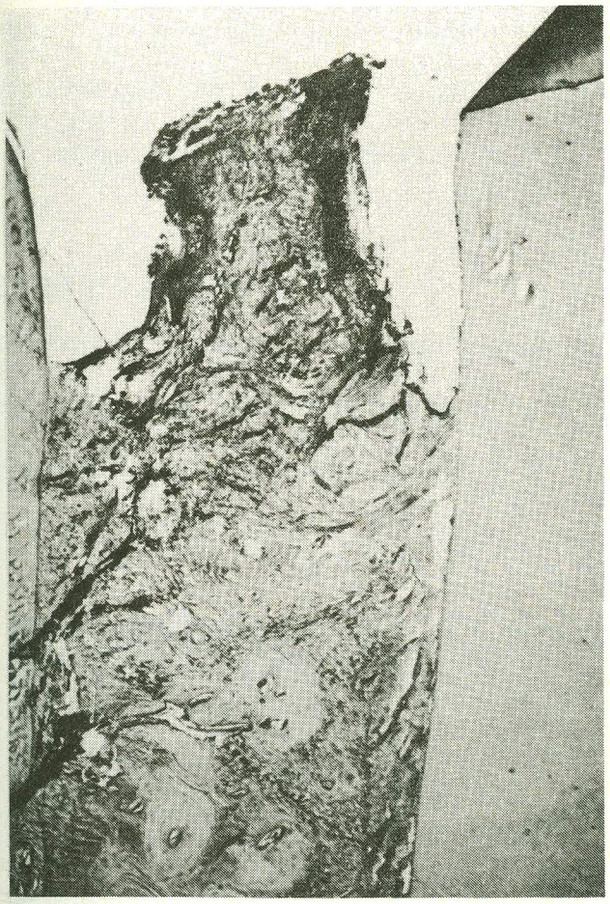


Fig. 18: Mesio-distal section of a maxillary P3 - M1, part showing total epithelial destruction. H.E. 48 x (Merkx splint).

However, the epithelial remnants which are intact have no Keratin layer.

On the average, the mesial and distal aspects are equally affected, although in some sections the distal sulcular epithelium is more affected at places where wire ligatures passed.

72% of all the sections sulcular epithelium does not show any rete pegs. Although there is no evidence of apical migration of the junctional epithelium, in 83% of the sections there is loss of epithelial attachment.

2.1.6 Gingival connective tissue

The integrity of the connective tissue is generally maintained and destruction is seen only in those areas where wire ligatures passed through the gingiva (Figs. 19 and 20).



Fig. 19: Mesio-distal section of a maxillary P3 - M1 interdental part showing the whole through which wire ligatures passed. Around this hole gingival fibres disintegrate. H.E. 75 x (Merkx splint).

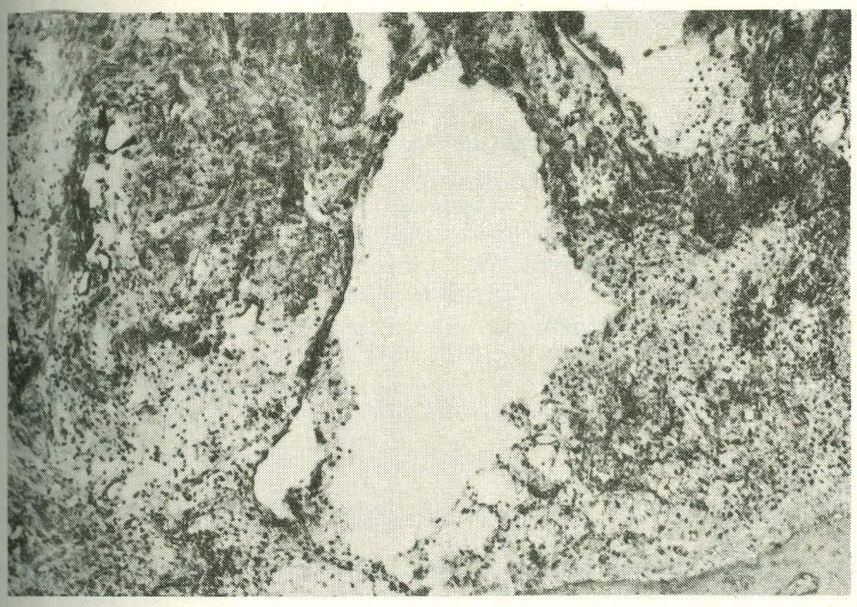


Fig. 20: The same section as above magnified to show the inflammatory cells and gingival fibre disorganisation around the hole through which wire ligatures passed. H.E. 120 x (Merkx splint).

It is interesting to note that even in case of total epithelial destruction, the connective tissue under it in most cases remains intact.

In all the sections there is either a severe or a moderate cellular infiltrate which, on the whole, is evenly distributed throughout the connective tissue.

Around blood vessels more inflammatory cells are seen.

In isolated cases the infiltrate is localized around the areas of ligature placement.

The mesial and distal aspects of the sections show an even distribution of cellular infiltrate.

Red blood cells are seen in the connective tissue in all the sections.

65% of the sectional sites show a very slight proliferation of small blood vessels which are generally equally distributed in the mesial and distal aspects of the sections.

These small blood vessels are, on the average, evenly distributed throughout the connective tissue.

Blood vessels, in all the sections are largely dilatated and in most cases inflammatory cells are seen around them. Again the dilatated blood vessels are evenly distributed throughout the connective tissue.

Gingival fibres are disorientated in all the sections, and the areas mostly affected are those close to the lesions and close to the alveolar bone.

2.1.7 Alveolar bone

The contour of the alveolar bone is normal and no osteoclasts are seen either on the crest or anywhere else in the alveolar bone. In all sections no inflammatory cells are seen anywhere in the narrow spaces of the alveolar bone.

2.1.8 Periodontal membrane

The orientation of periodontal fibres is undisturbed and therefore it can be said that the periodontal membrane is intact, although in 44% of the sections already inflammatory cells are seen scattered in the most coronal part of the periodontal membrane.

In 50% of the sections the most coronal part of the periodontium shows a moderate proliferation of blood vessels, although the rest show a normal diameter.

(c) M-D sections of P2 - P3 (maxilla) P3 - P4 (mandible)

Interdental part:

2.1.9 Epithelium

In all the sections continuity of the epithelium is disrupted, and in 65% of the cases there is total destruction such that no epithelium remains to be seen. In the majority of cases even the surviving epithelium, which is always the juntional part, is in the process of disintegration (Fig. 21).

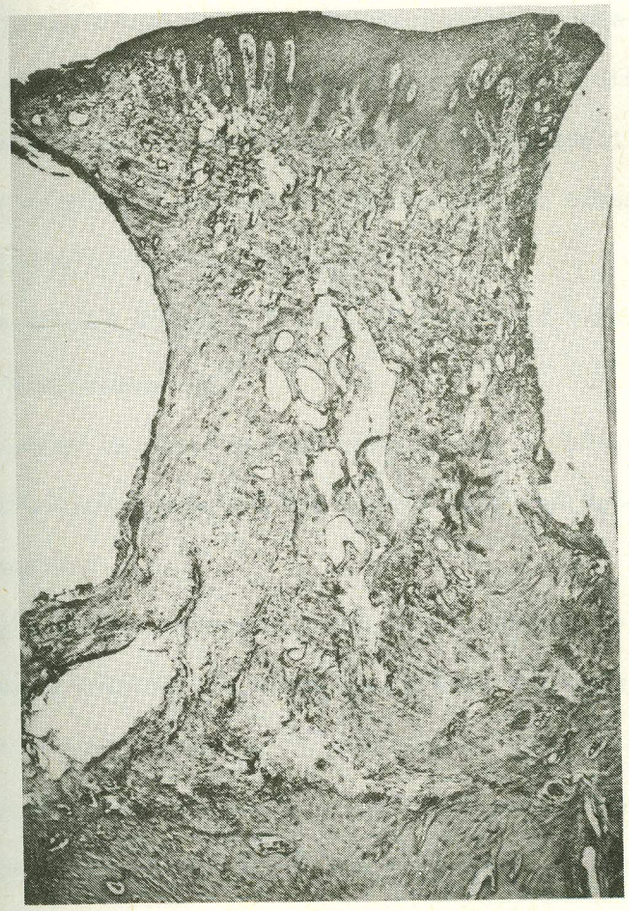


Fig. 21: Mesio-distal section of a mandibular P2 - P3 interdental part showing the generalized epithelial destruction which extends beyond areas of splint placement. H.E. 75 x (Merkx splint).

In all the sections there is an abundant accumulation of plaque in the interdental space.

Loss of epithelial attachment is seen in all the sections.

2.1.10. Gingival connective tissue

In all the sections the connective tissue has been affected in varying degrees.

In 65% of the sections at least 1/3 of the connective tissue has undergone necrosis, in this way covering areas beyond ligature and splint placements. In the rest of the sections destruction was more or less localized.

In the majority of cases even the connective tissue which appears to be intact is actually in the process of disintegration as evidenced by the disorganisation of the gingival fibres.

In all the sections, throughout the connective tissue are seen inflammatory cells in varying quantities.

65% of the sections show a severe cellular infiltrate while the rest show a moderate one.

Different sites in each section show either a moderate or severe inflammatory process suggesting that the process is highly localized.

40% of the sections show red blood cells especially around those areas where ligature wires penetrated the gingiva.

A very slight proliferation of small blood vessels is seen in 65% of the sections. Although in isolated cases these small blood vessels tend to be localized in certain sectional sites, on the whole they are found in the majority of cases distributed throughout the connective tissue.

Vasodilatation is seen in all the sections. (Fig. 22).

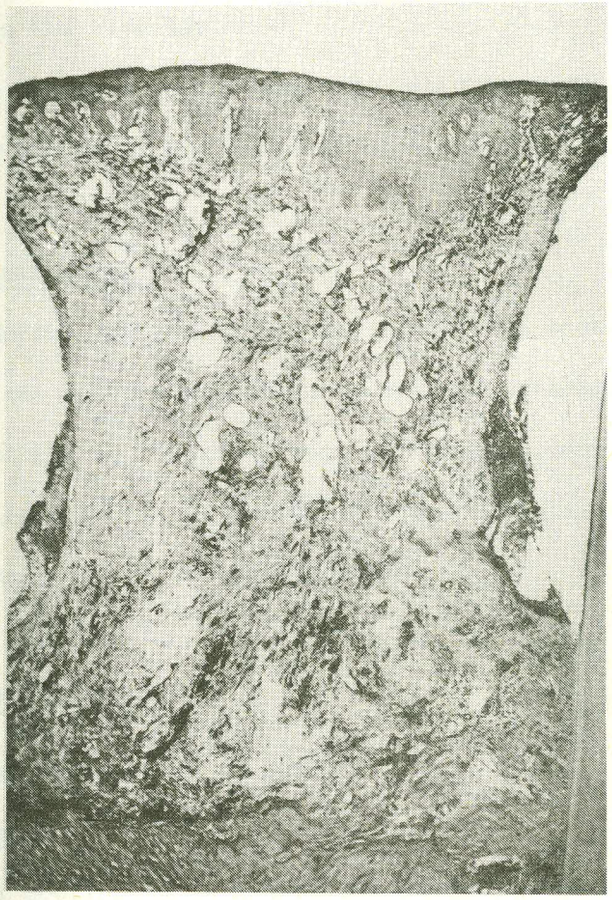


Fig. 22: Mesio-distal section of a maxillary P2 - P3 interdental part showing vasodilatation in the connective tissue. H.E. 75 x (Merkx splint).

These vasodilatated blood vessels tend to be evenly distributed throughout the connective tissue without so much tendency of localization. All the sections show a disorientation of the gingival fibres especially in the sites close to lesions produced either by the splint or the wire ligatures.

2.1.11. Alveolar bone

The contour of the alveolar bone is normal.

No osteoclasts are seen either on the alveolar crest or anywhere in the alveolar bone.

In all the sections no inflammatory cells are seen anywhere in the marrow spaces of the alveolar bone.

2.1.12. Periodontal membrane

The periodontal membrane is intact in all the sections although in 59% of the cases isolated and scattered inflammatory cells are seen in the most coronal part of the periodontal membrane.

A slight increase in the number of blood vessels is seen in the most coronal part of the periodontium.

2.2. OS fixed for 48 hours

(a) frontal sections of M1 part:

2.2.1 Epithelium

Epithelial continuity is maintained except in those areas where the splint was located (Fig. 23).

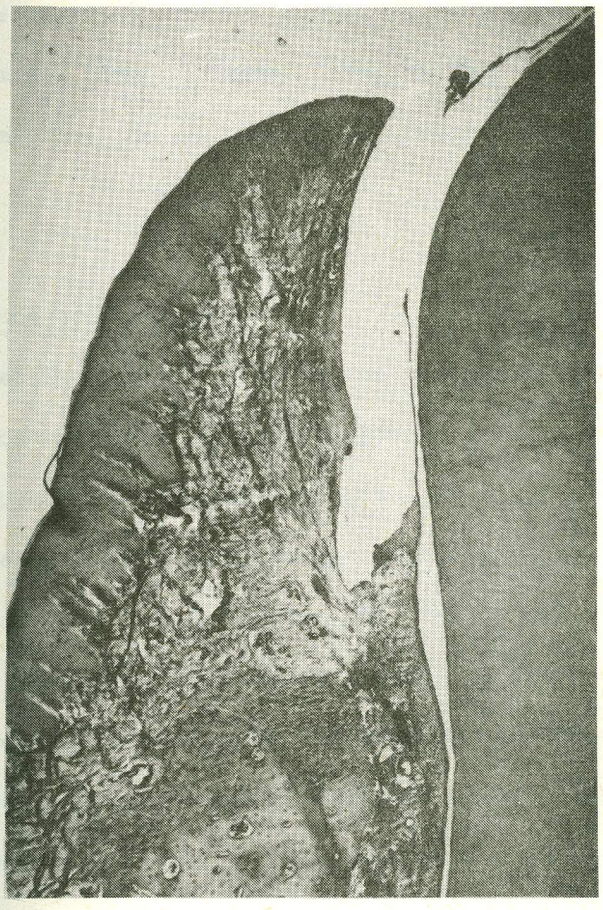


Fig. 23: Frontal section of a mandibular M1 part showing the localized epithelial destruction due to splinting. H.E. 75 x (Obwegeser splint).

In some cases a mere depression of the epithelium is seen. It can be said that also here epithelial destruction is highly localized.

In all the sections the buccal and lingual/palatal epithelia are equally affected.

The epithelial keratin layer is intact in all the sections.

Sulcular epithelium remains intact except in those areas of splint location, and only in 2% of the sections are seen some

epithelial proliferations.

In all the sections neither apical migration of junctional epithelium nor loss of epithelial attachment are seen.

Only a quater of the sections show plaque accumulation in the sulcus.

2.2.2 Gingival connective tissue

All the sections show a gingival connective tissue integrity except those areas of splint placement.

Both the buccal and lingual/palatal aspects of the sections show a moderate to severe cellular infiltrate more or less in equal quantities.

Although, generally cellular infiltrate is found almost throughout the connective tissue it tends to be more close to areas of lesions, around the slightly dilatated blood vessels.

No proliferation of small blood vessels is seen at all, and the diameter of the blood vessels remains almost normal in all the sections.

In all the sections gingival fibre orientation is almost normal.

2.2.3 Alveolar bone

In all the sections osteoclasts are not seen either on the crest of the alveolar bone or anywhere else in the alveolar bone.

No inflammatory cells are seen anywhere in the marrow spaces of the alveolar bone.

2.2.4 Periodontal membrane

The integrity of the periodontal membrane is maintained as the

orientation of the periodontal fibres is undisturbed.
There are no inflammatory cells seen in the periodontium.
The number and diameter of the blood vessels is within the normal limits.

(b) M-D sections of P3 - M1 (maxilla) P4 - M1 (mandible

Interdental part:

2.2.5 Epithelium

Epithelial continuity is disrupted in some parts of the sections and 29% of them show a complete epithelial destruction.
30% of the sections show already a functional epithelial destruction, while in 35% of the cases sulcular epithelium has developed some rete pegs.
In some sections epithelial destruction is no longer localized as evidenced by the extent of epithelial necrosis which extends beyond the place where the splint passes (Figs. 24 and 25).

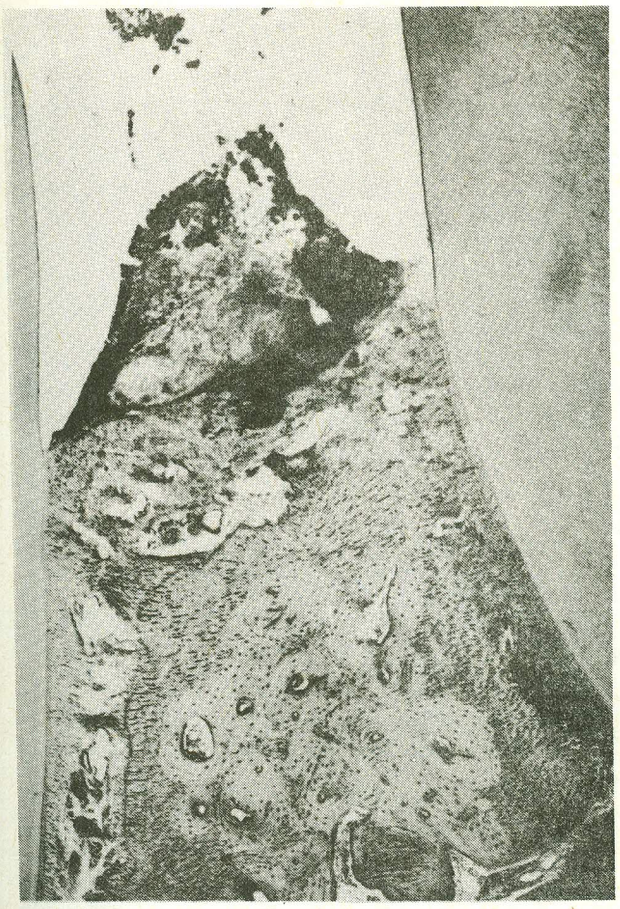


Fig. 24: Mesio-distal section of a mandibular P2 - P3 interdental part showing total epithelial destruction. H.E. 48 x (Obwegeser splint).

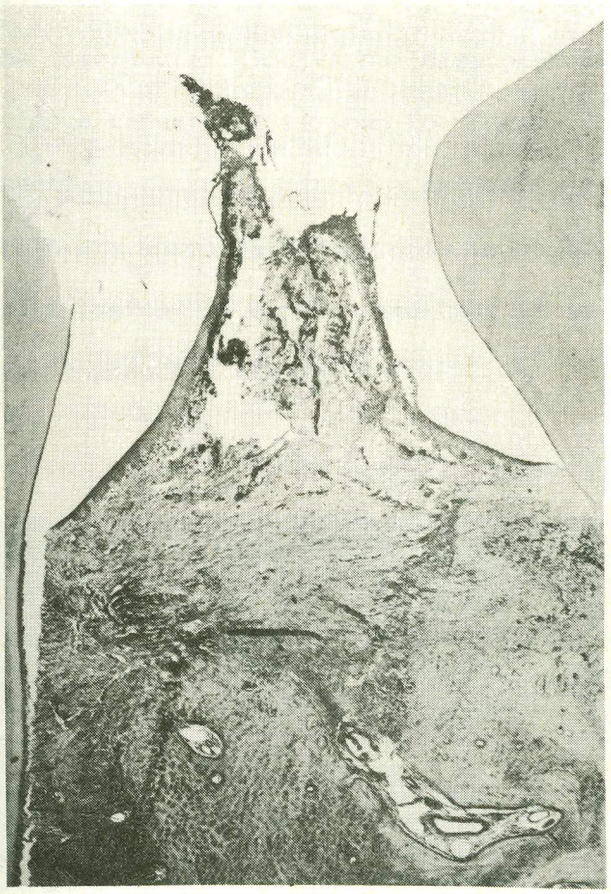


Fig. 25: Mesio-distal section of a mandibular P4 - M1, interdental part showing the place of wire placement and the associated area of tissue destruction. H.E. 48 x (Obwegeser splint).

In all the sections there is no evidence of either juntional epithelial apical migration nor loss of epithelial attachment.

2.2.6 Gingival connective tissue

Generally connective tissue destruction is seen around areas of splint placement although in cases where the whole epithelium on top of the interdental gingiva has disappeared, part of the connective tissue beneath it is seen to undergo destruction. In all the sections there is either a severe or a moderate cellular infiltrate which although tends to be more severe around areas of lesion, it can be said that it is distributed throughout the connective tissue (Fig. 26a).

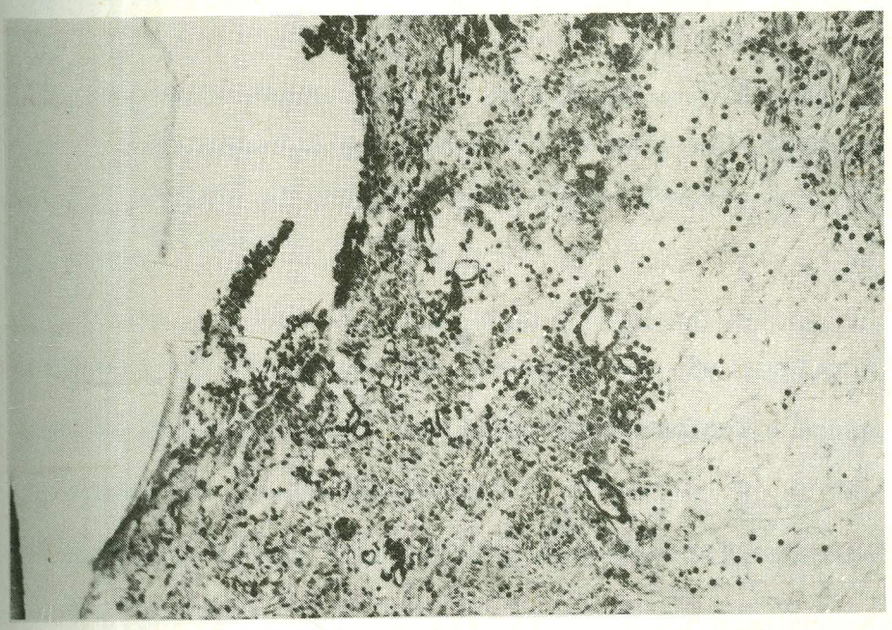


Fig. 26a: Mesio-distal section of a maxillary P2 - P3 interdental part showing inflammatory cells and gingival fibre disorganisation around the area of splint placement H.E. 120 x (Obwegeser splint).

Compare with normal situation in Fig. 26b.



Fig. 26b: Mesio-distal section of a maxillary P2 - P3 interdental part from the control side showing a normal arrangement of gingival fibres. H.E. 189 x (Obwegeser splint).

The mesial and distal part of the sections are equally affected.

Only 24% of all sections show some red blood cells scattered throughout the connective tissue.

62% of the sectional sites show a slight proliferation of small blood vessels which are generally equally distributed in the mesial and distal aspects.

In 65% of the sections the blood vessels are largely dilatated while the remaining though not normal, are very close to normal diameters.

Inflammatory cells are seen around the dilatated blood vessels. Generally, the dilatated blood vessels are evenly distributed throughout the connective tissue.

Gingival fibre orientation is distorted in all the sections though in varying degrees and areas with lesions are the most affected.

2.2.7 Alveolar bone

The contour of the alveolar bone is normal and no osteoclasts are seen either on the crest or anywhere else in the alveolar bone.

In all the sections no inflammatory cells are seen in the marrow spaces of the alveolar bone.

2.2.8 Periodontal membrane

The orientation of periodontal fibres is undisturbed and it can therefore be said that the periodontal membrane is intact.

No inflammatory cells are seen anywhere in the periodontal membrane.

The number and diameter of blood vessels remains normal in all the sections.

(c) M-D sections of P2 - P3 (maxilla P3)- P4 (mandible)

Interdental part:

2.2.9 Epithelium

In all the sections continuity of the epithelium is disrupted, and in 38% of the sections there is total destruction such that no epithelium remains to be seen.

In all the sections the surviving sulcular epithelium shows some rete pegs.

There is neither loss of epithelial attachment nor apical migration of junctional epithelium.

The epithelial keratin layer is intact.

Only one third of the sections show plaque accumulation above the interdental papilla.

2.2.10 Gingival connective tissue

In all the sections the connective tissue has been affected in varying degrees.

In 56% of the sections at least 1/3 of the connective tissue in the coronal part has undergone necrosis, in this way covering areas beyond those of splint placement. The rest of the sections show a localized destruction.

In 38% of the sections not only that there is complete destruction of the epithelium but at least the coronal 2/3 of connective tissue has undergone necrosis. In these sections no blood vessels are to be recognized, the connective tissue itself is not recognizable and it is impossible to see gingival fibres.

In all the sections, throughout the connective tissue are seen inflammatory cells in varying quantities.

44% of the sections show a severe cellular infiltrate while the rest show a moderate one.

Only 6% of the sections show some red blood cells in the connective tissue.

A slight proliferation of small blood vessels is seen in 81% of the sections. Generally these small blood vessels are found distributed all over the connective tissue although it is not uncommon to find them more concentrated around areas of splint placement.

Vasodilatation is seen in all the sections. These dilatated blood vessels tend to be evenly distributed throughout the connec-

tive tissue.

Gingival fibre disorientation is seen in all sections although at varying degrees. The areas more affected are those of splint placement.

2.2.11 Alveolar bone

The contour of the alveolar bone is normal.

There are no osteoclasts seen in all the sections.

In all the sections no inflammatory cells are seen in marrow spaces of the alveolar bone.

2.2.12 Periodontal membrane

In 38% of the sections some periodontal ligaments in the most coronal part of the periodontal membrane have been disrupted.

In 13% of the sections isolated and scattered inflammatory cells are seen in the most coronal part of the periodontal membrane.

There is no increase in the number and size of blood vessels in this part of the periodontal membrane.

2.3 MS fixed for 6 weeks

(a) frontal sections of M1 part:

2.3.1 Epithelium

In all the sections, continuity of the epithelium is interrupted and in 53% of the sections the buccal epithelium is either partly or totally missing (Fig. 27).

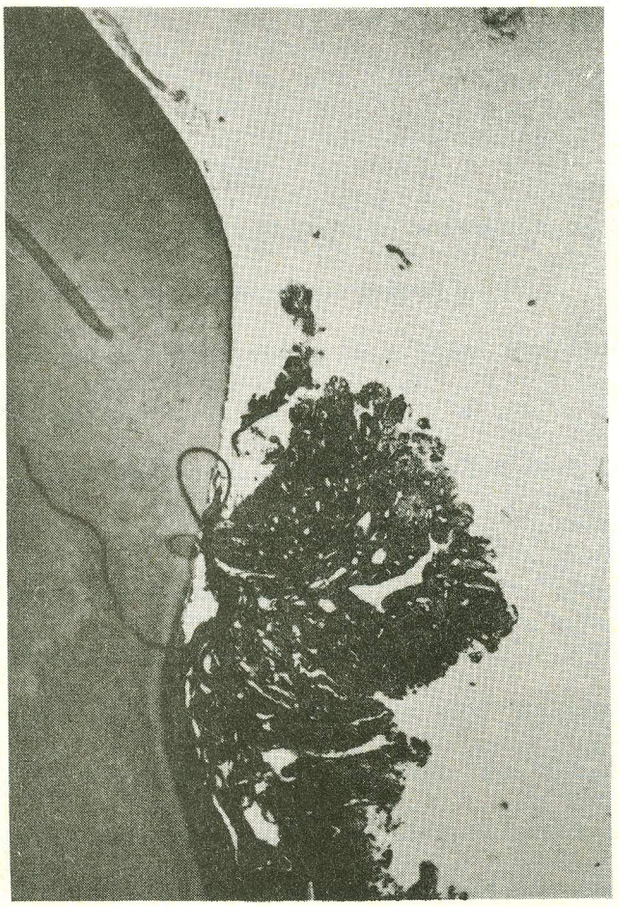


Fig. 27: Frontal section of a maxillary M1 part showing total epithelial destruction in the buccal aspect.

H.E. 75 x (Merkx splint).

The areas where the splint was located can easily be recognized as there is local epithelial ulceration or a depression.

Sulcular epithelium is intact in all the sections except in those areas of splint placement.

All the sections show a lot of epithelial proliferations (rete pegs) under the sulcular epithelium.

Only 12% of all the sections show the disappearance of the epithelial keratin layer.

In 41% of all the sections the junctional epithelium has undergone an apical migration and only in a few cases there is loss of epithelial attachment.

2.3.2 Gingival connective tissue

In all the sections, the connective tissue beneath the ulcerated buccal epithelium has undergone necrosis.

Otherwise in other cases there is a localized tissue destruction due to the splint.

In all the sections cellular infiltrate is seen troughout the connective tissue and it tends to be more in areas of lesion.

The lingual/palatal aspect is 1-4 times more affected with cellular infiltrate than the buccal aspect as revealed by scores obtained from the sectional sites of both aspects.

82% of all the sections show a heavier proliferation of small blood-vessels on the buccal than on the lingual/palatal aspect.

In all the sections the diameter of blood vessels is normal.

In all the sections, the gingival fibres are disorientated and completely destroyed in those areas under ulcerated epithelium.

2.3.3 Alveolar bone

In 41% of the sections are seen osteoclasts on the creastal margin and most coronal part of the alveolar bone. The total number counted is 15.

There are inflammatory cells in the marrow spaces of the alveolar bone in 18% of the sections.

2.3.4 Periodontal membrane

In all the sections the integrity of the periodontal membrane is maintained as the orientation of the periodontal fibres is undisturbed.

No inflammatory cells are to be seen in the periodontal membrane.

The number and size of the blood vessels are within the normal limits.

(b) M-D sections of P3 - M1 (maxilla) P4 - M1 (mandible)

Interdental:

2.3.5 Epithelium

Continuity of the epithelium is disrupted in all the sections and in 30% of the cases there is a complete epithelial destruction.

In the case of incomplete but big epithelial destruction, the most coronal part of the gingiva is mostly affected.

In the remaining sections with partial epithelial loss, the destruction is localized.

All the epithelium which remains intact has lost its keratin layer.

In all the cases where sulcular epithelium is intact, there is a heavy epithelial proliferation (rete pegs).

In 41% of the sections there is an apical migration (shift) of the junctional epithelium.

There is no loss of epithelial attachment.

2.3.6 Gingival connective tissue

In all the sections the connective tissue has been affected as evidenced by the disorganization of the gingival fibres. Beneath the necrotic (ulcerated) epithelium the connective tissue is also necrotic. The worst affected areas are the top of the interdental papilla.

In all the sections there is either a moderate or a severe cellular infiltrate which, on the whole is distributed throughout the connective tissue.

93% of the sectional sites show a severe cellular infiltrate while a moderate one is seen in the rest of the sections.

In almost all of the sections the whole connective tissue is equally affected without any preference for the mesial or distal aspects of the papilla.

Only in 6% of all the sections red blood cells are seen.

All the sections show a heavy (severe) proliferation of small blood vessels throughout the connective tissue (Fig. 28).



Fig. 28: Mesio-distal section of a mandibular P4 - M1 inter-dental part showing a severe proliferation of small blood vessels. H.E. 120 x (Merkx splint).

Also amongst the small proliferated blood vessels, are seen, in 35% of the sections slightly dilatated ones. These are distributed throughout the connective tissue.

In all the sections gingival fibres are disorientated especially in those areas of lesions, and under the necrotic epithelium no gingival fibres can be recognized as the whole connective tissue has undergone necrosis also.

2.3.7 Alveolar bone

65% of all the sections show osteoclasts either on crestal margin of the alveolar bone or in the marrow spaces of the most coronal part of the bone.

27 osteoclasts are counted in these sections.

All the sections show inflammatory cells in the marrow spaces of the alveolar bone.

2.3.8 Periodontal membrane

Only in 12% of the sections there is disorientation of the peridontal fibres while in the rest the periodontal membrane is intact.

The number and size of the blood vessels in the periodontal membrane is within the normal limits.

(c) M-D sections of P2 - P3 (maxilla) P3 - P 4 (mandible)

Interdental part:

2.3.9 Epithelium

Although in all the sections, continuity of the epithelium is interrupted, there is no complete epithelial destruction.

In some cases the splint leaves a depression only on the epithelium.

In all the sections destruction of the sulcular epithelium remains strictly localized only in those areas where the splint ligatures were located.

In 65% of all the sections there are rete pegs developed under the sulcular epithelium.

Only 35% all the sections show an apical migration of juntional epithelium.

There is no loss of epithelial attachment.

30% of the sections show no epithelial keratin layer.

No plaque accumulation is seen.

2.3 10 Gingival connective tissue

In all the sections the connective tissue has been affected in the sense that there is gingival fibre disorientation, but there is no connective tissue necrosis at all. Damage to the connective tissue is strictly localized, being almost exclusively found in areas of splint and ligature placement. The rest of the tissue shows only gingival fibre disorientation.

Throughout the connective tissue is seen cellular infiltrate either moderate or severe which is on the whole distributed throughout the connective tissue.

55% of all the sectional sites show a severe cellular infiltrate while a moderate one is seen in the rest of the sections.

In almost all of the sections the whole connective tissue is equally affected without any preference for the distal or mesial aspects of the papilla.

No red blood cells can be seen in all of the sections.

All the sections show a heavy (severe) proliferation of small blood vessels throughout the connective tissue.

No dilatated blood vessels are seen.

2.3.11 Alveolar bone

41% of all the sections show osteoclasts on the crestal margin of the alveolar bone or in the marrow spaces of the most coronal part of the bone.

25 osteoclasts are seen in these sections.

All the sections show inflammatory cells in the marrow spaces of the alveolar bone (Fig. 29).

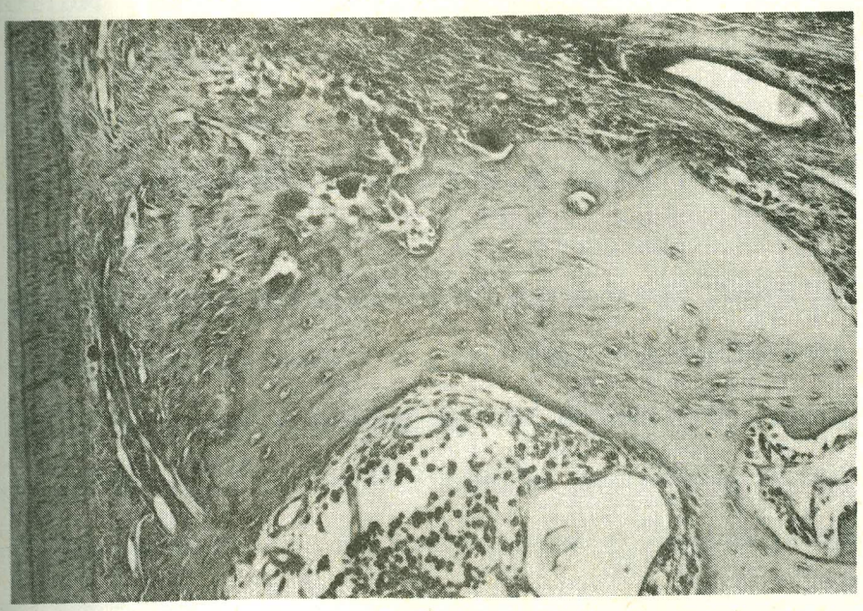


Fig. 29: Mesio-distal section of a mandibular P3 - P4 inter-dental part showing some osteoclasts on the surface of the alveolar bone and some inflammatory cells in the marrow spaces of the bone. H.E. 120 x (Merkx splint).

2.3.12 Periodontal membrane

All the sections show the periodontal ligament orientation to be normal.

The number and size of the blood vessels are within the normal limits.

2.4. OS fixed for 6 weeks

(a) Frontal sections of M1 part:

2.4.1 Epithelium

In all the sections except a part of one of them, continuity of the epithelium is maintained.

81% of the sections show a depression of epithelium at the area of splint placement.

Sulcular epithelium is intact in all the sections except in areas of splint placements where there are often seen depressions.

In 50% of the sections sulcular epithelium shows rete pegs.

Therefore it can be said that epithelial destruction is strictly localized. No preference for either buccal or lingual/palatal sides can be demonstrated.

In all the sections there is epithelial keratin layer.

There is an apical migration of junctional epithelium in 31% of all the sections, and no loss of epithelial attachment is seen.

2.4.2 Gingival connective tissue

In all the sections, the gingival connective tissue is intact as evidenced by the arrangements and orientation of the gingival fibres which is normal. Only in areas where the splint was located, there is a bit of disturbance in the fibre arrangement.

14% of the sectional sites show a normal tissue without any cellular infiltrate while in 11% of the sites there are seen scattered inflammatory cells throughout the connective tissue.

In 52% of the sites there is a moderate cellular infiltrate, while a severe infiltrate is seen in 23% of the sites.

Therefore it can be said, that a moderate inflammatory process dominates in this region.

The buccal aspect is 1.4 times more affected with cellular infiltrate than the lingual/palatal aspect as revealed by scores obtained from the sectional sites of both aspects.

75% of the sections show a proliferation of small blood vessels in the connective tissue, of which only 44% show a heavy (severe) proliferation the rest being moderate.

In all the sections the diameter of the blood vessels is normal.

In all the sections the gingival fibre orientation is distorted, although no area showed their total destruction except those areas where the splint passed through.

2.4.3 Alveolar bone

In 38% of the sections are seen osteoclasts on the crestal margin and most coronal part of the alveolar bone. The total number counted is 17.

There are inflammatory cells in the marrow spaces of the alveolar bone in 13% of the sections.

2.4.4 Periodontal membrane

In all the sections the integrity of the periodontal membrane is maintained as the orientation of the periodontal fibres is undisturbed.

No inflammatory cells are seen in the periodontal membrane.

The number and size of the blood vessels are within the normal limits.

(b) M-D sections of P3 - M1 (maxilla) P4 - M1 (mandible)

Interdental

2.4.5 Epithelium

Continuity of the epithelium is disrupted in all the sections and in 35% of the cases there is a complete epithelial destruction (Fig. 30).

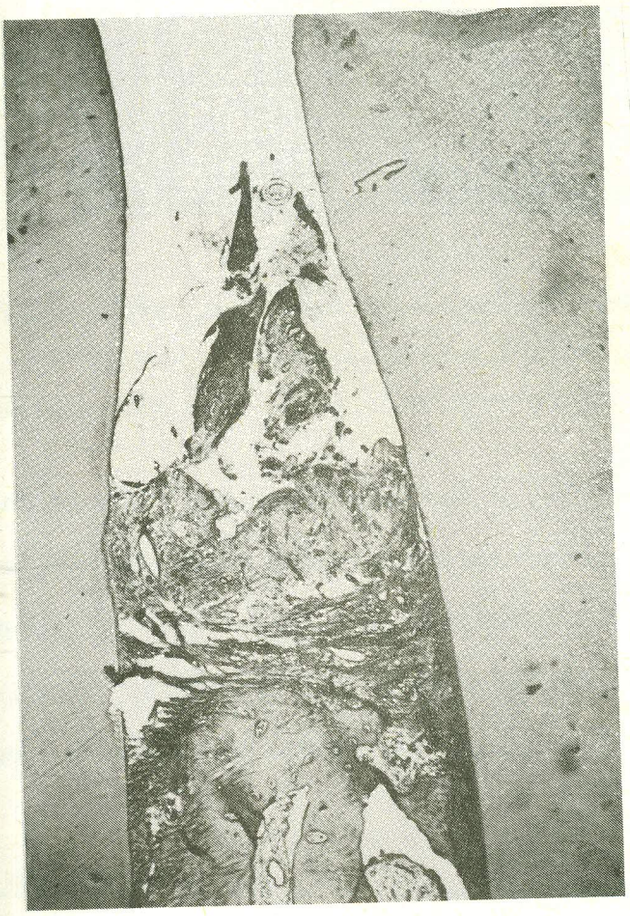


Fig. 30: Mesio-distal section of a maxillary P3-M1, interdental part showing complete epithelial destruction. H.E. 120x. (Obwegeser splint).

The rest of the sections show a strictly localized disruption in epithelial continuity involving the areas of splint placement (Fig. 31).

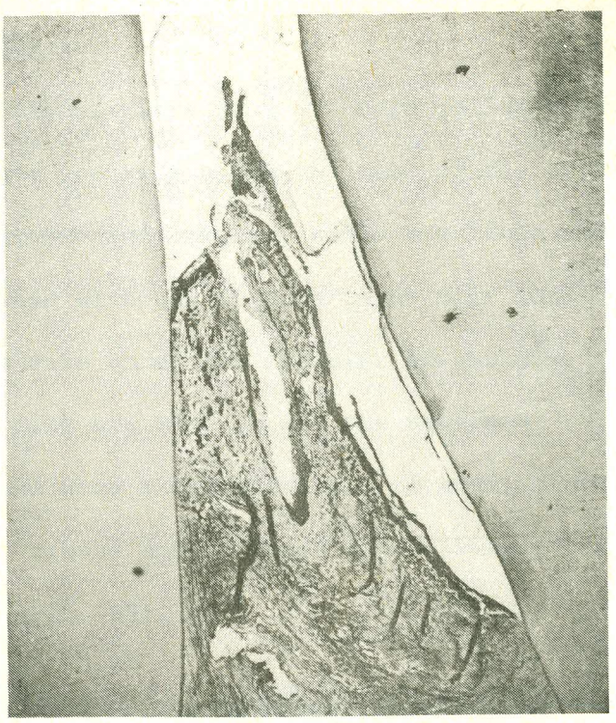


Fig. 31: Mesio-distal section of a mandibular P4-M1, interdental part showing a localized epithelial and connective tissue destruction. H.E. 48x. (Obwegeser splint).

The epithelium which remains intact has no keratin layer.

55% of the sections where sulcular epithelium is intact show a heavy (severe) proliferation of epithelium (rete pegs).

Apical migration is seen in 35% of all the sections, and 1/3 of the sections show loss of epithelial attachment.

2.4.6 Gingival connective tissue

In the 35% of the sections which show complete epithelial destruction, also approximately 1/3 of connective tissue beneath it has undergone necrosis.

29% of the sections show a localized connective tissue ulceration corresponding to the localized epithelial destruction areas.

The remaining sections show an intact connective tissue.

In all the sections there is either a moderate or a severe

cellular infiltrate.

71% of the sectional sites show a severe cellular infiltrate while a moderate one is seen in the rest of the sections.

In all the sections inflammatory cells are found throughout the connective tissue although in some cases more inflammatory cells tend to concentrate under the sulcular epithelium.

No red blood cells are seen in all the sections.

All the sections show a proliferation of small blood vessels in the connective tissue and of these approximately 3/4 show a severe proliferation (Fig. 32).

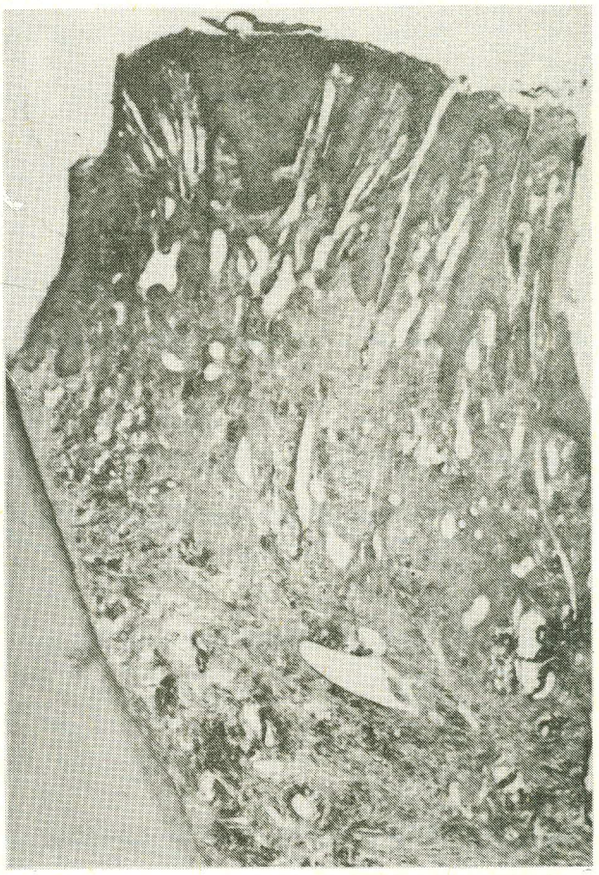


Fig. 32: Mesio-distal section of a maxillary P3-M1 interdental part showing a moderate proliferation of blood vessels in the connective tissue. H.E. 120x (Obwegeser splint).

Also amongst the small proliferated blood vessels are seen in 30% of the sections, slightly dilatated ones. These are distributed throughout the connective tissue.

In all the sections gigival fibres are disoriented.

Under the necrotic epithelium no gingival fibres can be recognized as part of the connective tissue has undergone necrosis also.

2.4.7 Alveolar bone

82% of all the sections show osteoclasts either on the crestal margin of the alveolar bone or in the marrow spaces of the most coronal part of the bone.

105 osteoclasts are counted in these sections.

All the sections show inflammatory cells in the marrow spaces of the alveolar bone.

2.4.8 Periodontal membrane

53% of the sections show a disorientation of the periodontal ligaments.

The number and size of the blood vessels in the periodontal membrane is within the normal limits.

(c) M-D sections of P2 - P3(maxilla) P4 - P4 (mandible) Interdental part:

2.4.9 Epithelium

50% of the sections show disruption in the continuity of the epithelium, while the rest show an intact epithelium. The epithelial disruptions are strictly localized (Fig. 33).

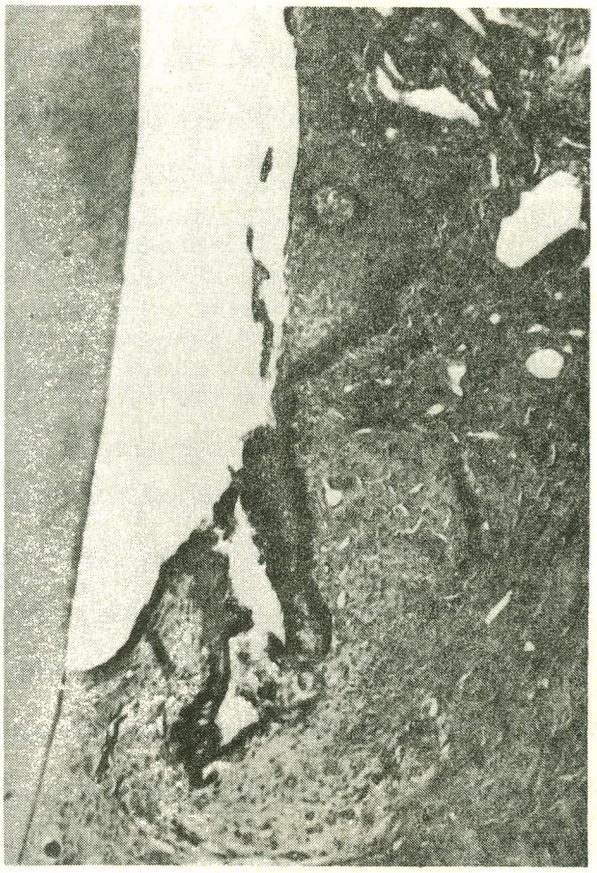


Fig. 33: Mesio-distal section of a mandibular P3-P4 interdental part showing a strictly localized epithelial destruction. H.E. 120 x (Obwegeser splint).

Sulcular epithelium remains intact except in those areas of splint placement.

In 67% there are rete pegs developed under the sulcular epithelium.

In all the sections, there is no apical migration of the junctional epithelium.

There is no loss of epithelial attachment.

33% of the sections show no epithelial keratin layer.

About 1/3 of the sections show plaque accumulation in the interdental space.

2.4.10. Gingival connective tissue

In all the sections there is gingival fibre disorganisation, but there is no connective tissue necrosis.

Damage to the connective tissue is strictly localized being exclusively found in areas of splint placement. The damage in these areas is very superficial.

Throughout the connective tissue is seen cellular infiltrate ranging from a few scattered inflammatory cells to massive (severe) infiltrate.

43% of all the sectional sites show a severe cellular infiltrate, 46% a moderate one while in 11% of the sections only scattered inflammatory cells are seen in the connective tissue.

No red blood cells are seen in the connective tissue.

All the sections show proliferation of small blood vessels in varying degrees distributed throughout the connective tissue.

56% of the sectional sites show a severe proliferation of blood vessels, 10% a moderate one and in 34% there are no inflammatory cells in the tissue.

Vasodilatation is seen in 33% of the sections.

2.4.11 Alveolar bone

67% of all the sections show osteoclasts in the crestal margin or in the marrow spaces of the most coronal part of the alveolar bone (Figs. 34 and 35).

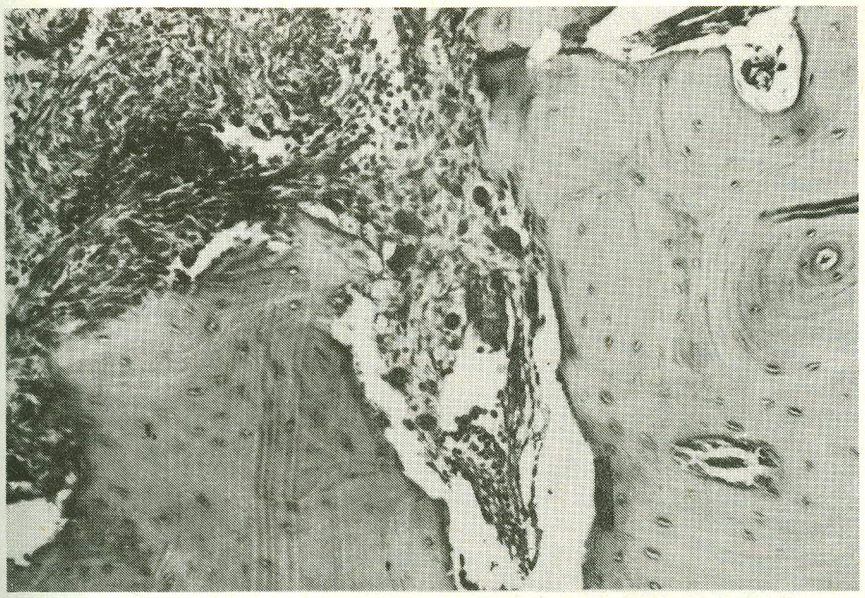


Fig. 34: Mesio-distal section of a mandibular P3-P4 interdental part showing osteoclasts on the alveolar bone. Note also inflammatory cells in the marrow spaces of the alveolar bone. H.E. 120 x (Obwegeser splint.)



Fig. 35: The same section as above magnified. 189x (Obwegeser splint).

46 osteoclasts are seen on these sections.

56% of all the sections show inflammatory cells in the marrow spaces of the alveolar bone.

2.4.12 Periodontal membrane

Periodontal ligament disorganisation is seen in 33% of the sections.

The number and size of the blood vessels are within the normal limits.

3. Integration of the histological results

48 hour group

(a) Epithelium:

1. Buccal and lingual/palatal aspects of M1.

Some of the sections show either a localized epithelial destruction or a mere epithelial depression at the place of splint location or wire ligature placement.

In the other sections continuity of the epithelium is maintained.

Thus there is no complete epithelial destruction for both types of splints.

The buccal and lingual/palatal aspects are equally affected.

Sulcular epithelium is intact in all the sections and there are no epithelial proliferations (rete pegs) under it.

In all the sections there is neither apical migration of junctional epithelium nor loss of epithelial attachment.

Epithelial destruction tends to be highly localized in the sense that it is seen only in those areas where wire ligatures or splints are located.

2. Interdental papillae.

In all the sections continuity of the epithelium is disrupted.

An average of up to 60% of the sections show a complete epithelial destruction in the case of the MERKX splint and 30% in the case of the OBWEGESER splint.

Some of the sections show already development of rete pegs in case of OBWEGESER splint while none at all are seen in the case of the MERKX splint.

In all the sections there is neither apical migration of junctional epithelium nor loss of epithelial attachment.

Epithelial destruction in the interdental papillae is generalized as it extends to cover those areas beyond splint placement especially in the case of total epithelial destruction.

SUMMARY:

1. Splinting affects the gingival epithelium be it locally or generally;
2. Splinting affects more the interdental papilla epithelium than the buccal and lingual/palatal epithelium;
3. MERKX splint does twice as much damage on the epithelium than the OBWEGESER splint, but the latter seems to provoke epithelial proliferations (rete pegs) under the sulcular epithelium at a greater speed;
4. There is no apical migration of the junctional epithelium;
5. There is no loss of epithelial attachment.

(b) Connective tissue:

1. Buccal and lingual/palatal aspects of M1.

The integrity of the gingival connective tissue is maintained except in those areas of splint or wire ligature placement, suggesting a localized destruction by splinting for both types of splints.

A moderate to severe cellular infiltrate is seen evenly distributed for both types of splints in the connective tissue although sometimes it tends to be more severe around areas of lesions.

54% of the sections show proliferation of small blood vessels in case of the MERKX splint while non at all is seen in case of the OBWEGESER splint.

50% of the sections show vasodilatation in the connective tissue in case of the MERKX splint while very few are seen in case of the OBWEGESER splint and even in these few cases vasodilatation is slight.

While 40% of the sections show gingival fibre disorientation in case of MERKX splint, in case of the OBWEGESER splint gingival fibre orientation is almost normal.

In this respect splinting with the MERKX splint affects the gingival connective tissue in the buccal and lingual/palatal aspects more than with the OBWEGESER splint.

2. The interdental papillae.

The integrity of the gingival connective tissue in most cases is maintained except in those areas of splint and wire ligature placement.

In P2 - P3 or P3 - P4 sections connective tissue destruction tends to be generalized as at least 1/3 of the coronal connective tissue is destroyed in 65% of the sections in case of the MERKXsplint and 56% in case of the OBWEGESER splint.

In all the cases a moderate to severe cellular infiltrate is found to be evenly distributed throughout the connective tissue. In P2-P3 or P3 -P4 sections, 65% of the sections show a severe cellular infiltrate, in the case of MERKX splint while the percentage is 44% in the case of OBWEGESER splint.

All the sections show red blood cells scattered all over the connective tissue.

An average of 65% of the sections show a proliferation of small blood vessels in case of the MERKX splint and 70% in case of the OBWEGESER splint. But it should be noted that the proliferation

in the latter case is less severe than in the former. These small blood vessels are found all over the connective tissue.

Vasodilatation is seen in all the sections, except the P3 - M1 or P4 - M1 ones of the OBWEGESER splint, where it is seen only in 65% of the sections.

Gingival fibre disorientation is seen in all the sections.

SUMMARY:

1. Splinting with either of the two splints affects the gingival connective tissue be it locally or generally;
2. The interdental papillae connective tissue is more affected (generalized) than that in the buccal and oral aspects, where it is localized;
3. Although the MERKX splint is seen to do more damage than the OBWEGESER splint, in the majority of cases, the difference is small.

(c) Alveolar bone:

The contour of the alveolar bone is normal in all the sections.

In all the sections no osteoclasts are seen either on the crest of the alveolar bone or anywhere else in the bone.

No inflammatory cells are seen anywhere in the marrow spaces of the alveolar bone.

At this stage the alveolar bone is unaffected by both types of splints.

(d) The periodontal membrane:

1. Buccal and lingual/palatal aspects of M1.

The integrity of the periodontal membrane is maintained as the orientation of the periodontal fibres is undisturbed.
There are no inflammatory cells and the number and diameter of the blood vessels remains within the normal limits.

2. The interdental papillae.

The integrity of the periodontal membrane is maintained in all the sections except in 38% of sections taken from P2- P3 or P3 - P4 area in case of the OBWEGESER splint.
An average of 50% of the sections show some isolated scattered inflammatory cells in the most coronal part of the periodontal membrane in case of the MERKX splint while in case of the OBWEGESER splint the percentage is only 4.
In all the sections, the number and size of blood vessels remains within the normal limits.
It can therefore be said that splinting affects the periodontal membrane of the interdental part at a very small scale and of the two, MERKX splint does more harm.

SUMMARY:

1. Splinting with either of the two types of splints does not affect the alveolar bone;
2. Splinting,slightly affects the periodontal membrane of the interdental part, and of the two, MERKX splint does more harm.

6 week group

(a) Epithelium:

1. Buccal and lingual/palatal aspects of M1.

In all the sections continuity of the epithelium is disrupted in the case of MERKX splint while it remains intact in the case of OBWEGESER splint.

In the former splint in 53% of the sections the buccal epithelium is partly or totally missing. In the OBWEGESER splint in 81% of the sections mere epithelial depressions are observed at places of splint placement.

Sulcular epithelial involvement remains strictly localized.

Epithelial proliferations under sulcular epithelium are seen in all sections in case of the MERKX splint while only ½ of the sections in case of the OBWEGESER splint show such proliferations.

About 40% of the sections show apical migration of the junctional epithelium in case of the MERKX splint while the percentage is 30% in case of the OBWEGESER splint.

There is no loss of epithelial attachment.

2. The interdental papillae.

Epithelial continuity is disrupted in all the sections and in approximately 1/3 of the sections there is a complete epithelial destruction for both of the splints.

In 65% of P2 - P3 or P3 - P4 sections there is a considerable epithelial proliferation (rete pegs) under the sulcular epithelium in the case of MERKX splint while the same process is observed in 67% of the sections in case of the OBWEGESER splint.

Apical migration of the junctional epithelium is seen in approximately 1/3 of the sections except the P2 - P3 or P3 - P4 sections where in case of OBWEGESER splint there is no migration.

There is no loss of epithelial attachment.

SUMMARY:

1. New features appear in the 6 week group
   i) sulcular epithelial destruction
   ii) sulcular epithelial proliferations
   iii) apical migration of junctional epithelium
   iv) epithelial destruction which was strictly localized in the lingual/palatal and buccal aspects of the 48 hour group, here we find also being generalized in some sections.
2. Splinting for 6 weeks affects the interdental papillae and the buccal and oral epithelium more or less equally for both types of splints;
3. On the average, the MERKX splint does a bit more damage to the epithelium than the OBWEGESER splint.

(b) Connective tissue:

1. Buccal and lingual/palatal aspects of M1.

The integrity of the connective tissue is maintained except in those areas of splint and ligature placement. In case of the MERKX splint the connective tissue beneath the ulcerated epithelium has undergone necrosis while in case of the OBWEGESER splint there is a slight disturbance ingingival fibre arrangement in areas where the splint was located.

All sections in case of the MERKX splint show cellular infiltrate

in the connective tissue ranging from moderate to severe, while in 86% in case of OBWEGESER splint is seen a moderate inflammation.
In 82% of the sections there is a severe proliferation of small blood vessels in case of MERKX splint while it is 75% in case of OBWEGESER splint of which only half is severe.
The diameter of blood vessels remains normal in all the sections.
In all sections there is gingival fibre disorientation especially in those areas where either a splint or wire ligatures passed through.
In case of MERKX splint gingival fibres are completely destroyed in those areas under ulcerated epithelium.
In case of OBWEGESER splint there is no total destruction seen.

2. The interdental papillae.
In the P3 - M1 or P4 - M1 sections there is gingival fibre disorganization and beneath necrotic epithelium also the connective tissue has undergone necrosis. The situation is worse in case of OBWEGESER splint where necrosis extends to 1/3 of the connective tissue.
All the sections show a moderate to severe cellular infiltrate distributed throughout the connective tissue.
An average of 70% show severe cellular infiltrate in case of MERKX splint while the percentage is 55 in case of OBWEGESER splint.
In all the sections there are no red blood cells, and there is a severe proliferation of blood vessels.
Vasodilatation is seen in only 30% of the sections.
Splinting causes a considerable damage to the connective tissue

of the gingiva.

SUMMARY:

1. Connective tissue destruction is still localized in the lingual/palatal and buccal aspects. It is generalized in the interdental papilla like in the 48 hour group;
2. The interdental papilla connective tissue is affected more by both types of splints than thelingual/palatal and buccal aspects of the gingiva;
3. Vasodilatation is reduced from 50% of the sections in the 48 hour group to 30% in the 6 week group;
4. There are no red blood cells seen in the gingival connective tissue;
5. Both quantitatively and qualitatively the MERKX splint slightly does more damage than the OBWEGESER splint.

(c) alveolar bone:

1. Buccal and lingual/palatal aspects.

   41% of the sections in case of MERKX splint and 38% in case of the OBWEGESER splint show osteoclasts on the crestal margin and most coronal part of the alveolar bone.

   Inflammatory cells are found in the marrow spaces of the alveolar bone.

2. Interdental papillae.

   More sections show osteoclasts in case of OBWEGESER splint than MERKX splint.

   All sections show inflammatory cells in MERKX splint while the percentage is only 13% in case of OBWEGESER splint.

It can therefore be said that in this respect splinting provokes alveolar bone inflammation and resorption. Although MERKX splint provokes much more bone inflammation, OBWEGESER splint provokes more alveolar bone resorption.

(d) Periodontal membrane:

1. Buccal and lingual/palatal aspects.

   Integrity of periodontal membrane is maintained and the number and size of blood vessels is normal.

2. Interdental papillae.

   Disorientation of periodontal membrane is seen in twice as many sections in case of OBWEGESER splint than the MERKX one.

SUMMARY:

1. After 6 weeks of splinting there is a slight effect on the periodontal membrane;
2. There is a reversal in effect of the two types of splints on the periodontal membrane. While the MERKX splint does more harm on the periodontal membrane in the 48 hour group, the OBWEGESER splint does more harm in the 6 week group.

V. DISCUSSION

1. Review of the literature

A few studies have been undertaken to assess the effect of splints on the periodontal tissues.

Härle et al. (1977) studied clinically the effect of Stout-Obwegeser splint on the marginal periodontium in 12 patients with jaw fractures and 7 patients with jaw osteotomies. The periodontium of these patients was examined before splinting, 1 week after splinting, 6 weeks after splinting and finally 6 weeks after splint removal.

All the patients were instructed to maintain a thorough oral hygiene during the period they had splints. The parameters used to determine the periodontal health status were:

- tooth mobility. This was examined clinically and graded as follows:
  grade 0 = no mobility
  grade 1 = mobility which can be felt by fingers
  grade 2 = mobility which can be felt and the tooth movement seen also
  grade 3 = mobility which can be effected by lip and tongue.
- pocket depth. This was measured with the help of a calibrated periodontal probe.
- sulcus bleeding index used as a parameter for inflammation according to Mühlemann and Son (1971).

It was found that of the 233 ligated teeth, only 2 showed grade 2 mobility after one week. The tooth mobility decreased with time and

eventually disappeared 6 weeks after splint removal. Pocket depth and sulcus bleeding indices increased with time during the 6 weeks of splinting, although 6 weeks after splint removal, initial values were reached. The authors conclude that the effect of Stout-Obwegeser splint on marginal periodontium is minimal and need not be considered.

Bienengräber et al. (1973) performed clinical studies and an animal experiment on the effect of wire splinting on the marginal part of the periodontium.

Clinical studies:

The effect of splinting was studied in 100 patients with jaw fractures splinted with an arch bar (type not further specified) without intermaxillary fixation for 6 weeks. The periodontium of these patients was examined prior to splinting, 3 weeks after splinting and after 6 weeks when the splint had just been removed. The parameters used were the same as those used by Härle et al. (1977). It was found that splints induced gingivitis which heals after splint removal almost in all the patients.

Animal experiments:

7 dogs were splinted with wire ligatures (exact type of splint is not mentioned) for 4 weeks without oral hygiene measures. After 4 weeks mild gingivitis could be seen, but 6 weeks after splint removal, it disappeared. Histological preparations from the dogs showed lymphatic cellular infiltrate, together with plasma cells under the gingival epithelium. Collagen fibres and ground

substance were disintegrated. 24 weeks after splint removal, a slight subepithelial fibrosis and a small epithelial atrophy is seen. The authors conclude that the damage done by splints on the periodontium is minimal, and the animal experiment excludes a serious permanent periodontal damage.

Fröhlich and Gäbler (1981) performed clinical investigations to find out the effect of splints on the periodontium. 24 patients with jaw fractures were splinted with semi-roundish arch bars fixed with wire ligatures. The same procedures for periodontal assessment were used except that for the first time, as parameters, were used the plaque index system according to Silness and Löe (1964), the gingival colour and swelling, and the gingival recession. It was found that splinting produces gingivitis which was not considered by the authors to be a serious problem. However there was no follow up after splint removal.

It will be noted that with the exception of one, all the above mentioned investigations so far have been restricted to clinical observations. The knowledge as to what actually happens, at microscopical level, to the periodontal tissues remains limited. Although all the authors conclude in these observations that periodontal damage done by splints is minimal it remains unclear as to how minimal it is in terms of histological assessment.

The studies done by Härle et al (1977) and von Bienengräber et al (1973) omit plaque index as a parameter in assessing the periodontal health status. As a close relationship between plaque and periodontal disease has been established (Saxe et al, 1967; Waerhaug, 1975;

Hamp et al, 1972; Hamp et al, 1973; Lindhe and Löe, 1973; Schroeder et al 1975; Schroeder & Lindhe, 1975) it seems that information obtained with the exclusion of such an important parameter is far from complete. However, factors such as individual oral hygiene and intermaxillary fixation make objective assessment more difficult anyway in a clinical study.

In the study of von Bienengräber et al.(1973) the exact type of splints used is not mentioned and it is assumed that the periodontal tissues react in the same manner for whatever type of splint. Since the basis of such assumption is not established, the information obtained from these studies remains incomplete.

2. The present investigations

i) Short term effect of splinting (48 hours)

The present investigation has demonstrated that in dogs, splinting with the Merkx or Obwegeser splints affects adversely the periodontal tissues both at clinical and histological levels when oral hygiene measures are neglected.

The fact that already after 48 hours the splinted sides show plaque accumulation suggests that during that time the splints have acted as plaque retentive devices in the oral cavity. This is confirmed by the statistical analysis which shows a significant rise in the plaque index as is expressed in the splint effect.

Clinically, the Merkx splint seems to accumulate more plaque than the Obwegeser splint. The greater plaque-retentive nature of the Merkx splint is also demonstrated by its higher splint effect on the plaque index. This is not unexpected as the larger cross-sectional size of the Merkx splint would be expected to give more plaque

retention. However, the difference between the two types of splints as far as the splint effect on the plaque index is concerned is not statistically significant. This is caused by the large standard deviations of the means as shown in graph 1. The clinical observations that irrespective of the type of splint, on the experimental sides the gingiva is swollen, reddish in colour and bleeds easily on probing, and the fact that statistical analysis shows a significant rise in the gingival index and pocket depth, suggests that an inflammatory process is taking place in the gingiva due to splinting. This is confirmed by the presence of cellular infiltrate and vasodilatation in the connective tissue in histological sections.

The splints provoke gingival inflammatory reaction through two mechanisms:

1. Directly

This mechanism in turn operates in two ways:

a) traumatic effect: the act of splinting itself is injurious and causes damage to the gingiva. This is confirmed by the histological sections where localized destruction of the gingival connective tissue is seen in places where the splint ligatures passed.

The injurious effect of the splints is further histologically demonstrated by the presence of red blood cells in the gingival connective tissue suggesting a haemorrhagic process taking place in the tissue. The constant presence of the splint would prevent healing and hence the persistence of injury and therefore inflammation. It is important to note that the traumatic factor, in respect of the two types of splints tested is not a significant variant as in either case the gingiva is perforated by wire ligatures. However, the skill of the surgeon in splinting would be an important factor.

b) irritation: the constant presence of the splint provides an external constant mechanical irritating factor to the gingiva, which in turn reacts through inflammation. This factor would depend on the size, shape and degree of smoothness of the splints. The Merkx splint has a larger contact surface area than the Obwegeser splint and this seems to explain, at least in part, the greater effect the Merkx splint has on the gingiva.

2. Indirectly

Through this mechanism the splint allows plaque accumulation which in turn provokes gingival inflammation. The pathogenicity of plaque bacteria in the development of gingivitis has already been established (Lövdal et al, 1958; Schei et al, 1959; Löe et al, 1965; Theilade et al, 1966; Saxe et al, 1967; Socransky et al, 1970; and Lindhe et al, 1975).

On the other hand, since in the process of splinting wire ligatures perforate the gingival epithelium at several places, the places of perforation would act as portal of entry for bacteria and toxins in the gingival connective tissue and cause septic gingival inflammation.

Comparing splint effect on plaque and gingival indices for each dog we see the following:

Dog no. 1: The Merkx splint has more adverse effect on the plaque. Obwegeser splint has more adverse effect on the gingival index (an indicator of gingival inflammation).

Thus, although the Obwegeser splint has less plaque effect, it brings about more inflammatory response than the Merkx splint (compare graphs 1 and 2).

Dog. no 2: The Obwegeser splint has more adverse effect on the plaque. The Merkx splint has more adverse effect on the gingival index.

Thus although the Merkx splint brings about less plaque it brings about more inflammatory response.

Dog no. 3: The Merkx splint has more adverse effect on the plaque.

The Obwegeser splint has more adverse effect on the gingival index.

Thus, although the Obwegeser splint brings about less plaque effect, it results into more inflammatory response.

Apparently the amount of inflammation does not depend on the amount of plaque in this animal model. It seems therefore, that at this stage, the direct traumatising factor plays a major role in provoking inflammatory response of the gingival tissues.

Sometimes the Merkx splint provokes more inflammation, sometimes the Obwegeser splint does.Probably this depends on the act of splinting rather than the splint itself as sometimes the process has to be repeated several times before it succeeds at a particular point.

Whatever the case, we see that even though not at a significant level, the Merkx splint compared to the Obwegeser splint allows more damage to the gingival epithelium (having up to 60% of the sections with total epithelial destruction compared to only 30% in case of the Obwegeser splint) and it does more damage to the gingival connective tissue.

However, these results in the present investigation should be extrapolated to man with care, taking the following into consideration:

- in the animal experiment, the splints were fixed without jaw fractures and intermaxillary fixation.

  Normally the splints are applied to human beings in case of

jaw fractures or in case of immobilising jaws after osteotomy; and usually intermaxillary fixation is done.

- In the course of splinting wire ligatures perforated the interdental papilla in most cases. This can usually be avoided in the humans.
- In the experiments the animals were not subjected to oral hygiene measures normally instructed to patients with splints.

Nevertheless, the present investigation has demonstrated how the periodontal tissues react to splints after 48 hours in the absence of oral hygiene measures.

Since both types of splints were subjected to the same testing conditions it is possible to compare them. In this respect it can be concluded that Merkx splint after 48 hours would do more damage to the periodontal tissues than the Obwegeser splint if oral hygiene is neglected.

The clinical periodontal index scores suggest, that for both types of splints, the interdental papilla is more affected than the buccal and lingual/palatal aspects of the gingiva. This is confirmed by the histological results which show more gingival epithelial and connective tissue destruction in the interdental papilla than in the buccal and lingual/palatal aspects. This is not unexpected since in the course of splinting, wire ligatures pass through the interdental papilla. Furthermore, the interdental space is more plaque retentive.

For both types of splints we see that the alveolar bone is not yet affected and only 38% of the sections show a few scattered inflammatory cells in the periodontal membrane.

We can therefore say that since the inflammatory response remains almost confined to the gingiva, the short term effect of the splints

is to cause gingivitis.

Gingivitis, which was observed in patients by Härle et al. (1977) after one week in the present investigation is seen as early as 48 hours after splinting. Most likely, even in humans the first gingival response to the splints could be demonstrated earlier than one week at least at a subclinical level.

ii) Long term effect of splinting (3 and 6 weeks)

After 3 weeks of splinting, clinically there is an increase in plaque accumulation around the splints. The Merkx splint seems to accumulate more plaque. This is confirmed by the statistical analysis which shows that the Merkx splint has a significant greater effect on the plaque index than the Obwegeser splint. If the indirect way of provoking gingival inflammation would dominate, we should find more damage to the periodontal tissues by the Merkx splint.
Since at that moment no histological evaluation took place, we have to rely on the results after 6 weeks.
At this stage also the other indices (gingival and sulcus/pocket depth indices) remain raised to a significant level. This suggests that after 3 weeks of splinting there is still an inflammatory process taking place in the gingiva.
It is not possible to tell which mechanism predominates in provoking gingival inflammation.
After 6 weeks of splinting, the same clinical picture of plaque accumulation as that after 3 weeks could be seen. Statistically, it appears that still the periodontal indices are raised up to a significant level for both types of splints compared to the normal situation.

The Merkx splint has a significantly greater effect on plaque index than the Obwegeser splint.The fact that all the indices at this time remain raised at a significant level suggests that still there is an inflammatory process taking place in the gingiva.
Histological results confirm and reveal new features in periodontal destruction which could not be seen after 48 hours of splinting and these are:

- total epithelial destruction
- sulcular epithelial destruction
- sulcular epithelial proliferations
- apical migration of junctional epithelium
- epithelial destruction which was strictly localized in the lingual/palatal and buccal aspects of the gingiva in the 48 hour group, here it is generalized in some sections.

These histopathological features which appear for both types of splints, show that after 6 weeks of splinting gingival epithelial destruction has increased. We can therefore conclude that with increasing time of splinting up to 6 weeks, both types of splints increase the damage to the gingival epithelium.
One interesting finding is that the splint effect on the plaque index for both types of splints seems to decrease between 3 and 6 weeks of splinting. Probably this is due to the fact that by this time the animals had got used to the splints in their mouths, got back to their original or closer to original chewing habits and therefore self-cleansing mechanisms became more efficient.
However, histologically it has been found that despite the apparent decrease in plaque the inflammatory response becomes severe. That is

just like in the 48 hour group, it has been shown that the inflammatory response does not depend on the amount of plaque even in the 6 week group. Perhaps there is a critical level of plaque accumulation which, once reached, further increase in the amount of plaque will not make any difference. This requires further investigation.

In this investigation the fall in the splint effect on the plaque index is not significant. Since the standard deviations of the means are large (graph 4) a bigger number of animals would be required to generalize these findings.

Statistically there is a general slight increase in the splint effect on the gingival and sulcus/pocket depth indices for both types of splints (graphs 5 and 6).

This would suggest that there is an increase of gingival inflammatory response at this time (6 weeks).

However, histologically we find that the inflammatory process in the connective tissue is more or less the same as that seen in the 48 hour group except that there is now less vasodilatation and no red blood cells. The severe proliferation of small blood vessels suggests the inflammatory process is already taking a chronic picture. At this stage it must be assumed that the mechanisms through which the splints would operate in bringing about and maintaining inflammation is the direct and indirect ways. The presence of the splint provides a constant irritating factor which prevents any healing of the traumatized gingival tissues.

The radiological findings suggest, that there is alveolar bone resorption taking place. This is confirmed by the presence of osteoclasts seen on the bone in the histological sections.

The presence of inflammatory cells in the marrow spaces of the alveolar bone indicates that the inflammatory process has reached the bone.

The disorientation of the periodontal fibres shows that the periodontal membrane is also affected.

It can therefore be stated that after 6 weeks of splinting for both types of splints, the periodontal inflammatory response has extended into deeper tissues and the reaction has changed from gingivitis to periodontitis.

For the same reasons as those given for the 48 hour group, it is possible to compare the two types of splints.

On the average we find that, statistically the Merkx splint has more effect on the plaque index (there is a significant difference between the two types of splints) and also on the gingival and sulcus/pocket depth indices.

This, would suggest that the Merkx splint seems to cause more harm to the periodontal tissues than the Obwegeser splint. This is partially confirmed by the histological results which show that the Merkx splint does slightly more damage to the gingival epithelium and connective tissue.

However, it is worthy noting that while the Merkx splint seems to provoke more alveolar bone inflammation, the Obwegeser splint seems to provoke more alveolar bone resorption. As one would expect that the splint that causes more bone inflammation would also cause more bone resorption, this finding remains to be explained.

It is also interesting to note that the effect of the two types of splints on the periodontal membrane is reversed at the end of the experimental period.

While the Merkx splint does more harm on the periodontal membrane after 48 hours, the Obwegeser splint does more harm after 6 weeks of splinting, for unknown reasons.

These results show that the Merkx splint causes more gingivitis while the Obwegeser splint causes more periodontal breakdown (periodontitis) after 6 weeks of splinting.

The results of the present investigation agree with those of Härle et al. (1977), von Bienengräber et al. (1973) and Fröhlich and Gäbler (1981) who found gingival inflammation in splinted patients despite the thorough oral hygiene measures they were instructed to undertake. However, the present investigation reveals through the histological results that what is seen clinically as gingivitis actually after 6 weeks of splinting has changed to periodontitis. It is expected that also in humans within 6 weeks of splinting, the periodontal tissues will react by developing periodontitis.

The histological results in the present investigation agree with those of von Bienengräber et al (1973), only that while the authors found mild gingivitis in the dogs after 4 weeks, in the present investigation as early as 3 weeks after splinting severe gingivitis could be seen.

While all the above mentioned authors finally conclude that the damage done by splints to periodontal tissues is minimal, we feel that the damage is serious for both types of splints tested. Whether or not the damage is reversible after splint removal remains to be studied as it has not been within the scope of this investigation.

As repeatedly pointed out, direct conclusions cannot be drawn and applied to the situation in human beings.

Three obvious differences have to be considered as to their effects:

- In humans splints can be placed in a considerably less traumatic way due to the more favourable tooth morphology and the contact points usually present. Usually the ligature will pass besides the papilla and only exceptionally it has to pierce it. In the animals this could not be avoided and wire ligatures had to pierce the papilla.
- Normally, oral hygiene measures are carried out in splinted patients, but this was not carried out in the experimental animals. Plaque inhibition (Renggli, 1983) or at least reduction in plaque accumulation will certainly have a favourable influence on the condition of periodontal tissues.
  This may lead to less marked damage or difference in reactions to different types of splints used. On the other hand one could also imagine that one type of splint could benefit more from oral hygiene measures, than the other.
- Finally intermaxillary fixation which very often has to be applied to humans will have an influence too. It is considered that this could have an adverse effect on oral hygiene.
  Also even immobilisation as such and the change in eating habits are factors which we were not able to study.

VI. CONCLUSIONS

Based on this animal experiment the following conclusions are drawn:

1. Throughout the splinting time, the Merkx and Obwegeser splints act as plaque retentive devices.

2. The amount of plaque which the splints retain increases up to 3 weeks of splinting. Thereafter there seems to be a decrease (though not significant in the present investigation) in plaque accumulation .

3. Of the two types of splints, the Merkx splint causes more plaque retention than the Obwegeser splint whether it is applied for a short time (48 hours) or longer time (6 weeks).

4. Both types of splints cause gingivitis when applied for a short time (48 hours). With increase in splinting time, the periodontal tissue reaction changes from gingivitis to periodontal breakdown (periodontitis).
   Also concomitantly the gingival connective tissue inflammatory response changes from acute to chronic. The reaction of periodontal tissues becomes most severe with increase in time of splinting.

5. The effect of splints on periodontal tissues is serious enough for further consideration.

6. Comparing the two types of splints, the Meerkx one seems to cause more damage to the gingival epithelium and more gingivitis as a short term (48 hours) effect as well as a long term effect (6 weeks) of splinting.
   However, the Obwegeser splint seems to cause more periodontitis in the long run (after 6 weeks of splinting).

7. Since it has been demonstrated in this investigation that the periodontal tissues react differently to the two types of splints tested, it is possible that for every type of splint the periodontal tissues will react differently. This requires further investigations extending the comparative studies to cover other types of splints as well.

8. On selecting splints for treatment, serious consideration should be given concerning the effect of each type of splint on the periodontal tissues and suitable corresponding measures taken to curb this effect. These measures incluse in any case vigorous oral hygiene.

9. Although this experiment sheds light on the behaviour of periodontal tissues under splinting conditions, direct conclusions may not be drawn concerning the application in humans because only a limitied number of factors could be evaluated.

VII. SUMMARY

Modern technology has developed more splints for the use in the treatment of jaw fractures and immobilization after jaw osteotomies than ever before. Often in the literature one comes across a situation where an author advocates the use of a particular type of splint while another abdocates another type for the same case without due reasons. Occasionally, the advantages and disadvantages of the splints have been cited in the literature.

However, the splints have mainly been evaluated from technical point of view and the biological aspect has not been given the weight it deserves. Mostly a splint is considered advantageous if it can be applied with ease, is not time consuming and not expensive.

No note is taken considering its side effects on the patient.

The effect of the splints on periodontal tissues has not been given sufficient consideration. There is no doubt that splints act as plaque retentive appliances and therefore must have an effect on periodontal tissues. Clinical studies have suggested however, that splints are tolerated without leaving significant scars.

It is in the light of this that an animal experiment was set in order to answer the following question:

1. What is the effect of splinting on the periodontal tissues (Both at clinical and histological levels)?
2. Is the effect of splinting on the periodontal tissues the same for whatever type of splint?
3. Is the splint effect related to splinting time?

6 beagle dogs were used for the experiment. They were divided into 2 groups of threes. The first group was splinted for 48 hours to assess the short term effect of splinting, while the second one was splinted for 6 weeks to see the long term effect of splinting. Two types of splints, one of the arch bar type (Merkx, unpublished) and one of the loop wiring type (Obwegeser, 1952) were evaluated. On each dog the two types of splints were fixed one on the upper and the other on the lower jaw. On each jaw one quadrant was used as experimental while the opposite one served as a control. Clinical and radiological assessment of the dog's periodontium was done before and after splinting for each group except that for the 6 week group these procedures were done also after 3 weeks of splinting. For clinical evaluation the following periodontal indices were used:

1. The plaque index system (Silness and Löe, 1964)
2. The calculus index (Ramfjord, 1959)
3. The gingival index system (Löe and Silness, 1963)
4. Sulcus (pocket) depth.

The data obtained from these periodontal index scores was transferred to an IBM 4341 computer and analysed as follows:
In order to obtain periodontal index for one tooth, indices for each side (buccal, mesial, palatal/lingual and distal)were added together. For each tooth then the effect of the splint was calculated as follows: (index at the end of splinting - index before splinting) - (control index at the end of splinting - control index before splinting) = splint effect.

For each group mean values and standard deviations were calculated for each index.

At the end of the splinting time the dogs were sacrificed for routine histological preparations for sections which were stained with H.E. and examined with the aid of a light microscope.

Short term effect of splinting (48 hours)

The present investigation demonstrates that, in the dogs, splinting with the Merkx or Obwegeser splints adversely affects the periodontal tissues both at clinical and histological levels.

Both types of splints act as plaque retentive devices, the Merkx splint being more retentive than the Obwegeser splint.

The clinical observations that irrespective of the type of splint, on the experimental side,the gingiva is swollen, reddish in colour and bleeds easily on probing and the fact that statistical analysis shows a significant rise in the gingival index and pocket depth suggests that an inflammatory process is taking place in the gingiva due to splinting. This is confirmed by the presence of cellular infiltrate and vasodilatation in the connective tissue in histological sections.

It is suggested here that splints would provoke gingival inflammatory reaction through 2 mechanisms:

1. Directly -the splint provoking inflammation through its traumatic effect or irritation.
2. Indirectly -the splint allowing for plaque accumulation which in turn provokes gingival inflammation. Finally as the wires perforate the gingival epithelium, the places of perforation act as portal of entry for bacteria and toxins.

Comparing the two types of splints it has been demonstrated that even though not at a statistically significant level, the Merkx splint allows more plaque accumulation and does more damage to the gingival epithelium and connective tissue.

Comparing the sites of splinting, it has been shown that for both types of splints the interdental papilla is more affected than the buccal and lingual/palatal aspects of the gingiva.

Since the inflammatory response for both types of splints remains almost confined to the gingiva, it can be said that the short term effect of the splints is to cause gingivitis.

Long term effect of splinting

After 3 weeks of splinting all the periodontal indices remain raised indicating that the inflammatory process is still persisting.

After 6 weeks of splinting the histopathological features that appear show that for both types of splints gingival epithelial destruction worsens with time (up to 6 weeks) and gingival inflammation seems to change from acute to chronic.

The present investigation demonstrates also that with time (up to 6 weeks of splinting) for both types of splints the imflammatory process changes from gingivitis to periodontitis, as revealed by alveolar bone resorption and inflammatory cells in the marrow spaces of the bone.

It has been shown further that the Merkx splint causes more gingivitis while the Obwegeser splint causes more periodontal breakdown (periodontitis) after 6 weeks of splinting.

In conclusion although for most parameters statistically significant differences could not be demonstrated, the tissue reactions on the

two splints are not exactly the same. Every splint used should therefore be evaluated carefully.

The most important questions arising are whether the changes described at histological level are completely reversible; and whether oral hygiene can reduce the damage sufficiently enough as to continue with the use of these devices without being forced to search for alternatives.