ALVEOLAR OSTEITIS: A REVIEW

A treatise submitted in partial fulfilment of the requirements for the degree of Master of Dental Surgery

by

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For
Deborah
Christopher
and
Stephen
Acknowledgements

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Introduction

The aim of this treatise is to delineate current thinking as to the cause, prevention and treatment of alveolar osteitis. "Dry socket" has probably been around since the practice of exodontia began. The disease has no boundaries. It afflicts all races equally and is remarkably consistent in its overall incidence.

The pathogenesis of alveolar osteitis is as elusive now as it was when first documented in 1896. A number of hypotheses have emerged in recent times, however, clinical findings, laboratory data and treatment outcomes have often been contradictory. The conflicting nature of this evidence seriously questions the validity of most arguments.

Treatment of alveolar osteitis has traditionally been empirical, although nowadays attempts are being made to treat the cause as well as the symptoms. By and large, the observation that an extraction socket afflicted with alveolar osteitis need only be covered to effect a cure still holds true today. This understanding provides the basis for the effectiveness of most, if not all, "dry socket" dressings currently in use.

Mandibular third molar extraction sockets are almost legendary in their predilection for alveolar osteitis. Reported incidence rates of 20% - 60% are not uncommon in the dental literature. Even so, an elevated
incidence for the mandibular third molar site is to be expected, given the considerable iatrogenic trauma associated with such surgery.

Although alveolar osteitis is benign in the sense that it is not life-threatening, it nevertheless causes an inordinate amount of suffering to those afflicted. Our role as dentists is to relieve the suffering caused by dental disease and in no other discipline of dentistry is there as much appreciation as when the incessant, dull, throbbing pain of "dry socket" is finally relieved. A thorough understanding of alveolar osteitis is perhaps our single most potent armamentarium.
Chapter 1

Definition, Clinical Features and Diagnosis of Alveolar Osteitis.

1.1 Definition.

It is generally agreed that the first documentation of alveolar osteitis was by a Dr. J.Y. Crawford (1896) who coined the term 'Dry Socket'. In a regular meeting of the Dental Society of the State of New York, U.S.A. in August 1896, J.Y. Crawford related a peculiar condition he termed dry sockets. Two cases were presented: one of 12 months duration! The other a more conventional type which had been present for two weeks. He was singularly impressed by the perfectly clean, dry, open socket and the severe pain this caused.

Crawford's (1896) definition of a dry socket is one where a patient experiences severe pain which affects the whole side of the face, head and ear. There is no blood, no suppuration, no granulation, but a perfectly dry, clean socket. This definition was given in relation to a patient who suffered the onset of symptoms within 24 hours of having had her left lower third molar removed. Crawford's definition is based solely on a description of the clinical findings and symptoms of alveolar osteitis. Nearly a century later, the accepted definition is essentially the same.

Silverman (1935), Williams (1935), Podolin (1936) and Schram (1947) considered alveolar osteitis to be localised osteomyelitis.

Alling and Kerr (1957) described alveolar osteitis in terms of a concept
where the following features should be present:

i) Severe pain arising from a tooth socket postoperatively, usually two to three days after extraction.

ii) The blood clot is either disintegrating or has totally broken down.

iii) A foul odour is present.

iv) Infection and suppuration of the socket may or may not be present.

v) Histologically, the picture is one of a localised osteomyelitis or osteitis.

Archer (1966) preferred the term alveolalgia, meaning "pain in the alveolus". He considered that a history of pain is the basis for diagnosing alveolalgia. Archer also stated that the onset of pain may be from one to ten days post-extraction, it arises from the socket and may be referred to the ipsilateral ear. The presence of a partly broken down blood clot in the middle or apical third of the socket is taken as confirmation of alveolar osteitis.

Thoma (1969) described the condition of alveolar osteitis as a severe pain at the extraction site which occurs two or three days postoperatively with the presence of a necrotic odour and a foul greyish discharge.
Birn (1973) regards the following points as being pertinent to a definition of alveolar osteitis:

i) The condition usually starts two to three days after extraction, with the disintegration of the blood clot.

ii) The alveolus appears empty, with completely or partially denuded, very sensitive bony surfaces, covered by a greyish/yellow layer of detritus or necrotic tissue.

iii) The surrounding gingiva often shows inflammatory changes.

iv) The patient complains of "heavy" neuralgic type pain. In the mandible, the pain often radiates towards the ear and temporal region of the ipsilateral side. In the maxilla, the
pain is often referred to the eye and frontal region of the ipsilateral side.
v) Halitosis is often pronounced and the patient complains of a foul taste in the mouth.
vi) Systemic signs and symptoms such as an increase in temperature are rare unless there is superimposed infection.

vii) Malaise may be due to lack of sleep and appetite.

Birn (1973) also considered swelling of the regional lymph nodes (in cases of alveolar osteitis) to be an uncommon finding. In his experience, regional lymphadenopathy is infrequently seen and like an elevated temperature occurs only as a result of superinfection. This may be due to localised (e.g., suppuration of the socket) or generalised (e.g., a bout of influenza) causes, and may occur concomitantly in a patient suffering from alveolar osteitis.

1.2 Synonyms for Alveolar Osteitis.

Although alveolar osteitis is the preferred term for the purposes of this treatise, the following terms have been used in the literature:

- Avascular Socket
- Alveolalgia
- Alveolitis
- Alveolitis Sicca Dolorosa
- Dolor Post Extraction
- Dry Socket
- Fibrinolytic Alveolitis
- Localised Acute Alveolar Osteomyelitis
- Localised Osteitis
Localised Osteomyelitis
Necrotic Alveolar Socket
Necrotic Socket
Painful Socket
Post-Extraction Osteomyelitic Syndrome
Post-Operative Localised Osteitis
Post-Operative Osteitis
Sloughing Socket

1.3 Clinical Features of Alveolar Osteitis.

1.3.1 Incidence.
MacGregor (1968) in a statistical analysis of 10,199 intra-alveolar extractions carried out under local anaesthesia found the overall incidence of alveolar osteitis to be 3.2%. The occurrence following single extractions was 5% and multiple extractions was 2.1%.
Most authors agree that the incidence of alveolar osteitis from all types of extractions range from 2-4%, with an average of 3% [Krogh (1937), Millhon et al. (1943), Adkisson and Harris (1956), Hansen (1960), Turner (1982) and Field et al.(1985)].
In a local study, Buchanan (1961) found an incidence of 0.79% over a 27 month period. His study was conducted in the Exodontia Department of the United Dental Hospital of Sydney and involved 50,386 extractions under local anaesthesia, of which 400 cases developed alveolar osteitis. In a retrospective analysis of patients records available from the Departments of Oral Surgery and Exodontia at the United Dental Hospital of Sydney, for an eleven year period 1971 - 1981 (Table 1), this author found the overall incidence of alveolar osteitis to be 1.6% (extraction and surgical cases included).
Table 1.

<table>
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<td>27,300</td>
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<td>2,814</td>
<td>28,395</td>
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<td>26,320</td>
<td>237</td>
<td>1,931</td>
<td>2,458</td>
<td>30,946</td>
<td>0.96%</td>
</tr>
<tr>
<td>1979</td>
<td>372</td>
<td>24,392</td>
<td>252</td>
<td>1,741</td>
<td>2,429</td>
<td>28,814</td>
<td>1.29%</td>
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<tr>
<td>1980</td>
<td>525</td>
<td>24,498</td>
<td>260</td>
<td>1,769</td>
<td>2,521</td>
<td>29,048</td>
<td>1.80%</td>
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<tr>
<td>1981</td>
<td>473</td>
<td>24,693</td>
<td>242</td>
<td>1,923</td>
<td>2,390</td>
<td>29,248</td>
<td>1.61%</td>
</tr>
</tbody>
</table>

Overall Incidence: Total A.O cases/Total No. Extractions

\[
\frac{5,325}{332,142} = 1.60\%
\]

Legend:  
A.O. = Alveolar Osteitis.  
Ext = Simple extractions in the Exodontia clinic.  
S.R. = Surgical removal.  
(E) = Exodontia Clinic.  
(O) = Oral Surgery Clinic.  
Total = Total number of extractions that year.  
Incid. = Incidence of alveolar osteitis.
The difference in the incidence rates between the two studies (above) may be explained by the fact that Buchanan's work was confined to simple extractions, whilst this author's survey included surgical extractions of third molars. It should be borne in mind that different diagnostic criteria may seriously (unwittingly) bias the results of one study when judged against others. The inherently high incidence rate of alveolar osteitis after third molar surgery - up to 30%, [Quinley, Royer and Gores (1960)], is the probable explanation for the increase in the overall incidence rate (1.6%) of the latter study. However, 1.6% is still significantly lower than the accepted average of 3%, as noted from the work of various authors worldwide.

1.3.2. Age.
MacGregor (1968) found alveolar osteitis to be age related, from a low incidence of 2.7% at 15 - 19 years to a maximum of 8.6% at 30 - 34 years. At age 50 - 54, the incidence decreases to 2.9%. Field et al. (1985) found a peak incidence in the third decade (21 - 30 years). This finding was similar to the results of their earlier studies in 1971 and 1983. Lechner (1958) and Heasman and Jacobs (1984) reported that alveolar osteitis occurs most frequently in the 20 - 40 years age group.

However, Rood and Murgatroyd (1980) recorded a peak incidence in a slightly older group of patients (40-44 years).
Alveolar osteitis is almost non-existent in children. Only Gustafson and Wallenius (1961) have reported alveolar osteitis occurring after extractions of primary teeth (seven cases out of 10,000 deciduous teeth extracted; an incidence of 0.07%).
The reasons for this apparent link with age remain unclear. Field et al. (1985) hypothesised that the relative thickness of alveolar bone is in
proportion to the patient's age. In the third and fourth decade of life, alveolar bone resorption is generally not advanced and the bone has lost the resilience of youth - thereby making extractions more difficult in this age group. In support of this hypothesis, it has been argued that alveolar osteitis rarely occurs in relation to severe periodontal disease where the bulk of alveolar bone has been lost [Turner (1982)]. The consensus is that, statistically, the 20 - 40 yrs. age group is the one most prone to alveolar osteitis.

1.3.3. Sex Distribution.
MacGregor (1968) was the first to report a significant difference in alveolar osteitis incidence between the sexes. In his study, the male : female ratio was 2:3. The only explanation offered by MacGregor was the possibility that women were more likely to return with a complaint should pain persist after a tooth extraction. If one accepts MacGregor's explanation, then the difference noted would not show a disparity between the sexes as such, but rather a difference in clinic attendance rates between males and females in his study.

However, Schow (1974), Lilly et al. (1974), Butler and Sweet (1977), and Sweet and Butler (1977), all noted an increased incidence of alveolar osteitis following mandibular third molar surgery in patients who were taking the contraceptive pill. Sweet and Butler (1977) recorded a three to fourfold increase in these women and attributed this to interference (unspecified) of the clotting mechanism caused by the high levels of oestrogens and progesterones present in these patients. Gersel-Pedersen (1977) showed elevated fibrinolytic activity postoperatively in women who used oral contraceptives and suggested this increased level of fibrinolytic activity in the circulation may predispose such women to the development of alveolar osteitis.
Field et al. (1985) found a male to female ratio (in alveolar osteitis incidence) of 3:5 and suggested that the reason for this difference may be the wider use of the contraceptive pill with its known ability to enhance systemic fibrinolytic activity [Heasman and Jacobs (1984)]. In contrast to these reports, earlier workers [Krogh (1937), and Lechner (1958)], found no sexual bias in the incidence of alveolar osteitis. As well, Rood and Murgatroyd (1980) and Turner (1982) were unable to show any differences in incidence on the basis of sex.

It appears from contemporary reports that there may well be a predisposition, of women taking oral contraceptives, to the development of alveolar osteitis. This is supported by Birn's (1973) theory that increased fibrinolytic activity at the extraction site is the cause of alveolar osteitis. Also, reports which showed an increased incidence of alveolar osteitis in women coincide (in timing) with the introduction of the contraceptive pill and the subsequent popularity of this method of birth control. If indeed the contraceptive pill is responsible, one may assume that pregnant women are at least as susceptible to developing alveolar osteitis as are those taking oral contraceptive medication. To date, no study of this kind has been reported in the dental literature.

1.3.4. Dental Arch Distribution.

The most frequent site of alveolar osteitis is the mandibular molar region. This is confirmed by the work of Krogh (1937), Adkisson and Harris (1956), Lechner (1958), Hansen (1960), Archer (1966), MacGregor (1968), Rood and Murgatroyd (1980), Turner (1982) and Field et al. (1985). An analysis of figures from these authors reveals a predilection of alveolar osteitis for the mandibular molars at two to three times the frequency of the maxillary molars.
Lechner (1958), MacGregor (1968) and Turner (1982) considered the mandibular first permanent molar as the site of highest incidence, if surgical removal of mandibular third molars is excluded. Most authors rank the mandibular third molar site (following mucoperiosteal flap elevation and bone removal) as the one with the highest incidence of alveolar osteitis.

The consensus of opinion (in decreasing order of frequency) is:

- Mandibular molars
- Mandibular premolars and canines
- Maxillary premolars and canines
- Maxillary molars
- Mandibular and maxillary incisors.

Buchanan (1961) found the following pattern (from highest to lowest frequency):

- Mandibular third molar
- Mandibular second premolar
- Mandibular second molar
- Mandibular first premolar
- Mandibular first molar
- Maxillary second premolar
- Maxillary canine
- Mandibular canine
- Maxillary first premolar
- Maxillary second molar.

It is interesting to note that in Buchanan's (1961) work, the mandibular second premolar displaced the mandibular first molar to fifth place. However, despite minor specific differences the general pattern is similar.

The incidence of alveolar osteitis after the surgical removal of
mandibular third molars is generally acknowledged to be highest due to special factors such as pre-existing pericoronial infection, surgical trauma (soft tissue reflection and bone removal), and the tendency of food debris to collect in the retromolar region. These factors and other aspects of mandibular third molar surgery are reviewed in Chapter 5.

1.3.5. Single vs Multiple Extractions.

Krogh (1937) and MacGregor (1968) reported that when adjacent teeth were removed at any one operation, there was less chance of alveolar osteitis developing. They considered alveolar osteitis to be more frequent after single extractions. Rood and Murgatroyd (1980) also found the incidence of alveolar osteitis in single extractions to be "significantly greater" than in multiple extractions.

In contradiction to the above, Heasman and Jacobs (1984) could find no significant difference between the incidence of alveolar osteitis following single or multiple extractions.

Field et al. (1985) recorded, in 1971, an incidence of alveolar osteitis after multiple extractions of 2.5% (out of 2543 extractions) as against 3.6% (2095 extractions) in single extraction cases. In 1983, Field et al. (1985) reported a reverse finding: they found an alveolar osteitis incidence after multiple extractions of 4.2% (1201 extractions) compared with 3.8% in single extractions (1586 extractions).

The evidence appears to be so conflicting as to make it unlikely that there is any real difference in incidence between single and multiple extraction cases. Certainly, the difficulty in excluding other aetiological factors such as trauma, age, difficulty of extraction, haematological status of the patient, general health and the limited sample sizes in such studies, if taken into account, would tend to diminish the significance of any differences found.
1.3.6. General Anaesthesia vs Local Anaesthesia.

Clinical observations by Killey and Kay (1965) and Howe (1966) indicate that the incidence of alveolar osteitis in mandibular third molar surgery is low when the procedure is done under general anaesthesia.

Meyer (1971) compared a non-infected group to a pericoronitis group of patients who required the surgical removal of a single impacted mandibular third molar under either local anaesthesia or general anaesthesia. Of the 559 patients in the non-infected group, there were 149 local anaesthetic cases, seven of which developed alveolar osteitis (4.7%); of the general anaesthetic cases (410), 21 developed alveolar osteitis (5.1%). No significant differences could be seen. However, in the pericoronitis group, the incidence of alveolar osteitis was significantly less in general anaesthesia cases (10.2% of 256 patients) than in local anaesthesia cases (16.1% of 127 patients). Meyer (1971) felt that the anaesthetic type is insignificant in non-infected patients but significant in those with concomitant pericoronitis. He concluded that general anaesthesia was associated with a decreased incidence of alveolar osteitis in patients with concomitant infection. However, the overall rise in alveolar osteitis incidence of the pericoronitis group compared with the non-infected group suggests that the presence of infection is a significant factor as well.

Buchanan (1961) reviewed 200 cases of extractions under general anaesthesia and found three cases of alveolar osteitis, an incidence of 0.095% (3,161 extractions and 116 surgical removals of third molars). This is in contrast to an alveolar osteitis incidence of 0.79% of 50,386 extractions under local anaesthesia. However, Buchanan attributed this difference to the fact that multiple extractions were done under general anaesthesia as opposed to single extractions with the local
anaesthesia cases. He felt that the type of anaesthetic had very little, if any, influence on alveolar osteitis incidence. With the paucity of evidence and the conflicting nature of it, no conclusion can be drawn as to whether the type of anaesthesia has any influence on the incidence of alveolar osteitis.

1.3.7. Onset and Duration.
Krogh (1937), Adkisson and Harris (1956), Hansen (1960) and Field et al. (1985) considered the onset of alveolar osteitis to be within the first 48 hours after extraction. Field et al. found that 30% of cases returned within three days of extraction and 88% within one week postoperatively.

The shortest recorded onset of alveolar osteitis is that reported by Hansen (1960) where symptoms were apparent immediately the local anaesthesia wore off (i.e. a few hours after extraction). Field et al. (1985) noted (in one patient) a 16 day interval between the date of dental extraction and time of presentation. Whether or not symptoms were present before the 16th day was not made clear, merely that the patient presented with alveolar osteitis on the 16th postoperative day. Possibly the patient had endured the symptoms until the pain finally overcame her, compelling her to seek treatment. The 16th postoperative day was more likely to be a reflection of her tenacity to withstand pain rather than a true indication of the onset of alveolar osteitis.

Most cases of alveolar osteitis (88%) present within the first post-extraction week. The longer periods of onset (greater than one week) may be due to the patient's increased pain threshold, the patient seeking dental treatment only when the pain becomes overwhelming. The duration of alveolar osteitis is dependent on the severity of any
given case, but is normally in the range of 7 to 14 days [Krogh (1937), Adkisson and Harris (1956) and Waite (1957)]. Field et al. (1985) documented the mean duration of treatment as being 7.45 days. This duration was calculated from the day of presentation to the final visit, or until the patient failed to attend, since it is assumed that the condition had resolved and there was no longer any incentive for the patient to return. Field et al. (1985) noted a duration range of one to 25 days. It is interesting to note that Crawford (1896) recorded the longest case of alveolar osteitis where "the socket remained open and dry for 12 months!" This unusual length of time may have been due to over enthusiastic treatment of the socket, as alveolar osteitis is a self-limiting condition.

1.3.8. Pain in Alveolar Osteitis.

Krogh (1948) considered the pain of alveolar osteitis to be so typical that it may be evaluated objectively and used as a basis for diagnosis. He described the clinical assessment of the pain as one in which the intensity is moderate to severe, character dull and throbbing, and often radiating to the ear, temporal or frontal regions of the ipsilateral side. The pain originates from and is focused on the tooth socket or sockets, this usually occurs within 72 hours of the extraction. Birn (1973) similarly described the pain as severe in character, occurring at the extraction site, and often radiating to the ear, orbit, frontal and temporal regions of the ipsilateral side of the face.

Hall, Bildman and Hand (1971), Birn (1973), Belinfante et al. (1973) and Turner (1982) have described the pain of alveolar osteitis as being:

i) Constant.

ii) Moderate to severe.

iii) Refractory to analgesics.
iv) Neuralgiform in nature.

v) Dull and throbbing, like a bad toothache often being worse than the original.

Matthews (1982) described the typical picture of alveolar osteitis as being characterised by intractable, throbbing post-extraction pain, commencing between 24-72 hours after tooth removal the pain being unresponsive to common analgesics.

The nature of pain in alveolar osteitis is so characteristic that there is universal agreement by authors in its description. Indeed, this pain is pathognomonic of alveolar osteitis.

1.3.9. Clinical Presentation of the Alveolar Osteitis Socket.

The socket in alveolar osteitis is characterised by:

i) A blood clot which has either disintegrated or is in the process of disintegration [Alling and Kerr (1957), Birn (1973), and Ritzau and Therkildsen (1978)].

ii) An alveolus which is empty with completely or partially denuded, very sensitive bony surfaces [Hansen (1960), MacGregor (1964), and Birn (1973)].

iii) A socket covered to a varying level by a greyish-yellow layer of detritus and necrotic tissue [Archer (1966), Birn (1973)].

iv) A socket which is extremely painful to touch [Butler and Sweet (1977), Gersel-Pedersen (1979), Matthews (1982), Turner (1982), and Field et al. (1985)].

v) A socket which has a distinct and fetid odour [Krogh (1947), Alling and Kerr (1957), Birn (1973) and Schow (1974)].

vi) Occasional concomitant infection of the socket with the presence of suppuration and sequestra [Alling and Kerr (1957), Birn (1973) and Turner (1982)].
vii) Inflammation of the surrounding gingiva of the socket, even in the absence of overt infection [Birn (1973)].

1.3.10. Histology of the Alveolar Osteitis Socket.
At the histological level (in human and mammalian models), remains of the blood clot are heavily infiltrated with inflammatory cells. Large areas of the lamina dura are necrotic with empty osteocyte lacunae in the bone tissue. The inflammatory process spreads into the surrounding marrow spaces including the periosteum. Necrotic tissue is often found in the marrow spaces close to the alveolus. The appearance is typical of an acute or subacute osteomyelitis, with thrombosed vessels and a dense infiltrate of polymorphonuclear and mononuclear leucocytes in the marrow spaces.
Because of the violent inflammatory reaction, the reparative processes are delayed and extensive osteoclastic activity (which may lead to sequestra formation) can be seen prior to any repair [Birn (1973)].
See Chapter 2 for a more detailed discussion.

1.4. Diagnosis.
Diagnosis of alveolar osteitis, like any other condition, is based upon a combination of patient history, symptoms and clinical findings. Pain is the outstanding feature of alveolar osteitis and is so characteristic that one should be suspicious of alveolar osteitis in any patient who complains of severe pain (within 48 - 72 hours) post-extraction. This pain often radiates to the ear, temporal or frontal regions of the ipsilateral side.
Many authors consider alveolar osteitis as a syndrome, the relevant diagnostic features being:
   i) Severe pain originating from the extraction socket, which may
be referred to the ear, temporal or frontal regions of the ipsilateral side of the face. The pain is often described as being worse than the original toothache: dull, throbbing and unresponsive to analgesics.

ii) A disintegrated or disintegrating blood clot in the extraction socket, which is extremely painful to touch.

iii) The bony walls of the socket may be exposed. Sequestra may be present if there is an overlying infection.

iv) In almost all cases of alveolar osteitis, there is a characteristic fetid odour arising from the affected socket or sockets.

v) In an otherwise healthy individual, the resulting osteomyelitis is localised to the affected extraction socket or sockets, and the patient is devoid of any general signs of infection such as temperature elevation or regional lymphadenopathy.

Pain alone is not sufficient, without other symptoms (as above) to confirm a diagnosis of alveolar osteitis. Conversely, a necrotic socket without an associated history of severe pain cannot be considered as alveolar osteitis.

In some cases of alveolar osteitis, the gingival mucosa may partially cover the socket, giving the superficial appearance of a normally healing socket. Upon gentle probing, it will be seen that the socket is "empty" with necrotic debris at its base and the presence of a fetid odour emanating from the socket. In the majority of cases, the surrounding alveolus and the afflicted socket is tender to touch. Visual examination of the socket is required before a diagnosis of alveolar osteitis can be made with any confidence.

1.4.1. Differential Diagnosis.
The fact that alveolar osteitis is a complication of tooth extraction simplifies the differential diagnosis to:
Acute suppurative osteomyelitis following extraction.
Delayed healing of an extraction socket.
Osteoradionecrosis following extraction on a susceptible patient.

*Acute suppurative osteomyelitis* is characterised by:

i) Inflammation and necrosis of the alveolar marrow with overt suppuration.
ii) Infection is not limited to the socket walls and if unchecked, may involve large areas of the surrounding alveolus [Soames and Southam (1985)].
iii) The maxilla is rarely involved.
iv) Sequestration and sinus formation is common.
v) Clinical symptoms include pain, swelling, lip paraesthesia (mandible), pyrexia and mobility of adjacent teeth.
vi) Radiological examination may show a "moth eaten" appearance around the socket walls with destruction of the lamina dura.

The features that distinguish alveolar osteitis from acute suppurative osteomyelitis are:

i) Facial swelling is not a feature of alveolar osteitis.
ii) Pyrexia is not seen in alveolar osteitis.
iii) Alveolar osteitis rarely, if ever, results in lip paraesthesia.
iv) Infection in alveolar osteitis is limited to the socket walls.
v) Suppuration and sequestration are not common in alveolar osteitis, although there may be a superinfection of acute suppurative osteomyelitis in some cases.
vi) Sinus formation does not occur in alveolar osteitis.

*Delayed healing* of an extraction socket may result from:

i) Systemic diseases (pre-existing), e.g., diabetes mellitus,
endocrine disorders, leukaemia, anaemia, immunosuppressed states, nutritional and vitamin deficiencies.

ii) Age. It is well documented that tissue healing is in an inverse proportion to age; as one ages, the ability of one's tissues to repair decreases significantly.

iii) Chemical suppression of the reparative process, e.g., patients on long-term steroid therapy show a tendency toward impaired healing. This is due to the constant suppression of the inflammatory response so crucial to the repair process. Another example is cancer chemotherapy, where the medication is cytotoxic to all cells, resulting in a compromised host defence. Wound healing is prolonged and the patient is at risk of developing an infection for a period of 6 weeks post-therapy.

iv) Excessive surgical trauma during the extraction process may lead to delayed healing. This is probably the commonest cause of delayed socket healing.

v) Local factors such as ill-fitting dentures, poor oral hygiene, oral candidiasis may predispose to impaired healing.

vii) Chemical trauma, e.g., aspirin burn of the socket's gingiva either before or after extraction may hinder socket healing. Aspirin application to the aching tooth is a common folk cure for toothache, with most patients oblivious to its ability to cauterise the mucosa.

viii) Social habits, e.g., smoking, alcohol and continual spitting (clot dislodgement) after extraction may lead to delayed healing. In delayed healing remnants of blood clot and granulation tissue are evident. Small sequestra may be present and these are normally exfoliated as healing progresses. No suppuration is seen. Pain is not a presenting symptom. In most cases, the patient's main concerns are the slowness of wound closure, food entrapment within the socket and the
presence of small sequestra.
The features that distinguish alveolar osteitis from delayed healing are essentially the ones that define the condition:

i) Severe socket pain.

ii) Disintegrated or disintegrating blood clot.

iii) Denuded socket walls giving an "empty" appearance.

iv) Fetor oris arising from necrotic debris within the socket.

**Osteoradioneurosis** following tooth extraction may result in patients who have undergone therapeutic radiation for oral malignancy. Radiation therapy affects the vascularity of alveolar bone by causing a sloughing of the endothelium and subsequent fibrotic scarring of the intima of the blood vessels (endarteritis obliterans) which leads to thrombosis of blood vessels, especially the inferior dental vasculature. The resultant reduction in blood supply is asymptomatic but the jaws become extremely susceptible to infection from the teeth, periodontium and trauma; be it ill-fitting dentures, exodontia or oral surgical procedures [Soames and Southam (1986)]. This elimination of the fine vasculature within the bone restricts the turnover rate of bone to a point where self repair becomes ineffective [Peterson et al. (1988)].

If adequate antibiotic prophylaxis is not given prior to exodontia in susceptible patients, post-extraction infection may spread rapidly through the irradiated bone since the inflammatory response is restricted due to the diminished blood supply. Infection leads to extensive, painful necrosis of the bone, often with sloughing of the overlying oral and, occasionally, facial soft tissues. Thankfully, present day methods of radiation therapy have greatly reduced the incidence of osteoradioneurosis. This, combined with aggressive
antibiotic therapy, has made osteoradionecrosis less of a problem than in the past. However, despite these improvements, cases of osteoradionecrosis following dental extraction are by no means rare. Osteoradionecrosis as a result of dental extraction may be differentially diagnosed from alveolar osteitis by:

i) A history of radiotherapy to the head and neck.
ii) Socket infection eventually spreads into the cancellous bone. The appearance is reminiscent of acute suppurative osteomyelitis with extensive bone necrosis, sloughing and sequestration.
iii) Infection is rapid, aggressive and difficult to contain.

1.5. Conclusion.

Some aspects of alveolar osteitis are relatively constant:

i) Incidence of between 1.5% to 4.0% after all extractions.
ii) The pain associated with alveolar osteitis is so characteristic that it is pathognomonic.
iii) The appearance of the socket is characteristic.
iv) Dental arch distribution is clearly defined.

However, the following questions remain unclear:

i) Is the condition sex linked? Is oral contraceptive therapy significant?
ii) Is there a greater incidence after single or multiple extractions?
iii) Are individuals, having had one episode of alveolar osteitis, doomed to future episodes after subsequent extractions?

Diagnosis is easy (in most cases) as the history, pain and clinical features of the socket are characteristic.
Chapter 2.

Normal and Disturbed Healing

2.1. Normal Healing of Extraction Sockets.
The normal healing of extraction sockets has been studied for many years in both the human and animal models. The events, as seen by light microscopy, are now well known, although the controlling factors at the biochemical level are yet to be fully elucidated.

Euhler (1923), in his experimental work on extraction socket healing in dogs, found the following sequence:
- Haemorrhage
- Coagulation
- Thrombosis of alveolar wall vessels
- Organisation of the fibrin clot
- Proliferation of the epithelium over the surface of the wound
- Resorption of the damaged tissue
- Formation of new bone.

Clalnin (1936) in his histological study of dog extraction wounds, noted the following reparative changes in the healing of undisturbed wounds:
- Day 1 - Blood clot formation.
- Day 2 - Beginning of clot organisation.
- Day 3 - First appearance of osteoclasts at the crest of the alveolus.
- Day 5 - First appearance of bone formation in the
fundus of the alveolus.
Day 7 - Completion of epithelialisation over the socket.
Day 28 - New bone formation complete.

The chronology of bone repair was documented as:
Day 3 - First appearance of osteoclasts.
Day 5 - Start of bone formation.
Day 11 - New bone reaches alveolar crest.
Day 15 - Socket is filled with new bone except for the central portion.
Day 28 - Bone levels off at the crest.
Day 31 - Osteoclasts still present at the fundus.

The findings of Euhler (1923) and Claflin (1936) correspond very closely. However, there are two significant differences:

i) Claflin (1936) found new bone in a 5 day old socket whereas Euhler (1923) observed this at day 8.

ii) Claflin's specimens indicated complete epithelialisation at day 7-9, while Euhler observed this on the eleventh day. Both series of experiments used dogs as the model.

The differences may be explained by the small sample size in both studies, where individual socket healing was highlighted, although general trends were similar.

Claflin (1936) also studied a 6 week old lower second molar socket in a human specimen obtained at necropsy, the patient having died of pneumonia. He concluded that the reparative changes in man are similar to those seen in the dog, the only difference being that healing in man progresses more slowly. He also pointed out that since most human patients are usually very ill before death, a degree of delayed healing is to be expected in such specimens.
Mangos (1941) studied six human biopsies of upper lateral sockets and two necropsy specimens (an upper central and a lower cuspid socket), his conclusions being:

i) The first stage of extraction wound healing is haemorrhage, coagulation and clot formation.

ii) Fibroblast proliferation at the edges of the clot may be seen after 3 days.

iii) The upper third of the socket is the first to organise, due to the greater vascularity of the submucosa as opposed to the alveolar bone.

iv) Osteoblastic and osteoclastic activity is first noticeable after 10 days.

v) After 15 weeks, new bone has completely filled the socket.

vi) Resorption of the alveolar crest is minimal and is in the form of a "rounding off" process of the sharp alveolus.

vii) Bone regeneration is remarkably even throughout the socket - suggestive of an evenly distributed blood supply.

viii) The epithelium has proliferated completely across the wound in 2 weeks. Epithelial healing is complete at 21 days.

ix) There is evidence of round cell infiltration in the submucosa at 5.5 weeks, but this infiltration of inflammatory cells disappears by the eighth week.

x) No keloid scarring occurs due to the absence of muscle tissue in the alveolar epithelium. Scar formation is more pronounced when muscle tissue is
replaced by fibrous connective tissue as a result of the healing process.

xi) Bone repair in man takes three times longer than in the dog.

xii) Epithelial and submucosal repair in man takes approximately twice as long as in dogs.

Mangos (1941) considered the role of the periodontal membrane and found that it plays no part in the repair process. Histological evidence showed that remnants of the periodontal membrane underwent degeneration and appeared to be absorbed into the granulation tissue of the socket.

The ligamentum circulare (a circular band of fibrous tissue surrounding the neck of each tooth) was found to play a vital role in the initial stages of extraction wound healing by physically retaining the blood clot in the socket. Mangos noted that upon tooth extraction, the ligamentum circulare contracts, producing an infolding of the mucous membrane over the surface of the wound, thus affording protection to the newly formed blood clot.

Radiographs were found to give only an approximate idea of the bony changes involved in socket healing. Mangos judged the earliest radiographically detectable change to be at about 3 weeks, when the socket shadow appears a little denser and the lamina dura becomes less well defined. Bony healing was considered complete at 105 days when the radiopacity of the socket equalled the surrounding alveolar process. It must be said that the subjects of Mango's (1941) study were afflicted with systemic diseases ranging from rheumatic fever, prostate disorder, Parkinson's disease, tuberculosis and Potts disease. This may have prolonged the healing process seen in the biopsy specimens and unwittingly prejudiced his work.
Amler, Johnson and Salman (1960) essentially confirmed the work of previous authors. They carried out a histological and histochemical analysis of undisturbed alveolar socket healing by utilising sequential post-extraction biopsies at intervals of 2 to 3 days for a period of 50 days. All tissue samples were human. The volunteer patients were carefully screened to exclude systemic diseases, e.g., cardiovascular, endocrine and nutritional deficiencies.

Amler, Johnson and Salman's observations of alveolar socket healing were:

i) **Blood clot.**

Upon completion of the extraction, the clot fills the entire alveolar socket. The ratio of red to white blood cells is the same as circulating blood and on haemostasis, fibrin formation is initiated.

ii) **Granulation tissue.**

This is composed of red cells, increased white cells, cells of the reticuloendothelial system, elaboration of capillaries, metachromatic ground substance, glycoprotein and the presence of alkaline phosphatase. Granulation tissue first appears peripherally 2 to 3 days after extraction and completely replaces the blood clot by the seventh day.

iii) **Young connective tissue.**

Connective tissue is characterised by the appearance of spindle-shaped cells, collagen fibres, disappearance of granulation tissue and increased vascularity. Connective tissue cells appear peripherally on the fourth day, rapidly increase and by the twentieth day have replaced the last remnants of granulation tissue. Histochemically, the presence of young connective tissue is marked by a corresponding increase in metachromatic ground substance, alkaline phosphatase and glycoprotein.
iv) Bone Formation.

Uncalcified bone spicules are evident by the seventh day after tooth extraction. They appear as either isolated patches or attached to old spicules of bone, continuous with the peripheral alveolar portion of the socket. Bone spicules first appear at the base and lateral aspects of the socket, interspersed with the connective tissue.

v) Epithelialisation.

Peripheral proliferation of the epithelium was seen on the fourth day post extraction. The final stage of fusion was not reached until at least 24 days postoperatively. Mangos (1941) reported complete epithelialisation across the socket at day 21. A significant finding was the presence of ragged edges of damaged or traumatised epithelium slough, whereas there was minimal sloughing in areas where a clean incision had been made.

Amler, Johnson and Salman (1960) concluded that complete epithelialisation of the socket is dependent upon the following factors:

a) Non-pathological factors.
Socket diameter.
Age.
Gingival laceration.
Alveolar crest height.
Presence of foreign bodies in the socket (e.g. root fragments, sequestra, amalgam).

b) Pathological factors.
Preoperative periodontal condition.
Local postoperative infection.
Nutritional and systemic factors (e.g. organic diseases).
Amler, Johnson and Salman (1960), as a result of their investigation, suggested the following sequence in the healing of an alveolar socket:

i) Clot formation occurs immediately after tooth extraction.

ii) Granulation tissue appears 2-3 days after extraction and replaces the blood clot by the seventh day.

iii) Young connective tissue encroaches on the granulation tissue (day 4) progressively and replaces it by the twentieth day.

iv) Bone formation is signalled by the appearance of osteoid tissue at day 7 and by the thirty-eighth day, at least two thirds of the socket fundus is filled with new bone. Bone development is considered complete by 100 days, when the density of the socket is identical to the surrounding alveolus (as indicated by radiographs).

Epithelialisation is evident peripherally at day 4, while fusion of the epithelial ingrowth is variable (depending on the factors mentioned above); the earliest was observed at day 24.

It is interesting to note that Amler, Johnson and Salman (1960) reported essentially the same time sequence as Mangos (1941) despite the latter's study on patients with concomitant systemic disease. The differences were minor, e.g.,

i) Both authors agreed that osteoid formation was evident by day 7.

ii) Epithelial fusion - Mangos 21 days, Amler et al. 24 days.

iii) Completion of bone formation - Mangos 105 days, Amler et al. 100 days.

It would appear that systemic diseases such as those included in Mangos's study (rheumatic fever, Parkinson's disease, tuberculosis and Pott's disease) have no significant effect on alveolar socket healing.
Undisturbed human alveolar socket healing may be schematically represented as (Fig. 1.):

**Undisturbed human alveolar socket healing**

<table>
<thead>
<tr>
<th>Day of Extraction:</th>
<th>2-3 Days Post - extraction:</th>
<th>4 Days Post - extraction:</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>7 Days Post - extraction:</th>
<th>20 Days Post - extraction:</th>
<th>40 Days Post - extraction:</th>
</tr>
</thead>
</table>

**Fig 1.** Adapted from Amler (1969)

2.2 Changes in Alveolar Healing as seen in Alveolar Osteitis.

Few studies of disturbed extraction socket healing (alveolar osteitis included) have been published in the English literature. However, Amler (1973) studied 11 biopsy specimens from patients diagnosed clinically as having developed alveolar osteitis, taken at intervals ranging from 3 days to 9 days postoperatively.

The histological findings were:
- Clot degradation; the dissolution of erythrocytes and fibrinolysis were the most noticeable features.
- Haemosiderin deposits were present only in the early stages.
- Ghost cells having erythrocyte morphology, inflammatory cells, miscellaneous debris and amorphous material provided the typical picture.

- There was no sign of granulation tissue or young connective tissue.

Amler (1973) concluded that alveolar osteitis results in the disruption of healing between stage 1 (clot formation) and stage 2 (granulation tissue formation). See Fig. 2.
**Stage 1.**
Day 1.
i) Blood clot formation.

**Stage 2.**
Day 2-3.
i) Blood clot
ii) Granulation tissue.

**Stage 3.**
Day 4.
i) Blood clot
ii) Granulation tissue
iii) Connective tissue
iv) Epithelium.

---

**Normal Healing of Alveolar Sockets**

**Stage 4.**
Day 7.
i) Granulation tissue
ii) Connective tissue
iii) Osteoid
iv) Epithelium.

**Stage 5.**
Day 40.
i) Connective tissue
ii) Bone
iii) Epithelium.

---

**Healing in Alveolar Osteitis**

**Stage 2.**
Day 2-3.
i) Blood clot degradation
ii) No Granulation tissue.

**Stage 3.**
Day 4-7.
i) Complete disintegration of the blood clot
ii) Socket debris.

**Stage 4.**
Day 7.
i) Socket debris
ii) "Empty" socket.

---

**Fig.2.** Adapted from Amler (1973).
Birn (1973) made the following observations regarding alveolar osteitis:

i) Blood clot remains were heavily infiltrated with inflammatory cells.

ii) Large areas of lamina dura were necrotic with empty osteocyte lacunae in the bony tissue.

iii) The inflammatory process was evident in the surrounding marrow spaces including the periosteum.

iv) Necrotic tissue was often found in the marrow spaces close to the socket walls.

v) The histological picture is typical of an acute or subacute osteomyelitis with thrombosed vessels and a dense infiltrate of polymorphonuclear and mononuclear leucocytes in the marrow spaces.

Because of the violent inflammatory reaction, the reparative processes were delayed and extensive osteoclastic activity (which may lead to sequestra formation) can be seen prior to any repair.

At varying times after the onset of alveolar osteitis, with or without symptomatic treatment, granulation tissue starts to grow into the alveolus through the perforations of the lamina dura.

The alveolar socket is gradually filled, from the bottom up, with granulation tissue and subsequently, epithelialisation across the socket begins.

2.3. Conclusion.

Since Amler (1973) published his findings based on human samples, a lot of research has been done on wound healing in the rat model. Whilst our knowledge of the minutiae of oral wound healing has increased exponentially [McMillan (1973, 1975, 1978, 1980), Smith (1974),
Leibovich and Ross (1975), Knighton et al. (1982), Repesh, Fitzgerald and Furcht (1982), Clark et al. (1982), Niall, Ryan and O'Brien (1982), Kurita et al. (1985), Lawman et al. (1985) and Bodner et al. (1991)], the sequence of events at the clinical level remains as described by Amler.
Chapter 3.

Aetiology of Alveolar Osteitis.

From the time Crawford (1896) first coined the term 'dry socket', there have been numerous attempts made to explain the pathogenesis of the condition. The fact that there is no universal agreement as to the aetiology of alveolar osteitis is evidenced by the plethora of theories put forward by various authors.

An attempt will be made here to clarify the situation by presenting the theories currently in vogue, and examining the scientific data upon which they are based.

It has been traditional to divide aetiological factors into:

  i) General
  ii) Local.

3.1 General Factors.

3.1.1. Decreased Resistance to Infection.

A depressed immunological state may be caused by systemic disease and/or nutritional deficiencies e.g., protein, vitamin, calcium, phosphorus etc.

Zimmer (1929), Pell (1934), Eman (1944) and Harang (1948) considered systemic diseases, such as cardiac disease, uncontrolled diabetes, liver dysfunction, syphilis, anaemia, haemorrhagic disorders and endocrine disturbances to be important to the development of alveolar
osteitis.
Nutritional, vitamins (A, B, C, D), calcium and phosphorus deficiencies were thought to be influential in the development of alveolar osteitis [Armstrong (1940), Whitfield (1941), Shea (1943), and Sinclair (1943)]. Whitfield (1941) observed a reduction in the incidence of alveolar osteitis with the use of thiamine chloride intramuscular injections at the time of extraction. However, his clinical trials were poorly controlled, e.g., mandibular third molar sockets were packed with dressings (unspecified) as well as the patient having been given an intramuscular injection of thiamine chloride. Also, he used records of 100 cases of alveolar osteitis as his controls, rather than a concurrent series of extractions. Hence, the validity of his study is in some doubt. Ringsdorf and Chereskin (1975) compared the response to the glucose tolerance test of "normal" (no history of alveolar osteitis) patients and those with a history of alveolar osteitis. They found elevated serum glucose levels in the alveolar osteitis group, thereby implying a possible link between diabetes and alveolar osteitis. MacGregor (1985) felt that any deviation from normal in the health of the patient is an invitation to untoward postoperative sequelae. Although this general rule of surgery undoubtedly applies in dentoalveolar surgery, specific causes such as hypovitaminosis C or B, hypoproteinaemia, diabetes, corticosteroid and anticoagulant therapies have been identified by inference from studies of healing in general rather than experimental evidence derived from studies of extractions in particular.
On the other hand, Gardner (1929) was unable to find any correlation between systemic diseases and the frequency of alveolar osteitis. Erickson, Waite and Wilkinson (1960) also found no correlation between the occurrence of alveolar osteitis and the patient's general health.
However, their sample size of 98 patients was perhaps too small to reveal any connection, if indeed, there was any to be found. There appears to be more circumstantial evidence in favour of systemic disease/nutritional deficiencies being a factor in the development of alveolar osteitis than against it. It must be borne in mind that most of this evidence is speculative, having been inferred from studies of healing in general. The danger of theoretical inferences of course, is that the reality may fall far short of expectations. Systemic disease and nutritional deficiencies will not necessarily lead to the development of alveolar osteitis. It is felt that these factors predispose the patient rather than cause the condition. The consensus is that in medically compromised patients, one or more local factors must be present in order for the development of alveolar osteitis to take place.

3.1.2. Sex.

MacGregor (1968) was the first author to indicate that women suffered an increased incidence of alveolar osteitis compared with men; he noted a male:female ratio of 2:3. However, the possibility was raised, by MacGregor, that females complain of pain more readily and are therefore more inclined to seek treatment than men! Hence, his results may not indicate a true sex difference. Sweet and Butler (1977) reported a three to fourfold increase in the incidence of alveolar osteitis in women undergoing mandibular third molar surgery, who were taking the contraceptive pill at the time. They hypothesised that the clotting mechanism was interfered with by the increased levels of oestrogens and progesterones. Also, Gersel-Pedersen (1977) observed an elevation in fibrinolytic activity postoperatively in women using the contraceptive pill who underwent
mandibular third molar surgery, and suggested that the increased level of fibrinolytic activity predisposed these women to alveolar osteitis. Field et al. (1985) found a male:female ratio of 3:5, and felt that this ratio reflected the use of the contraceptive pill (i.e. increased fibrinolytic activity in the circulation) rather than an innate difference of the sexes. These studies confirmed the earlier work of Lilly et al. (1974) and Schow (1974) who initially showed that the contraceptive pill predisposed women to the development of alveolar osteitis. It appears that being female per se, is not the predisposing factor, but rather the hormonal imbalance imposed by the contraceptive pill seems to be the causal agent. The increased fibrinolytic activity in the circulation that results from oral contraceptive use is thought to be the mechanism by which these women are put at risk of alveolar osteitis development. This deduction is intimately linked to Birn's (1973) hypothesis of "Fibrinolytic Alveolitis" [See 3.2.12(b)].

3.2 Local Factors.

3.2.1 Inadequate Alveolar Blood Supply.
It is thought that the alveolar blood supply may be compromised in one of two ways:

i) Genetically, inasmuch as the normal anatomy is in itself the limiting factor.

ii) Pathological changes, e.g., periapical infection, cyst formation or sclerosis which may lead to a structural change in the bone, thereby restricting the normal alveolar circulation.
Sinclair (1943), Lechner (1958), Gustafson and Wallenius (1961), Rud, Baggensen and Moller (1963) and Rothenberg and Landman (1965) were of the opinion that the reason for the frequent occurrence of alveolar
osteitis in the mandibular molar region is the insufficient blood supply caused by the dense layer of bone which surrounds the roots of these teeth and the relative paucity of blood vessels in this region.

Lechner (1958) found that out of 51 cases of alveolar osteitis (in a survey of 2,500 extractions), 20 cases occurred in patients with sclerotic bone or thickened lamina dura. From this he suggested that sclerosing osteitis is an aetiological factor in alveolar osteitis. Presumably the underlying rationale is that sclerotic bone is less vascularised than non-sclerosed bone.

None of the above authors carried out any scientific studies to verify this hypothesis, rather they extrapolated from clinical experience and anatomical observations regarding the distribution of solid (cortical) bone in human jaws. There is no doubt that the mandible is very much denser than the maxilla, whether it is less well supplied by blood vessels is questionable.

Birn (1966), in an elaborate experiment, showed that the blood supply actually increases evenly in a posterior direction and is greatest in the mandibular second molar region. He studied 84 dried specimens of Indian jaws, calculating the area of the alveolar wall and the area of the lamina dura perforations (assumed to be representative of the blood supply to the teeth). The relationship between the lamina dura perforations and the dental blood supply was examined in 5 rats. The vessels of these rats were injected with India ink before they were sacrificed. Serial sections of the alveoli were made and a direct relationship between the lamina dura perforations, the number and size of alveolar blood vessels was noted. This relationship was then extrapolated to the observations previously noted on the alveoli of the Indian jaws.

Birn's (1966) results failed to support the clinical impressions of the
earlier mentioned authors. Where the bone was thickest, (the mandibular molar region) the blood supply was greatest; conversely, where the alveolar wall was thinnest, (the incisive region) the blood supply was poorest.

The hypothesis that the mandibular molar region is poorly vascularised and therefore prone to alveolar osteitis development cannot be supported in the light of Birn's (1966) work, which shows the same region to be richly endowed with blood vessels.

3.2.2. Vital/Non-vital Teeth.

Krogh (1937) and Archer (1939), noted that alveolar osteitis develops more frequently after extraction of vital teeth than teeth with a necrotic or inflamed pulp. Krogh (1937) found that the sockets of retained roots with vital pulps developed alveolar osteitis in 18.6% of all extractions, whereas erupted teeth which are often extracted as a result of periapical pathosis show an incidence of only 1.2%.

Similarly, Archer (1939) found that 62% of the 226 cases of alveolar osteitis in a sample of 23,886 extractions occurred after extraction of vital teeth and 38% after extraction of non-vital teeth. However, it is unclear whether he corrected for the ratio of vital to non-vital teeth in his sample. The result may conceivably be skewed because of this.

MacGregor (1968) reported an incidence of 5.2% (159/3,080) in extractions of teeth with pulpitis, compared with 4.5% (41/921) in extractions of teeth without pulpitis. The result was equivocal and MacGregor dismissed the finding as statistically insignificant.

In summary, there is insufficient evidence to show whether tooth vitality (or lack of it) has any influence on the development of alveolar osteitis.
3.2.3. Pre-existing Infection (Marginal Periodontitis, Pericoronitis).
There is some evidence to show that extraction of teeth (viz., wisdom teeth) with concomitant pericoronitis gives rise to a substantial increase in the incidence of alveolar osteitis. Kay (1966) found that alveolar osteitis developed in 24% of all cases with pericoronitis in his study. It did not matter whether pericoronitis was acute or chronic. Kay also noted that the incidence of alveolar osteitis was much higher (88%) when the pericoronitis had spread into the bone: so-called deep pocketing, as judged by periodontal probing.
Rud (1970) confirmed Kay's (1966) result; he found an incidence of 31% in acute and 20% in chronic pericoronitis cases. The earlier work of Adkisson and Harris (1956), Lechner (1958), and later MacGregor (1968) also supported the findings of Kay (1966). All of the authors felt that the presence of infection around the tooth contributed to the increased incidence of alveolar osteitis. They reasoned that bacteria in the pericoronial tissues inevitably find their way into the alveolus and blood clot postoperatively.
The apparent correlation between pericoronitis and alveolar osteitis is presumptive upon the hypothesis that oral bacteria have a causal role in the pathogenesis of alveolar osteitis.

3.2.4. Bacterial Contamination of the Dental Alveolus.
One of the most widely held beliefs of the aetiology of alveolar osteitis is that it is caused by the infection of the extraction site as a result of bacterial contamination by the oral commensals. This is supported by the association of alveolar osteitis with pre-existing gingival infections such as acute necrotising ulcerative gingivitis (ANUG) and pericoronitis [Adkisson and Harris (1956), Kay (1966), Rud (1970) and Brown, Merrill and Allen. (1970)].
The dental literature is replete with reports showing a link between antibiotic (local and systemic) therapy and a diminished incidence of alveolar osteitis. Quinley, Royer and Gores. (1960), Rud, Baggensen and Moller. (1963), Swanson (1966), Hall, Bildman and Hand. (1971), Tjernberg (1979) and Krekmanov and Hallander (1980) all reported success with various antiseptic mouthwash regimens, systemic and local antibiotic preparations.

Claflin (1936) demonstrated that "dry socket" can be experimentally produced in the dog by the immediate socket placement of a tampon saturated with a mixed culture of streptococci and staphylococci strains. The culture was derived from infected human dental pulp. However, this failed to show whether the bacteria in the tampon or the physical mass occluding the socket was the principal factor. In any case, the gross contamination required in Claflin's experiments were hardly representative of the conditions under which alveolar osteitis normally occurs.

A few years later, Archer (1939) considered streptococci to be important in the development of alveolar osteitis, as he found streptococci in 80% of the 226 cases of alveolar osteitis in his study. Unfortunately, he did not identify the specific strains of streptococci implicated.

More recently, Brown Merrill and Allen (1970) found that patients with high preoperative counts of bacteria (especially streptococci) in the third molar region and saliva were prone to the development of alveolar osteitis postoperatively.

Nitzan (1983) provides some rather convincing arguments why he considers Treponema denticola (an anaerobic bacterium with strong plasmin-like fibrinolytic activity and a normal oral commensal) to be the aetiologic agent in the development of alveolar osteitis.
He considered the following conditions needed to be fulfilled in order to show whether a bacterium in low concentrations would be capable of producing alveolar osteitis:

i) The bacterium must be isolated from a socket afflicted with alveolar osteitis.

ii) It must belong to the blood lysing bacterial group - consistent with Birn's (1973) fibrinolysis theory.

iii) It must be non-pathogenic, as defined by the criteria for inflammation, viz., it must not produce swelling, redness or suppuration. Most cases of alveolar osteitis are devoid of these inflammatory signs.

iv) The conditions in the extraction wound must be conducive to the incubation and growth of the bacterium.

v) The bacterium must be anaerobic - an anaerobe would explain the malodour and bad taste so characteristic of alveolar osteitis.

vi) The bacterium must be an oral commensal; it must remain quiescent until the appropriate conditions arise.

vii) The bacterium should be absent from the oral cavity of children. Alveolar osteitis is rarely documented in children.

Nitzan found that Treponema denticola fulfilled all of these requirements.

Although the arguments were convincing, on balance, the evidence is largely circumstantial; no direct "cause/effect" relationship was shown experimentally with T. denticola. Because of the complexity of
the pathogenesis of alveolar osteitis, it may well prove to be impossible to identify any one specific cause, be it bacterial or otherwise.


Grandstaff (1935) used bacteriological techniques to investigate 40 extraction wounds and found no difference in bacterial types in normally healing alveoli and alveolar osteitis. MacGregor and Hart (1969), MacGregor (1970) and MacGregor and Hart (1970) carried out extensive and well supported investigations on bacterial numbers and types in normally healing extraction wounds and alveolar osteitis sockets. No significant differences were found and they concluded that bacterial infection is not an aetiological factor in alveolar osteitis. They considered bacterial colonisation of extraction wounds to be opportunistic and saprophytic.

Despite the somewhat contradictory findings of the above authors, the weight of the evidence would suggest that bacterial infection is of some importance in the pathogenesis of alveolar osteitis. The exact nature of its role is yet to be elucidated.

3.2.5. Ischaemia of the Alveolus caused by using Local Anaesthetics containing Vasoconstrictors.

Seldin (1933), Harang (1948), Harnisch (1949) and Huebsch (1958) have implicated the use of infiltration techniques with local anaesthetics containing vasoconstrictors as being an aetiological factor in alveolar osteitis. The authors reasoned that the blood supply to the extraction socket is diminished, thus predisposing it to alveolar osteitis. Central
to this argument is the belief that alveolar osteitis is a result of the physical lack of a blood clot from the outset, i.e., the socket is devoid of any blood clot from the moment of extraction.

Lechner (1958) in support of the above, reported a twofold increase in the incidence of alveolar osteitis when infiltration anaesthesia was used compared with general anaesthesia cases. He concluded that infiltration anaesthesia with vasoconstrictors gives rise to a temporary "ischaemia", i.e., a decreased alveolar blood supply.

A temporary ischaemia of the soft tissues and alveolus can often be visualised clinically as "blanching" of the area whenever infiltration anaesthesia with vasoconstrictors are employed. Vasoconstrictor levels as low as 1:100,000 adrenaline are sufficient to cause this phenomenon.

At the time Seldin (1933) advanced this idea, adrenaline levels being used were 1:20,000 [Cutler (1983)]. The clinical impression of the resultant tissue "blanching" must have been very marked and it was logical to think that such severe deprivation was incapable of sustaining a healthy blood clot. However, Birn (1973) notes that this ischaemia lasts for an hour or so and is followed by a reactive hyperaemia. Nitzan (1983) also points out the fact that vasoconstrictors, especially at the levels being used in modern dentistry (e.g., 1:100,000 to 1:200,000 adrenaline) do not entirely occlude the blood vessels; thus permitting a diminished blood supply to reach the alveolar socket and hence allow clot formation.

Krogh (1937) found that the incidence of alveolar osteitis is far higher in the mandibular molar region where block anaesthesia is used as opposed to infiltration. He concluded that the ischaemia of infiltration techniques do not account for this: local anaesthetics containing vasoconstrictor agents and infiltration procedures are insignificant in
the pathogenesis of alveolar osteitis.

Kay (1966) found a minor increase in alveolar osteitis incidence when using local anaesthesia compared with general anaesthesia. He ascribed this difference to other factors such as operative technique and difficulty of extraction rather than the type of anaesthetic. Meyer's (1971) investigation supported Kay's (1966) conclusion.

Hahn (1962) and Schilli, Scharp and Lange (1967) compared the use of local anaesthetics without vasoconstrictor added, with those containing vasoconstrictor and found an equal number of alveolar osteitis in each group.

MacGregor (1985) compared the effect of different amounts of the same local anaesthetic but failed to find any differences in outcome with regard to alveolar osteitis.

In conclusion, there is a lack of evidence to substantiate the contention that ischaemia resulting from an infiltration injection leads to the development of alveolar osteitis. The hypothesis that alveolar osteitis arises because of a lack of bleeding to provide a blood clot cannot be condoned, especially since in most cases of alveolar osteitis remnants of necrotic blood clot can be seen within the socket.

3.2.6. Root, Bone Fragments or Foreign Bodies left in the Alveolus after Extraction.

Ward (1932) and Eman (1944) both considered this complication of extraction as one cause of alveolar osteitis. They suggested that root or bone fragments, small pieces of amalgam, calculus and other small foreign bodies may become the foci of infection, leading in some cases to the sequestration of bone and concomitant alveolar osteitis.

Brown, Merrill and Allen (1970) and MacGregor and Hart (1970) found that clinically, the integrity of the blood clot may be affected by the
presence of foreign bodies which may lead to secondary infection by commensal micro-organisms thus predisposing to alveolar osteitis. Birn (1973) contends that if root, bone fragments or foreign bodies (e.g., pieces of amalgam) are left in the socket, they may give rise to alveolar osteitis by way of infection of the alveolus. Indirectly, such fragments may indicate a particularly traumatic extraction, the trauma being the principle aetiologic factor. However, Simpson (1960, 1961, 1969) as a result of his work on Rhesus monkeys showed that small bone and tooth fragments inevitably occur after normal extraction or surgical removal of teeth and do not give rise to any healing complications. Postoperative complications appear as a function of the size of the fragment: small pieces are dealt with by the body whilst larger or contaminated fragments cause problems. Indeed, it has been noted clinically, that root fragments without periapical pathosis seldom give rise to symptoms postoperatively. By and large, clinical experience shows that small pieces of amalgam are well tolerated by the oral tissues, manifesting only as amalgam tattoos in the majority of cases. In conclusion, it seems highly unlikely that root fragments, bone remnants or foreign bodies per se are capable of causing alveolar osteitis. Inasmuch as they are vehicles of infection (either as a result of a primary periapical infection or secondarily by bacterial contamination from the oral microflora) are they considered to predispose to alveolar osteitis. This would support the hypothesis that bacterial contamination is a possible cause of alveolar osteitis.

3.2.7. Trauma to the Alveolar Bone during Extraction.

Exodontia, by definition, is a traumatic process whether executed with forceps or elevators or by a surgical approach. The trauma essentially
differs only in degree.

Surveys carried out by Krogh (1937) and MacGregor (1966) related the difficulty of extractions (by subjective operator assessment) to postoperative healing. These showed clearly that the more trauma induced during an extraction, the greater was the probability of the patient developing alveolar osteitis.

Experimentally induced alveolar osteitis was produced by Harrison (1943) in the sockets of sheep incisors by simply burnishing the socket wall after the extraction. Presumably this was to simulate the trauma of a difficult extraction. Alling and Kerr (1957) successfully produced alveolar osteitis in the Rhesus monkey using much the same technique. Heasman and Jacobs (1984) have also documented the relationship between the occurrence of alveolar osteitis and the time taken to extract the tooth. Their findings were as shown in Table 2.

Table 2.

Alveolar osteitis incidence Vs Extraction difficulty.

<table>
<thead>
<tr>
<th>Difficulty</th>
<th>Time</th>
<th>Extractions</th>
<th>A. Osteitis</th>
<th>Incidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Easy</td>
<td>Under 1 min.</td>
<td>1082</td>
<td>27</td>
<td>2.5%</td>
</tr>
<tr>
<td>Moderate</td>
<td>1 - 5 min.</td>
<td>1007</td>
<td>24</td>
<td>2.4%</td>
</tr>
<tr>
<td>Difficult</td>
<td>Over 5 min.</td>
<td>274</td>
<td>18</td>
<td>6.6%</td>
</tr>
<tr>
<td>Total (n=)</td>
<td></td>
<td>2363</td>
<td>69</td>
<td>3.8% (mean)</td>
</tr>
</tbody>
</table>

Heasman and Jacobs concluded that trauma was indeed significant in alveolar osteitis incidence.

Brekke, Bresner and Reitman (1986) considered operative trauma delivered during an extraction to be of two parts:

i) The intensity of the trauma was judged by the
authors to be proportional to such factors as bone density, root curvature, degree of impaction and tooth vitality (on the assumption that a non-vital tooth is inherently more difficult to extract than a vital one).

ii) The length of time over which the trauma is sustained.

In their study of mandibular third molar wounds (of various impactions), 38 patients out of 227 suffered alveolar osteitis. Because trauma itself is virtually impossible to measure with any degree of accuracy, the duration of the operation was taken as being indicative of the surgical trauma induced. Thus out of a total of 38 cases of alveolar osteitis, the trauma/incidence ratio was as shown in Table 3.

**Table 3.**

<table>
<thead>
<tr>
<th>Duration of Operation</th>
<th>Alveolar Osteitis Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Under 2 minutes</td>
<td>3</td>
</tr>
<tr>
<td>2 to 4 minutes</td>
<td>7</td>
</tr>
<tr>
<td>Over 4 minutes</td>
<td>28</td>
</tr>
<tr>
<td>Total (n=)</td>
<td>38</td>
</tr>
</tbody>
</table>

The time was taken from the start of incision to final wound closure. Brekke, Bresner and Reitman (1986) concluded that any mandibular third molar extraction wound which has received more than 4 minutes of "average" surgical trauma is at considerable risk of developing alveolar osteitis. As most impacted mandibular third molars need 4 or more minutes of surgery, trauma may be one reason why the mandibular third molar socket is the site most afflicted with alveolar osteitis.
As a corollary, surgical removal of teeth as distinct from simple extractions, have been shown to be related to a marked increase in alveolar osteitis incidence. Erickson, Waite and Wilkinson (1960) found an incidence of 1.5% after simple extractions and a massive 60.4% after surgical extractions. Hansen (1960) also noted an increase in incidence of alveolar osteitis after surgical extractions. In a sample of 1079 extractions, 2% of alveolar osteitis occurred after simple extractions whereas 7.3% occurred after surgical extractions.

Despite the weight of evidence supporting trauma as an aetiological factor, some authors have found to the contrary. In an analysis of 23,886 extractions, Archer (1939) found 226 episodes of alveolar osteitis (0.9%), of these, trauma was implicated in only 56% of cases. From this, he concluded that trauma does not play as important a role as previously thought. Whilst the assessment of the level of trauma induced during any given procedure is, by definition, subjective, it is difficult to see how any extraction can be performed without at least some degree of trauma.

In a study of only 47 third molar extractions, Swanson (1968) found an incidence (after minimal trauma) of 62.5% and in cases of substantial trauma - 28.6%. Swanson concluded that trauma is inconsequential to alveolar osteitis development. A major criticism of Swanson's study is the small sample size, which is probably too small from which to draw any meaningful conclusions.

Trauma associated with tooth removal is defined (by most authors) as:

i) Raising of a mucoperiosteal flap.

ii) Burnishing of the socket walls by excessive buccolingual and rotatory movements during the extraction procedure.

iii) Removal of bone with bur or chisel to facilitate
the tooth's delivery.
iv) Tooth sectioning.
v) Soft tissue laceration and trauma (crush injury).
vi) Length of time taken for the procedure viz., the longer the time taken, the more trauma the patient suffers.

In summary, the evidence is that trauma is an important factor in the incidence of alveolar osteitis. Most cases of alveolar osteitis can be linked to a history of difficult tooth extraction. Cases where alveolar osteitis follows a simple "atraumatic" extraction appear to be the exception rather than the rule.

3.2.8. Postoperative Bleeding.
Harang (1948) and Commissionat (1964) considered postoperative bleeding as a cause of alveolar osteitis. They hypothesised that with persistent bleeding a healthy blood clot is not sustained and subsequent breakdown occurs. However, both authors relied on clinical impressions rather than scientific investigation.
On the other hand, McIntyre et al. (1959) in a series of extractions on 164 haemophiliacs did not mention a single case of alveolar osteitis. It is not clear how postoperative bleeding from an extraction socket might precipitate alveolar osteitis, especially since the clinical picture is one of a denuded socket, i.e., the socket is devoid of a blood clot or "empty". Presumably, the argument is that because of persistent postoperative bleeding, what little blood clot there is has no time in which to organise and therefore is apt to be washed out of the socket. There is a paucity of data to support the above contention and it must be concluded that postoperative bleeding is very unlikely to cause or predispose to alveolar osteitis.
3.2.9. Excessive Irrigation or Curettage of the Socket after Extraction.

Ward (1932), Whitfield (1941) and Elwell (1944) felt that over vigorous irrigation and/or curettage of the socket after extraction may lead to destabilisation of the blood clot and subsequent alveolar osteitis. Indirectly, they are supported by Heasman and Jacobs (1984) who noted a greater predisposition to alveolar osteitis in extraction wounds that displayed poor immediate postoperative filling with blood clot. In a sample of 2,363 extraction sockets, 1,982 were judged to be 2/3 full of blood clot; of these 54 developed alveolar osteitis, an incidence of 2.7%. In 304 cases where the blood level was between 1/3 to 2/3 of the socket height, 8 cases of alveolar osteitis were noted (2.6% incidence). Seventy seven extraction sockets showed a blood level of less than 1/3 the socket height, of these 7 cases developed alveolar osteitis (9.1%).

The rationale that repeated irrigations and/or curettage of the socket might lead to defective clot formation appears feasible, on the basis of a relative diminution of available clotting factors (excessive irrigation) and increased socket trauma (curettage). Certainly, not all instances where irrigation might be labelled excessive or curettage unduly vigorous lead to alveolar osteitis. However, in some surveys of socket healing the predisposition appears to be there. Further experimental work is needed to either confirm or refute this observed tendency.

3.2.10. Heavy Sucking or Spitting Postoperatively.

Heavy sucking or spitting postoperatively have been reported by Gardner (1929) and Whitfield (1941) to cause the detachment of the blood clot from the alveolus and the consequent development of
alveolar osteitis. Gardner reasoned that patients who sucked the extraction clots and spit, often develop a negative pressure within the oral cavity which leads to the dislodgment of the blood clot from the socket. Furthermore, he suggested this as an explanation of why alveolar osteitis occurs more frequently in the molar and premolar regions of the maxilla and mandible, as this is where the greatest amount of negative pressure is produced.

Elwell (1944) included manipulation of the blood clot with fingers, tongue and toothpicks as well as sucking and spitting as possible causes of alveolar osteitis.

Birn (1973), however, dismissed the above hypothesis for want of scientific evidence.

Whilst it is true that no experimentation has been carried out to investigate whether excessive sucking or spitting postoperatively causes alveolar osteitis, it is nonetheless conceivable that anything which might lead to a destabilisation of the blood clot at the very least, predisposes the patient to alveolar osteitis. In any case, it is sound clinical practice to advise patients to refrain from sucking the blood clot or spitting postoperatively.

3.2.11. Cigarette Smoking.

Sweet and Butler (1978) observed a fourfold increase in the incidence of alveolar osteitis in smokers compared with non-smokers. Nine out of 142 smokers (6.4%) compared with 5 of 362 non-smokers (1.4%) suffered alveolar osteitis postoperatively. The authors hypothesised that contaminants in the smoke, heat from the burning tobacco and/or systemic effects (unspecified) associated with smoking have an effect on the stability of the alveolar blood clot.

Later, the same authors [Sweet and Butler (1979)] carried out a well
controlled study of the effects of smoking on the incidence of alveolar osteitis) in 200 patients who underwent bi-mandibular third molar removal. They found:

i) Alveolar osteitis occurred in 11 out of 92 surgical sites (12%) of patients identified as smokers, compared with only 8 out of 308 (2.6%) surgical sites of non-smokers.

ii) Patients identified as heavy smokers (a half packet or more per day) had a significantly greater incidence of alveolar osteitis.

iii) Alveolar osteitis occurred in 11 of 76 (14.5%) surgical sites of patients who were smokers and who continued to smoke postoperatively. On the other hand, patients who did not smoke postoperatively (including some smokers who refrained from smoking postoperatively) recorded an incidence of 8 out of 324 (2.5%).

iv) Alveolar osteitis occurred in 10 of 38 patients (26.3%) who smoked postoperatively. However, there was no occurrence of alveolar osteitis in seven patients identified as smokers preoperatively who refrained from smoking postoperatively.

v) Of the 10 patients in the smokers group who developed alveolar osteitis, 40% smoked on the day of surgery and all 10 smoked the day after surgery. This corresponds to the period of time in which the blood clot is most susceptible to lysis [Sweet and Butler (1978)]. As a result of this study, the authors recommend that patients should stop smoking for at least 5 days postoperatively to guard against the development of alveolar osteitis. This advice, soundly based on scientific study as it is, would nevertheless be totally ignored by the overwhelming majority of smoker patients!

Several possible explanations have been advanced for this predisposition of smokers to alveolar osteitis:

i) Chemical irritation from nicotine, tar and other smoke
contaminants at the extraction/surgery site disrupts the integrity of the blood clot.

ii) Suction applied while 'dragging' on the cigarette may physically dislodge the blood clot or parts thereof.

iii) Tobacco ingredients are toxic and may affect the clotting mechanism systemically. It has been shown that tobacco smoke contains at least a dozen noxious gases [U.S. Department of Health (1977)] and over 3,000 chemicals - some of which have been identified as causing specific diseases e.g., lung cancer [Gori (1976)].

iv) Carbon monoxide (1-5% of tobacco smoke) can alter the morphology of arterial walls, making them more permeable: possibly having an adverse effect on the blood clotting mechanism [Gori (1976)].

v) Levine (1973) has shown that the platelet response to disodium adenosine diphosphate (ATP), a stimulus to platelet aggregation, is changed by the inhalation of cigarette smoke. One cigarette is enough to produce a significant retardation of platelet aggregation.

vi) Hawkins (1972) showed that blood coagulation, rate of initial clot formation and clot retraction all showed changes when the blood of smokers were compared with that of non-smokers. All three events were retarded in smokers.

Meechan et al. (1988) also found a significant increase in the incidence of alveolar osteitis in heavy smokers (4.4%, n = 963) compared with non-smokers (2.7%, n = 1620). They defined heavy smokers as those having 20 cigarettes or more daily. No significant differences were found between light smokers and non-smokers.

Although the mechanisms of wound healing in smokers have not been fully elucidated, there appears to be a body of evidence to indicate that smoking postoperatively, at best, predisposes to alveolar osteitis.
3.2.12. (a). Fibrinolysis.

*Background:* The fibrinolytic mechanism is a regulator of blood coagulation: preventing it when it is undesirable and when it occurs, removing blood clots to allow normal healing. Fibrinolysis is best considered as a series or cascade of interdependent biochemical reactions which may be initiated by tissue damage (trauma) or infection [Marsh (1981)].

Taussig (1979) provides a schematic diagram of fibrinolysis (Figure 3).

![Diagram of Fibrinolysis](image)

**Fig. 3.** Clotting and Fibrinolysis Cascades [Taussig (1979)].

Human fibrinolytic activity was first observed in cadavers; at post-mortem, blood became fluid and this blood incubated with various agents, e.g., alcohol, ether and chloroform gave rise to fibrinolytic activity [Konttinen (1968), Astrup (1969)].

Tillet and Gardner (1933) reported rapid fibrinolysis of human blood clot by filtrates of haemolytic streptococci cultures. The causative streptococci strain(s) was not identified.

Further investigation by Tillet (1938) confirmed the fibrinolytic
activity of the β haemolytic streptococci (especially Lancefield group A). Fibrinolytic activity was found to be high in humans, as distinct from other mammalian species.

Milstone (1941), in commenting on the fibrinolytic activity of the β streptococci, hypothesised that a precursor of the fibrinolytically active agent was present in human blood. This supposition was based on the observation that a streptococcal filtrate alone will not cause fibrinolysis, however upon addition of small amounts of the euglobulin fraction (that part of plasma containing plasminogen activators, plasmin and fibrinogen, obtained by acetic acid precipitation) pronounced fibrinolysis was seen.

Mullertz and Lassen (1953) postulated the existence of a proactivator in human blood. This was based on their observation that bovine plasminogen was fibrinolytically activated by a mixture of streptokinase and human globulin.

Other authors, notably Ablondi and Hagan (1957), Kline and Fishman (1957), Sherry, Fletcher and Alkjaersig (1959) considered proactivator not as a separate entity but rather a quality - "proactivator quality"; caused by the action of streptokinase on human plasminogen.

Birn (1970) found that kinases from staphylococci, coli bacilli and pseudobacilli in addition to streptococci are capable of forming activator from proactivator.

The fibrinolytic property of most of these bacteria is based on the conversion of plasminogen to plasmin. Tissue activators have been identified and their importance highlighted by the following authors: Lewis and Ferguson (1950), Astrup (1952), Albrechtsen (1959), Lack and Ali (1964).

Schematically, human tissue activators and their actions on plasmin may be represented as shown in Fig. 4.
Fig. 4. The components of the Fibrinolytic system and their relations [Birn (1973)].

Two types of tissue activators have been described:

i) A labile, saline soluble activator which loses its activity when heated at low pH.

ii) A protein bound, sparingly soluble activator which is stable and retains its activity when heated at low pH.

Rasmussen and Albrechtsen (1960) contended that the labile activator originates from the blood and as such is not a true tissue activator. It has been clearly demonstrated that fibrinolytic activity in tissues is triggered by damage to cells, as seen in inflammation and necrosis [Tagnon and Petermann (1949), Lewis and Ferguson (1950), Ende and Auditore (1961), Tice and Worth (1968) and Astrup (1969)].

Albrechtsen and Thaysen (1955) found plasminogen activators in human saliva. Also, a number of proteolytic enzymes in serum have been shown to activate plasminogen [Jacobson (1953)]. As well, other substances such as chloroform, ether, alcohol and adrenaline are known to activate
plasminogen *in vivo*. [Biggs, Macfarlane and Pilling. (1947)].

It must be stressed that fibrinolysis is a normal physiological process that removes fibrin deposits by the action of plasmin: an integral part of the tissue repair process. Normally, fibrin is constantly being laid down and removed as injury and repair occur. Hence, surgical trauma may give rise to an increase in fibrinolytic activity in the blood [Anderson, Nilsson and Olow. (1962)].

In the normal individual, fibrinolytic activity is controlled by the production of antiplasmin, a strong inhibitor of plasmin. This self regulation ensures that fibrinolysis is only allowed to proceed in areas where it is required and for only as long as required. In certain diseases this balance is disrupted, which may lead to either an increase or decrease in fibrinolysis. These effects may be generalised or localised, depending on the site and extent of injury.

3.2.12. (b). Fibrinolysis and Alveolar Osteitis.

In 1943, Sinclair suggested that fibrinolysis, or some other proteolytic activity capable of destroying blood clots, may be important in the aetiology of alveolar osteitis.

This concept received further impetus when, in 1964, Gabler *et al.* reported that patients awaiting oral surgery were found to have increased levels of circulating fibrinolysins.

Doku, Schklar and Bugbee (1966) showed that an antifibrinolytic agent, epsilon amino caproic acid (EACA) when used locally, improved extraction wound healing in hamsters.

Bjorlin and Nilsson (1968) detected an increase in fibrinolytic activity in the alveolar bone of patients who underwent various tooth extraction procedures. However, this local activity was not reflected systemically.
Birn (1970) showed that assayed fibrinolytic activity in the alveolar bone of patients suffering from alveolar osteitis was high in comparison to a control group with normally healing sockets. Furthermore, a direct correlation between fibrinolytic activity and the severity of symptoms was noted. Birn's (1970) findings were:

i) Fibrinolysis was moderate at the onset of symptoms (day 2), but as the pain and clot breakdown intensified, the fibrinolysis increased correspondingly.

ii) A peak (of fibrinolytic activity) was reached at day 10 - 11, after which there was a sudden decline in fibrinolytic activity as regenerative processes started to heal the socket.

iii) This decrease continued until healing was complete (Fig. 5).
Fig. 5. Mean fibrinolytic activity in saliva from patients with alveolar osteitis (unbroken line) (n = 20) and from patients with normally healing extraction wounds (broken line) (n = 19). F.A. = fibrinolytic activity in mm². ▲ = onset of alveolar osteitis. ▼ = cessation of alveolar osteitis.

The activity of saliva in patients with alveolar osteitis is a little higher than in the control patients, corresponding to 10-12 days after extraction. This corresponds to the time of maximum activity in the alveolus of alveolar osteitis patients.


Megquier (1971) also studied the fibrinolytic activity of human extraction sockets and found similar results as Birn (1970). Megquier showed that fibrinolytic activity in intra-oral wounds was increased in all postoperative complications, with the most pronounced increases in alveolar osteitis cases. He also noted plasmin activity in normally healing alveolar bone five days after the surgical removal of mandibular third molars.

Birn (1973) concluded that fibrinolysis is found in all postoperative complications as well as in normally healing extraction sockets. Only in alveolar osteitis does it reach a level which precipitates the
disintegration of the incumbent blood clot. Whether this disintegration is partial or complete depends on the fibrinolytic activity in the alveolus and the concentration of antiplasmin in the blood clot. Birn's (1973) investigations certainly provide a plausible explanation for one of the most important features of alveolar osteitis, namely, the partial or complete disintegration of the blood clot.


i) Saliva.

Albrechtsen and Thaysen (1955) initially reported the presence of proactivator (plasminogen) and activator (plasmin) in saliva. Birn (1970) compared the fibrinolytic activity of saliva in alveolar osteitis patients with a group of normally healing controls. He found a low level of fibrinolytic activity in both groups and concluded that this activity was not sufficient in quality and quantity to precipitate alveolar osteitis. Birn noted the existence of plasmin in saliva but ascribed the low level of fibrinolytic activity (and low plasmin levels) to a leakage of fibrinolytically active material from the alveolar bone rather than to the salivary contents per se (Fig. 6 and Fig. 7).

However, investigations by Schulte and Gewalt (1964) and Schulte and Goens (1967) recorded a high level of fibrinolytic activity in mixed saliva. The above authors concluded that this level of salivary fibrinolytic activity was significant to alveolar osteitis development. In summary, the presence of some fibrinolytic activity in saliva postoperatively, is to be expected; however, its significance to alveolar osteitis development is speculative. Other factors, e.g., alveolar bone fibrinolytic activity and bacterial contamination may well be of far greater significance.
Fig. 6. The mean fibrinolytic activity in the alveolus (unbroken line) and saliva (broken line) in patients with fibrinolytic alveolitis (n = 20). F.A. = fibrinolytic activity in mm². ▲ = onset of fibrinolytic alveolitis. ▼ = cessation of fibrinolytic alveolitis. The fibrinolytic activity rises sharply in the first days to reach a peak 10-12 days after extraction. Then it rapidly decreases. Saliva fibrinolytic activity varies daily, but shows a slight increase corresponding to the peak of activity in the alveolus.

Fig. 7. Mean fibrinolytic activity in the alveolus in fibrinolytic alveolitis (unbroken line, n = 20) and in the alveolus in normally healing extraction wounds (broken line, n = 19). F.A. = fibrinolytic activity in mm². ▲ = onset of fibrinolysis. ▼ = cessation of fibrinolytic alveolitis. Analysis of variance gave a significant p value (p < 0.001).


**ii) Bacteria.**

In 1970, Birn investigated the fibrinolytic potential of bacteria isolated from the alveolar bone of 10 patients suffering from alveolar osteitis. The bacteria were cultured aerobically and anaerobically in liquid and solid media. Using the fibrin plate method, Birn (1970) was unable to show any fibrinolytic activity arising from these bacteria. Human plasma was added to disclose a possible lysokinase activity. This too proved negative. Birn (1970) concluded that bacteria found in the alveolus of alveolar osteitis extraction sockets were not
responsible for the fibrinolytic activity normally seen in this condition.

However, Nitzan (1983) presented strong circumstantial evidence to the contrary. Nitzan, Sperry and Wilkins (1978) found the spirochaete Treponema denticola to be fibrinolytically active. As well, this bacterium was implicated in alveolar osteitis because of the following:

i) It has been isolated from alveolar osteitis extraction sockets.

ii) It belongs to the blood-lysing bacterial group (fibrinolytic activity).

iii) It is non-pathogenic in the sense that it does not produce inflammation.

iv) Its incubation and growth is facilitated by conditions found in the extraction wound.

In 1983, Nitzan highlighted the following properties of T. denticola as further evidence of a strong link with alveolar osteitis development:

i) An anaerobic infection would explain the foul odour and bad taste so characteristic of alveolar osteitis.

ii) T. denticola is found in abundance in some gingival conditions, including pericoronitis. This may partially explain the high incidence [88%, Nitzan (1983)] of alveolar osteitis in extractions associated with pericoronitis.

iii) Alveolar osteitis is never seen in childhood, this corresponds with the fact that T. denticola and alveolar osteitis make their first appearance in late adolescence [Nitzan, Sperry and Wilkins (1978)].

iv) The use of antibiotics either locally or systemically usually leads to a reported decrease in alveolar osteitis incidence [Versnel (1953), Rud, Baggensen and Moller (1963), Mourfield and
Barron (1968), Hall, Bildman and Hand (1971), Goldman et al. (1973), and Krekmanov and Hallander (1980).

By way of support for Nitzan's (1983) hypothesis, Catellani (1979) had earlier postulated a more subtle mechanism relating bacterial contamination of the extraction wound and fibrinolysis. Bacterial pyrogens have been identified as indirect activators of fibrinolysis, producing sustained increases in fibrinolytic activity when injected intravenously [Meneghini (1958)]. Catellani contended that certain species of the normal oral microflora secrete pyrogens at a basal rate. With trauma, e.g., tooth extraction, these bacteria multiply as cellular defences are weakened and their nutritional source expanded. The ensuing increase in bacterial population results in an increase in pyrogen production, increased fibrinolytic activity and subsequent development of alveolar osteitis.

Although Catellani's view is appealing, he does not provide experimental evidence to support his hypothesis, relying instead on extrapolations of previous studies by other investigators. Extraction wounds are contaminated by the oral microflora almost as soon as the extraction is completed and it seems reasonable to assume that these bacteria have some influence on the subsequent course of events. Whether this influence is sufficient to initiate the development of alveolar osteitis is unclear. On balance, the evidence suggests that the normal oral flora is a significant factor in alveolar osteitis.

iii) Tissue fibrinolysis originating from the maxillary/mandibular alveolar processes.

Birn (1971) demonstrated large amounts of tissue activators in extracts of alveolar bone from the mandibular third molar and incisor regions.
Two types of tissue activators were identified:

i) Labile (saline extract).

ii) Stable (potassium thiocyanate extract).

Also, it was shown that the concentration of stable tissue activators was the same in different areas of the jaws.

Following on from this, Birn (1971-2) found that the alveolar bone in alveolar osteitis cases not only contain stable tissue activators and plasmin, but also a conspicuous lack of labile tissue activators. Birn suggested that this was so because the labile activator was likely to disappear shortly after it is released - due to its inherent volatile nature. Diffusion into the blood stream was considered the most likely clearance mechanism. On the other hand, stable tissue activators were found in abundance as they are localised due to protein binding. Megquier (1971) also reported the detection of tissue activators in alveolar socket blood after dental extraction. As well, plasmin was detected (on the fifth postoperative day) - indicating the presence of stable tissue activators.

Birn and Myhre-Jensen (1972) confirmed that normal alveolar bone has a high level of fibrinolytic activity initiated by plasminogen activators. They also hypothesised that the stable activators are possibly linked with the osteoblasts of the endosteum. Magnusson and Gustafson (1972) reached the same conclusion, although their work was based on the rat. According to Birn and Fejerskov (1971), the dissolution of the blood clot is initiated by the liberation of these tissue activators from the alveolar bone. These activators convert plasminogen to plasmin; thereby precipitating fibrinolysis.

Birn is convinced that the excess production of alveolar bone derived tissue activators is the cause of alveolar osteitis. Birn (1973) coined the term "Fibrinolytic Alveolitis" to encapsulate this concept.
3.2.13. Conclusion.

Traditionally, the occurrence of alveolar osteitis has been ascribed to two principle causes:

i) The absence of a blood clot from the outset (immediately following dental extraction).

ii) Initial blood clot formation is not sustained.

Subsequent fibrinolysis precipitated by a variety of factors lead to the characteristic "empty, dry socket" [Nitzan (1983)].

The concept of a blood clot which fails to form initially is not consistent with the clinical findings and symptoms of alveolar osteitis. The total absence of a blood clot postoperatively does not account for the delayed onset of the characteristic, unbearable pain (generally, 2-3 days following extraction). Also, the typical picture of an "empty" socket with remnants of blood clot and necrotic debris at its base is incongruous.

Of the various theories explaining the disintegration of the blood clot as the aetiology of alveolar osteitis, one stands out, namely, Birn's (1973) hypothesis of localised fibrinolysis. For the first time in the literature, there is firm scientific evidence to support the theory. Especially convincing is the demonstration that fibrinolytic activity is closely linked to the clinical course of alveolar osteitis.

There is however, one reservation in my mind as regards Birn's "Fibrinolytic Alveolitis": fibrinolysis explains the mechanism of alveolar osteitis very elegantly, but the factors that instigate such a cascade of events have yet to be elucidated. Fibrinolysis is a normal physiological process of wound healing and as such cannot be regarded as the cause of disease. Excessive fibrinolysis is a response brought about by extraneous factors in certain conditions, it is these factors which should be viewed as the cause(s) of alveolar osteitis.
In Birn's defence, he did postulate the presence of a critical concentration of alveolar bone tissue factors above which fibrinolysis and hence, alveolar osteitis is likely to occur. Presumably, this critical concentration varies with different individuals and at times with the same individual, otherwise we would expect to see a higher incidence of alveolar osteitis in the elderly and debilitated and those with a history of alveolar osteitis.

Ritzau (1973) attempted to validate Birn's explanation of the role of tissue activators by using tranexamic acid (a proven tissue activator inhibitor) as a prophylactic against alveolar osteitis. Tranexamic acid failed in clinical trials to prevent the development of alveolar osteitis. The concept that the release of tissue activators is the sole precipitating factor in fibrinolysis appears now to be questionable.

However, Apennyl (a proprietary drug containing an antiplasmin component) was used successfully as a preventative against alveolar osteitis [Birn (1972)]. This result supports fibrinolysis as the process involved in the development of alveolar osteitis.

Nitzan (1983) proposed bacterial derived fibrinolysis as a cause of alveolar osteitis and promoted the bacterium Treponema denticola as an ideal candidate. Certainly, bacterial contamination of dental extraction sockets is a matter of course and it is not unreasonable to suspect an active role by the oral microflora in the development of alveolar osteitis. Although much circumstantial evidence is cited by Nitzan to advance his idea, evidence derived from experimentation along Koch's postulates (for pathology) would have been more convincing.
Chapter 4

Treatment.

4.1. Introduction.
A wide range of treatments are available for alveolar osteitis and these may be conveniently categorised as:

i) Antibiotic agents (systemic and local).
ii) Bland obtundent dressings, e.g., collagen paste and polylactic acid (PLA).
iii) Analgesic or anodyne dressings, e.g., zinc oxide eugenol packs, alvogyl.
iv) Antiseptic dressings, e.g., iodoform gauze, bismuth iodoform paraffin paste (BIPP).
v) Antifibrinolytic agents, e.g., apenyl cones and tranexamic acid.
vi) Surgical intervention to remove necrotic material and encourage new blood clot formation.
vii) Antiseptic mouth rinses prior to dental extraction in an attempt to reduce the oral microflora, e.g., chlorhexidine (0.2%).
viii) Lavage of the surgery site prior to wound closure, i.e., mechanical clearance of bacteria, with normal physiologic saline solution.
ix) Dextranomer granules (wound debridement).
ix) Fibrin sealants.
4.2. Treatment Modalities and Rationale.
The management of alveolar osteitis is aimed at its prevention as well as definitive treatment of the afflicted socket.
Traditionally, medication has been either systemic or local (topical). Systemic administration is best illustrated by the use of oral and parenteral antibiotics. Topical application is usually in the form of socket packs, surgical sponge, dental cones or pastes. A multitude of drugs have and are being used to treat alveolar osteitis. In this chapter, each treatment type will be examined individually and an assessment made of its relative merits.

4.3. Antibiotics.

4.3.1. Systemic Administration.
The rationale for using antibiotics is based upon the assumption that alveolar osteitis is a bacterial infection, a hypothesis that is yet to be proven.
Nevertheless, in the past, some eminent authors, [Guralnick (1968), Thoma (1969), Howe (1971) and Killey, Seward and Kay (1971)] have recommended the use of systemic antibiotics to reduce postoperative sequelae, including alveolar osteitis. Traditionally, penicillin, erythromycin, clindamycin and tetracycline have been the drugs of choice. In more recent times metronidazole and, to a lesser extent, tinidazole have become the new hope as attention is focused on the role of anaerobes in dental infections. Oral administration is usually the preferred method, with regimens varying from the first dose being taken one hour preoperatively to anything up to two days before surgery. Duration of therapy is usually 3 to 5 days. The recommended dose varies from 200mg to 400mg 8 hourly (t.d.s.).
The best evidence to date in support of the effectiveness of systemic antibiotics is the study by Krekmanov and Hallander (1980), where in a controlled, double blind, randomly allocated trial, penicillin V (Kavepenin) was compared with:

i) Scopolamine (an antialluge).

ii) A control group where no medication was given.

One hundred and twenty patients, who were matched for age and sex, underwent the removal of at least one mandibular third molar. Cases with concurrent pericoronitis were included. As well, the patients were distributed equally amongst the three treatment groups. Alveolar osteitis occurred in 13 of 40 patients in the control group, 2 of 40 in the penicillin group and 1 of 40 in the scopolamine group. Cultures taken of the alveolar blood clots 48 hours postoperatively showed significant diminution of aerobes and anaerobes in the penicillin group compared with the other groups. The authors concluded that penicillin and antialluge medication inhibited the development of alveolar osteitis. However, no specific micro-organism responsible for producing alveolar osteitis was demonstrated. Krekmanov and Hallander (1980) suggested the possibility of microbial synergism in alveolar osteitis development to explain their findings.

This evidence, though statistically significant, is nevertheless circumstantial. No causative micro-organism was isolated and a strong placebo effect would have been in operation, since the control group received no medication. Although microbial synergism in cases of alveolar osteitis was postulated, the result could just as easily have been interpreted as opportunistic colonisation of the socket by oral commensals.

MacGregor and Addy (1980) studied the value of penicillin in preventing postoperative sequelae (pain, swelling and trismus) following the
removal of ectopic mandibular third molars and concluded that giving systemic antibiotics was only "superior at the lowest level of statistical significance". However, penicillin therapy showed to advantage in difficult cases, where the amount of surgical trauma was considerable. MacGregor and Addy cautioned against the indiscriminate use of antibiotics, pointing to such untoward effects as sensitisation, development of resistant strains and, less commonly, anaphylaxis. Despite this, the authors did feel that systemic antibiotics were warranted in difficult extraction cases. Bystedt, Nord and Nordenram (1980) investigated the effectiveness of penicillin (Azidocillin), erythromycin, clindamycin and doxycycline on postoperative complications after surgical removal of mandibular third molars. Patients (n=140) were randomly distributed in a double blind study (placebo vs antibiotic) in which medication was given orally, preoperatively and for seven days postoperatively. Antibiotic concentrations in serum, alveolar serum and mandibular bone were assayed and the postoperative course (pain, swelling, trismus, alveolar osteitis, wound healing) recorded. It was found that no correlation existed between antibiotic concentration and postoperative complaints. However, the antibiotic group (80) responded significantly better in terms of wound healing than the placebo group (60). Alveolar osteitis occurred in 5 patients of the placebo group (8% incidence) compared with 3 patients in the antibiotic group (4% incidence). Bystedt, Nord and Nordenram concurred with MacGregor and Addy in concluding that systemic antibiotics offered only a marginal advantage in routine third molar operations and should be reserved for use in particularly traumatic procedures where they have proven to be of value. Since the discovery of an abundance of anaerobic bacteria in painful
extraction wounds (including alveolar osteitis) by Schroff and Bartells (1929) and Belding and Belding (1934), there has been growing interest in the possible role of anaerobes in the aetiology of alveolar osteitis. In 1983, Nitzan hypothesised that Treponema denticola (an oral anaerobe) may be the culpable pathogen of alveolar osteitis.

In line with this thinking, metronidazole has been evaluated for its effectiveness in the prevention and treatment of alveolar osteitis. Rood and Murgatroyd (1980) trialed oral metronidazole tablets (200 mg t.d.s. for 3 days) in 1096 patients (2035 extractions). Five hundred and fifty-five patients received metronidazole, whilst 541 were given a placebo tablet. Six patients from the metronidazole group developed alveolar osteitis compared with 23 patients from the placebo group (1%: 4.2% respectively). On this basis, the authors concluded that oral metronidazole was effective in the prophylaxis of alveolar osteitis.

Mitchell (1984) drew attention to two advantages of metronidazole over other antibiotics such as penicillin:

i) Metronidazole eliminates the pathogenic anaerobes whilst not disturbing the normal oral aerobic flora.

ii) Microbial resistance does not appear to develop among anaerobes with metronidazole use. [Chow, Bednorz and Guze (1977), Finegold (1977), Sutter and Finegold (1977)].

In 1981, Rood and Danford reported success in treating (as distinct from prophylaxis) alveolar osteitis with oral metronidazole - 400 mg t.d.s. for 5 days. They treated 60 patients, of whom 45 returned for review, whence a subjective evaluation was undertaken to determine if there was any symptomatic improvement. Although conceding that subjective bias and a placebo effect would have influenced the patients, Rood and Danford (1981) were convinced that oral metronidazole aided the resolution of alveolar osteitis. They observed
that patients treated with oral metronidazole were symptom-free in 3 days, on average, compared with 6-8 days when zinc oxide/eugenol socket dressings were used [MacGregor (1964)]. Two clinical advantages impressed the authors:

i) Early resolution as judged by the "symptom-free" criteria.

ii) The socket itself need not be interfered with in any way.

Other authors have opposed the use of systemic antibiotics in an attempt to prevent postoperative complications. Moore (1965) and Kruger (1968) rejected routine systemic antibiotic prophylaxis following third molar surgery, condemning it as potentially harmful and without scientific basis.

Paterson, Cardo and Stratigos (1970) studied a group of maxillofacial trauma patients who required major surgical intervention. They concluded that systemic antibiotic therapy postoperatively, as a prophylactic measure, was questionable and may lead to complications in the management of those patients who do develop postoperative infections. Development of resistant bacterial strains was seen as being more detrimental than the perceived benefits of prophylactic systemic antibiotics.

Rud (1970) found no significant increase in complications in cases of acute pericoronitis operated on without antibiotic prophylaxis compared to those with prophylaxis. He advocated systemic antibiotics only in cases where generalised symptoms (e.g., an elevated body temperature, enlarged regional lymph nodes) are present or where extractions proved to be difficult.

Zallen and Strader (1971) after assessing antibiotic usage in mandibular osteotomy, concurred with the view that antibiotic
prophylaxis is unnecessary as a routine.
Curran, Kennett and Young (1974) conducted a double blind study of 133 impacted mandibular third molar surgery cases where one group was given antibiotic prophylaxis against a control group (no antibiotics). The authors were unable to show any reduction in alveolar osteitis incidence in the antibiotic group compared with the control group. Curran, Kennett and Young felt that since the majority of patients undergoing third molar surgery are healthy young adults, whose response to surgical trauma is generally good, it is illogical to submit such patients to the risk of sensitising them to an extremely valuable drug for a questionable improvement in their postoperative recovery.
Van Gool, Bosch and Boering (1977), after a study of 932 mandibular third molar extractions, concluded that prophylactic administration of systemic antibiotics was of no value in ameliorating the postoperative course.

Conclusion:
Whilst the routine administration of systemic antibiotics after every extraction is to be discouraged, there is good evidence to show that antibiotics are of benefit in cases where extraction is difficult. Here, increased iatrogenic trauma is seen as a predisposing factor in the development of alveolar osteitis.
The decision must, of course, be a clinical one. The perceived benefit of antibiotic therapy must be weighed against the risk of sensitisation and other adverse effects, e.g., nausea, abdominal cramps, diarrhoea and candidal infection.
4.3.2. Local Application of Antibiotics.
Local application, either immediately after extraction (as a preventative) or as treatment for the condition, is quite popular in Europe and the United States of America.
Various vehicles have been tried, including: glycerin, gelatin sponge (Gelfoam), ribbon gauze and carboxymethylcellulose paste (Orabase).
It appears that Sinclair (1937) started the trend to local implantation of antibiotic preparations into extraction sockets with his report which commended the local use of sulphonamides in the treatment of alveolar osteitis. Enthusiasm for sulphonamides reached a peak in the 1940s with a host of authors extolling its virtues, including: Sinclair (1938), Sinclair and Barker (1938), Dent (1940), DeWolf (1940), Stern (1941), Greiss (1942), Krogh (1948), Davis et al.(1955), Rud, Baggensen and Moller (1963). Claims of sulphonamide success (as with other local antibiotic preparations) are based on two assumptions:

i) Antibiotics reduce the intra-socket bacterial population thereby removing the major cause of alveolar osteitis, i.e., the aetiology of alveolar osteitis is bacterial.

ii) The mechanical displacement of the central portion of the coagulum physically removes the bulk of bacteria and is, at least, of equal importance as the bactericidal effect of the drug. Because the clot volume is reduced, healing is aided - on the premise that a smaller blood clot needs less time to organise [Millhon et al. (1943)]. This assumption is debatable, because the dead space left after the absorption or expulsion of the used dressing will still need replacement with granulation tissue to effect completion of wound healing.

It is interesting to note that, with the exception of metronidazole in Orabase [Mitchell (1984)], all local antibiotic preparations have been
advocated for use prophylactically. Indeed, as early as 1943, Millhon et al. made the observation that sulfathiazole was of no value once symptoms of alveolar osteitis had developed.

Rud, Baggensen and Moller's (1963) investigation into the efficacy of 'sulpha' cones used after mandibular third molar surgery to reduce postoperative pain serves as an example of a clinical trial which justified the use of such preparations. The authors used pain as the only criterion for postoperative complications, including alveolar osteitis. No record of the actual incidence of alveolar osteitis was made in the study. One thousand, six hundred and twenty impacted mandibular third molars were removed and 'sulpha' cones of varying consistency were placed prior to wound closure. A series of placebo cones were used (lactate sugar) as well as a control series of 425 patients where no cones were used. The presence or absence of pain was judged by patients on a subsequent postoperative appointment. After statistical analysis, the authors came to the following conclusions:

i) Sulphonamide containing cones were effective in preventing postoperative pain.
ii) The addition of penicillin (5,000 IU and 25,000 IU) did not enhance the performance of the 'sulpha' cones.
iii) Placebo cones of lactate sugar had no effect on alveolar osteitis compared with controls.
iv) 'Sulpha' cones were effective in reducing the incidence of pain in acute pericoronitis cases (30% incidence in the control group compared with 15% incidence in the 'sulpha' cone group).

The authors equated this reduction in pain as a reduction in the incidence of postoperative infection. This may be misleading; although
pain often accompanies infection, it is not always so. On the strength of these conclusions, Rud. Baggensen and Moller (1963) recommended the use of locally implanted 'sulpha' cones immediately after removal of mandibular third molars as a safe and effective means of reducing the incidence of postoperative complications, including alveolar osteitis.

Authors who are convinced of the effectiveness of locally applied sulphur compounds (as a prophylactic against alveolar osteitis) point out the following advantages:

i) The implanted sulphonamide and its vehicle are capable of being absorbed as part of the healing process [Millhon et al. (1943)].

ii) Sulphonamides are well tolerated by the oral tissues [Rud, Baggensen and Moller (1963)]. A histological study by Frandsen and Pindborg (1961) which investigated wound healing in rat dental sockets implanted with 'sulpha' cones, found no evidence of delayed healing or foreign body reaction.

iii) Minimum clinical time is required for cone insertion prior to closure.

iv) Marked reductions in the incidence of alveolar osteitis have been reported in a number of clinical trials.

However, despite the apparent success of locally implanted sulphonamides, quite a few remain unimpressed. Ostrander and Hartman (1942) reviewed 72 previously published reports in the dental and medical literature and concluded that the use of sulphonamides in extraction wounds on a routine basis is of doubtful value. Their criticisms concerned the structuring of the clinical trials and included:

i) The lack of objective controls.
ii) The use of subjective criteria in the evaluation of clinical performance.

iii) The frequent combination of therapeutic agents, making the identification of the active ingredient difficult.

iv) The insufficient number of cases included in such trials.

Ostrander and Hartman (1942) felt that there was insufficient evidence to justify the claimed effectiveness of sulphonamides. This opinion was supported by Krashen (1940), Devine (1944), Boothe (1945), American Medical Association (1947), Pennington (1947) and the Council of Dental Therapeutics, American Dental Association (1948). Krashen (1942) considered the local use of sulphonamides as having no merit. He noted that such preparations delayed wound healing and were no substitute for proper surgical technique and careful wound debridement. Despite this, he noted some clinical advantage in using sulphonamides in acute/subacute cases. Krashen deplored the use of cones and tablets; he claimed that the physical mass actually delayed granulation of the socket.

Versnel (1953) examined the histological effects of implanting a penicillin cone and compared these to a sulphonamide cone and oxidised cellulose gauze. Dogs were the experimental model. Four dogs were used; the socket of one was left untreated as a control. Versnel found that the sulphonamide cone aided the breakdown of the coagulum, to the extent that it may have prevented stable clot formation. The same was true of the oxidised cellulose socket. He felt that both substances retarded wound healing from the time of implantation. Penicillin came closest to normal with new bone evident on the fourth postoperative day - similar to the control socket. Foreign body reaction was seen in
all test sockets.
Olech (1953) clinically evaluated the efficacy of sulfanilamide (0.25 gm), penicillin G (5,000 units), sulfanilamide and penicillin G (0.25 gm and 5,000 units) against a control series where none of the sockets were interfered with. The test substances were applied in powder form to mandibular third molar sockets immediately following removal. Olech found that, although locally applied antibiotic preparations gave the clinical impression of promoting healing and lessening postoperative morbidity, analysis of objective data did not support this. He concluded that the use of such medications was hard to justify. MacGregor and Hutchinson (1975) evaluated sulfanilamide/sulfathiazole/potato starch cones (100 mg) following the reported success of Rud, Baggensen and Moller (1963), and Brown, Merrill and Allen's (1970) contention that there was a significant statistical association between an increase in the intra-socket streptococci population and the incidence of alveolar osteitis. Ninety four patients were included in the double blind study in which 'sulpha' cones were trialed against a placebo and control group simultaneously. MacGregor and Hutchinson concluded that the local application of sulphonamide cones was not an effective means of reducing postoperative complications (including alveolar osteitis) following mandibular third molar removal. The hypothesis that streptococci strains are responsible for alveolar osteitis is, at best, debatable.
Other antibiotic agents have been tried since the advent of the sulphonamides, no doubt clinicians were encouraged by their reported effectiveness. Notable amongst these are penicillin, erythromycin, tetracycline, lincomycin, and more recently, topical metronidazole.

Penicillin. Gwinn and Grimm (1948) compared the use of penicillin
and sulfathiazole as socket implants after extraction. They concluded that both drugs were effective in aiding wound healing.

Epstein and Kaufman (1951) tested pure crystalline penicillin G in the form of 50,000 unit tablets. The sample size was 250 tooth extractions. There was no control group. Uniformly good healing was reported in all cases.

However, Holland and Tam (1954) conducted a double blind experiment where penicillin tablets (50,000 units) were placed in 274 uncomplicated extraction sockets and compared with 286 uncomplicated extraction sockets treated with a placebo tablet of sterile lactose. No significant difference in healing was found between the two groups.

Similarly, Linn (1954) compared 147 mandibular third molar sockets after surgery which had two penicillin 50,000 unit tablets implanted immediately after the procedure, with 114 like sockets which received no medication. Again, no significant differences were found between the groups.

Local use of penicillin in dental sockets has been abandoned in more recent times because of the well known risks of hypersensitivity and anaphylactic shock.

**Erythromycin.** Mourfield and Barron (1958) considered erythromycin dental cones to be superior to any other in decreasing the incidence of alveolar osteitis. In a series of 1,471 multiple extractions, erythromycin cones were placed in 917 sockets and one socket in each patient was left untreated as a control. A reduction in alveolar osteitis incidence was seen in the erythromycin group (1.2%) compared with 13.3% in the control group.

Erythromycin, as with penicillin, has been discarded as a local implant
due to problems with drug sensitisation.

**Tetracyclines.** The tetracyclines, although initially a poor relative to the sulphonamides in popularity, have nevertheless, sustained enough interest to see their continued local use today - especially in the U.S.A. Stewart and Roth (1950) first drew attention to the use of 25 mg chlortetracycline hydrochloride (Aureomycin) cones in extraction sockets of acute alveolar abscess cases. Their report was not particularly encouraging; foreign body reactions were noted in all 13 cases. It was thought that this foreign body reaction was due to the non-absorbable nature of the cone material.

Verbic (1953) tested the efficacy of 50 mg soluble Aureomycin tablets, convinced that the foreign body reaction noted by Stewart and Roth (1950) was due to the composition of the cone and not the chlortetracycline HCl *per se*. Two sets of 103 patients were divided into control and experimental groups. The 50 mg Aureomycin tablets were implanted immediately after extraction. Verbic concluded that Aureomycin did significantly reduce the incidence of alveolar osteitis (6 cases of alveolar osteitis in the Aureomycin group compared with 14 cases in the control group). With regard to the foreign body reaction, the author detected Aureomycin particles in the healing sockets after the first postoperative week - but judged this not to be a foreign body reaction. No histological examination was performed to definitively rule out a foreign body reaction to the Aureomycin tablets.

Davis *et al.* (1955) reported a 60% decrease in postoperative symptoms of the oxytetracycline (5 mg) group compared with controls. No adverse effects from the oxytetracycline cones were reported. Only two instances of alveolar osteitis were recorded, but the authors did not indicate to which of the treatment groups they belonged.
Quinley, Royer and Gores (1960) using Achromycin tablets (50 mg tetracycline hydrochloride) first noted a marked foreign body reaction which manifested as an insoluble, black tarry substance which sloughed off from the treated socket. After some experimentation, they concluded that this was due to the crystallisation of the free base of the tetracycline hydrochloride molecule, which follows initial dissolution into the alveolar blood. An unspecified number of cases developed this unsightly foreign body reaction; the authors merely stated an incidence of 'several' cases. However, despite this complication, Quinley, Royer and Gores (1960) concluded that oxytetracycline HCl was an effective prophylaxis against alveolar osteitis. In a series of 510 mandibular third molar sockets, an alveolar osteitis incidence of 0.78% was achieved using oxytetracycline HCl.

In a study of 99 patients, Hall, Bildman and Hand (1971) reported on the use of Gelfoam (gelatin sponge) as a vehicle for the local application of tetracycline solution. Gelfoam was used in an attempt to prevent the foreign body reaction reported earlier by Quinley Royer and Gores (1960). In a double blind, randomised study, Hall, Bildman and Hand noted an incidence of 7% (alveolar osteitis) in the tetracycline treated side compared with 19% on the side treated with placebo. No tarry residue was seen in any of the tetracycline treated sockets. The authors concluded that soluble tetracycline in combination with gelatin sponge is a safe and effective means of reducing the incidence of alveolar osteitis.

A retrospective study by Davies et al. (1981) of their use of a granular gelatin/tetracycline compound in mandibular third molar sockets over a period of 20 years, showed an alveolar osteitis incidence of 2.67% (out of 860 sockets treated with this medication). The authors felt that the routine use of such a compound consistently provided a firm blood clot
free of bacterial infection, thus increasing the likelihood of uneventful socket healing.

Gelfoam saturated with Terra-Cortril Ophthalmic suspension (5 mg oxytetracycline and 15 mg hydrocortisone acetate) was reported by Julius et al. (1982) to reduce alveolar osteitis morbidity to 6.6% in a double blind study of 90 impacted, bilateral mandibular third molars. No significant side effects were seen and the clinicians were impressed by the observation that the Terra-Cortril-Gelfoam dressing appeared to have worked best in the most traumatic cases.

Rutledge and Marcoot (1987), whilst confirming the effectiveness of Terra-Cortril-Gelfoam dressings, also drew attention to the rather disturbing complication of myospherulosis. Myospherulosis is a foreign body reaction which manifests as indurated swellings of muscular and subcutaneous tissue [McClatchie, Warambo and Bremner (1969)]. Earlier, Dunlap and Barker (1980) had described a case of myospherulosis following the placement of a Terra-Cortril-Gelfoam socket dressing into a mandibular third molar wound. In the jaws, pain and swelling often accompanies myospherulosis, but in some cases it may be asymptomatic. Investigations by Kyriakos (1977), Bright, Russel and Keyes (1982) and Ide et al. (1984) strongly suggested that myospherulosis is an iatrogenic condition which follows the implantation of petroleum based products into extraction wounds and other tissue spaces. Histologically, the picture is one of frustrated bone repair, with a proliferation of tan to brown spherical structures admixed with erythrocytes. Radiographically, the lesion presents as a radiolucent image which persists for an indefinite period after the extraction/implant site has clinically healed.

Belfiglio, Wonderlich and Fox (1986) reported a case of myospherulosis following the use of a Terra-Cortril-Gelfoam socket dressing in a
mandibular third molar wound six years earlier. Upon surgical investigation, a black tar-like substance with the consistency of petroleum jelly was found. Belfiglio, Wonderlich and Fox advised against the use of Terra-Cortril type ointments; especially since a significant portion of cases are asymptomatic and hence, would go undetected by most patients.

Recently, the use of tetracycline in tablet/cone form has been abandoned, due to the incidence of foreign body reactions and associated delayed healing in treated sockets [Hall (1987)].

Despite this, Sorensen and Preisch (1987) advocated the use of tetracycline in powder form as a socket dressing after third molar surgery. They based their opinion on a study where they achieved a reduction in alveolar osteitis incidence from 28.7% (controls) to 14.6% (tetracycline treated). Two hundred and nineteen mandibular third molar sockets were treated with the tetracycline powder.

Hall (1987), although discouraging the use of tablets or cones, advocated the use of Gelfoam soaked in a tetracycline solution as a socket dressing/implant.

Advocates of local tetracycline application, cite the following advantages:

i) Safety. Tetracyclines are claimed to be the least sensitising of all the antibiotics available.

ii) Convenience of treatment delivery.

iii) No patient compliance is required in respect of postoperative self medication.

iv) Documented efficacy in the prevention of alveolar osteitis.

Recently, Fridrich and Olson (1990) in a large, well controlled study (476 patients - 952 mandibular impacted third molar surgery sites),
confirmed the effectiveness of Terra-Cortril/Gelfoam socket implants as a preventative of alveolar osteitis.

**Lincomycin.** Goldman *et al.* (1973) advocated the use of lincomycin solution in a vehicle of gelatin sponge as an effective socket dressing in the prevention of alveolar osteitis. Two groups of 90 patients were treated in a double blind study which used either lincomycin/gelatin sponge or saline/gelatin sponge (placebo). The lincomycin group scored an alveolar osteitis incidence of 1.1% compared with 7.8% in the placebo group. The authors concluded that lincomycin appeared to be superior to other antibiotic preparations, including tetracycline. Fridrich and Olson (1990) also found that lincomycin/Gelfoam was superior to oxytetracycline HCl-hydrocortisone acetate (Terra-Cortril)/Gelfoam in reducing the incidence of alveolar osteitis after mandibular third molar surgery. However, both were significantly better than Gelfoam alone (control).

Trieger and Schlagel (1991) reported earlier studies by the authors which showed that clindamycin (a semi-synthetic derivative of lincomycin) used in combination with Gelfoam as a socket implant was significantly effective in preventing alveolar osteitis. Trieger and Schlagel were so impressed that they billed the clindamycin/Gelfoam therapy as "a simple procedure that works"!

**Metronidazole.** Until 1984, antibiotic preparations for local application had been aimed at prevention rather than cure. Mitchell (1984), studied the potential of 10% metronidazole in a carboxymethylcellulose gelatin (Orabase) vehicle as a means of treatment of the afflicted socket. The sample was 55 cases, which, although small, was double blind and placebo controlled. Under these
conditions, metronidazole was judged superior to placebo, reducing the cure time from a mean of 8 days to 5 days. Mitchell saw the use of metronidazole as advantageous because resistant anaerobes are almost unknown. Pain relief was reported to occur within hours of dressing placement and the material appeared to leach out of the socket progressively, making removal unnecessary. The apparent effectiveness of the metronidazole/Orabase dressing is supportive of the hypothesis that anaerobic bacteria are contributory to the pathogenesis of alveolar osteitis.

**Conclusion:**
There is ample clinical evidence to suggest that locally applied antibiotic preparations do lower the incidence of alveolar osteitis. However, indiscriminate use of such antibiotics has recently been frowned upon due to the very real risks of hypersensitivity and the development of resistant strains. Alveolar osteitis, although exquisitely painful, is known to be self limiting, i.e., will resolve on its own, even if left untreated. From a medico-legal standpoint it would be extremely difficult to defend exposing a patient to the attendant risks of such powerful drugs in order to prevent a condition which has a low incidence (approx. 3% overall) and is self limiting anyway. This is probably the reason why, despite the favourable reports, locally applied antibiotics have not enjoyed the same popularity as more empirical formulations such as zinc oxide and AlvoGyl socket dressings. Another consideration is that the use of topical antibiotic preparations (or any other preparation for that matter) cannot guarantee freedom from alveolar osteitis in any given patient.
As a clinician, I consider that the risks associated with the routine use of topical antibiotics significantly outweigh the potential benefits to
be gained. However, in patients with a history of alveolar osteitis such therapy may well be justified.

4.4 Bland Obtundent Dressings.
Clinical experience has shown that any dressing or foreign material (provided it is non-toxic) which occludes an alveolar osteitis socket, will reduce the pain and allow wound healing by secondary intention [Turner (1982)].

4.4.1. Polylactic Acid.
Polylactic acid (PLA) is a biodegradable ester polymer developed by Kulkarni (1966) for use as an inert surgical implant with haemostatic properties. Its reputed biocompatibility and safety, [Kulkarni (1971), Cutright and Hunsuck (1971) and Cutright et al. (1974)] prompted Brekke et al. (1983) to investigate its value as a post-extraction implant for the prevention of alveolar osteitis. The rationale for using PLA is that any substance with the ability to absorb blood and act as a clot matrix would be an adjunct against alveolar osteitis. It was implied that an artificial clot matrix adds strength to the original blood clot. The fact that PLA is biodegradable enhanced its appeal.
A study of 228 patients who required bilateral mandibular third molar removal was conducted where one surgery site was implanted with a PLA cube whilst the other site (control) was left untreated after simultaneous left/right side extraction. An alveolar osteitis incidence of 16.2% (27/228) was reported for the controls compared with 3.5% (8/228) for the PLA sites. Clinically, the PLA mesh was well tolerated by the socket tissues and showed no untoward effects on the healing process. A 3 month recall of patients showed no discernible differences between the treated and untreated sockets. Brekke et al. concluded that
PLA was effective in providing an alternative medium for granulation tissue support within the original blood clot. This was thought to be responsible for the reduced incidence of alveolar osteitis seen in the PLA treated sockets.

Two subsequent studies by the same team confirmed their initial findings. Brekke, Bresner and Reitman (April 1986) found an incidence of 2.2% (PLA sites) compared with 18.1% (control) in 227 patients. Similarly, in a sample of 215 patients, the following frequencies were found: 2% (PLA) compared with 19% (control) [Brekke, Bresner and Reitman (July (1986)].

In a comparative study of the effect of polylactic acid (PLA), gelatin sponge (Gelfoam) and oxidised cellulose (Surgicel) dressings on healing extraction sites in dogs, Olson, Roberts and Osbon (1982), found that PLA demonstrated excellent tissue tolerance and less inflammatory reaction than either gelatin sponge or oxidised cellulose. However, PLA did elicit a mild foreign body reaction. Also, PLA was the slowest of the three to be completely biodegraded; it remained in the tissues for approximately three months.

Despite the reported effectiveness of PLA cubes, there are a number of disadvantages associated with its use:

i) The PLA cube must be fashioned to fit the socket. A scalpel must be used so as not to collapse the interstices of the cut surface. If the interstices are collapsed, an impenetrable wall would result - precluding the complete saturation of the mesh with alveolar blood. If this were so, then the wound would fail to heal by primary intent.

ii) Allowance must be made for its absorbency, the cube must fit to a maximum of 75% of the socket by volume. Otherwise, pain due to the PLA's expansion will result postoperatively.
Accurate sizing of the cube is important as it may be ineffective if too small.

iii) Placement of the cube must be done without undue force so as to prevent interstitial collapse.

iv) In the event that a PLA treated socket develops alveolar osteitis, the remnants must be curetted out under local anaesthesia prior to instigating conventional therapy.

v) PLA cubes are of no value in the treatment of established alveolar osteitis as its effectiveness is dependent on being saturated with alveolar blood.

**Conclusion:**

PLA appears to be very effective as a prophylactic agent against alveolar osteitis. It is well tolerated, non-toxic, biodegradable and results in healing which is indistinguishable from normal control sockets. However, it does elicit a mild foreign body reaction, is slow to resorb (three months) and is technique sensitive in application. Its greatest appeal is its inherent biological 'inertness' and relative biocompatibility. My opinion is that no socket dressing should be placed routinely prior to wound closure; as, in the vast majority of cases, there is no substitute for the original (whole) blood clot. However, PLA may be of value in a number of clinical situations.

**4.4.2. Collagen Paste.**

Mitchell (1986) trialled the use of collagen paste (formula K) for the treatment of established alveolar osteitis. In his search for a suitable formulation, Mitchell considered these to be the ideal properties of an alveolar osteitis socket dressing:

i) Early and effective pain relief.
ii) Non irritant to surrounding tissues.
iii) Absorbed or incorporated into the healing tissues.
iv) Capable of close bony contact.
v) Bactericidal.
vi) Stable in oral fluids.
vii) Dimensional stability (no change in volume when exposed to blood and saliva).
viii) Ease of application.
ix) Single visit treatment.
x) Low cost.

A study of 100 patients treated with 'formula K' collagen paste was undertaken. Another 51 patients received the standard zinc oxide eugenol dressings as controls. The results showed that the collagen treated patients experienced less pain during treatment, were less likely to return for review, showed less tissue reaction (delayed healing) and required fewer dressings than their zinc oxide counterparts. It was not clear whether the collagen paste washed out or became incorporated into the healing tissues. Mitchell's rationale for using collagen is to provide a biologically compatible matrix which encourages the ingrowth of fibroblasts; thus ensuring secondary granulation. The lack of bactericidal properties was not seen as a major handicap of 'formula K'.

Mannai et al. (1986) evaluated the biological response to collagen sponge in the extraction sockets of mongrel dogs at three days, one week and six months intervals. Histological sections showed a slight lymphocytic reaction but no foreign body reaction (as distinct from the foreign body reactions noted with gelatin sponge, polylactic acid and oxidised cellulose). They concluded that collagen was non-toxic and biologically well tolerated. Also, Mannai et al. indicated that collagen
sponge (Collaplug) may be useful as a preventative of alveolar osteitis. In this case, the collagen would be implanted in the socket immediately after extraction. This feeling was based upon the observation that collagen sponge is a good haemostatic agent (encourages platelet aggregation), forms a matrix for the blood clot and initiates osteogenesis [DeVore (1977)]. To date, no clinical evaluation using collagen sponge as a means of preventing alveolar osteitis development has been documented in the literature.

**Conclusion:**
Collagen appears to be the best tolerated dental implant to date. However, because it is of porcine origin, there is a potential risk of hypersensitivity associated with its use. Nevertheless, its efficacy as a treatment and prophylactic agent for alveolar osteitis needs further clinical evaluation to verify the promising results obtained by Mitchell (1986).


4.5.1. Zinc Oxide/Eugenol.
The ubiquitous zinc oxide/eugenol first made its appearance to general dentistry in 1875 when Dr. Foster Flagg demonstrated its usefulness as a cavity liner [Molnar and Skinner (1942)]. Its use as a surgical dressing was popularised with the introduction of a zinc oxide/eugenol 'postoperative pack' by Ward (1932). His formulation was marketed as Ward's 'WondrPak'. Commercially, it was very successful. However, its acceptance by the profession was not without its problems. In 1934, the Council on Dental Therapeutics of the American Dental Association declared it unacceptable on account of the secrecy
over its composition [Molnar and Skinner (1942)]. Despite this, it became popular with clinicians because of its remarkable success in alleviating the major symptom of alveolar osteitis - pain. The formulae of commercial preparations of zinc oxide/eugenol pastes such as 'WondrPak', 'Kirland Kaiser' and 'Box' are essentially the same and consist of varying proportions of the following:

i) Powder
   - zinc oxide (sedative to tissues)
   - resin [pine resin or abietic acid (reinforcement)]
   - tannic acid (astringent)
   - asbestos fibre (reinforcement)
   - zinc acetate or stearate (setting accelerator).

ii) Liquid
   - eugenol (anodyne, bactericidal)
   - cottonseed oil (paste plasticity)
   - thymol (antiseptic).

[Smith (1970)].

The clinical success of zinc oxide/eugenol dressings is dependent upon the anodyne properties of eugenol and the occlusive nature of the set pack. Because free eugenol is constantly leaking from the set material, the anodyne effect is continuous. Water (saliva) tends to extract eugenol from the zinc eugenate thereby encouraging further eugenol release [Molnar (1967)].

Criticisms against the use of zinc oxide/eugenol dressings include:

i) Tissue necrosis and delayed wound healing caused by the irritant nature of the compound [Colman (1962), Radden (1962), Summers and Matz (1976)].

ii) Some of its constituents are toxic, e.g., tannic acid - direct absorption leads to liver necrosis [Smith (1970)]. Asbestos
fibres are known to cause mesothelioma - the greater danger here is to the dentist/assistant who mixes the preparation rather than the patient [Dyer (1967)].

iii) The taste of zinc oxide/eugenol is unpleasant.

iv) There is the need for a review appointment to remove the zinc oxide pack.

v) After pack removal, an open cavity remains which traps food debris and encourages the growth of colonising bacteria. Regular syringing with warm salt water is required to maintain oral hygiene [Summers and Matz (1976)].

Pharmacologically, eugenol is regarded as an irritant, rubefacient and mild analgesic. When packed into extraction wounds, it prevents clot formation, initiates inflammation and retards healing. Summers and Matz (1976) considered that eugenol accentuates the inflammation already present in an alveolar osteitis socket and that pain relief is brought about by the secondary destruction of nerve endings.

On the credit side, zinc oxide/eugenol reduces the number of organisms present in the sockets [MacGregor (1970)]. This is consistent with in vitro findings that zinc oxide/eugenol is bactericidal [Turkheim (1955) and McKnight (1967)]. Although zinc oxide/eugenol is caustic and delays healing, it does not prevent granulation of the socket [Summers and Matz (1976)]. Treatment is consistently effective clinically, in that the pain of alveolar osteitis is relieved and wound healing is achieved. Healing takes between 2 to 3 weeks after initial socket dressing. Another advantage of zinc oxide/eugenol is that the set dressing forms an effective barrier against the ingress of food and debris, thus maintaining a clean wound.

MacGregor (1967) surveyed British dental practitioners and found zinc oxide/eugenol to be the most popular. Despite the drawbacks of zinc
oxide/eugenol as a socket dressing, it has become the benchmark against which other treatments are judged. This is probably due to its long established use and resultant familiarity as much as its clinical effectiveness. MacGregor (1970), whilst acknowledging the shortcomings of zinc oxide/eugenol, could find no alternative and no ethical reason for not using it.

There are perhaps as many methods employed in using zinc oxide/eugenol dressings as there are commercial brands available. The following is a list of some traditional techniques:

i) Ward's 'WondrPak'. Carefully remove debris (by syringing with physiologic saline) and fill the affected socket with a measure of the 'WondrPak' liquid. Remove this liquid after 10 minutes. Mix a small quantity of the powder and liquid to a thin paste and apply onto a strip of ribbon gauze. Place this gauze lightly into the socket so that the dressing is flush with the surface. Leave in situ for between 3 - 7 days. After 7 days, replace/remove the dressing as required.

ii) Howe's Method (1974). The socket should be irrigated with warm, normal saline and all necrotic blood clot removed. Sharp bony spurs should be excised. A loose dressing of zinc oxide/eugenol on cotton wool is tucked into the socket. It must not be packed tightly or it will be difficult to remove. An appointment for pack removal is made in three days time.

iii) United Dental Hospital of Sydney. The socket is irrigated with sterile, physiologic saline to remove debris. A suitable length of 1 cm. ribbon gauze is soaked in Dentalone (chlorbutol compound BP 33%, oil of cloves 55%, oil of cassia, methylsalicylate, cinnamic aldehyde QS), excess liquid is sponged off and the impregnated gauze is placed in the socket under biting pressure for 10 minutes. This procedure alone is responsible for the pain relief (either
total or partial - in some cases). The 'WondrPak' paste is mixed to a light consistency and a suitable piece of sterile ribbon gauze is incorporated to add body. This dressing is lightly packed into the socket where excess zinc oxide/eugenol is removed and the pack left in situ for 3 - 7 days. At recall, the pack is removed, socket irrigated and either:

i) a piece of iodoform gauze is placed for two days (after which it is removed), or

ii) an irrigating syringe is issued to enable the patient to wash out the socket of debris after meals.

Socket healing is by secondary intent, usually achieved within three weeks of presentation.

To the best of this author's knowledge, 'WondrPak' has been used in the United Dental Hospital of Sydney since its introduction in the 1930s. However, since 1986 'WondrPak' has been discontinued because of its asbestos fibre content. Zinc oxide/eugenol dressings are now made using sedative (restorative) materials such as 'Kalsinol' and 'Kalsogen'.

**Conclusion:**

Despite zinc oxide/eugenol's strong tendency to retard wound healing, it is still regarded as homoeopathic. It is second only to local anaesthesia in its ability to obtund the pain of alveolar osteitis. It still has a place in the treatment of alveolar osteitis, although therapy must be strictly monitored so that the chemical cauterity associated with its use is not needlessly prolonged.

**4.5.2. Alvogyl.**

Alvogyl is a proprietary compound first marketed in the 1970s.

Its formula is:
tri-iodomethane 15.8 g
butyl paraminobenzoate 27.7 g
volatile oil of peppermint 9.0 g
eugenol 13.7 g
excipient to 100.0 g (Septodont, Paris).

Septodont (the manufacturer) claims that Alvogyl relieves post-extraction pain, including the pain of alveolar osteitis and provides a clean environment in which normal healing is allowed to proceed. It is promoted as a preventative (for use immediately after extraction), as well as an effective treatment for established alveolar osteitis. Alvogyl is particularly popular in Europe and Australia, although this may reflect the marketing skills of Septodont, rather than its clinical efficacy. The lack of any competition (with the demise of 'WondrPak') in the form of a pre-packaged, commercially available medication for alveolar osteitis is probably the biggest factor in its popularity.

Application of Alvogyl is simple: after thorough debridement of the socket with normal saline, a measured quantity of the Alvogyl is lightly packed into the socket. In some cases, one application is sufficient to alleviate the symptoms of alveolar osteitis although two or more dressings are average therapy. Dressings should be changed weekly, more frequently if the pain proves to be intractable. In general, treatment should continue until granulation is well advanced and the patient pain free. After this time, an irrigation syringe is invaluable in keeping the granulating socket free of debris. Alvogyl is non-resorbable and if left in situ (as recommended by the manufacturer), will eventually wash out.

Pain relief is provided by the eugenol and peppermint oil, while butyl paraminobenzoate and tri-iodomethane (iodoform) are antiseptic in action.
Summers and Matz (1976) studied the effects of socket implantation of Alvogyl and zinc oxide/eugenol compared with untreated (control) sockets in six mongrel dogs. They found that Alvogyl and zinc oxide/eugenol prevented blood clot formation, caused severe inflammation and retarded healing as a result. Further, the presence of bracken fern fibres (Cibatum barometz) in the Alvogyl formulation elicited a foreign body reaction - these fibres were incorporated into the healing scar tissue.

Photo 2. 'Alvogyl' in situ following 'Dentalone' therapy (38 socket).

Syrjanen and Syrjanen (1979) undertook a study of Alvogyl in extraction sockets in humans. Biopsies of the treated sockets were taken at one and two week intervals postoperatively. Eight healthy volunteers underwent two extractions simultaneously. One socket was
packed with Alvogyl and the other left untreated as a control. As expected, histological examination of the control sockets showed the well defined sequence of normal socket healing. In the test sockets, signs of delayed wound healing were encountered, i.e., acute inflammatory infiltrate, persistent granulation tissue, failure of connective tissue scar formation and foreign body reaction. An unexpected finding was that, whilst Alvogyl gave immediate pain relief; pain reappeared after one week in all patients. This pain was thought to be a result of the cessation of butyl paraminobenzoate activity. Syrjanen and Syrjanen concluded that Alvogyl as a prophylactic and cure cannot be recommended.

In this author's experience, Alvogyl is clinically effective in reducing the pain of alveolar osteitis, although it is not as good an anodyne as zinc oxide/eugenol. The advantage is that one application is usually sufficient to reduce pain to a bearable level. Unless accidentally avulsed, enough paste remains in situ to obtund the socket over a period of a week. Granulation time and socket epithelialisation appears not as delayed as with zinc oxide/eugenol.

Conclusion:
Alvogyl, like zinc oxide/eugenol is far from ideal. That it is effective clinically, as judged by the amount of pain relief, there is no doubt. However, its use as a prophylactic agent (i.e., as an immediate post-extraction socket implant) is questionable; it retards rather than aids wound healing and in approximately 97% of cases, the socket is better off left untreated. Although its use in established cases of alveolar osteitis is empirical with very little scientific basis, its proven performance justifies its continued use until a more suitable alternative is found.

Fry and Goldman (1958) introduced Biosone G.A. dental paste as a panacea for alveolar osteitis. Its formulation is:

- active isomers of glycyrrhetinic acid 1.0 %
- cinchocaine 2.0 %
- amethocaine 2.0 %
- acetylsalicylic acid 3.3 %
- neomycin sulphate 0.5 %
- polythene base excipient to 100.0 %

Glycyrrhetinic acid is an anti-inflammatory agent and relieves pain through prostaglandin release inhibition. Cinchocaine and amethocaine are topical local anaesthetic preparations. Salicylic acid (aspirin) is a well known NSAID (Non-steroidal anti-inflammatory drug) with proven analgesic and anti-pyretic effects. Neomycin sulphate is a topical antibiotic particularly active against gram negative bacteria, especially streptococci [MacGregor and Hutchinson (1975)]. Neomycin is an aminoglycoside, being of the streptomycin group. In high doses, it may cause VIII cranial nerve damage and impair hearing and balance. Its use as a topical antibiotic has since been discouraged. Its vehicle is a polythene base, with special properties of plasticity and resistance to wash out by oral fluids.

Fry and Goldman (1958) reported that a "blind investigation" was conducted over a six months period, where Biosone proved its clinical efficacy. Details of this investigation were not published, which cast serious doubts on the authors' claims.

Application is simple. The socket is syringed with sterile saline to remove any debris, dried with sterile gauze and a suitable amount of paste is carefully inserted under finger pressure. Paste retention is good, sufficient to occlude the socket for 48 hours. Pain relief has been
reported to occur within an hour of application.
Although not too well known in Australia, Biosone was popular in the United Kingdom, being second only to zinc oxide/eugenol in dentist preference [MacGregor (1967)]. It was used in the United Dental Hospital of Sydney during the 1960s, but was discontinued due to its poor analgesic properties [Assoc. Professor N.H.H. Smith (private communication 1988)].

Reservations about the use of Biosone include the risk of allergy and sensitisation to aspirin, cinchocaine, amethocaine and neomycin sulphate. Also, the wisdom of using antibiotics and anti-inflammatory drugs locally in an open healing wound has been questioned by Schwarz (1958).

Benigni and Franco (1958) confirmed the reported antimicrobial activity of glycyrrheticin acid. Colman (1962) tested Biosone for its antimicrobial activity and tissue compatibility in a culture of chick chorio-allantoic membrane. He found antimicrobial activity to a number of stock bacterial strains, including staph. aureus and klebsiella. On the basis of the contact reaction with the chorio-allantoic membrane of the chick embryo; tissue tolerance was judged to be good.

Further, the claimed anti-inflammatory action of glycyrrheticin acid was verified by Finney and Somers (1958). However, a side effect of this anti-inflammatory action was that it reduced the amount of granulation tissue formed under experimental conditions. This tendency would result in delayed wound healing [Finney and Tarnoky (1960)].

**Conclusion:**
From the limited number of reports on Biosone, it appears that it offered no advantage over zinc oxide/eugenol or Alvogyl in terms of pain relief. In the considered opinion of Assoc. Professor N.H.H. Smith
(1988) Biosone was clinically inferior. It is no longer available in Australia.

4.6. Antiseptic Dressings.

4.6.1. Iodoform gauze (3% iodoform in vaseline on ribbon gauze).

Iodoform is a medicament of some antiquity. It was used in medicine during the nineteenth century as an internal and external antiseptic dressing [Cottle (1879)]. Despite its age, iodine is still considered to be one of the best antiseptics available [Goodman and Gilman (1980)]. Iodoform has no analgesic properties; its use in alveolar osteitis management is to obtund the healing socket and keep it aseptic. Placement of iodoform usually follows treatment with an anodyne dressing such as Dentalone for 10 minutes or zinc oxide/eugenol for 5 to 7 days.

The rationale is to provide an infection free environment in which healing takes place uneventfully. Colman (1962) showed that iodoform is active against a selection of oral commensals including staph. aureus and candida albicans. Allergy to iodine or petroleum products are the only contraindications to its use. Iodine is otherwise well tolerated by the body's tissues [Goodman and Gilman (1980)].

Iodoform on vaseline gauze provides effective antisepsis for only 2-3 days. After this time the iodoform fouls and needs to be changed in order to avoid sepsis of the socket. This loss of antiseptic activity is linked to the amount of iodoform lost through saliva washout following placement. With the loss of iodoform from the ribbon gauze, the meshwork begins to act as a sieve trapping plaque and food debris, thereby predisposing to bacterial contamination after three days in situ.
Conclusion:
In this author's experience, iodoform is well tolerated and clinically effective in providing a "clean" environment in which granulation is promoted. The disadvantages are the less than pleasant taste of the iodoform and the frequency of recall appointments for dressing renewals.

4.6.2. Bismuth Iodoform Paraffin Paste (BIPP)
Morison (1916) is generally credited with the creation of BIPP. He combined bismuth subnitrate and iodoform with liquid paraffin for use as an antiseptic dressing in open wounds. The antibacterial action of BIPP was studied by Chambers and Goldsmith (1917) and Fleming (1919-20); both were able to culture bacteria from open wounds previously treated with BIPP. Chambers and Goldsmith attributed the clinical effectiveness of BIPP to bacteriostasis, whilst Fleming thought correct surgical technique was the critical factor, rather than any antisepsis from the BIPP. Saint (1937) reported on its effectiveness in the treatment of acute osteitis, claiming a bacteriostatic action of the iodine component. Recovery of iodine from the urine of treated patients over a period of 3-4 weeks was seen as an indication of the continuous chemical action of BIPP. Of course, urinary iodine more accurately indicates systemic absorption from the site of dressing. Chambers and Goldsmith (1917) found that iodoform has decreased antibacterial activity when suspended in paraffin compared with an ether combination. Colman (1962) confirmed this decreased potency of iodoform in BIPP compared with iodoform alone. Also, Colman found that the bismuth subnitrate was devoid of any antibacterial activity. Bismuth subnitrate is an astringent (dries up weeping wounds).
Radden (1962) found that BIPP is well tolerated by the tissues, some evidence of delayed wound healing was found in BIPP treated sockets, but compared with zinc oxide/eugenol, these were minimal. Treatment of alveolar osteitis with BIPP is the same as for iodoform gauze. BIPP on ribbon gauze is preferred, as BIPP alone lacks body to resist saliva washout. Removal/renewal after 2-3 days is advised, to avoid fouling of the socket. Use of iodoform gauze and BIPP as an immediate socket implant after third molar surgery has fallen from favour, especially since no significant advantage (in doing so) has been documented.

Conclusion:
BIPP appears clinically to be no better or worse than iodoform gauze. Both are petroleum based and carry a risk of myospherulosis with use. BIPP achieved its zenith in popularity in the Great War of 1914-18, the pre-antibiotic era. Its use has waned so much that most hospitals now do not stock it.

4.7. Antifibrinolytic Agents.

4.7.1. Apernyl.
Apernyl is an alveolar socket implant in the form of a cone, made up as follows:

- acetylsalicylic acid 32 mg
- propyl-hydroxy-benzoate 3 mg
- unknown tablet mass 20 mg

Apernyl is manufactured by Bayer Pharmaceuticals (West Germany) specifically for the treatment of post-extraction pain, including alveolar osteitis. It is unavailable in Australia. Apernyl has been well
received in Europe following favourable initial reports by Menzel (1969) and Neuner and Schegg (1969). Menzel found that all patients treated with Apernyl were pain free 30 minutes after placement. However, a slight burning sensation of the gingiva where the cones had extruded from their sockets, was also reported. This was thought to be due to the aspirin. Neuner and Schegg treated 204 cases of alveolar osteitis with Apernyl. Eighty two per cent of patients required only one treatment to be pain free. Their technique included socket curettage to ensure fresh bleeding prior to apernyl placement. Neuner and Schegg also placed Apernyl into fresh sockets after the removal of 136 impacted wisdom teeth. The trial was not double blind and a low alveolar osteitis incidence of 1.5% (2/136) was reported.

Schulte and Worner (1970) demonstrated Apernyl's high antifibrinolytic activity on full blood in vitro. Further to this, Schulte (1971) identified propyl-hydroxy-benzoate as the major antifibrinolytic component of Apernyl. He postulated the inhibition as being on two levels:

i) Inhibition of the proactivator and activator activity.

ii) Inhibition of the plasmin activity.

Birn (1972) sampled 15 alveolar osteitis sockets, added various concentrations of either Apernyl or acetylsalicylic acid and tested these combinations for antifibrinolytic activity using the fibrin plate method. He found that plasmin activity was completely inhibited whilst activator inhibition was nearly complete with Apernyl. The acetylsalicylic acid showed weak inhibition. Birn confirmed that propyl-hydroxy-benzoate was the major inhibitor.

The effects of acetylsalicylic acid (aspirin) seem paradoxical. Carroll and Melfi (1972) showed that acetylsalicylic acid in contact with bone causes an increased and prolonged inflammatory response which accelerates the liberation of tissue activators and hence, plasmin. This
elevation in plasmin levels increases the likelihood of fibrinolysis. This finding was substantiated by Rud (1972) who found the use of Apernyl as an immediate socket implant caused serious wound inflammation which often resulted in alveolar osteitis.

Keskitalo and Persson (1973), using alleviation of pain as the criterion, compared the effectiveness of Apernyl against Ward's 'WondrPak' dressing. In a sample of 51 alveolar osteitis cases, they found that Apernyl achieved analgesia significantly faster (3-4 days) than 'WondrPak'. However 'aspirin burn' was noted around the wound margins of 33% of the Apernyl group (8/26 patients).

Kjellman (1973) in a randomised, placebo controlled study found a significant reduction in alveolar osteitis incidence in the Apernyl group (1/50) compared with the placebo group (4/50). He was of the opinion that using only one Apernyl cone per socket circumvented the gingival burning seen when multiple cones were used.

Vedtofte, Ritzau and Donatsky (1974), following Birn's (1972) suggestion that acetylsalicylic acid be dropped from the Apernyl formulation, tested the efficacy of cones which contained 3 mg of the propylic ester of p-hydroxy-benzoic acid (PEHB) in a 52 mg tablet against cones containing placebo tablet mass only. Three cones of either PEBH or placebo were implanted immediately post-extraction in 144 consecutive third molar patients. The authors concluded that PEBH, under certain conditions, in vivo, may have an antifibrinolytic effect that may prevent the breakdown of the blood clot. There was a significant difference in alveolar osteitis incidence in the male groups where the PEBH group had 6% compared with 29% in the placebo group. No statistical difference was demonstrated in the females.

Schatz, Fiore-Donno and Henning (1987) conducted a double blind, randomised clinical trial on 300 extraction cases with no preselection
of extraction sites. The sample was divided into three numerically identical groups:

i) Group A - gelatin sponge only.
ii) Group B - gelatin sponge, 20% by weight of Solcoseryl (a protein free, standardised diasylate of calf blood containing a mixture of glycolipids) and 0.0084% by weight of p-hydroxy-benzoic acid propyl ester (PEHB).
iii) Group C - gelatin sponge, 20% Solcoseryl and 8.4% PEHB.

The gelatin sponge (15mm X 10mm X 10mm) was adapted to fill the volume of the individual alveolus, then placed in situ prior to wound closure. All patients were screened to exclude haemostatic problems prior antibiotic therapy and possible allergy to the test materials. The authors concluded that the addition of PEHB was insufficient to prevent the development of alveolar osteitis. This supported the earlier findings of Vedtofte, Ritzau and Donatsky (1974) and Ritzau and Swangsilpa (1977). As part of their trial, Schatz, Fiore-Donno and Henning (1987) assayed blood from the alveoli of treated sockets but could find no antifibrinolytic activity. They concluded that PEHB was not antifibrinolytic in vivo.

Although PEHB has been clearly shown to be antifibrinolytic in vitro, the predicted in vivo results have not been forthcoming. From this we may reasonably conclude:

i) Fibrinolysis is not the only mechanism of note in the development of alveolar osteitis.
ii) Fibrinolysis may, in fact, be only a by-product and not the aetiology of alveolar osteitis.
iii) In vivo factors, as yet unrecognised, may inhibit
the antifibrinolytic activity of PEHB.

**Conclusion:**
Allowing for individual variations between authors as to the diagnostic criteria used in determining the incidence of alveolar osteitis, it appears that PEHB, or more specifically, Apennyl is not significantly better than zinc oxide/eugenol tamponade in the treatment of alveolar osteitis. Its use as a prophylactic agent is difficult to justify.

**4.7.2. Tranexamic Acid (Trans-4 amino-methyl-cyclohexane acid) - AMCA.**
AMCA is a potent antifibrinolytic drug which has been used in general surgery for some time [Kjellman (1974)]. Its antifibrinolytic activity is brought about by the inhibition of plasminogen activation. Gersel-Pedersen (1979) tested the efficacy of AMCA (applied into third molar sockets) against a placebo in a double blind fashion. One hundred and twenty healthy patients who required bilateral removal of impacted mandibular third molars made up the sample. Both sides were operated on simultaneously and AMCA (160 mg/site) or placebo was applied in each socket prior to closure. The incidence of alveolar osteitis was 7.5% with AMCA and 5.0% on the placebo side. The author concluded that local inhibition of plasminogen activation with AMCA is insufficient to prevent the development of alveolar osteitis. Alveolar blood samples showed concentrations of 743 µg AMCA/g clot after 24 hours and 9 µg AMCA/g clot after 3 days per 4 AMCA cones (average per socket). Theoretically, the concentrations of AMCA should have been sufficient to cause some inhibition of plasminogen production by the tissue factors so pertinent to Birn's (1973) fibrinolysis hypothesis. The fact that no such result was seen throws some doubt on the role of
tissue factors in localised fibrinolysis. Indeed, this finding embarrasses the hypothesis that tissue factors are central to the fibrinolysis seen in alveolar osteitis.

Conclusion:
The value of antifibrinolytic (plasminogen inhibition) medication in the prevention of alveolar osteitis is seriously questioned following the findings of Gersel-Pedersen (1979). To be fair, more clinical work needs to be done to fully evaluate the role of these types of antifibrinolytics in alveolar osteitis management. The state of knowledge to date sees AMCA as experimental at best.

4.8. Surgical Intervention.
Podolin (1936) was one of the first to advocate surgical curettage as an effective means of treating a socket with alveolar osteitis. The rationale is to physically clean away the necrotic blood clot and bone tissue so that a fresh wound is created, giving the socket every chance to heal normally. It was thought that this necrotic layer of bone cells was the cause of the intense pain so characteristic of alveolar osteitis and its removal would alleviate the pain as well as speed up the healing process.

Turner (1982) in a pilot study of the surgical approach (mucoperiosteal flap reflection and debridement), treated 12 cases of alveolar osteitis. Eleven cases resolved uneventfully after intervention giving the clinical impression of rapid pain reduction and healing. One case developed localised suppuration following surgery. As a consequence, a second operation was performed, which proved successful. Turner felt that the success of the surgical method was due to the removal of traumatised inflamed (necrotic) bone. This bone has been credited with
the release of kinins which are responsible for the intense pain of alveolar osteitis [Birn (1972)]. Also, removal of necrotic debris allowed the initiation and stabilisation of a fresh blood clot, which led to uneventful healing.

Earlier, Osterloh (1945) had advocated the surgical removal of all teeth showing radiographic evidence of condensing osteitis as a preventative measure. He was mistaken in the belief that condensing osteitis invariably gave rise to alveolar osteitis after the extraction of the involved tooth. There is no evidence to show that teeth associated with condensing osteitis are in any way more prone to alveolar osteitis.

As a treatment alternative to medication, there are a number of considerations to keep in mind:

i) Surgery involves bone removal, either with a bur or chisel, and by definition, is inherently more traumatic than a simple forceps extraction. The concept that bone treated in this way is less traumatised and would release few, if any, pain causing kinins is paradoxical to say the least.

ii) A surgically treated socket is subjected to the full range of postoperative morbidity (infection, pain, swelling, trismus, alveolar osteitis) that a medicated socket is otherwise free of.

iii) The use of surgery as a preventative measure in the first instance, for a condition with an overall incidence of 3% is both excessive and unnecessary.

iv) Surgery tends to reduce the ultimate height of the alveolar ridge by virtue of its greater bone removal and subsequent resorption compared with forceps extraction. Thus, due consideration must be given to other alternatives if preservation of alveolar ridge height is of paramount importance.

v) Surgery as a form of treatment, compared with
other methods, is costly and inefficient.

Conclusion:
From the patients' point of view, surgery as an alternative to medication looks decidedly unappealing. Surgery has not been shown to be any more effective than conventional medication and there is the theoretical risk that the procedure may spread infection from the necrotic debris into surrounding healthy alveolus, leading to an osteomyelitis. All things considered, surgery should be reserved as a last resort should conventional therapy fail.

4.9. Antiseptic Mouthwashes.

4.9.1. Aqueous Chlorhexidine Digluconate 0.2%.
For the qualitative and quantitative reduction of oral microflora, chlorhexidine digluconate mouthwash has been shown to be particularly effective. The rationale for using an antiseptic mouthwash pre or postoperatively lies in the belief that oral commensals are significant in the development of alveolar osteitis.
Chlorhexidine is an antiseptic with proven activity against a wide spectrum of gram positive and gram negative bacteria and fungi [Loe and Schiott (1970) and Olsen (1975)]. Also, it has significant plaque inhibiting qualities in vivo and prevents superficial gingivitis [Gjermo, Baastad and Rolla (1970) and Yanover et al. (1988)].
Tjernberg (1979) sampled 60 patients who underwent the surgical removal of mandibular third molars. The test group had their teeth scaled and polished five days preoperatively and then used a 0.2% aqueous chlorhexidine digluconate mouthwash twice daily (b.d.) before the operation and during the first postoperative week. One patient in
the test group developed alveolar osteitis compared with five in the control group. Tjernberg concluded that chlorhexidine mouthwash pre- and/or postoperatively may be a simple and effective way of reducing the incidence of alveolar osteitis. Earlier, Altonen et al. (1976) had shown that chlorhexidine gluconate 0.2% (w/v) was effective in removing potentially pathogenic microbes from saliva. MacFarlane, Ferguson and Mulgrew (1984) showed the same was also true for the gingival sulcus. On this basis, Martin and Nind (1987) clinically trialed the use of chlorhexidine gluconate for the preoperative disinfection of apicectomy sites. They applied chlorhexidine gluconate 0.2% (w/v) solution to an area approximately 0.5 cm square at the incision site preoperatively. Application was for one minute. The control sites were swabbed with cotton wool only. Both areas were sampled bacteriologically preoperatively and 7-10 days postoperatively. This latter sampling was to test the finding by Bonesvoll, Lokken and Rolla (1974) that chlorhexidine persists in the oral mucosa for days after topical application (a characteristic known as Sustantivity).

Chlorhexidine gluconate was found by Martin and Nind (1987) to reduce the recoverable flora from apicectomy sites by 94.4% immediately after application. After 7-10 days, the flora present was reduced by 78.3% compared with controls. Twenty four patients comprised the test group, a further 10 made up the controls. On this basis, the authors suggested that chlorhexidine gluconate would be a useful preoperative antiseptic prior to minor oral surgery.

MacGregor and Hart (1971) tested the efficacy of local application of chlorhexidine gluconate (0.5% in 70% alcohol) in the mandibular third molar region prior to surgery. In a study of 41 patients who underwent the simultaneous removal of bilateral, impacted mandibular third
molars; a randomly selected site was bathed in chlorhexidine gluconate (0.5%) for a period of 30 seconds preoperatively. The contralateral side was left untreated. A reduction in the number of surface bacteria to negligible levels was noted on the side treated with chlorhexidine. This confirmed the earlier findings of Cawson and Curzon (1959) and Blake and Forman (1967). However, when the sockets were sampled immediately postoperatively, no significant differences in bacterial count were seen between the treated and non-treated sites. Obviously, contamination of the treated areas by saliva and tongue movements re-established the status quo.

MacGregor and Hart (1971) concluded that chlorhexidine used at the operation site only, was of no value as a preoperative antiseptic in oral surgery; sockets became as contaminated as untreated sites during the course of the procedure. They suggested that a pan-oral approach (a mouth rinse) may be more effective as a preoperative prophylactic measure. Yanover et al. (1988) conducted a six months trial of a once daily mouth rinse with 10 mL chlorhexidine 0.2% (w/v) solution by the institutionalised elderly. His results showed that chlorhexidine was well accepted and effective in significantly reducing plaque and gingivitis scores in the test population. Yanover et al. also noted that some species, e.g., S. mutans, may be suppressed for at least six weeks after the discontinuance of the chlorhexidine mouth rinse.

Field et al. (1988) demonstrated that the preoperative irrigation of the gingival crevice and mouth rinsing with 0.2% (w/v) chlorhexidine gluconate significantly reduced the incidence of alveolar osteitis. Larsen (1991) found a significant 60% reduction in the incidence of alveolar osteitis compared with placebo. The study (278 bilaterally impacted mandibular third molars in 139 patients) was randomised, double blind and placebo controlled. Chlorhexidine gluconate 0.12% was
used as a mouth rinse twice daily, for one week, both preoperatively and postoperatively. Schiott et al. (1970) established the safety of 0.2% chlorhexidine mouth rinse as a daily routine. A group of 61 dental students used chlorhexidine daily for a period of two years. Regular blood, urine, liver-function and kidney-function tests were performed to detect any abnormalities. No systemic or local side effects of a serious nature could be attributed to the use of chlorhexidine. However, minor local side effects such as loss of taste, burning sensation of the oral mucosa, dryness of the mouth, teeth and tongue discoloration were noted. These effects were reversible: discontinuance returned the patient to normal.

In respect of these side effects, Hepso and Bjornland Tard Skoglund (1988) showed that the use of a 0.1% (w/v) chlorhexidine solution minimised the incidence and intensity of such effects without significantly reducing its clinical effectiveness.

Conclusion:
Chlorhexidine gluconate (0.2% or 0.1%) has been shown to be safe and well tolerated as a mouth rinse. Clinical efficacy against alveolar osteitis has been demonstrated. Its use is non-invasive and results in a reduction of the oral microflora qualitatively and quantitatively (a desirable postoperative outcome). Chlorhexidine pre- and postoperatively is recommended for those undergoing third molar surgery or those with a predisposition for alveolar osteitis.

4.10. Wound Debridement with Saline prior to Closure.
Saline lavage (flushing of the intra-oral wound with sterile, physiologic saline) physically removes debris and minimises bacterial
contamination. The rationale for this strategy is based on the implication that bacteria and alveolar debris (tissue factor release - initiation of fibrinolysis) are prominent in alveolar osteitis. Previous workers [Bhaskar, Cutright and Gross (1969), Gross *et al.* (1970) and Gross *et al.* (1971)], had shown that the use of water lavage in experimental wounds (in rats) significantly reduced the incidence of infection. Following on from their earlier findings, Gross *et al.* (1971) studied the possibility of inducing a postoperative bacteraemia after the use of water lavage delivered at 70 lbs/sq in. for a period of 30 seconds in 75 rats. They concluded that the probability of such a bacteraemia occurring was negligible.

Wound lavage is an extension of good surgical technique and ensures thorough debridement prior to closure. It has long been accepted that the cleaner the wound, the greater are the chances of uneventful healing.

The use of normal, sterile saline for wound lavage is not a recent innovation. Favourable reports have been documented as early as the late 1930's and early 1940's. Mason (1937), Estes (1940) and Koch (1941) all encouraged the use of saline irrigation as a means of wound debridement. Petersen (1945) found that wound healing and the incidence of postoperative infection was directly proportional to the amount of isotonic, sterile saline used for irrigation. The greater the volume of saline used, the less the incidence of postoperative complications.

Sweet, Butler and Drager (1976) compared the effectiveness of a mechanical irrigation device to the conventional hand syringe. The volume of saline used was 350 mL in all cases. One case of alveolar osteitis (of 103 surgical mandibular third molar sites) occurred in the hand syringe group. No cases of alveolar osteitis occurred in the
mechanical device group. Both types of irrigation were used simultaneously on opposite sides at the time of the operation. An overall alveolar osteitis incidence of 0.49% (of 206 cases) compares very favourably to the 25-30% incidence which is often quoted for the mandibular third molar site [Krogh (1937) and Adkisson and Harris (1956)]. Sweet, Butler and Drager (1976) concluded that there was no significant differences between the use of a mechanical device and a hand syringe. They felt that the amount of saline used was the crucial factor; 350 mL ensured total debridement of the surgical wound. Visual judgement was the only gauge used in the assessment of wound 'cleanliness' prior to closure.

Subsequent to their 1976 study, Butler and Sweet (1977) tested the efficacy of smaller volumes of saline (25 mL to 175 mL) with respect to the incidence of alveolar osteitis. Their findings were:

i) An overall incidence of 8.3% (32/422 sites - mandibular third molars).

ii) 25 mL gave an incidence of 10.9% (23/211 sites).

iii) 175 mL gave an incidence of 5.7% (12/211 sites).

Butler and Sweet (1977) concluded that a minimum of 350 mL saline is needed for maximum reduction in the incidence of alveolar osteitis.

**Conclusion:**
Irrigation of surgical wounds with copious quantities of sterile, physiological saline is a logical extension of proper wound toilet and should be encouraged.

4.11. Dextranomer Granules.
Dextranomer granules are dehydrated, porous, spherical beads (0.1 mm to 0.3 mm in diameter) composed of a highly hydrophilic dextran
polymer capable of absorbing up to five times its own weight in water. It is used in general medicine as a wound cleanser where bacteria, tissue exudates and toxins are removed from the wound by hydration of the dextran beads. Dextranomer granules (Debrisan) have been used successfully in the treatment of burns [Paavolainen and Sundell (1976)], penile ulcers [Lassus, Karvonen and Juvakoski (1977)], leg ulcers [Floden and Wikstrom (1978), Groenewald (1980)], septic wounds [Goode, Glazer and Ellis (1979)] and decubitus ulcers [McClemont, Shand and Ramsay (1979)].

Matthews (1982) evaluated the efficacy of dextranomer granules in the treatment of established alveolar osteitis. He treated 58 sockets with dextranomer granules and compared these with 24 sockets treated with zinc oxide/eugenol (control group). For mandibular sockets, the beads were dispensed directly using a spoon curette, then covered with a generous amount of Orabase gel. Maxillary sockets required the addition of glycerine as a base to counter the effects of gravity and were similarly covered with Orabase. Matthews conceded that the addition of glycerine reduced the absorbency of the granules, but was assured by the manufacturer that this reduction was in the order of 20%. Sequential postoperative visits were arranged two days apart, the patients being discharged only when pain-free and with the clinical impression of a well granulating socket.

Matthews reported that, compared with zinc oxide/eugenol, dextranomer granules proved very effective. Duration of treatment to achieve a pain-free, well granulating socket averaged three dressings (4-5 days after initial treatment); zinc oxide/eugenol required more dressings to produce the same result. Pain control during treatment was judged to be similar in both materials. Significantly, the clinical impression of dextranomer treated sockets was one of healing
promoted, rather than the characteristic delayed healing of zinc oxide/eugenol.

Conclusion:
Although Matthew's (1982) preliminary report suggested a promising future for dextranomer granules as an alveolar osteitis treatment option, there is a question as to which agent was responsible for promoting the socket healing observed. Was it the dextranomer granules, Orabase or a combination of both? Both materials are listed as inert and non-irritating to tissues. Would a similar result or better have been achieved if carmellose sodium (Orabase) was the sole dressing used? In view of this, more clinical studies need to be done to fully evaluate dextranomer granules per se.
There are two obvious disadvantages associated with the use of dextranomer granules in the treatment of alveolar osteitis:

i) Dextranomer granules are not an effective anodyne. Pain relief was not obtained until 3 to 5 days after initial treatment. As pain relief is the primary objective of anyone suffering from alveolar osteitis, dextranomer fails badly from the patient's point of view.

ii) Dextranomer granules or beads (0.1 mm to 0.3 mm in diameter) may be inadvertently inhaled during application. Mixing the beads with glycerine does lessen the chances of aspiration, but conceivably, particles will work loose from the socket dressing during the course of treatment and present a real inhalation risk. This risk is heightened by the fact that the majority of alveolar osteitis cases occur in the posterior mandible, an area where the chances of aspiration are greatest.
Fibrin sealants composed of highly concentrated human fibrinogen, factor XIII, albumin, insoluble globulin, thrombin, calcium chloride and a fibrinolysis inhibitor (Aprotinin) have been available to the medical profession for some years. It has been used with success in face to face sealing of tissues, wound healing and establishing haemostasis [Seelich and Redl (1979), Wepner, Bukal and Beck-Mannageta (1979), Skjoldborg (1982) and Petersen (1985)]. Moller and Petersen (1988) tested the efficacy of a two part fibrin sealant (Tisseel) as a wound cover after mandibular third molar surgery. The sample was 122 mandibular third molar sockets: the test groups were fibrin sealant alone, fibrin sealant with phenoxyethyl penicillin and no treatment (control). The fibrin sealant was applied as soon as possible after tooth removal. The authors found that fibrin sealant with or without penicillin was no better than leaving the sockets to heal unaided. There was no significant reduction in postoperative morbidity (including alveolar osteitis) compared with the control group.

Conclusion:
Although fibrin sealants have been shown to be of little value as a prophylactic against alveolar osteitis, they may be effective as a treatment for established cases. Used as an obtundent dressing, its neutral effect on wound healing [Staindl (1979) and Staindl, Galvan and Macher (1981)] would, theoretically, result in the promotion of granulation within affected sockets.
Chapter 5

Alveolar Osteitis and Third Molar Surgery.

Most authors agree that the mandibular third molar has the highest incidence of alveolar osteitis [Hansen (1960), MacGregor (1968), Birn (1973), Tjernberg (1979), Heasman and Jacobs (1984), Field et al. (1985)].

Alveolar osteitis in the mandibular third molar site has been documented from a low 0.5% [Belinfante et al. (1973)] to a high of 68.4% [Erickson, Waite and Wilkinson (1960)]. A possible explanation for this wide range may be found in the lack of uniform diagnostic criteria employed by the various authors. Birn (1973) reviewed the incidence of alveolar osteitis after the surgical removal of mandibular third molars and settled on a mean incidence of 20%. This is a more realistic figure, which nevertheless, reflects a 7:1 incidence ratio compared to any other socket site. The two most common explanations as to why the mandibular third molar site is the most affected by alveolar osteitis are:

i) Increased trauma.

ii) Increased risk of bacterial contamination.
5.1 Increased Trauma.

Zimmer (1929), Paine (1937), Krogh (1937), Gottlieb and Crawford (1943), Russell and Charlotte (1944), Erickson, Waite and Wilkinson (1960), Hansen (1960), MacGregor (1968), Lilly et al. (1974) and Bruce, Frederickson and Small (1980) are convinced that trauma is the principle cause of alveolar osteitis, and the increased trauma of mandibular third molar surgery is responsible for the high incidence at that site.

Krogh (1937) in a sample of 6,403 extractions found an incidence of 0.7% in extractions categorised as "easy" and 23.5% in those considered "difficult". Similar findings have been reported by Erickson, Waite and Wilkinson (1960): 1.5% (easy), 60.4% (difficult) and Hansen (1960): 2% (easy), 7.3% (surgical cases).

MacGregor (1968) in a series of 10,199 extractions showed a
discrepancy of 7% between the simple cases (3.3%) and the complicated (10.3%).

Alling and Kerr (1957) were able to induce alveolar osteitis in rhesus monkeys by traumatically injuring the alveolus with a metallic instrument. From this rather crude experiment they concluded that trauma is the cause of alveolar osteitis.

Others have shown the opposite: Archer (1939) in a series of 23,886 extractions, estimated that trauma was integral in 56% of cases. He reported no significant difference in alveolar osteitis incidence between the 'traumatised' and 'non-traumatised' groups. The averaged incidence was 0.9%. Swanson (1966), in a sample of 47 third molar extractions, found an incidence of 62.5% in cases of minimal trauma compared with 28.6% in cases of considerable trauma.

In more recent times, there have been consistent reports implicating trauma as a fundamental cause of alveolar osteitis. Lilly et al. (1974) in a series of 2,195 mandibular third molar extractions (359 non-surgical and 1,836 surgical), concluded that alveolar osteitis was more than twice as common in surgical extractions than in non-surgical (simple) extractions. Van Gool, Bosch and Boering (1977) compared 430 simple mandibular third molar extractions with 502 surgical mandibular third molar extractions and found an incidence of 2% in the simple group and 5% in the surgery group. This difference was attributed by the authors to the increased trauma inflicted by the surgery.

Osborn et al. (1985) evaluated the surgical and post surgical problems of 9,574 patients (16,127 mandibular third molars) of varying ages. They found the incidence of alveolar osteitis was five times greater following surgery for complete bony impactions than for erupted teeth, four times greater for partial bony impactions than for erupted teeth.
and twice as great for tissue impactions than for erupted teeth. Osborn et al. concluded that an increased incidence of alveolar osteitis may be anticipated as a result of soft tissue manipulation and bone surgery.

Sisk et al. (1986) reaffirmed the findings of Osborn et al. (1985). Sisk et al. compared the incidence of alveolar osteitis in 500 patients following mandibular third molar surgery by experienced faculty staff, with 208 patients similarly treated by inexperienced oral surgery residents. Their findings: staff 6.4% (54/843 extractions), residents 19.5% (70/359 extractions). Also, complete bony impactions gave the highest incidence of alveolar osteitis in both groups.

Birn (1973) considered trauma to be axiomatic to his fibrinolysis hypothesis - the more trauma, the more tissue activators released from the alveolar bone, the greater the chance of fibrinolysis. Cases where alveolar osteitis develops after an 'atraumatic' extraction are explained by the possibility that the patient may be more susceptible, at the time, by virtue of an elevated level of systemic fibrinolytic activity.

The consensus of opinion is that trauma is very significant to the development of alveolar osteitis. In respect of third molar surgery, it has been shown that surgical trauma is directly proportional to:

i) the technical difficulty of the impaction and

ii) the experience of the operator [Sisk et al. (1986)].

5.2 Bacterial Contamination.

The retromolar triangle is an area where food debris is channelled by the opposing actions of the cheek musculature and tongue. A wound near the retromolar triangle will naturally become a catchment of materia alba and bacteria. Given that an extraction socket is ideal culture medium for a plethora of oral commensals, it follows that the patient
is at a substantial risk of postoperative infection unless strict oral hygiene measures are adhered to.

Dental literature is replete with investigations supporting infection as the cause of alveolar osteitis. A sampling of authors include Schroff and Bartels (1929), Belding and Belding (1934), Paine (1937), Brown, Merrill and Allen (1970), Rozanis, Schofield and Warren (1977), Catellani (1979), Tjernberg (1979), Krekmanov and Hallander (1980) and Krekmanov (1981).

A number of infective pathways have been proposed:

i) Direct inoculation. Schroff and Bartels (1929) found fusobacteria and spirochaetes in the dental sockets of eight patients with alveolar osteitis. Archer (1939) found streptococci in 80% of the 226 cases of alveolar osteitis he investigated. Similarly, Brown, Merrill and Allen (1970) isolated streptococci, including the β-haemolytic group, from the sockets and saliva of patients suffering from alveolar osteitis. Meyer (1924) and Claflin (1936) were able to produce alveolar osteitis and delayed healing respectively, in dogs, by simply inoculating cultures of staphylococci and streptococci into fresh dental sockets.

ii) Bacterial synergism. Krekmanov and Hallander (1980) suggested the possibility of such symbiosis after failing in their quest to find a specific pathogen for alveolar osteitis. The concept is, presently, one of speculation with no experimental evidence to support it.

iii) Bacterial pyrogens. Catellani (1979) postulated that the pyrogens released by specific oral commensals stimulate fibrinolysis by promoting the conversion of proactivator to activator, thus initiating the formation of plasmin from plasminogen. Catellani reasoned that the ideal culture medium provided by a traumatised
alveolus would allow these bacteria, amongst others, to thrive. This proliferation (if unchecked) would precipitate fibrinolysis of the coagulum.

iv) Specific pathogen. Nitzan (1983) hypothesised that Treponema denticola, a spirochaetal anaerobe, is dominant in the genesis of alveolar osteitis. The basis for this hypothesis is the isolation of T. denticola (in abundance) from the dental sockets of patients suffering from alveolar osteitis. As well, T. denticola is known to be fibrinolytically active.

v) Alveolar osteitis is an extension of pre-existing infection, e.g., pericoronitis. Adkisson and Harris (1956), Kay (1965), Kay (1966), and Meyer (1971) showed that the incidence of alveolar osteitis increased when mandibular third molar surgery was performed whilst the patient was suffering concomitant pericoronitis. Nitzan et al. (1985) postulated a link between pericoronitis and alveolar osteitis. They found an anaerobic spirochaete (Treponema denticola) was dominant in both the pericoronitic microflora and alveolar osteitis socket. It was reasoned that after extraction of a third molar with concomitant pericoronitis, T. denticola becomes entrapped in the coagulum. As this is an ideal growth medium, T. denticola’s multiplication is unrestrained, resulting in fibrinolysis of the blood clot.

Others have also linked this bacterium to acute necrotising ulcerative gingivitis (ANUG) [Schroff and Bartels (1929), Box (1930), Birn (1970) and Nitzan et al. (1985)]. Because some cases of acute pericoronitis resemble localised ANUG, Nitzan et al. believed the causative microorganism of all three conditions (alveolar osteitis, ANUG and pericoronitis) to be T. denticola.

As yet, it is unclear whether T. denticola is an instigator of alveolar
osteitis or merely a saprophyte - an opportunistic, infective bacterium capitalising on an already broken-down socket.

5.3. Prophylactic Measures employed in Third Molar Surgery

5.3.1. Surgical Technique.
Van Gool, Bosch and Boering (1977) echoed the sentiments of many when they concluded: the least traumatic surgical technique gives the best result statistically in terms of alveolar osteitis incidence. They observed that postoperative complications (including alveolar osteitis) are less frequent when mucoperiosteal flap reflection, bone removal, elevator use and socket manipulation are avoided or minimised. Earlier, Lilly et al. (1974) noted an incidence of alveolar osteitis twice as common for surgical extractions of mandibular third molars as for simple extractions of the same. They concluded that alveolar osteitis incidence is directly proportional to the amount of trauma delivered during tooth removal.

Catellani (1979) advocated the use of the least traumatic means possible when extracting teeth in order to avoid alveolar osteitis. Osborn et al. (1985) stated that an increased incidence of alveolar osteitis is to be anticipated after tissue manipulation and bone surgery during mandibular third molar removal. Most authors recommend that, where possible, the following should either be avoided or minimised:

i) Mucoperiosteal flap reflection.

ii) Bone removal.

iii) Soft tissue manipulation.

5.3.2. Timing of the Procedure.

Ygge et al. (1969) reported high levels of fibrinolytic activity in women taking oral contraceptives. He showed that this fibrinolytic activity
reached a maximum at the middle of the tablet cycle and decreased to normal after one drug free week. Hedlin and Monkhouse (1971) confirmed the work by Ygge et al. (1969) and demonstrated that an increase in fibrinolytic activity usually starts within 24 hours of the first dose of oral contraceptive.

Gersel-Pedersen (1979), was one of the first to observe a connection between oral contraceptive medication and an increase in alveolar osteitis. Catellani (1979) surmised that oestrogens are indirect activators of fibrinolysis and contribute to the occurrence of alveolar osteitis because of this.

Investigations by Schulte and Goens (1967) found that plasminogen proactivators and activators in saliva increase and reach a peak just before, during and immediately after menstruation. Following this work, Nordenram and Grave (1983) found that not only was there an increased incidence of alveolar osteitis in women who were taking oral contraceptive medication at the time of the surgery, but also that there was a significant increase in alveolar osteitis incidence in women treated during menstruation. However, Gersel-Pedersen (1979) was unable to demonstrate any connection between surgical timing within the menstrual cycle and alveolar osteitis incidence.

While it is unrealistic to insist that women withdraw from oral contraceptives prior to, and for a short time after third molar surgery; it may be worthwhile considering in an individual with a history of alveolar osteitis who is keen to avoid a recurrence.

Al-Khateeb, El-Marsafi and Butler (1991) in a prospective study of 412 patients (642 impacted third molar surgery sites) found that the probability of developing alveolar osteitis is significantly greater if surgery is delayed until the tooth has partly erupted and been exposed to pericoronal inflammation. Further, an unerupted wisdom tooth socket
is conducive to primary closure compared with a tooth that is partly erupted. They advocated 'prophylactic' removal of unerupted wisdom teeth as a means of avoiding alveolar osteitis.

It seems logical that a primarily closed wound (where the clot is afforded physical protection) is less likely to develop alveolar osteitis than one left to heal by secondary intent.

5.3.3. Age Considerations.

Age, in relation to mandibular third molar surgery, is indirectly contingent upon surgical trauma.

Lechner (1958) found that 39% of alveolar osteitis cases occurred in the 18-25 years group whilst 47% occurred in the 25-45 years group. The sample size was 100 alveolar osteitis cases from all sites of the dental arches.

Alveolar osteitis before the 18th year and after the 50th year is rare [Lechner (1958), MacGregor (1968), Chalifour (1969)]. MacGregor is of the opinion that relatively young age groups (20-30 years) experience alveolar osteitis more frequently compared with older age groups. One explanation for this may lie in the fact that the 20-30 years is where the vast majority of third molar surgery is performed.

Bruce, Frederickson and Small (1980) compared three equal groups of 330 patients in the following age categories: group 1 (14-24 years), group 2 (25-34 years), group 3 (35-81 years). Postoperative morbidity associated with mandibular third molar surgery was assessed, alveolar osteitis included. They found that group 3 incurred the highest incidence of alveolar osteitis (17.0%), compared with group 2 (15.5%) and group 1 (7.6%). Bruce, Frederickson and Small recommended mandibular third molar surgery (when indicated) on patients less than 25 years old as a means of reducing postoperative morbidity.
It is a long held view that given a particular type of mandibular third molar impaction; the procedure is less traumatic and recovery less eventful if surgery is undertaken in the 20-30 years age group rather than the over 35 years.

5.3.4. Pericoronitis.
Kay (1966) reported an alveolar osteitis incidence of 24% in mandibular third molar surgical cases with concomitant pericoronitis. According to Kay, the nature of the pericoronitis (whether acute or chronic) did not influence the incidence. However, cases of pericoronitis where the infection had spread to bone: the so-called 'deep pocket' cases gave an incidence of 88%. The view that pericoronitis contributes towards the development of alveolar osteitis is shared by Pell (1934), Parfitt (1938), Whitfield (1941), Conza (1953), Waite (1957), Schilli, Scharp and Lange (1967) and Nawara (1969).
Birn (1973) after a literature review, concluded that pericoronitis is often of sufficient intensity to precipitate alveolar osteitis by spreading into the bone marrow and blood clot following mandibular third molar surgery. Krekmanov and Hallander (1980) in a study of 120 patients, concluded that patients with pericoronitis and gingivitis before third molar surgery showed a greater predisposition to alveolar osteitis than those with healthy mucosa and gingivae. In a later study of 200 patients, Krekmanov (1981) found an alveolar osteitis incidence of 21.5% in acute pericoronitis cases compared with 13% in pericoronitis-free patients.
Osborn et al. (1985) confirmed Krekmanov's results (1980, 1981) in an extensive study of 16,127 mandibular third molar cases. They concluded that surgery in cases of active pericoronitis results in twice the incidence of alveolar osteitis than in non-pericoronitis cases.
There appears to be strong clinical evidence to suggest that pericoronitis is contributory to alveolar osteitis, based on a whole host of studies over the years [Fridrich and Olson (1990)]. However, this implication has never been tested in a manner which would satisfy Koch's postulates for establishing bacterial aetiology.

A number of authors, Kulenkampff (1941), Krogh (1951), Feldman (1951), Harnisch (1961), Freitag (1967) and Rud (1970), advocated immediate surgical intervention in acute pericoronitis cases; stating that it is good surgical practice to evacuate pus and remove the offending tooth at the earliest opportunity. Most quote studies where the postoperative morbidity (including alveolar osteitis) was judged to be similar to that in non-pericoronitis cases.

Rud (1970) compared a series of 776 acute pericoronitis surgery cases with 1620 previous cases where 90% were non-acute [Rud, Baggensen and Moller (1963)]. He found that the alveolar osteitis incidence was identical in both groups. Further, the use of systemic antibiotics was held to a minimum; penicillin was only prescribed when systemic symptoms of infection were present or if the swelling extended submandibularly, sublingually, parapharyngeally or if the procedure proved difficult. Rud estimated this use of antibiotics to be in the order of 7% of cases.

Belinfante et al. (1973) in an analysis of 400 mandibular third molar extractions, found that patients with pericoronal infection at the time of surgery did as well postoperatively as their asymptomatic counterparts. Lilly et al. (1974) found a greater incidence of alveolar osteitis following mandibular third molar surgery in teeth without pericoronitis (10.2%), compared with acute pericoronitis cases (7.2%). Pericoronitis was dismissed as a predisposing factor to alveolar osteitis.
In conclusion, I take the view of Thoma (1969), who advocated immediate removal of the offending wisdom tooth if the extraction is easy, i.e., without the need for raising a mucoperiosteal flap. However, if surgery is required, then the procedure should be delayed until the acute inflammation has resolved. The risk of alveolar osteitis may be equivocal between pericoronitis and asymptomatic cases, but the risk of a debilitating cellulitis following an elective surgical procedure in a systemically ill patient is difficult to justify.

5.3.5. Chlorhexidine Mouthwash.

Schiotte et al. (1970) showed that twice daily (b.d.) mouth rinses with a 0.2% (w/v) chlorhexidine solution reduced the number of microorganisms present in the saliva by 80%. Subsequently, Legarth, Munster and Swedensen (1977) investigated the effect of postoperative mouth washes with 0.2% chlorhexidine on the incidence of alveolar osteitis following mandibular third molar removal. The results showed that chlorhexidine mouth washes b.d. for the first postoperative week significantly reduced the incidence of alveolar osteitis from 13.7% (control, n= 160) to 7.5% (test, n= 254).

Tjernberg (1979) in a study of 60 mandibular third molar surgery patients, compared:

i) dental prophylaxis, plus pre- and postoperative 0.2% chlorhexidine mouth rinses with,

ii) a control group where none of the above was used.

A reduction in alveolar osteitis incidence from 17% (control) to 3% (test) was noted.

Krekmanov and Nordenram (1986) compared patient groups who received:

i) Oral systemic penicillin.
ii) Dental prophylaxis, gingival sulcus irrigation with 0.2% chlorhexidine preoperatively and 0.2% chlorhexidine mouth rinses postoperatively.

iii) No medication or preoperative treatment - a control group.

Sample size was 100 patients. Alveolar osteitis was noted in 19.4% of the control group, 5.4% of the antibiotic group and 8.1% of the chlorhexidine group.

Field et al. (1988) compared the incidence of alveolar osteitis (in the extraction of 324 lower premolars and molars) between a chlorhexidine treated group and a saline treated group. The gingival sulcus was irrigated for two minutes with either a 0.2% chlorhexidine solution or normal, sterile saline prior to extraction. A significant reduction in alveolar osteitis was seen in the chlorhexidine group (3.7%) compared with saline (11.1%) and control (9.3%). The authors felt that this reduction was achieved as a result of the antiseptic action of chlorhexidine, not the mechanical cleansing usually associated with an inert solution, e.g., saline. Field et al. recommended gingival irrigation preoperatively and postoperative mouth rinsing as an effective means of prophylaxis against alveolar osteitis.

On the other hand, MacGregor and Hart (1971) saw no advantage in the use of a 30 seconds preoperative mouth rinse of 0.5% chlorhexidine in 70% alcohol. A marked reduction in the oral microflora was noted but no decline in the incidence of alveolar osteitis was seen.

Sweet and Macynski (1985) studied 400 surgical cases of mandibular third molars, comparing the use of normal saline, chloramine-T, povidone iodine and sodium bicarbonate as preoperative and postoperative mouth washes. They concluded that there was no advantage in the use of antimicrobial mouth washes, either
preoperatively or postoperatively, in terms of preventing alveolar osteitis development. Further, Sweet and Macynsksi dismissed the antibacterial action of chlorhexidine as being of no significance clinically; the fact that chlorhexidine was not used in their evaluation (unavailable in the U.S. at the time) was of no consequence!

Berwick and Lessin (1990) supported Sweet and Macynski's (1985) contention that chlorhexidine was no more effective at reducing the incidence of alveolar osteitis than saline rinses. However, Berwick and Lessin used 0.12% chlorhexidine rather than the more conventional 0.2%.

Larsen (1991) in a prospective, randomised, double blind study of 139 patients (278 bilaterally impacted mandibular third molars) found a statistically significant 60% reduction in the incidence of alveolar osteitis in the chlorhexidine treated group compared with the placebo group. Chlorhexidine 0.12% was used twice daily as a preoperative and postoperative mouth wash; one week before and one week after surgery. The use of 0.2% chlorhexidine mouth washes pre- and postoperatively still provokes controversy amongst clinicians, however, there are a number of factors favouring its use:

i) It has proven antibacterial properties.

ii) It is relatively safe and cost effective.

iii) Some studies indicate a reduction in the incidence of alveolar osteitis following postoperative use.

iv) Chlorhexidine mouth washes maintain good oral hygiene postoperatively.

v) Sustantivity. Chlorhexidine has the ability to bind to oral mucous membranes and gingivae, thus providing a sustained antibacterial action which may last up to 12 hours after mouth rinsing [McIntyre (1991)].
Chlorhexidine mouth washes should be adopted as routine postoperative practice because of its ability to greatly reduce the oral microflora, thus considerably improving oral hygiene following third molar surgery.

5.3.6. Normal Sterile Saline Lavage.

Sweet, Butler and Drager (1976) reported a decrease in alveolar osteitis incidence in 206 cases of mandibular third molar surgical extractions following the use of high volume (350 mL) normal saline irrigation of the wound before closure. The procedures were carried out under pentobarbital (Nembutal) intravenous sedation and the patients hospitalised for 5 days postoperatively. An alveolar osteitis incidence of 0.49% (1/206) was reported. No differences were seen in the use of either a mechanical irrigator or a hand operated syringe. The authors ascribed the effectiveness of such large volume saline irrigation, to the mechanical removal of surgical debris, and the cleansing (removal of bacteria) of the wound prior to closure.

Butler and Sweet (1977) tested the use of 175 mL of normal saline against 25 mL of the same. They found the larger volume irrigation to be twice as effective in preventing the incidence of alveolar osteitis. Incidence rates of 5.7% and 10.9% respectively were recorded.

Sweet and Butler (1978) then compared the effectiveness of 175 mL with 350 mL of normal saline irrigation and found no significant differences in alveolar osteitis morbidity. The authors concluded by recommending the use of at least 175 mL normal saline irrigation as a means of gentle debridement prior to wound closure. Fridrich and Olson (1990) have suggested that using 60 mL saline irrigation is as effective as using 175 mL.
The use of plentiful irrigation as part of the operative procedure is nothing more than an extension of good surgical technique. It is well established that bone fragments, debris, pieces of detached soft tissue and pathological lesions within the surgery site are potentially infectious at worst, irritant at best. It makes good sense to thoroughly clean the wound before closure. Normal sterile saline irrigation is an effective means of debridement which is atraumatic as well.

5.3.7. Systemic Antibiotics.

Much has been written, both advocating and deploring the routine use of systemic antibiotics as a means of reducing the incidence of alveolar osteitis. For a full discussion, please refer to chapter 4.1 of this treatise.

In recent times, Rood and Murgatroyd (1980), Rood and Danford (1981) and Fazakerley and Field (1991) advocated metronidazole 200 - 400 mg 8 hourly (t.d.s.) for 3 - 5 days, as the antibiotic of choice for the prevention and treatment of alveolar osteitis following third molar removal.

Barclay (1987) questioned the value of metronidazole as a prophylactic agent. MacGregor (1990) cautioned against the routine use of any systemic antibiotic postoperatively, but favours penicillin in cases where extraction was particularly traumatic and/or the patient immunosuppressed in some way.

Because the relationship of intra-oral bacteria to alveolar osteitis is unclear, the practice of routine systemic antibiotic prophylaxis is contentious. Further, it is difficult to justify the blanket administration of antibiotics with all their attendant risks of sensitisation and development of resistant strains, in the hope that a small percentage of patients might be saved from suffering alveolar
osteitis.

However, systemic antibiotics may be warranted in a patient with a history of alveolar osteitis, or who is immunosuppressed. The decision is clinical, the treatment empirical.
Chapter 6.

A Summary of Current Knowledge.

6.1. Incidence.
Alveolar osteitis follows 3-4% of all extractions. The mandibular third molar site has an averaged incidence of 20% of wisdom tooth extractions. [Birn (1973)].

Still unclear. Birn's (1973) "Fibrinolytic Alveolitis" theory is impressive and has a lot of support. It remains contentious due to the clinical failure of antifibrinolytic medications, e.g., Apernyl, p-hydroxy-benzoic acid propyl ester (PEHB) and Tranexamic acid (AMCA), in the prevention of alveolar osteitis.
There is no doubt that fibrinolysis is an integral part of alveolar osteitis development. However, it appears that fibrinolysis is the mechanism rather than the cause of alveolar osteitis. Whatever triggers off this process of fibrinolysis may be regarded as an aetiological factor. Birn's (1973) rather simplistic explanation of excessive trauma and/or infection (Fig. 8) is only part of the puzzle, as excessive trauma/infection do not always precede alveolar osteitis.
Nitzan (1983) suggested Treponema Denticola (an anaerobe) as a possible cause of alveolar osteitis and noted the absence of both T. denticola and alveolar osteitis in childhood. Nitzan also linked T. denticola with Birn's (1973) fibrinolysis theory by virtue of the anaerobe's potent plasmin-like fibrinolytic activity. The fact that T. denticola is only one of a multitude of saprophytes found in the sockets of established alveolar osteitis cases tends to diminish its appraisal as being the cause of alveolar osteitis. Although metronidazole (an anti-anaerobic antibiotic) has been shown to lessen the incidence of alveolar osteitis in clinical trials [Rood and Murgatroyd (1979) and Rood and Danford (1981)], it does not prevent alveolar osteitis completely - as one would expect if T. denticola is the
only aetiological factor. The inconsistencies found in these theories point to a multifactorial aetiology.

6.3. Predisposing Factors.

i) Sex.

MacGregor (1968) showed a higher incidence of alveolar osteitis in females. In recent years an increased incidence has been linked with the widespread use of oral contraceptives [Catellani (1979) and Gersel-Pedersen (1981)]. It is thought that oral contraceptives, by increasing the systemic fibrinolytic activity, predispose women to the development of alveolar osteitis. Further, Heasman and Jacobs (1984) omitted women who were on oral contraceptive medication from their study and found no significant differences in incidence between the sexes. This appears to add weight to the oral contraceptive hypothesis.

ii) Age.

A number of studies record the highest incidence of alveolar osteitis in the third and fourth decades of life [MacGregor (1968) and Field et al. (1985)]. Alveolar osteitis is virtually unknown in childhood [Nitzan (1983)].

iii) Extraction Site.

Mandibular molars have the highest incidence of alveolar osteitis, followed in descending order by: premolars, upper molars and incisors [MacGregor (1968)]. Birn (1973) reviewed the literature and found an averaged incidence of 20% for the mandibular
third molar site.

iv) Trauma/difficulty of Extraction.

There is a plethora of evidence to suggest that trauma is an important predisposing factor [Krogh (1937) and Heasman and Jacobs (1984)]. MacGregor (1968) concluded that patients were more likely to suffer alveolar osteitis following difficult extractions or root fracture during exodontia. Birn (1973) considered trauma as one of two precipitating factors which initiate fibrinolysis in the alveolar socket.

v) Smoking.

Clinical observations suggest that smokers are more prone to alveolar osteitis than non smokers [Sweet and Butler (1979)]. No mechanism has been proposed to explain this phenomenon. Smoking in the first postoperative day appears to increase the incidence of alveolar osteitis.


The primary aim of treatment is pain relief. Empirical treatment with a variety of socket dressings is the mainstay of management. Anodyne dressings are popular because of their clinical effectiveness in alleviating the pain of alveolar osteitis. Some practitioners give a long acting local anaesthetic injection, e.g., 0.5% bupivacaine (Marcain) as a routine prior to socket irrigation. From the patient’s viewpoint; treatment success is directly proportional to the amount of pain relief gained.

In Australia, Alvogyl has become very popular as the treatment of choice. This is not surprising given that it is effective, easily applied, requires a minimum of chair time and is very cost effective. Although far from ideal, dressings such as Alvogyl are recommended until
superior treatment systems are developed. Bland, non-irritant, obtundent dressings such as collagen paste [Mitchell (1986)] and fibrin sealants [Moller and Petersen (1988)] show promise; as these do not retard socket healing in the same way as zinc oxide/eugenol and Alvogyl. The disadvantage, of course, is the lack of any analgesic properties in collagen or the fibrin sealants. Perhaps combinations with a proven topical anaesthetic agent are the future for such medications.

A notable clinical failure has been the equivocal results obtained with antifibrinolytic medications, e.g., tranexamic acid, Apernyl, PEHB [Kestitalo and Persson (1973), Ritzau (1973), Vedtofte, Ritzau and Donatsky (1974), Ritzau and Swangsilpa (1977) and Schatz, Fiore-Donno and Henning (1987)]. After the high expectations generated by the "Fibrinolytic Alveolitis" theory, it is disappointing to say the least. If nothing else, it highlights the multifactorial nature of alveolar osteitis aetiology.

6.5. Prevention of Alveolar Osteitis.

A number of clinical measures have been shown to lower the incidence of alveolar osteitis:

i) Good 'atraumatic' surgical technique in which trauma to the hard and soft tissues is minimised reduces postoperative morbidity, including alveolar osteitis [MacGregor (1968)].

ii) Thorough saline irrigation prior to wound closure in third molar surgery is effective [Sweet and Butler (1978)].

iii) 0.2% chlorhexidine mouthwashes b.d. as a postoperative regime is advocated by Krekmanov and
iv) Oral metronidazole 200 mg - 400 mg t.d.s. for 3-5 days postoperatively, significantly reduces alveolar osteitis incidence [Rood and Murgatroyd (1979), Rood and Danford (1981), Fazakerley and Field (1991)].
v) The avoidance of smoking postoperatively has been shown to decrease the incidence of alveolar osteitis [Sweet and Butler (1979)].
vi) Studies have shown that women on oral contraceptive medication are predisposed to the development of alveolar osteitis [Fridrich and Olson (1990)].

There is no panacea for the prevention of alveolar osteitis, even if there was, would it be ethical to subject every patient to the potential risks of such a medication in order to save 3-4% from alveolar osteitis?

6.6. Conclusion.
Our understanding of alveolar osteitis has advanced enormously since Crawford (1896) described the first documented case of “dry socket” [Swanson (1991)].
Birn (1973) explained the mechanics of the disease process and suggested a few aetiologies. In addition, Nitzan (1983) provided a bacterial pathogenesis which neatly elucidated some aspects of alveolar osteitis. However, despite these insights, the complete story of alveolar osteitis is yet to be told.
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