THIS CRITICAL REVIEW OF THE LITERATURE CONCERNING INFECTIONS OF THE JAWS IS SUBMITTED IN SUPPORT OF THE CANDIDATURE FOR THE DEGREE OF MASTER OF DENTAL SURGERY.

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# Infections of the Jaws

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CHAPTER ONE

ALVEOLAR OSTEITIS

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1. **INTRODUCTION**

Alveolar osteitis, or "dry socket" as the condition is commonly called, is the most frequent and the most unpleasant complication of extraction wound healing. The condition has been referred to as alveolitis, localized osteomyelitis, alveolar osteitis, localized acute osteomyelitis, postoperative osteomyelitis and postoperative aveolalgia by various authors, each has a good reason for his own particular choice but they all recognize the term "dry socket" which will be used throughout this review.

Although there have been a great number of papers published and experimental studies carried out, there is still no universal agreement on the etiology or treatment of the condition. However it is obvious that trauma during extraction is probably the greatest predisposing cause of "dry sockets", and today the conscientious dental surgeon, faced with a difficult extraction, will take steps to minimize the risk of producing this condition by surgically removing the tooth. Even so, there still occur occasions when traumatic extraction is proceeded with in the hope of saving time, or with the excuse that should a "dry socket" eventuate, the pain can soon be relieved.

The condition, which is very often unpredictable, is characterized by a socket containing a blood clot in various stages of disintegration, a foul odour, and almost
constant pain of variable intensity over part, or all the distribution of the trigeminal nerve.

2. **INCIDENCE**

The incidence of "dry sockets" following tooth extraction has been investigated by Krogh (1937) and Archer (1939). Krogh (1937), in recording 138 "dry sockets" in 6,403 extractions, found an incidence of 1.2%; Archer (1939), in recording 224 "dry sockets" in 23,886 extractions, found an incidence of .9%.

This author, in an analysis of patients treated in the Exodontia Department of the United Dental Hospital of Sydney has found 400 "dry sockets" in 50,386 extractions, an incidence of .79%. According to Krogh (1937), male and female are equally susceptible, and Archer (1939) found 79% "dry sockets" in the mandible and 21% in the maxilla. Analysis of this author's figures agrees with Krogh's (1937) statement on the male - female ratio, but a lower incidence was found in the mandible, 72% as compared with Archer's (1939) 79%.

Fry and Goldman (1958) note that "dry sockets" seldom occur after multiple extractions or in cases where there has been considerable loss of alveolar bone due to periodontitis, and Krogh (1937) found in his survey that the more adjacent teeth removed at one operation, the less the danger of "dry socket" development. On clinical observation, this author agrees with both these statements.

3. **HISTOPATHOLOGICAL FEATURES**

A brief review of the steps in normal healing of extraction
sockets is necessary before the histopathological features of alveolar osteitis are considered.

(a) Normal Healing of Extraction Wounds: Histological examination of the healing process in extraction wounds has been carried out by many workers, both on humans and animals. Claflin (1936) with dogs, Simpson (1960) and Radden (1959) with Rhesus Macacus monkeys, Huebsch and associates (1952) with normal male rats (Long-Evans strain) and Mangos (1941) with human specimens, have all published well illustrated articles. It was stated by the latter author, and agreed with by the others, that histologically there was no difference in the healing processes in humans and animals. Mangos (1941) pointed out that healing in humans differed only from dogs by taking twice as long for epithelium to heal and three times as long for bone. Consequently the steps in normal healing of an extraction socket can be summarized as follows (Weinmann and Sicher, 1955; Mangos, 1941; Shafer, Hine and Levy, 1959).

1. Formation of blood clot, with red cells being entrapped in a fibrin meshwork and the ends of torn blood vessels in the periodontal membrane becoming sealed off.

2. Within the first 24 hours there is vasodilatation and engorgement of the blood vessels in the remnants of the periodontal membrane. The surface of the clot is covered by a thick layer of fibrin and the unsupported gingival tissue collapses into
the mouth of the socket as the clot contracts.

(3) On the third day there is proliferation of fibroblasts from connective tissue cells in the remaining periodontal membrane and these cells begin to grow into the clot around the entire periphery.

(4) After five days, evidence of organization of the clot by young connective tissue is found. This occurs more rapidly in the upper half of the socket, due probably to the greater vascularity of the submucosa than the alveolar bone.

(5) By the seventh day, new delicate capillaries have penetrated to the centre of the clot, remnants of the periodontal membrane which have been undergoing degeneration are no longer recognizable and epithelial proliferation over the surface of the wound is well advanced and, in small sockets may be complete.

(6) Ten days after extraction, evidence of osteoblastic and osteoclastic activity is seen gradually replacing the young connective tissue with course fibrillar bone. At the end of fifteen weeks, the socket is completely filled with new bone and resorption and rounding off of the sharp alveolus is taking place.

(7) The epithelium has proliferated completely
across the wound in two weeks.

From clinical observation and these studies, it can be seen that if the blood clot within an extraction socket survives for approximately ten days, the danger of a "dry socket" developing, is past.

(b) **Disturbed Healing in Extraction Wounds**: The histopathological picture of sections taken from a "dry socket" is, according to Faiello (1948), much the same as in any necrotic bone. There is cellular infiltration of phagocytes, inflammatory cells and giant cells, with bacteria and dead bone present. The dead bone may become detached in the form of a sequestrum, or it may be wholly absorbed by the action of giant cells if it is microscopic in size.

4. **Bacteriology**

The invasion of a tooth socket by microorganisms following extraction is considered by Di Conza (1953) as a possible cause of "dry socket" formation, and Helmore (1958) claims that two organisms, *Streptococcus haemolyticus* and *Bacillus fusiformis* can always be demonstrated in these cases. Other organisms such as staphylococci and spirillum vincenti are present according to Alling and Kerr (1957) and Di Conza (1953). Claflin (1936) found that "dry sockets" could be produced in dogs by the introduction of a mixed culture of staphylococci and streptococci from infected human sockets. Grandstaff (1935), on the other hand, reported that bacteriological findings in twenty instances
of normal healing and twenty instances of delayed healing sockets were not significantly different.

Although this author does not agree with Di Conza (1953) when he claims that pre-existing infection, which is permitted to infect the organizing clot, or a secondary infection, are the only real factors in alveolar osteitis, he does feel these types of infection are definitely a factor in the production of a "dry socket" but, without trauma or some other factor to lower the clot resistance there is little chance of infection becoming established.

5. CLINICAL FEATURES
The patient suffering from a "dry socket" usually presents complaining of a dull, constant, deep seated pain which may increase at night. This pain, which is unrelieved by the use of ordinary sedatives (Di Conza, 1953) is sometimes referred to other branches of the trigeminal nerve and frequently, when the lower premolars and molars are involved, the pain is felt in the region of the ear as well as the socket. On rare occasions the patient may manifest toxic absorption and malaise. Patients usually return for treatment on the third or fourth day following extraction although anytime up to ten days may elapse, apparently depending on the severity of the condition and each individual's pain threshold. Osterloh (Sept. 1945) states the pain is the result of inflammatory irritation of the highly sensitive nerve endings in the bony walls of the socket. A complaint of foul breath and taste, as well as pain, is usually made
by the patient.

Examination of the socket reveals the degree of disintegration of the clot which may be partial or total; there is no sign of suppuration and the clot, if still present, is easily removed by gentle irrigation with normal saline. The bony wall of the socket is now exposed and, according to Gibson (1937), when dried is of a darker colour than normal bone and appears glazed. Exploration of the socket either before or after the loss of the clot will cause increased pain if the instrument is allowed to touch these bony walls.

6. **ETIOLOGY**

The etiology of "dry sockets" has been attributed to many factors, either singly or in combination. These factors will be considered separately.

(a) **Inadequate Blood Supply to the Socket Following Extraction:**

Huebsch (1958) states that in order for normal healing to take place in an extraction socket, it is necessary for the blood clot to organize. This is only possible when an adequate blood supply is present, derived from the capillaries in the periodontal membrane, the apical arterial blood supply, and the rich capillary network in the gingiva and mucoperiosteum. The loss of one or more of these supplies, plus the shrinkage of the clot may lead to the formation of a "dry socket". This theory is substantiated by the fact that "dry sockets" are rare following alveolectomies, removal of teeth from fracture lines, or teeth around which
considerable periodontal bone loss has occurred. Where the blood supply is adequate and "dry sockets" still occur, they can be attributed to various systemic conditions such as diabetes mellitus, anaemia, vitamin deficiency, etc.

In experiments with white rats, Huebsch (1958) produced a decrease in the incidence of "dry sockets" following extractions, by punching holes through the lamina dura into the more vascular regions of the cancellous bone, thereby increasing the blood supply to the clot within the socket.

It is felt by this author that Huebsch (1958) in seeking the etiology of "dry sockets", has overlooked the fact that there is always a predisposing cause for the lack of adequate blood supply. This may be either a local factor, possibly trauma or dense bone enveloping the socket, or a systemic factor. Consequently the true etiology of the condition is probably more fundamental than an inadequate blood supply to the socket.

(b) Loss of Periodontal Membrane: Radden (1959), Alling and Kerr (1957) and Huebsch (1958) consider any periodontal membrane remaining after an extraction in which considerable force and manipulation is necessary, is so damaged as to be unable to contribute anything to the regenerative process. Consequently, if an extraction is difficult and the tooth is extracted with a large amount of periodontal membrane attached, these authors expect a
"dry socket" to develop.

(c) **Loss of Blood Clot:** The loss of the blood clot from an extraction socket can be caused by infection, the patient sucking the wound, or early presence of saliva in the socket.

1. **Infection:** It is considered by Di Conza (1954) that pre-existing infection, which is permitted to invade the clot, and secondary infection, are the only real factors in the formation of dry sockets. He based this claim on a survey carried out on the removal of 220 impacted lower third molars. Half the patients received Terramycin, 250 mgms. sixth hourly for twenty four hours immediately after surgery, the other half acted as controls. No cases of "dry socket" developed in the patients using Terramycin, and as this drug was the only variant between the groups, it was concluded that infection was the main predisposing cause.

Eman (1944) and Faillo (1948) also feel that organisms introduced into an extraction wound and multiplying in an area of lowered resistance such as traumatized bone or soft tissue, can be the cause of "dry sockets". Eman (1944) considers that proof of this fact is seen in the reduction in "dry socket"
incidence achieved by the application of strict asepsis during the extraction. Helmore (1958) and Thoma (1958) claim traumatic extraction combined with infection, either introduced or pre-existant, are the main etiological factors. These types of infections, and the presence of an unduly thick layer of compact bone lining the socket, are considered by Stones (1954) the cause of "dry sockets".

It is felt by this author that the work of Di Conza (1953) is partly invalidated by the small number of cases studied and also by the short duration of antibiotic therapy. It is difficult to understand how the antibiotic, given for twenty four hours post-operatively, can have any beneficial effect on the blood clot which does not begin to vascularize until after the third day. This author agrees with Thoma (1958) and Helmore (1958), and feels from clinical experience that "dry sockets" are due to infection of a blood clot having inadequate blood supply due to trauma at the time of extraction and sometimes to thick compact bone lining the socket as described by Stones (1954).

(2) Patient Sucking the Socket: "Dry sockets" are undoubtedly caused, on rare occasions,
by patients forcing their tongues into recent extraction sites or by sucking the sockets. In these cases, the loss of blood clot is mechanical and due to self-inflicted trauma and can hardly be included in the discussion of etiology.

(3) Early Presence of Saliva: Russell (1944) is of the opinion that ingress of saliva into an extraction wound is the principal cause of "dry sockets", and claims to have reduced their incidence by immediate postoperative application of "silver adhesive foil" over the socket. In 100 cases used as controls, 14 "dry sockets" developed; where the foil was used and left in place for two hours, only three cases developed in 100 simple extractions. Although there is a marked reduction in the incidence of "dry sockets", even Russell's (1944) improved figures of 3% do not compare favourably with those of other authors. Osterloh (Sept. 1945) does not agree with this theory, feeling that, if it were correct, we would be faced with a greater number of "dry sockets".

(d) Trauma: Most writers believe a combination of factors is responsible for delayed healing in extraction sockets, but conclude that one agent or another is paramount. It has been found in the literature that
trauma is considered the principal cause by many authors (Gibson, 1937; Paine, 1937; Krogh, 1937; Eman, 1944; Alling and Kerr, 1957; Thoma, 1958; Helmore, 1958 and Shafer, Hine and Levy, 1959).

Alling and Kerr (1957), having burnished socket walls during extraction of lower molars from monkeys, found histological and clinical evidence indicating a condition, analogous to "dry socket" in man, had been produced. Control sockets in the same animals healed uneventfully. The burnishing or compression of socket walls by prolonged rocking of teeth or misuse of elevators during extraction, has been observed by this author as a common predisposing cause of the condition.

(e) Excessive Sponging of Bone During Extraction: Although Osterloh (Sept. 1945) considers exposure of the operative field for any great length of time, has little effect on healing, this author feels that continual drying out of a socket in searching for retained root apicies does eventually produce a bloodless socket, which on completion of the extraction may not entirely refill with blood, thus exposing alveolar bone to infection from the saliva.

(f) Prolonged Vasoconstriction with Local Anaesthetics: Gibson (1937), Krogh (1937) and Osterloh (Sept. 1945) consider from clinical experience that the use of local anaesthetic solutions containing vasoconstrictors has no adverse effect on socket healing, and therefore cannot be cited as a cause of "dry sockets". Eman (1944) on the
other hand feels that their use may delay socket healing if care is not taken to use sterile, isotonic, warm solutions slowly injected, preferably away from the extraction site (conduction anaesthesia).

Although it has been suggested in the past that the use of general anaesthesia will reduce the incidence of "dry sockets", it is felt by this author that this reduction is not due to the absence of vasoconstrictors in local anaesthetics, but to the fact that extractions under general anaesthesia are usually multiple, and as emphasized by Krogh (1937) and Fry and Goldman (1958), this in itself reduces the incidence of "dry sockets". A survey carried out by this author on 200 patients who had multiple extractions under general anaesthesia revealed only three "dry sockets" in 3,161 extractions, an incidence of .095%.

(g) Age and Systemic Conditions: Many authors claim the condition is purely local, and age or systemic conditions play no part. Osterloh (Sept. 1945) states the higher incidence of dry sockets in elderly patients is understandable since the bone is more calcified and condensed, with consequent diminished circulation. Thoma (1958) feels that in any undernourished patient, or one suffering from some debilitating disease, the condition is more likely to develop than in a healthy person.

This author agrees with both these theories, firstly in elderly patients, not specifically on account of their age and denser bone, but mainly due to the increased trauma occasioned by the removal of their teeth. Secondly in
patients suffering from conditions such as diabetes mellitus or anaemia, receiving ACTH or cortisone, or having lowered nutritional states particularly lack of protein or Vitamin C, clinical observation reveals a definite predisposition to infection following any type of trauma.

(h) **Pre-Existing Causes:** Stones (1954), Osterloh (Sept. 1945), Millhon and associates (1943) and Schwarz (1933) claim that an unduly thick layer of compact or sclerotic bone, due to irritation from chronic infectious processes or trauma, is the cause of "dry sockets". Hence any attempt to preserve the blood clot formed after extraction is futile, unless the sclerotic bone is removed down to a level where there is adequate blood supply to nourish the clot. Osterloh (Sept. 1945) stresses the necessity for good radiographs so that the possibility of "dry socket" development can be recognized. It is felt by this author that radiographic recognition of bony sclerosis in the immediate vicinity of the tooth and/or root abnormality is an indication for surgical removal.

7. **PREVENTION**

(a) **Surgical Measures:** Surgical removal of the tooth in any case where there is the slightest possibility of a "dry socket" developing, is considered by this author as the only method of prevention. This view is also taken by Helmore (1958) who also suggests prophylactic antibiotic therapy, with which this author cannot agree, considering it unnecessary and contrary to the patient's best interests.
The use of antibiotics, where not absolutely necessary, is to be discouraged both from the aspect of sensitizing the patient, and the development of resistant strains of organisms. Admittedly there are extenuating circumstances when they should be used, as in diabetes mellitus, etc. but not routinely. Schwarz (1933) and Osterloh (Sept. 1945), besides advocating surgical removal of the tooth, state that any sclerotic bone surrounding the socket should also be removed down to the level where an adequate blood supply is obtained. This approach is to be commended. Huebsch (1958) suggests the perforation of the socket walls to allow a better blood supply to the clot. Although this method appears clinically to reduce the incidence of "dry sockets", it carried with it the dangers of introducing infection into cancellous bone if strict asepsis is not adhered to, nerve injury in the lower molar region, thermal necrosis with sequestration if burs are used, and excessive haemorrhage. The practice of treating already established "dry sockets" by perforation of the socket walls is definitely contraindicated owing to the danger of osteomyelitis.

(b) Local Measures: The technique, advocated by Millhon and associates (1943), of reducing the size of the blood clot in a socket with sterile removable dressings is logical. The amount of blood clot remaining between dressing and socket walls is small and, being protected from mouth fluids by the dressing, organizes readily. Although it is felt by this author that adequate clot
reduction can be obtained by surgical removal, the technique is applicable to large bone cavities resulting from the removal of unerupted teeth and cysts. This method is, I feel, contraindicated in simple extractions because of the frequent change of dressings necessary, whereas surgical removal of the tooth will achieve the same result in a much shorter period of time.

Evaluation of sulphanilamide and/or sulphathiazole, placed in sockets immediately after extraction, as a method of preventing "dry sockets", has been carried out by many workers. The results differ considerably, Meacham and Osgood (1941), Griess (1942), Millhon and associates (1943), Weiner (1944), Krogh (1948), Gwinn and Grimm (1948) and Davis and associates (1955) have all produced statistical evidence of the value of these drugs in the prevention of "dry sockets". Griess (1942) does however point out the danger, admittedly slight, of toxic manifestations resulting from absorption of the drug from a tooth socket. Krashen (1940, 1942), Olech (1953) and Osterloh (Jan. 1945) state the local application of sulphanilamide is of no benefit in the reduction of "dry socket" incidence, causes local irritation, postoperative pain and delay in healing time and is contraindicated in the light of possible toxic reactions. Versnel (1953) found histologically that sulphanilamide and sulphathiazole cause breakdown, prevent organization of the clot and retard healing by as much as three weeks. In the same series of experiments he found that penicillin had no adverse effect on the healing of ext-
raction wounds.

Gwinn and Grimm (1948) found that penicillin introduced into extraction wounds considerably reduced the incidence of "dry sockets", whereas Linn (1954) and Holland and Tam (1954) found no significant difference in the healing rate when compared with control groups.

Davis and associates (1955) with terramycin, Verbic (1953) with aureomycin, Mourfield and Barron (1958) with erythromycin and Quinley and associates (1960) with achromycin, all found a decrease in the incidence of "dry sockets" following immediate postoperative application of the drugs, although there was some tendency for achromycin to be rejected as a foreign body. This same reaction was noted by some observers in relation to sulphathiazole if excess amounts were used.

This author feels that the local application of sulphophenamides and antibiotics should be discontinued for the following reasons.

(1) It has been shown by careful trials that they are, in many instances, useless in preventing postoperative complications.
(2) It has also been shown that some of these drugs actually irritate the tissues and delay healing.
(3) There is a danger of the drug being absorbed, with possible toxic manifestations.
(4) Local application is conducive to the formation of resistant strains of bacteria.
(5) Even small doses of antibiotics can sensitize a patient to the drug.

(6) If we accept the theory that "dry sockets" result from a loss of blood clot, due to trauma or dense bone limiting the blood supply, it is difficult to see how the application of these drugs can be of any use. In other words antibiotics are not a substitute for atraumatic surgery and asepsis.

(c) Systemic Measures: Systemic use of antibiotics to prevent "dry sockets" is advocated by Cash (1951), Di Conza (1953) and Helmore (1958), the latter author combining it with surgical removal of the teeth. Once again this author feels that the use of antibiotics is unnecessary and contraindicated in the majority of cases. Molt (1936) stresses the importance of a balanced diet in the systemic prevention of "dry sockets", the value of a high Vitamin C intake being mentioned in particular.

8. Diagnosis
The diagnosis of "dry socket" is entirely clinical, being based on the patient's description of the pain and their complaint of a foul taste and odour in the mouth. Examination reveals a foul smelling, degenerating blood clot in various stages of disintegration. Increase in pain when the socket walls are touched with an instrument confirms the diagnosis, the bone encountered having a characteristic
rough feeling.

9. **TREATMENT**

Surgical removal of the exposed bone in "dry sockets" is advocated by Schwarz (1933) but, as pointed out by Helmore (1958), any surgical intervention at that stage is definitely contraindicated owing to the danger of osteomyelitis. The local application of sulphonamides and antibiotics has been suggested by Sinclair and Barker (1938), Stern (1941), Zeff (1947) and Helmore (1958) and although some decrease in the bacterial flora of the socket is possible, this author feels that the resultant relief of pain claimed by these authors is probably due to the irrigation prior to insertion of the dressing or, in Helmore's (1958) cases, to the eugenol used as a vehicle for the sulphonamides. The local use of antibiotics is contraindicated for the same reasons as when used in the "prevention" of "dry sockets", namely the danger of toxic manifestations, promoting resistant strains of bacteria or sensitizing the patient to the drug.

The most universal, and most satisfactory method of treating "dry sockets" in this author's opinion, is the use of an obtundent such as "Dentalone" (Parke Davis Co.) following a thorough irrigation of the offending socket "Dentalone", consisting of chloretone, oils of cloves, cassia and wintergreen, produces almost immediate relief of pain by depressing the exposed nerve terminals. Its use is advocated by many authors including Gibson (1937)
Paine (1937), Eman (1944), Kemp (1951), Cash (1951) and Thompson and Morris (1958).

After recommending the use of "Dentalone" Thompson and Morris (1958) continue "At this juncture, a decision must be made as to the desirability of concurrent antibiotic therapy. Also, of great importance is the patient's nervous and physiological state. He may require only reassurance and a little additional medication with codeine or antibiotic therapy, ...". The use of systemic antibiotic therapy in the treatment of "dry sockets" as advocated by Thompson and Morris (1958) and Spiegel (1958), is most unnecessary and contrary to the patient's best interests, but to infer, as Thompson and Morris (1958) do, that it may be used to improve a patient's "nervous and physiological state", obviously needs no further comment.

Fry and Goldman (1958) claim "Biosone G.A. Dental Paste" has the advantage of not needing to be changed daily as "Dentalone" does, and yet relieves pain from "dry sockets" as effectively. This author cannot agree with such a claim as most patients he has treated with "Biosone" appear to obtain little or no relief from its use, whereas it is extremely rare to find the pain of a "dry socket" resistant to "Dentalone".

It is stated by Stock (1940) that "dry sockets" respond extremely well, with quick and lasting relief, to the use of Short-wave currents. They do not appear to act by virtue of any direct bacteriocidal action but probably
evoke some physiological response in the tissues.

Holland (1948) suggests the use of .5cc of absolute alcohol, combined with local anaesthesia, given as a mandibular block to overcome severe or prolonged pain in lower molar or premolar "dry sockets". It appears rather a drastic procedure to subject a patient, suffering from this condition, to anaesthesia or paraesthesia over the distribution of the inferior dental nerve for anything up to 18 months when the use of a long acting anaesthetic would suffice. "Efoeaine" has been recommended by Hayward (1954) and although there appears to be considerable danger of complications, it is apparently used extensively by the medical profession to alleviate pain resulting from malignant growths or their treatment. Seldin and associates (1955) reported a severe reaction following its use to relieve post-operative dental pain. Shapino and Norman (1953) and Bonica (1952) recorded serious neurological symptoms, and Angerer and associates (1953) reported a death following the use of "Efoeaine" to relieve severe pain. These latter authors also recorded tissue destruction with sloughing, severe neuralgia persisting for weeks and long lasting and probably permanent motor paralysis.

According to Faillo (1948), Anderson (1957), Toto (1958), Spiegel (1958) and Hansen (1960), the use of proteolytic enzymes in the treatment of "dry sockets" is
successful in removing necrotic tissue, promoting healthy granulation and relieving pain in a large proportion of cases. This author agrees that proteolytic enzymes such as trypsin do produce a very clean socket which heals rapidly, but has found as Anderson (1957) did, that pain is not greatly reduced, "Dentalone" still being necessary. However one must agree with Anderson (1957) who, finding a 39% faster healing rate, stated that although trypsin is a powerful adjunct in the treatment of "dry sockets", it obviously is not the final or complete answer to the problem.
CHAPTER TWO

SUPPURATIVE OSTEOMYELITIS

SYNOPSIS

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11. CHRONIC SUPPURATIVE OSTEOMYELITIS

12. TROPICAL OSTEOMYELITIS OF THE JAWS
1. **GENERAL INTRODUCTION**

Osteomyelitis of the jaws was, in the past, considered common, but with the advent of antibiotics it has now become a relatively rare condition. Indeed, the extensive lesions involving all or a greater part of the mandible, described so often in the past, are now almost non-existent.

Osteomyelitis is defined by Thoma (1953) as inflammation involving cancellous bone, bone marrow, cortex and periosteum, whereas Stones (1954) and Weinmann and Sicher (1955) divide the condition into osteomyelitis, inflammation of the soft tissues within the bone, and periostitis, inflammation of the periosteum. These latter authors consider the condition, involving all components of the bone, should be called osteitis, inflammation of a bone. Although this description is histologically correct, we find throughout the literature that Thoma's (1954) definition is generally accepted.

Waldron (1943) states that the condition occurs in the mandible five or six times more frequently than in the maxilla, usually between the ages of 10 and 40 years, being three times more prevalent in males than females.

Acute, subacute and chronic conditions are distinguishable although there is no clear demarkation between the clinical divisions (Thoma, 1954). This author agrees with this classification, feeling that the majority of cases begin as an acute osteomyelitis and progress through the
subacute stage to chronicity. However, there are cases in which the onset is insidious, the condition first becoming apparent as a subacute or chronic infection. Although the acute and subacute conditions are similar, the manifestations of a chronic condition which arises without a preceding acute stage, are sufficiently characteristic to warrant a separate description.

2. **Etiology**

Osteomyelitis of the jaws may arise from dental infections, furuncles of the skin, trauma or haematogenous transportation of bacteria (Thoma, 1954).

(a) **Dental Infections:** Dental infection is by far the most common cause of osteomyelitis and may occur spontaneously, or after dental treatment. Spontaneous infections develop from pulpal, apical, periodontal or residual infections spreading directly into the cancellous bone, or pericorononal infections involving the periosteum. Shuster and Graham (1954) record a case of osteomyelitis in the ascending ramus of the mandible which was secondary to a peritonsillar abscess.

Osteomyelitis following dental treatment may occur as a result of ill-timed or, indiscriminate surgical treatment of a suppurative lesion, or lack of asepsis, allowing infection to invade a previously unaffected area. The curettage of acutely inflamed sockets, surgical removal of partly erupted teeth still associated with acute pericoronitis, and injection of local anaesthetic solutions into
inflamed tissues are all procedures which, in the opinion of this author, are likely to result in osteomyelitis, or in the latter case, periostitis, which may extend to the bone, and thus should all be discouraged.

The extraction of teeth during the acute stage of dental infection has been and still is a matter of controversy. Many surgeons, such as Kayne (1941), refuse to extract an abscessed tooth until the acute symptoms have subsided, preferring to establish drainage by incision or through the root canal of the tooth. On the other hand, this author feels that Thoma (1954) is correct when he states that the simplest method to establish drainage is by extraction of the offending tooth, which can invariably be done without undue trauma, owing to partial disintegration of the periodontal fibres. Thoma (1954) further states that cases of osteomyelitis after tooth extraction are generally due to delayed extraction, application of external heat, or failure to maintain the drainage established. From clinical experience this author agrees with this statement.

(b) Furuncles of the Skin: Furunculosis of the chin is considered by Hoenig (1931) to be an important cause of mandibular osteomyelitis. Twelve out of the twenty-five cases of furuncle in this region, reported by Hoenig (1931), were complicated by osteomyelitis in some form. He claims that the skin is connected to the periosteum by strong connective tissue in which the lymph vessels lead directly
to the bone. Stones (1954), while considering this method of infection rare, states that it is usually the result of a blow on the furuncle, or too deep an incision of the abscess in which the scalpel involves the periosteum.

In this author's experience, two out of the three cases of osteomyelitis with wide spread bone involvement which he has observed, have resulted from this type of infection.

(c) Trauma: Small localized areas of osteomyelitis are commonly seen following traumatic extractions in which fragments of alveolar bone have been detached and become non-vital. These cases present little difficulty in treatment, for the sequestrum is readily removed and final healing is almost immediate.

Trauma resulting in mandibular fractures is an important cause of osteomyelitis. The condition usually arises as a result of delayed treatment, lack of adequate immobilization, or retention of a tooth in the line of fracture. Reduction of a badly displaced fracture may warrant the retention of a tooth involved but only if the patient is kept under close observation, treated with an antibiotic systemically, and the tooth removed at the earliest opportunity.

Englert and Pasqual (1958) report a case of wide spread osteomyelitis of the edentulous maxilla, traumatized by a laryngoscope during the administration of a general anaesthetic.

(d) Haematogenous Infection: Acute haematogenous
osteomyelitis is a disease occurring in growing bones and most commonly found in teenage boys. In adults the condition is rare and much less acute than in the lower age groups (Waldron, 1943; Fabe, 1950 and Weinmann and Sicher, 1955).

The organisms enter the blood stream from furuncles, infected tonsils, skin injuries or extraction wounds, and it is generally accepted that trauma is necessary to promote the pyaemia or septicaemia. Haematogenous infection commonly causes multiple lesions involving a number of bones, simultaneously or in succession, (Thoma, 1954 and Kallenberger, 1949) and the anterior part of the mandible being supplied by terminal arteries, is often the site of infection.

Waldron (1943), claims that haematogenous osteomyelitis in adults is sometimes seen at the site of unerupted teeth, particularly in the mandible, and agrees with Thoma (1954) and Stones (1954) that the condition may occur during, or after severe infections such as typhoid fever, smallpox, pneumonia, influenza, measles, malaria, scarlet fever or diphtheria.

3. HISTOPATHOLOGY

If infection commences in the cancellous bone it spreads through connecting blood vessels to the periosteum. Should it arise from the periosteum, the reverse process takes place. Stones (1954) and Weinmann and Sicher (1955) state that inflammation of bone is confined to the connective
tissue forming periosteum, marrow and that filling the Haversian canals, and reactions in the hard tissues are always secondary. The medullary spaces are filled with inflammatory exudate composed chiefly of neutrophilic polymorphonuclear leucocytes, and occasional lymphocytes and plasma cells. Later there is pus formation, the bony trabeculae losing their viability and undergoing slow resorption. This inflammation results in increased tension in the blood vessels leading to thrombosis and necrosis of the area normally supplied. The necrotic bone becomes separated from living bone by granulation tissue and is finally sequestrated.

According to Waldron (1943) the degree of bone loss is dependent on

(a) the type and virulence of the infecting organisms and the effectiveness of opposing antibodies,

(b) the amount of disturbance in blood circulation and thrombosis, and

(c) the character and degree of the consequent deprivation of bone cell nutrition, which determines the extent of bone necrosis.

Sequestration of the condylar and coronoid processes is rare (Waldron, 1943 and Cohen, 1954) due to the rareness of blood vessel involvement and the presence of collateral circulation in this region.

Drainage, either spontaneous or surgical, of the accumulated inflammatory exudate limits the pathological
changes (Cahn, 1955).

The osteogenic cells of the periosteum, although often separated from bone by pus, have great powers of vitality and commence regeneration by the formation of an involucrum. This may commence before all sequestra have been exfoliated, and the new bone formed is usually coarse and poorly developed.

Key (1942) claims that tissue death results from excretion of toxins by the staphylococci rather than static circulation.

4. **BACTERIOLOGY**

The most common infecting organism seen in osteomyelitis is Staphylococcus aureus according to Key (1942), Waldron (1943), Fabe (1950), Stones (1954), Weinmann and Sicher (1955), Lucas and Kramer (1959) and Shafer, Hine and Levy (1959). Most of these authors agree the condition can also result from infection with Staphylococcus albus, various types of streptococci, pneumococci and mixed infections. Kader and Christmas (1951) and Thoma (1954) feel that Staphylococcus aureus and Streptococcus haemolyticus are both common causes of osteomyelitis and also point out the importance of isolating the causal organism, both for purposes of recognition and antibiotic sensitivity tests. Stuteville (1950) on the other hand makes no attempt to isolate the causative organism in osteomyelitis before performing radical surgery.

Too often do we see antibiotics blindly used for treating infections of the jaws, with little or no attempt made to establish drainage and identify the causal organisms.
In cases of this nature antibiotics tend to mask the symptoms and actually prolong treatment.

5. **RADIOGRAPHIC APPEARANCE**
In osteomyelitis there is no radiographic change for approximately 10 days. After this time trabeculae, which are normally distinct and meet each other at sharp angles, become broken up and irregular radiolucent patches are seen (Stones, 1954 and Shafer, Hine and Levy, 1959). As the disease progresses, the picture is characterized by a diffuse, moth-eaten pattern with a tendency towards necrosis in parts. Cahn (1955) points out that this appearance, often taken for complete disintegration, is for the most part hyperaemic decalcification. According to Durbeck (1946), the necrotic areas of bone often contain a considerable amount of calcium deposition, which renders them highly radiopaque.

Sequestration and new bone formation, a defence mechanism of the patient, now take place, leading to a thickened irregular appearance in radiographs. The simultaneous destruction and production of bone is characteristic of the disease in the chronic stage. Durbeck (1946) points out that the radiographic appearance can be similar to some malignant areas, and although sequestration is not seen in malignancy, it can take place if there is secondary infection.

6. **CLINICAL FEATURES**
Acute or subacute suppurative osteomyelitis involving the maxilla usually remains fairly well localized, whereas in
the mandible there is a tendency to more diffuse and widespread involvement. Pain is often the first symptom manifested, intermittent at first but soon replaced by a dull gnawing ache. There is an increase in pulse rate and temperature combined with chills, fever and general malaise.

Swelling and redness of the mucosa or skin does not occur until the infection has reached the periosteum. Muscular trismus, due to involvement and inflammatory contraction of the masseter muscle, and regional lymphadenopathy, often occur and possible involvement of the inferior dental nerve produces anaesthesia or paraesthesia over its distribution.

Intraorally, the teeth involved in the area of osteomyelitis are loose and tender to percussion rendering mastication difficult, if not impossible. It has been found by this author that teeth so affected usually respond to thermal stimuliæ, and provided adequate drainage of the area is obtained, can be retained. Drainage often occurs spontaneously from gingivae or sulci, and sequestra when formed in these regions are sometimes shed. Those occurring at the lower border of the mandible invariably need surgical intervention for their removal.

If osteomyelitis occurs as a complication of a fracture or some operative procedure in which the wound is open, there may be little or no disturbance apart from local swelling, pain and discharge.
The use of antibiotics has, according to Cohen (1954), so altered the condition that it may be difficult to elicit any of the classical symptoms. The violence of the acute phase and length of treatment have been lessened to such an extent that sequestration may be prevented and the symptoms masked, thus rendering diagnosis difficult.

Once drainage has been established, either spontaneously or surgically, the acute symptoms subside, the patient feels vastly improved and the disease passes into the chronic state which persists until final sequestration takes place. During this period, subacute or even acute exacerbations, due to blockage of the sinuses or minor extensions of the condition, may develop.

7. PREVENTION

The importance of refraining from surgical procedures involving bone during acute infections is emphasized by Kayne (1941), Waldron (1943) and Helmore (1958). Actions such as curetting acute abscesses and removing teeth during acute pericoronitis are inviting the production of osteomyelitis. The performing of oral surgery or intra-osseous injections except under aseptic conditions is also contraindicated. Incomplete immobilization of an open fracture and/or the retention of teeth in the fracture line often lead to osteomyelitis which, even if localized in the vicinity of the fracture, greatly prolongs the treatment.

8. DIAGNOSIS

An accurate diagnosis of acute osteomyelitis is possible
from clinical and intraoral examination, together with consideration of the patient's history. As mentioned earlier, radiographic changes, although characteristic of the condition, are delayed approximately 10 days, consequently radiographs are of no value in establishing an early diagnosis and treatment plan. Laboratory tests, carried out during the acute stage will reveal a leucocytosis, 15,000 to 25,000 per cubic mm., with 85 - 95% polymorphonuclear neutrophils (Durbeck, 1946), and will also eliminate systemic conditions or specific infections. Blood cultures may be positive and if consistently so, are indicative of an unfavourable prognosis.

9. **PROGNOSIS**

In the pre-antibiotic era, the prognosis of osteomyelitis was considered grave (Kader and Christmas, 1951) and depended on the patient's response to treatment and the course the disease followed. When drainage and sequestration have been adequately accomplished an involucrum usually forms, and the patient recovers. Salama (1941) on the other hand found the mortality rate low, approximately 1% in 300 cases.

Kader and Christmas (1951) and Stones (1954) feel that the use of antibiotics has produced a more favourable prognosis but there still remains the danger of pathological fracture and mandibular deformity, particularly if radical treatment is used.

10. **TREATMENT**

(a) **Systemic Treatment:** All authors, in describing
the treatment of acute osteomyelitis of the jaws, recommend the use of an antibiotic to limit the infection and prevent complications. Although penicillin was, in the past, the antibiotic of choice, the increased development of resistant strains of Staphylococcus aureus to this drug has prompted clinicians to use other broad spectrum antibiotics.

If osteomyelitis is treated early, according to Tolhurst, Buckle and Williams (1955), complete resolution of the local lesion may be obtained without drainage. Karpawich (1949) reports a case of mandibular osteomyelitis treated successfully by the use of penicillin alone. Even though a large external swelling developed and there was radiographic evidence of sequestration, no surgery was carried out. It is felt by this author that when a case has developed to this stage, surgical drainage would have produced a more rapid result than the relying on penicillin to sterilize the infected area, then waiting for resolution to occur.

There is only slight reference in the dental literature to general supportive measures, which, in this author's opinion, play such an important part in the recovery of an acutely ill patient. However we do find Waldron (1943) and Thoma (1958) emphasizing the importance of rest and diet.

In the acute stage, care must be taken to prevent the patient becoming dehydrated, fluids should be given frequently and intravenous administration may become necessary in a
gravely ill patient. When a patient cannot masticate, because of tender teeth or perhaps immobilization of a fracture, it is important they be told, not only what to eat, but how to prepare it. A diet rich in protein, minerals and vitamins must be prescribed to aid wound healing and bone regeneration.

(b) **Local Treatment:** With regard to local treatment of osteomyelitis there are two schools of thought; firstly radical surgery on the affected area as soon as the acute symptoms have subsided, and secondly, the conservative approach of drainage when and where necessary and the removal of sequestra as their separation is indicated clinically and radiographically.

In this author's opinion, the radical approach, which is claimed by its adherents to produce a rapid cure of the condition, is contraindicated for two reasons;

1. the resultant deformity and loss of teeth.
2. the danger of spreading the infection even when surgery is carried out under extensive antibiotic cover. Cahn (1955) states that serious complications and death can occur following such treatment.

1) **Radical Treatment:** Mowlem (1945), Durbeck (1946), Wass (1949), Stuteville (1950), Moody (1958) and Thoma (1958) all recommend wide exposure of the infected bone, either intraorally or by an external approach,
removal of sequestra, curettage of the granulation tissue and saucerization of the resultant bone defect to allow obliteration of the cavity. Antibiotic therapy is continued postoperatively for 2 - 3 weeks and it is claimed that the wounds generally heal by primary intention.

Moody (1958) favours an intraoral approach with extraction of all teeth involved in the area of osteomyelitis and packing of the wound, thus allowing granulation from the base.

Where the entire thickness of the mandible is involved or bone removal produces deformity, Stuteville (1950) recommends the use of a metal plate to immobilize the bone fragments, and the filling of the defect with cancellous bone chips from the iliac crest. Although complications following this procedure seem inevitable to this author, Stuteville (1950) claims to have treated more than 100 cases successfully.

(2) **Conservative Treatment**: Conservative treatment in the form of establishing and maintaining drainage, antibiotic therapy to prevent further spread of the infection and complications, and the removal of sequestra as indicated radiographically, is recommended by Kayne (1941),

The importance of continuing with antibiotic therapy for at least three weeks after cessation of pain and abatement of fever is stressed by Cahn (1955), who states that recrudescence often takes place if the antibiotic is suspended too early, as a short course of treatment only prevents multiplication and does not cause death of the bacteria.

The need of close supervision of antibiotic therapy is emphasized by Kader and Christmas (1951) and Stones (1954). Frequent antibiotic sensitivity tests should be carried out on the causal organisms to guard against the production of resistant strains. It is felt by this author that a close match for antibiotic toxic effects must also be kept. In the prolonged use of these drugs there is always a danger of gastro-intestinal disturbances, depression of bone marrow function, allergic manifestations and oral fungal infections.

Kallenberger (1949) and Stones (1954) both point out the importance of splinting the mandible should there be any risk of pathological
fracture, and of conserving the teeth. It has been found that vital teeth involved in an osteomyelitic area, although loose, can usually be retained, moreover extraction would risk further spread of the infection.

This author feels that strict attention must also be given to oral hygiene, frequent irrigations of the infected area and, in cases of osteomyelitis complicating a fracture, complete immobilization of the fragments to allow undisturbed callus formation; any movement at the fracture site will result in a fibrous union.

11. CHRONIC SUPPURATIVE OSTEOMYELITIS

Chronic suppurative osteomyelitis usually follows the acute condition, but may arise from a dental infection without a preceding acute stage (Shafer, Hine and Levy, 1959). The clinical features are similar to, but milder than acute osteomyelitis; pain is only slight, or perhaps non-existent with the jaw not feeling perfectly comfortable. Suppuration often perforates the bone with the formation of fistulae on the skin or mucous membrane. Acute exacerbations may occur periodically and the treatment for these and the chronic condition is the same as previously outlined; drainage, antibiotic therapy and sequestrectomy when indicated.

Verbic (1958) presents a typical case in which osteomyelitis of the mandible had pursued a chronic course with
multiple fistulae over a period of five years. Sequestrectomy, débridement and saucerization produced a satisfactory cure.

The use of "Varadase" (Lederle), a combination of a catalytic agent, streptokinase, and an enzymatic complex, streptodornase, which liquefies clotted blood and thick purulent exudates, has been recommended in the treatment of chronic suppurative osteomyelitis by Nichols and Vande Mark (1954).

It is felt by this author that sequestrectomy followed by the use of "Varadase" locally instilled into the cavity is possibly the treatment of choice. The danger of spreading infection in the bone by curettage would thereby be eliminated, although with antibiotic cover the possibility of this complication is remote in a chronic condition.

12. TROPICAL OSTEOMYELITIS OF THE JAWS

It would appear, after reading the reports of Tratman (1934, 1938), Karim (1949) and Allwright (1953, 1958), that these authors are justified in referring to osteomyelitis of the jaws in Eastern peoples, particularly the Chinese, as a tropical disease. Although Allwright (1958) feels there is some doubt as to whether the severity of the condition is due to a racial susceptibility or to circumstances of undernourishment, neglect, overcrowding and bad climatic conditions, both Karim (1949) and Tratman (1938) have come to the conclusion that the Chinese have a high racial susceptibility to osteomyelitis.
Tropical osteomyelitis is characterized by rapid spread of the infection, often across the mid-line and into the rami, with the formation of numerous and/or massive sequestra and multiple sinuses. Treatment is basically the same as that employed in the other forms of suppurative osteomyelitis; Tratman (1934, 1938) and Karim (1949) adopting the conservative approach to allow formation of new bone prior to sequestrectomy, whereas Allwright (1953, 1958) favours the radical removal of sequestra and infected granulation tissue down to healthy bone.
CHAPTER THREE

INFANTILE OSTEOMYELITIS

SYNOPSIS

1. GENERAL INTRODUCTION

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8. INFANTILE OSTEOMYELITIS OF THE MANDIBLE
1. GENERAL INTRODUCTION

Osteomyelitis in infants, that condition which occurs in the first few weeks or months of life is rather uncommon. (Wilensky, 1932). Stones (1954) describes the condition as extremely rare and Shafer, Hine and Levy (1959) state that it is fortunately becoming extremely uncommon, due to the use of antibiotic therapy. Wilensky (1932) claims that 82.5% of cases occur in the first two months of life, the greatest number being in the second and third week.

In the literature, there appears some doubt as to who was first in describing this condition. Thoma (1959) and Cohen (1949) both state it was Klementowsky in 1876, whereas Asherson (1939), Stoy (1953), and Hitchin and Naylor (1957) quote Rees as reporting the first case in 1847.

The rarity of the condition is indicated by the figures of Asherson (1939), 70 cases, and Stoy (1953), 80 cases since the first description by Rees in 1847. Thoma (1954) and Cohen (1949) both state that approximately 80 cases have been recorded since Klementowsky's report in 1876. Asherson (1939) states that of the 70 cases recorded, there are only 42 with definite records. Of this number, 38 cases occurred in the first 12 weeks of life, and ten deaths were recorded.

Wilensky (1932) describes the complex character of osteomyelitis in infants and its various unusual manifestations which emphasize the reasons for it being considered a separate entity. He states that the disease is characterized by:

(a) pathological manifestations associated with the
osteomyelitis which are referable to the mouth, nose, nasopharynx and orbit.

(b) the clinical manifestations associated with an acute infection of severe intensity.

(c) sequestration and loss of the entire jaw and of the teeth which it customarily carries.

(d) the subsequent deformity associated with the loss in the fortunate cases which recover.

(e) a high mortality.

2. DISTRIBUTION

It is generally accepted by all authorities that the maxilla is far more commonly affected than the mandible. Stones (1954) claims 90% of cases occur in the maxilla.

3. ETIOLOGY AND PATHOGENESIS

Practically all writers agree that osteomyelitis of the jaws in infants, as with that of the long bones must be attributed to bacterial infection. Wilensky (1932), Stones (1954), Asherson (1939), Lucas and Kramer (1959), Stoy (1953) and Hitchin and Naylor (1957) all agree that Staphylococcus aureus is the most common infecting organism. This has become the accepted theory. However there are reports of Streptococci and Pneumococci causing osteomyelitis in infants. Wilensky (1932) quotes Allard and Sicard and Thoma (1954) quotes Self (1948) as finding these organisms more commonly associated with the condition in infants than staphylococci. Galli (1926) reports a case of infantile osteomyelitis of the maxilla caused by Bacillus coli.

The majority of observers believe that this form
of osteomyelitis is a primary lesion. The entrance of the organisms taking place through abrasions caused during delivery of the infant or by the cleansing of the mouth at some stage after birth. Trauma during delivery can result from a narrow pelvis, pressure due to the use of forceps or, in the case of a "face delivery", an attendant's fingers being inserted in the mouth and traction applied to the palate to assist the delivery.

Actual infection may take place during birth or later by contamination from the vagina or nipple of the mother or from feeding bottles. Hitchin and Naylor (1957) in recording four cases found three in which the mothers had a breast infection. Stones (1954) feels that the source of infection is not certain, it may be due to infection at, or immediately after birth as described above or in some cases may be haematogenus from infection of the umbilicus or from boils; such instances were observed by Self (1948) and Bass (1928).

Other theories include those of Zarfl (1913), Kelly (1904), and Marx (1922), who believe that the infection in the maxilla is secondary to a primary focus, such as erysipelas or stomatitis which has extended to the maxilla via the lymph channels. Paunz (1926) and Poncher and Blayney (1934) feel that nasal infections and sinusitis are an important cause of the disease. Asherson (1939) on the other hand does not agree with this latter statement. He claims that nasal or antral infections are usually caused by Streptococci and that infantile osteomyelitis of the maxilla is caused by a Staphylococcal infection of the deciduous first molar.
crypt, which at that stage is larger than the maxillary sinus. Fleming (1954) and Reed, Spiro and Wilson (1953) agree that the infection frequently begins in the crypts of one of the upper deciduous molars. Stoy (1953) records one case and traces two others of the condition which appear to be caused by exfoliation of a "natal tooth".

Wilensky (1932) believes that osteomyelitis in infants is in no way different from the haematogenous form occurring in other parts of the body, the infection lodging in the maxilla because of various forms of trauma associated with birth, and especially the cleansing of the mouth. The predilection of the upper jaw being due to its larger size and more rigid construction and attachment to the skull, which favours the more frequent reception of traumatisms. He further emphasizes that abrasions on the alveolar border are pure and simple sites of entry for organisms, and that transmission into the substance of the jaws is only made possible by way of the blood stream and not by simple extension by continuity.

It is extremely difficult to confirm or deny conceptions advanced by the various authors but in view of the delicate nature of oral mucosa in the newborn and the rigidity of the maxilla, it must be agreed that injuries as described could easily occur. The source of infection, whether by external contamination or from the infant itself is unimportant except from the aspect of prevention. I feel that the important predisposing factor must be the intraoral laceration:
and agree with Wilensky's theory on the mode of infection. It is felt that an infected surface laceration would tend to heal or at least be self-limiting, and the reason for infection within the bone substance must surely be a transmission of infected embolus via the blood stream.

In describing the mechanism of pathogenesis Wilensky (1932) feels that the fundamental cause of spreading from the original lesion (usually an infected oral laceration) is an infected thrombus lying in the original area of infection and communicating at some point with the freely circulating blood. Organisms growing on the surface of the thrombus, or parts of the thrombus itself break off, are discharged into the circulation and become lodged in other parts of the body giving rise to a secondary lesion. The actual point of lodgement depends on the blood vascular system in that part of the body. The occlusion of the blood vessel results in deprivation of blood supply and nourishment to part of the bone. The character of the resultant necrosis is in direct proportion to the number, size and importance of the vascular channels occluded and the amount of available collateral circulation.

Although the blood supply of the maxilla is abundant and anastomosis is free, practically all these blood vessels are derived from the one artery, the maxillary. Also a periosteal network is almost non-existent, a fact borne out by the lack of new bone formation in the maxilla after bone destruction. Consequently clinical extent of osteomyelitis in infants can be adequately explained by considering
the arteries affected.

(a) Lesion in the maxillary artery, when collateral circulation is at a minimum, results in a maximum osteomyelitis involving the entire bone with manifestations referable to the palatal, nasal and orbital surfaces.

(b) Lesion in the course of the posterior superior dental artery produces its effect in the alveolar process, the amount of bone involved depending on the collateral circulation.

(c) Lesion in the course of the junction of the posterior and anterior superior dental arteries results in involvement of the anterior part of the alveolar process.

Involvement of the maxillary sinus and tooth buds occurs in these first three cases.

(d) Lesion in the palatal artery results in involvement of the palatal surfaces, or this type of lesion may be an extension from an involvement of the alveolar process.

(e) Lesion in the nasal or anterior branches of the infra-orbital artery produces its effect in the nasal aspects of the bone and may be associated with empyema of the maxillary sinus.

(f) Lesion in the course of the infra-orbital artery produces its effect in the orbital aspect of the maxilla.

MacBeth (1952) agrees with the above classification of Wilensky. It is interesting to note that at birth the distance between orbital floor and palate is less than 10 mm. (Hitchin and Naylor, 1957) and consequently it is not surprising to find in acute maxillary osteomyelitis
of infants, the palate, tooth buds and nasal passages are all involved in the very early stages.

4. **GENERAL CLINICAL FEATURES**

Infantile osteomyelitis of the maxilla presents a clear-cut definite picture, one case report being very similar to another. The disease usually attacks healthy infants between the age of two and ten weeks. Wilensky (1932) states that the onset of the condition differs. In some cases the child cries and refuses nourishment, there may be diarrhoea, constipation or a rise in temperature or these symptoms may be absent. In other cases the child is violently ill, with high temperature, vomiting and convulsions. Asherson (1939) states that severe constitutional symptoms may include a temperature of 104–105°F, a pulse rate of 160 and a respiration rate of 60 together with loss of weight and a septicaemia lasting for 10 days. Following drainage the condition becomes chronic, with sequestra which may persist for years without adversely affecting the patient.

Ober, (1938) distinguishes between three types of osteomyelitis in children:

(a) Sudden onset with severe constitutional reaction characterized by high fever, rapid pulse, vomiting, delirium and prostration.

(b) Although quite ill, child appears to be in excellent physical condition.

(c) Slow onset, slight rise in temperature, and moderate amount of localized pain.
At this stage the swelling appears with associated trismus and facial anaesthesia (MacBeth, 1953). It may begin in the cheek or infra-orbital region or, according to Wilensky (1932) and Round (1936) palatal swelling may occur first. Asherson (1939) and Stones (1954) consider palatal swelling occurs later in the disease. Accompanying the swelling of the cheek there is almost always oedema of the lower eye lid, sometimes exophthalmus from oedema of the orbit, the sclera is inflamed, there is conjunctivitis and occasionally chemosis. It is for this reason that many cases are first seen by the ophthalmologist.

In the majority of cases according to Wilensky (1932), Cohen (1949), Stones (1954) and Hitchin and Naylor (1957) the swelling localizes below the inner canthus of the eye with the formation of a discharging sinus. Intraorally there is swelling, perforation, discharge of pus and sinus formation taking place in the alveolar process and palate. Numerous small sequestra discharge through these sinuses and in the mouth tooth buds may be shed. In practically all cases there is, at some time during the disease, discharge of pus from the nostrils causing difficulty in nursing and loss of appetite. If the condition goes unchecked the final stage may be a single large abscess cavity which harbours the entire or a major portion of the necrotic maxilla. Frequently the primary accumulation of pus is in the maxillary sinus or molar dental crypts.

**Mouth Symptoms.**

Wilensky (1932) states that 80% of the oral sinuses
are found on the alveolar process of which more than 50% are in the canine region. In 60% of cases, discharge of teeth and tooth buds occurs through the sinuses in the alveolar process and this presents the most characteristic phenomenon of the disease. Stones (1954) and Asherson (1939) both consider that the crypt of the unerupted first deciduous molar is almost always involved due to its comparatively large size. Confusion has arisen between the maxillary sinus and this crypt when a draining sinus was explored by means of a silver probe. The final result, if the child survives, is considerable deformity of the face and palate with absence of deciduous and permanent teeth.

**Nasal and Antral Symptoms.**

According to Wilensky (1932) a purulent discharge from the nose is a very common symptom, having a sinus within the nose leading to the necrotic bone. It is interesting to note in relation to the maxillary and ethmoidal sinuses that Asherson (1939) considers infection of these sinuses due to Streptococcal organisms, this fact he considers rules out acute sinusitis as the primary lesion.

**Eye Symptoms.**

Of the 35 cases tabulated by Marx (1922), 30 developed definite eye symptoms, the other five being doubtful in this respect. Eight of the patients sought treatment because of these symptoms. Symptoms of this nature are usually divided into those of the eyelids, conjunctiva, and orbital cavity. Swelling of the eyelids due to severe
inflammation of the bone is the first eye symptom to attract attention. The conjunctivitis follows and is a result of the same bone inflammation. Exophthalmus arises from the inflammation of the bony walls of the orbital fossa and is followed by a purulent exudation.

5. **DIAGNOSIS**

Wilensky (1932) states that differential diagnosis must be made from Ophthalmia neonatorum, Erysipelas, Dacryocystitis, Syphilis and Tuberculosis. Careful examination and history together with laboratory aids should result in a correct diagnosis. The phenomenon of exfoliation of teeth and tooth buds is characteristic of the condition. X-rays, according to Asherson (1939), are not warranted as they necessitate a general anaesthetic. In the later stages they may be helpful in discovering sequestra. Stones (1954), Thoma (1954) and Hitchin and Naylor (1957) all agree that the diagnosis must be based entirely on clinical findings; for radiographic changes occur only late in the condition and, even then are only slight. The small size of the maxillary sinus prevents any opacity being clearly seen.

6. **TREATMENT**

Treatment, prior to the advent of antibiotics, consisted of rigid adherence to the principles of surgery. Rest, warmth, fluids and adequate nutrition were also considered necessary. As fluctuation occurred, drainage was established and later sequestra and tooth germs were removed as indicated by clinical and radiographic examination. Asherson (1939) in stressing the need of immediate and adequate drainage claims,
that intraoral drainage in the canine fossa is not usually successful and incision should be made externally. Wilensky (1932) on the other hand feels that as much drainage as possible should be done intraorally or nasally. Marx (1922) apparently could adequately drain orbital abscesses from appropriately placed incisions in the mouth.

The use of antibiotics has considerably modified the course and sequelae of the disease and as a result Hitchin and Naylor (1957) feel that antibiotic therapy should be instituted immediately without waiting for bacteriological examination.

These writers, do however stress that as soon as pus is available the nature of the organisms and their antibiotic sensitivity must be determined.

The principles of surgical care, drainage and removal of sequestra are just as important now as they were prior to the discovery of antibiotics. With general acceptance of the fact that the majority of maxillary infantile osteomyelitis cases is caused by Staphylococcus aureus, it is to be expected that resistance of the organisms to some antibiotics will be met. Hitchin and Naylor (1957) record a case where bacteriological examination revealed Staphylococcus aureus resistant to penicillin and streptomycin. Terramycin was used successfully. Stoy (1953) also recorded a similar case and Reed, Spiro and Wilson (1953) warn of the likelihood of infecting organisms being penicillin resistant and suggest the routine use of some other antibiotic
while awaiting the results of sensitivity tests on the bacteria. Thomson (1959) states that in Australian Hospitals during the four years ending 1957 there has been an increased incidence of penicillin resistant neonatal staphylococcal infections. Some strains isolated have proved as much as 94% resistant to penicillin. It would appear from these figures that the substitution of some other antibiotic for routine use, prior to sensitivity tests, in infantile osteomyelitis is definitely indicated.

The importance of orthodontic supervision of the patient after recovery is stressed by Thoma (1954) particularly if there has been loss of tooth or bone structure.

7. PROGNOSIS

In the preantibiotic era the mortality rate of infantile osteomyelitis of the maxilla was high. White (1935) giving a figure of 26% and Asherson (1939) a figure of 25%.

In fatal cases according to Wilensky (1932) the infants died as a result of the virulence of the infection before secondary foci have time to develop. MacBeth (1953) does not completely agree with this statement but claims that septicaemia or intracranial complications may cause the death of the patient. Since the advent of antibiotics the condition has become rare and the course and prognosis of osteomyelitis of the maxilla in infants have changed from grave to benign (MacBeth, 1953). The rareness of the condition since the introduction of antibiotics has prevented the accurate assessment of a modern mortality rate.
However deaths do still occur: Hitchin and Naylor (1957) recording one death in their four cases. There can be no doubt however that the course of the disease has been shortened and the possibility of facial deformity and dental abnormalities, considerably reduced.

Prevention of the disease is also discussed by Hitchin and Naylor (1957) who advance evidence supporting the view that the source of the infection is the maternal breast. They advocate bacteriological investigation of the mother's nipples in obstetric cases with any symptoms suggesting breast lesions. This appears a sound and logical procedure, particularly in the light of Thomson's (1959) findings concerning neonatal staphylococcal infections.

8. INFANTILE OSTEOMYELITIS OF THE MANDIBLE

With regard to this condition which comprises 10% of the total osteomyelitis of the jaws in the newborn, Stones (1954), Bronner (1925) and Wilensky (1932) all state that the infections are more simple technically, and ordinary surgical principles should be used in their treatment. As in the maxilla all cases are similar and many give a history of a difficult delivery, particularly a "breech presentation". Ehrenfest (1944) states, "The severest injuries in the infant's mouth are produced in connection with the Mariceau-Weit-Smelli method of delivery in a breech presentation case in which the infant's head appears last. Here, one or two fingers are introduced into the baby's mouth and, if improperly used to exert strong traction, the resulting lesions may vary anywhere between mere
lacerations of the skin in the corner of the lips to complete evulsion of the lower jaw including fracture".

Zugsmith (1951) reported a case which he has had under observation for 18 years. The patient was first seen at the age of 9 weeks suffering from osteomyelitis of the mandible, considerable bone, and two lower deciduous teeth were lost. Periodic examination up to the age of 18 years revealed normal permanent tooth and bone development and no facial deformity.

It would appear from this case report and others concerning the maxilla, that basically the treatment is similar to that adopted for adult osteomyelitis. Provided adequate drainage is maintained, sequestra and teeth causing infection removed, and antibiotics used only to prevent further spread of the infection, the dentition and contour of the jaws can be largely maintained even though the treatment may be of long duration.
CHAPTER FOUR

SCLEROSING OSTEOMYELITIS

SYNOPSIS

1. GENERAL INTRODUCTION

2. CHRONIC FOCAL SCLEROSING OSTEOMYELITIS
   (a) Introduction
   (b) Clinical Features
   (c) Radiographic Appearance
   (d) Histological Features
   (e) Treatment

3. CHRONIC DIFFUSE SCLEROSING OSTEOMYELITIS
   (a) Introduction
   (b) Clinical Features
   (c) Radiographic Appearance
   (d) Histological Appearance
   (e) Treatment
1. GENERAL INTRODUCTION

Sclerosing osteomyelitis of the jaws may be described as a specific form of chronic osteomyelitis usually caused by a low-grade subclinical infection. Thoma (1954) states that the weak toxins produced by this type of infection stimulate osteoblastic activity, thereby causing increased density of bone. Although this is the generally accepted theory, it has been suggested that the cause of the condition is a circulatory disturbance of the nutrient arteries, or the formation of thrombi in smaller intraosseous vessels. Production of diffuse or localized forms depends on the area supplied by the artery.

The factors determining the type of bone response after injury are not all known. But tissue resistance, degree of injury, blood supply and age and general health of the patient must all be considered. Robinson (1956) has included chronic sclerosing osteomyelitis amongst the responses of bone to injury, collectively known as "osseous dysplasias".

The jaws, because of the teeth and associated pathological conditions, provide a site for trauma and infection not often found in other parts of the body. Consequently, chronic sclerosing osteomyelitis is relatively common although only slight reference to it is found in dental literature, probably due, I feel, to the fact that the condition is very often symptomless. Thoma (1954), Shafer, Hine and Levy (1959), Bell (1959), Robinson (1956) and
Shafer (1957) all differentiate between a diffuse and focal type of chronic sclerosing osteomyelitis, Thoma (1954) describing the condition as "ossifying osteomyelitis".

2. **CHRONIC FOCAL SCLEROSING OSTEOMYELITIS**

(a) **Introduction:** This response, according to Shafer (1957), may be regarded as a proliferative reaction of bone found in persons of high tissue resistance following periapical infection caused by microorganisms of comparatively low virulence. The dynamic reactivity of the tissues, coupled with their high resistance, prevents diffuse spread of the infection, and irritation by the infecting organisms is of such a low order that the osteoblasts are stimulated to produce bone rather than being destroyed.

(b) **Clinical Features:** The condition occurs almost invariably, in young persons before the age of twenty and when present is found at the apex of a tooth, usually a lower first molar which is grossly carious. Only occasionally does the patient exhibit symptoms other than those associated with the carious tooth.

(c) **Radiographic Appearance:** Chronic focal sclerosing osteomyelitis, known by some authors as "condensing osteitis", appears in an x-ray as a well circumscribed radiopaque mass of sclerotic bone surrounding, and extending beyond the apex of one or all the roots. The lamina dura surrounding the roots of the offending tooth has been destroyed and, the periodontal membrane widened and merged into the sclerotic
area. The border of the lesion may be smooth and distinct or may blend into the normal bone. The lesion remains after removal of the tooth and may be recognized on radiographs many years later, occasionally being diagnosed as a retained root.

(d) **Histological Features:** Osteoblastic activity may have completely subsided by the time the tissue is examined leaving a dense mass of bony trabeculae with very little interstitial marrow space (Shafer, Hine and Levy, 1959).

(e) **Treatment:** The offending carious tooth is extracted leaving the sclerotic bone which, according to Shafer, Hine and Levy (1959), is an indication that the body has been able to deal effectively with the infection; consequently surgical removal of this bone is not indicated.

This author agrees that the condition, particularly the focal type is common, and also with the treatment advocated by Shafer, Hine and Levy (1959) unless the patient has matured to the degree that removal of the tooth risks the production of a "dry socket". Fortunately this latter condition is not common in the age group usually allotted to chronic focal sclerosing osteomyelitis, but with the diminution in blood supply to the socket and the possibility of trauma due to bone density, consideration should be given to surgical removal of the tooth and associated sclerotic bone.

3. **CHRONIC DIFFUSE SCLEROSING OSTEOMYELITIS**

(a) **Introduction:** The pathological process is basically
the same for both diffuse and focal conditions, and chronic diffuse sclerosing osteomyelitis being endosteal in location, is analogous to the periosteal condition known as Garré’s osteomyelitis.

(b) **Clinical Features:** Chronic diffuse sclerosing osteomyelitis may occur at any age but, in contrast with the focal type, it predominates in middle, or old age. This fact according to Shafer (1957), may be accounted for by the decreased ability of the tissues at this age to contain or limit the infection as successfully as tissues in younger persons. Often the patient is unaware of the condition; other times its presence is discovered during investigation of a dull, poorly localized pain in the jaw, and on still other occasions there is an acute exacerbation of the dormant chronic infection. It is suggested, rightly so I feel, by Bell (1959) that infection is secondary to oral contamination of the osteosclerotic areas following tooth extractions and not necessarily due to pre-existing infection.

Shafer (1957) states that the condition occurs frequently in edentulous persons and, although usually involving the premolar and molar regions, may be found in any region and is not uncommonly found bilaterally symmetrical.

When acute symptoms occur there is mild suppuration with the formation spontaneously, of intraoral fistulae.

(c) **Radiographic Appearance:** The radiographic
appearance is of diffuse bony sclerosis, often involving a large portion of the body of the mandible and blending into the surrounding bone at the periphery. Sequestra of various sizes may be apparent in the case of long standing infections. The radiographic appearance can be very similar to osteitis deformans (Paget's Disease).

(d) **Histological Appearance:** Histological examination of a specimen from the affected area reveals, according to Shafer (1957), osteosclerosis and irregular proliferation of viable bone. The interstitial connective tissue is often somewhat more fibrous than usual and is typically infiltrated by small numbers of lymphocytes and plasma cells.

(e) **Treatment:** The treatment of chronic diffuse sclerosing osteomyelitis presents a difficult problem and it is generally accepted that surgical intervention should be avoided or at least minimized. Shafer, Hine and Levy (1959) and Shafer (1957) claim excision of the involved areas is contraindicated owing to the extensive nature of the disease, but Thoma (1958) considers that if pain becomes a dominant feature, it may be necessary to excise the sclerosed area of bone to decompress the inferior dental nerve. Bell (1959) states that the patients must be treated conservatively and extractions performed only as a last resort. If removal of a tooth becomes necessary, surgical approach is the method of choice, with wide bone removal for access and to expose bleeding cancellous bone. Antibiotic therapy is advocated for acute episodes by Thoma
(1958), Shafer, Hine and Levy (1959) and Shafer (1957) but Bell (1959) points out that it is difficult for parenteral antibiotics to disseminate through the osteosclerotic areas, consequently the local use of antibiotics such as bacitracin and/or neomycin is recommended. The treated condition shows no tendency to regress and further acute episodes are to be expected.

This author feels that we have no choice but to adopt the conservative approach in the treatment of chronic diffuse sclerosing osteomyelitis. If surgery is considered necessary the patient must be acquainted with the possibility of delayed healing, paraesthesia or anaesthesia over the distribution of the inferior dental nerve if it is likely to be involved, intraoral deformity and fracture. We must also agree with Bell (1959) in his statement on parenteral antibiotic therapy and consider it useless and therefore contraindicated, unless there is an associated soft tissue infection. The application of local intraoral heat, maintenance of oral hygiene, sequestrectomy as indicated by clinical and radiographic examination and insertion of adequately relieved dentures when the acute symptoms have subsided, must be considered the treatment of choice.

The importance of further radiographic examination such as x-rays of skull, pelvis and long bones, serological tests and blood analysis should, I feel, be stressed because
of their assistance in developing a differential diagnosis.

It should be noted that both osteitis deformans and syphilitic osteomyelitis can present similar radiographic pictures to chronic diffuse sclerosing osteomyelitis.
CHAPTER FIVE

GARRÉ’S OSTEOMYELITIS

SYNOPSIS

1. GENERAL INTRODUCTION
2. CLINICAL FEATURES
3. RADIOGRAPHIC APPEARANCE
4. HISTOLOGICAL FEATURES
5. TREATMENT AND PROGNOSIS
6. INFANTILE CORTICAL HYPEROSTOSIS (CAFFEY’S DISEASE)
1. **GENERAL INTRODUCTION**

This condition is a very distinctive type of chronic osteomyelitis, first described in 1893 by Carl Garré as a focal gross thickening of the periosteum of long bones with peripheral reactive bone formation resulting from a mild irritation or infection. It is felt by Bennett (1948) that the infectious agent becomes localized beneath the periosteum and spreads only slightly into the interior of the bone, which will become thickened if the cause is not removed.

Shafer, Hine and Levy (1959) and Pell, Shafer, Gregory, Ping and Spear (1955) consider, rightly so I feel, that the condition is essentially a periosteal osteosclerosis analogous to the endosteal sclerosis of chronic focal and diffuse sclerosing osteomyelitis. This chronic focal condition has been referred to as condensing osteitis, sclerosed bone or enostosis. In other parts of the body Garré's osteomyelitis is a well known entity recognised by orthopaedic surgeons and pathologists, being generally overlooked as a distinct condition affecting the jaws until the report of Pell and his associates was published in 1955.

2. **CLINICAL FEATURES**

Garré's osteomyelitis occurs almost entirely in young persons before the age of 25 and the area most frequently involved is the anterior edge of the tibia where it may or may not regress without surgical intervention. When the jaws are affected the condition invariably occurs in the mandible, still almost exclusively in children and young adults. Seldom is the max-
illa affected, although the reason for this is unknown. (Shafer, Hine and Levy, 1959).

With regard to the age distribution Pell and his associates (1955) feel that the clinical symptoms are caused by the "much more dynamic" reaction of young bone compared with old bone. When it is considered that the jaws are exposed to infection far more often than any other bone in the body, it is surprising that Garré's osteomyelitis has not been described more frequently as a dental complication.

The patient suffering from Garré's osteomyelitis usually presents, according to Shafer, Hine and Levy (1959) complaining of toothache or pain in the jaw and a bony hard swelling of the outer surface of the jaw. This mass is usually of at least several weeks duration. Ritvo (1955) lists the same symptoms, but considers the diagnosis more difficult, stating that the condition "— often is not diagnosed until persistence of symptoms leads to exploratory operation. Localized pain, tenderness and the absence of constitutional symptoms and significant laboratory findings should suggest the true nature of the condition".

Thoma (1958) considers the increase in mass of bone is probably due to mild toxic stimulation of the periosteal osteoblasts by an attenuated infection or a compensatory thickening as a mechanical adaptation to reinforce the bone weakened through disease.

3. **RADIOGRAPHIC APPEARANCE**

Thoma (1958) states that the radiographic appearance of
Garré's osteomyelitis is quite typical. The affected portion of bone showing an area of increased density and cortical thickening which in some cases may be localized. Pell and associates (1955) describe it as focal outgrowth or reduplication of the cortical layer of bone. Shafer, Hine and Levy (1959) adhere to this latter view and further state that intra-oral views will reveal a carious tooth opposite the hard bony mass. Ritvo (1955) extends the description by stating that the affected area may be smooth or irregular in outline, the periosteum is not elevated and rarefaction usually does not occur. There are no changes in the overlying soft tissues.

Pell and associates (1955) stress the care necessary to differentiate the condition from Ewing's sarcoma, a neoplasm occurring in the same age group and with a similar radiographic appearance.

4. **HISTOLOGIC FEATURES**

Histologically according to Pell and his associates (1955) the lesion consists of newly formed bone and osteoid tissue showing a mild chronic inflammatory reaction of the fibrous interstitial connective tissue. This immature bone formation in Thoma's (1953) case report exhibited osteophytic pattern. The formation of osteophytes according to Weinmann and Sicher (1955) is an emergency response to strengthen bone weakened through infection and destruction. A spongy bone of mature type is produced forming osteophytes to compensate rapid destruction of bone, which normally would outstrip the rather slow process of apposition of mature lamellated compact bone or even the less slow formation of mature
spongy bone.

5. TREATMENT AND PROGNOSIS

Garre's osteomyelitis is treated by removal of the carious infected tooth with no surgical intervention for the periosteal lesion (Shafer, Hine and Levy, 1959). Following removal of the offending tooth the bony swelling slowly subsides and the jaw contour returns to normal. The treatment outline by these authors is satisfactory if the surgeon is sure of his diagnosis, which would then be verified by the slow regression of the swelling. If there is any doubt as to the diagnosis a biopsy, I feel, is considered advisable at the time of tooth removal to eliminate firstly the possibility of a neoplasim (Ewing's sarcoma - Pell and associates, 1955) and secondly the waiting period for bony changes to be noticable clinically and radiographically as confirmation of the diagnosis of Garre's osteomyelitis.

It is noted that in the case reports presented in the dental literature, one by Pell and his associates (1955) and the other by Thoma (1953) that biopsies were carried out. In the case recorded by Pell and his associates (1955) removal of a carious infected tooth produced relief of pain and considerable regression of the bony swelling in six months. In Thoma's (1953) case extractions in the area had been carried out twenty years previously. Operation consisted of removal of the thickened periosteum and the bulging bone. No post-operative record is given, consequently efficiency of the treatment is hard to assess.

6. INFANTILE CORTICAL HYPEROSTOSIS (CAFFEY'S DISEASE)
This condition was distinguished from Garré's osteomyelitis by Caffey (1950), who claimed it occurred in very young infants involving a number of bones usually clavicles and ulnae and invariably the mandible. Caffey (1950) believes the disease is of infectious origin because of the accompanying high fever and high sedimentation rate, although no bacteria could be cultured from the tissues or fluids and the condition is uninfluenced by sulpha drugs or antibiotics. The disease is self-limiting and eventually regresses.
CHAPTER SIX

TUBERCULOUS OSTEOMYELITIS

SYNOPSIS

1. GENERAL INTRODUCTION
   (a) Tooth Apex and Socket Cases
   (b) Cases Involving Oral Mucous Membrane
   (c) Tuberculosis of the Jaws Resulting in Tuberculous Osteomyelitis

2. PATHOGENESIS

3. BACTERIOLOGY

4. HISTOPATHOLOGY

5. CLINICAL FEATURES

6. RADIOGRAPHIC APPEARANCE

7. DIAGNOSIS

8. PROGNOSIS

9. TREATMENT
1. **GENERAL INTRODUCTION**

Tuberculous lesions in the oral cavity are relatively uncommon and are generally accepted as being secondary to lesions in some other part of the body, particularly the lungs. It was formerly thought that primary lesions did not occur in the mouth but, although they are extremely rare, the reports of Spilka (1955), Miller (1953) and Galloway and Horne (1953) were of such lesions.

Darlington and Salman (1937) classified lesions into three groups:

(a) **Tooth Apex and Socket Cases**: In this group Brodsky (1942) reported an incidence of 8% positive tuberculous periapical granuloma in a study of tubercular patients. Prognosis of these cases is good and the treatment is thorough curettage following extraction.

(b) **Cases Involving the Oral Mucous Membrane**: These are usually a manifestation of a far-advanced pulmonary infection occurring most frequently in patients between 20 and 60 years of age and affecting men more commonly than women.

(c) **Tuberculosis of the Jaws Resulting in Tuberculous Osteomyelitis**

This condition is described as rare by most authors (Stones, 1954; Thoma, 1954; Spilka, 1955; Allen, 1956 - 7; Cohen, 1959 and Pekarsky, 1954). Invariably it is secondary to other lesions in the body and according to Stones (1954), Cohen (1959) and Canepa (1956) the incidence of the condition
decreases with the age of the patient, 60% of all cases of tuberculosis of the jawbone, according to Pilcher and Trauner (1948), occur in children under the age of 16 years.

The mandible shows a greater predisposition to the infection than the maxilla and bony involvement is thought, by Spilka (1955), to be due to extension from the investing soft tissues, although in the case he reported there was no evidence of a soft tissue lesion.

The condition, should it occur in the maxilla, runs a relatively mild course (Oppenheimer and associates, 1951).

2. PATHOGENESIS

Tuberculosis of the jaws may occur in the following ways:

(a) By direct extension from a tuberculous gingival lesion.

(b) From infection of the bone by infected sputum following tooth eruption or extraction. Removal of teeth with periapical granulomata containing tuberculous granulation tissue as reported by Brodsky (1942) may cause spread of the infection to the bone.

(c) From the haematogenous route. It is generally accepted that this is the most common mode of infection with lodgement of the tubercle bacilli in the bone from a primary focus elsewhere in the body. Trauma of any kind may cause localization of the organism at the site of injury.

Shengold and Sheingold (1951) feel that infection may be either endogenous or exogenous but that chronic trauma at the site of infection is the determining factor.
3. **BACTERIOLOGY**

In cases of tuberculous bone infection, smears of sputum or from the tuberculous lesion do not often reveal the tubercle bacillus, consequently a negative film does not exclude tuberculosis (Stones, 1954 and Lucas and Kramer, 1959). Material from the lesion may be cultured, a difficult procedure necessitating special media and at least two weeks for growth, or inoculation of a guinea-pig carried out, in which case four weeks elapses before the result is known.

4. **HISTOPATHOLOGY**

The histopathologic picture is characteristic of tuberculosis. In the lesion there is bone destruction causing gradual resorption of the bone trabeculae and formation of soft tuberculous granulation tissue. There is little tendency to bone repair, except for a certain amount at the periphery. The granulation tissue displays an inflammatory reaction with typical tubercle follicles in which acid fast bacilli can often be demonstrated (Lucas and Kramer, 1959 and Thilander and Wennström, 1956). These tubercles consist of epithelioid cells, which are large and pale staining with large nuclei, the cells being formed from the mononuclears of the reticulo-endothelial system after they have partially digested tubercle bacilli, and Langhans' cells. The latter are giant cells, formed by the fusion of many epithelioid cells, and have many nuclei which are gathered at the periphery, or at the poles (Stones, 1954). These tubercle follicles are surrounded by fibrous
connective tissue.

Bacterial toxins cause caseation in the centre of the lesion and subsequently, softening and liquefaction may take place with discharge through sinuses.

5. CLINICAL FEATURES

Tuberculosis of the bone is characterized by a chronic course and insidious onset. Pain, according to most authors is not usually acute and may be absent, occurring only on mastication. Shengold and Sheingold (1951) on the other hand, claim that cases of tuberculosis osteomyelitis are characterized by severe pain. It is felt that acute pain may be present if secondary infection becomes established or if the inferior dental nerve is involved. Swelling is also a feature of the condition, firm and relatively painless at first, later becoming softer and often rupturing spontaneously either intraorally or externally. Once drainage commences, secondary infection occurs, according to Stones (1954) and Cohen (1959) with freer discharge of pus. Local reaction is poor both in hard and soft tissues; little or no involucrum being formed although sequestration is a frequent occurrence. Trismus, particularly with lesions in the ramus, paraesthesia or anaesthesia from involvement of the inferior dental nerve, loosening of teeth and pathological fracture can all occur.

6. RADIOGRAPHIC APPEARANCE

The radiographic appearance varies with the extent of the condition, ranging from slight changes to extensive
involvement of the jaw. Rarefaction and necrosis are evident and characteristic lack of new bone formation is noted. Sequestra are usually evident in the latter stages and pathological fracture may be apparent.

7. **DIAGNOSIS**

The quickest method of obtaining a diagnosis, according to Lucas and Kramer (1959) is to obtain a biopsy and seek histopathological evidence of the condition. Allen (1956–7) feels that diagnosis of tuberculous osteomyelitis on biopsy alone is difficult to establish as the histological picture may be similar to other chronic granulomata. Thilander and Wennström (1956) claim that certain diagnosis is obtained by either biopsy or inoculation of a guinea-pig.

It is felt by this author that the association of any necrotic lesion with an insidious onset is suggestive of tuberculosis and investigation should include:

(a) radiographic examination of the chest.

(b) skin sensitivity tests with tuberculin, by means of either Mantoux or Heaf test, the latter apparently being more reliable.

(c) smears from the lesion.

(d) smears of sputum (if available).

(e) biopsy (if available).

(f) inoculation of a guinea-pig.

8. **PROGNOSIS**

The prognosis of tuberculous osteomyelitis of the jaws is
considered grave by Thoma (1954), Stones (1954), Pekarsky (1954) and Shafer, Hine and Levy (1959), the mortality rate being estimated at approximately 68%, whereas Thilander and Wennström (1956) feel that the new therapeutic materials coming into use during the last few years have greatly improved the prognosis of oral tuberculosis.

9. TREATMENT

It is universally accepted that general treatment of the patient is the primary consideration and antituberculous measures should be instituted. Streptomycin was the first antibiotic found to be of use in the treatment of tuberculosis and it remains the most effective (Lucas and Kramer, 1959). It is always given in combination with other chemotherapeutic agents such as isonicotinic acid hydrazide (I.N.A.H., isoniazid) or para-aminosalicylic acid (P.A.S.). Both drugs used must be effective singly against the organisms, their combination increasing the therapeutic effect and delaying the emergence of resistant organisms (Thilander and Wennström, 1956 and Rosenthal, 1955\(^2\)). In discussing the effect of streptomycin on tuberculous osteomyelitis, Lafond (1958) considers there is little change radiographically although there is slight, but definite, effect on the lesion clinically.

Local treatment should include rigid oral hygiene, removal of loose teeth, incision and drainage. Sequestrectomy should be carried out when the necrotic bone is
detached as indicated by clinical and radiographic examination (Stones, 1954 and Canepa, 1956). More radical treatment involving removal of infected tissue, curettage and partial resection, is advocated by Thoma (1953), Allen (1956 - 7) and Pekarsky (1954). Evans (1952) feels that elective surgery of tuberculous osteomyelitis need no longer wait for quiescence of the disease. If there is no improvement in the bone lesion after a trial period of chemotherapy alone, radical excision should be carried out.
CHAPTER SEVEN

SYPHILITIC OSTEOMYELITIS

SYNOPSIS:

1. GENERAL INTRODUCTION
   (a) Acquired Syphilis
       (1) Primary Stage
       (2) Secondary Stage
       (3) Tertiary Stage
   (b) Congenital Syphilis

2. BONE CHANGES
   (a) Acquired Syphilis
       (1) Gummatous Destruction of Bone
       (2) Syphilitic Osteomyelitis
       (3) Ossifying Syphilitic Osteomyelitis
   (b) Congenital Syphilis

3. RADIOGRAPHIC APPEARANCE

4. HISTOPATHOLOGY

5. DIAGNOSIS

6. TREATMENT
1. **GENERAL INTRODUCTION**

The incidence of syphilis, a centuries old infectious disease caused by the spirochaete, *Treponema pallidum* has, in recent years with the advent of antibiotic therapy and other epidemiologic control measures, been greatly reduced. Even though the condition has not been completely eradicated, cases showing the typical clinical features are becoming exceedingly rare but none the less important to the dentist when we consider that 35% of all chancre of the finger are reported to occur in this group (Bradlaw, 1958).

Syphilis is transmitted by direct contact in the majority of cases, though there have been instances in which the disease has resulted from contact with infected articles such as cutlery, towels and dental instruments (Lucas and Kramer, 1959).

Syphilis may be acquired or congenital.

(a) **Acquired Syphilis:** Acquired syphilis, if untreated, proceeds through three different phases.

(1) **Primary Stage:** The lesion develops at the site of infection after an incubation period generally of 3 - 4 weeks but, occasionally as long as three months. Shafer, Hine and Levy (1959) claim approximately 95% of these chancre occur on the genitalia but when they occur in the mouth the lips or tongue are the usual sites.
(2) **Secondary Stage:** If untreated the primary chancre heals in 6 - 8 weeks and is soon followed by the secondary or metastatic stage with the development of a generalized rash. The manifestations of this stage tend to disappear in a few weeks but may recur for months or several years.

(3) **Tertiary Stage:** The tertiary stage may follow immediately, or several years may elapse before the characteristic gumma appears. It is at this stage bone lesions occur, most frequently in the tibia, sternum, cranium, nasal bones and hard palate (Dible and Davie, 1945).

(b) **Congenital Syphilis:** Congenital syphilis results from transmission of Treponema pallidum through the placenta, from the mother to the foetus. If the mother is suffering from a recently contracted infection, she is likely to miscarry; when of longer standing, the child may be stillborn and in the case of an old infection, manifestation may be present at birth, develop later or the child may be perfectly healthy. This so-called latent congenital syphilis, which, according to Thoma (1954), occurs in early childhood or more frequently in adolescence, may produce lesions of the skull or facial bones.

2. **Bone Changes:**

   (a) **Acquired Syphilis:** The bone changes of acquired
syphilis, rarely seen until the tertiary stage, can be divided into three groups (Thoma, 1954).

(1) **Gummatous Destruction of Bone**: The periosteal gumma invades the underlying bone causing gradual destruction, as seen in the hard and soft palate. Gumma of the mandible and lymph gland involvement, following such lesions, are rare.

(2) **Syphilitic Osteomyelitis**: The mandible is usually affected although the condition has been reported in the maxilla (Cowden, 1944) where the symptoms are less acute. In the mandible, the clinical features are similar to pyogenic osteomyelitis but no cause of the infection may be apparent at first examination. In untreated cases, necrosis of the bone may occur with the formation of massive sequestra and the possibility of pathological fracture.

(3) **Ossifying Syphilitic Osteomyelitis**: This is the productive stage of syphilis with new bone formation which, according to Thoma (1954), may simulate osteogenic sarcoma.

(b) **Congenital Syphilis**: Bone lesions once again occur in the tertiary stage and, Alaroze (1929) claims that these lesions are manifested in three different forms.

(1) **Osteoperiostitis** which begins with swelling and pain and terminates in suppuration and
sequestration.

(2) Gummatous osteoperiostitis involving a small area.

(3) Gummatous osteomyelitis involving the greater part of the bone.

3. **RADIOPHGRAPHIC APPEARANCE**

Gumma of the bone is seen as a radiolucent area caused by osteolytic changes. Syphilitic osteomyelitis usually shows exactly the same picture as the purulent form, with the possibility of sequestra formation and pathological fracture. In the subsequent stage of ossifying syphilitic osteomyelitis, there is increased radiopacity, usually irregular in appearance, with thickened cortex due to the new bone formation.

4. **HISTOPATHOLOGY**

Syphilitic bone lesions are all related to the formation of gummata, localized granulomatous lesions being composed of lymphocytes and plasma cells (Stones, 1954). Obliteration of the lumen of the blood vessels interferes with proper nutrition of the bone, leading to a degenerative process and necrosis. The enlarging gumma may infect overlying epithelium and cause its breakdown. Spirochaetes are not easily demonstrated in the gumma, being present in small numbers only, but are generally abundant in the broken down mucosa and exudate (Lucas and Kramer, 1959 and Thoma, 1954). Healing is characterized by scarring whether in bone or soft tissues.
5. **DIAGNOSIS**

Diagnosis can be difficult unless a complete case history is taken and supplemented with laboratory investigations. Even negative serological tests, such as the Wasserman reaction and the Kahn Flocculation test, do not exclude tertiary syphilis and these should be repeated as long as clinical suspicion remains. The case reports of Bell and Arnim (1957) on congenital syphilis, and that of Cowden (1944) on syphilitic osteomyelitis both stress the confusion in diagnosis brought about by delaying serological tests, and both authors advocate the use of these investigations routinely in cases where syphilis could be a diagnosis.

It is felt by this author that in all cases of chronic ulcers, chronic swellings and bone infections, routine serological tests should be carried out. This applies to cases of osteomyelitis in which the cause appears obvious, as it is possible for the lesion to become secondarily infected with spirochaetes, or the condition to be syphilitic originally and then masked by acute infection which forces the patient to seek treatment.

6. **TREATMENT**

Intravenous injections of neoarsphenamine supplemented by intramuscular injections of bismuth were used to treat the disease systemically in the past, but the course of treatment was of considerable duration. According to Lucas and Kramer (1959), penicillin, to which Treponema pallidum is sensitive, has considerably altered the condition, effecting a cure in a much shorter period and with less risk of toxic effects.
Tolhurst, Buckle and Williams (1955) state that aureomycin and terramycin have been used with success but are not as reliable as penicillin. Although the institution of antisyphilitic measures is the essential feature of treatment, local treatment must not be overlooked. Incision, drainage, removal of sequestra, as indicated by clinical and radiographic examination, maintenance of good oral hygiene and adequate diet are as important in the treatment of syphilitic osteomyelitis as in pyogenic osteomyelitis.

In all cases of chronic syphilis, according to Tolhurst, Buckle and Williams (1955), examination of the cerebro-spinal fluid should be made and found negative before a cure is claimed.
CHAPTER EIGHT

CHEMICAL AND THERMAL NECROSIS

SYNOPSIS

1. OSTEOMYELITIS ARISING FROM MERCURY POISONING

2. OSTEOMYELITIS ARISING FROM ARSENIC NECROSIS

3. OSTEOMYELITIS ARISING FROM PHOSPHORUS NECROSIS
   (a) General Introduction
   (b) Clinical Features
   (c) Treatment

4. OSTEOMYELITIS FROM THERMAL NECROSIS
   (a) Electrocoagulation
   (b) Effect of Drilling Into Bone
It must be emphasized at the outset that these conditions arise primarily from chemical and thermal bone necrosis, the necrotic bone becoming infected from exposure to the oral fluids, thereby giving rise to osteomyelitis.

Chemical necrosis may be caused by mercury, arsenic, phosphorus or any caustic agent.

1. **OSTEOMYELITIS ARISING FROM MERCURY POISONING**

Thoma (1954) states that mercury poisoning often caused in the therapeutic use of the metal, affects the oral mucosa. This is followed by chemical necrosis of the bone and later, when infection sets in, by osteomyelitis. Schour and Sarnat (1942) consider it can be an oral manifestation of an occupational disease in bronzers (gun barrels), battery and paint makers, dentists, detonators, explosive and mercury salt workers due to contact with the chemical dust.

Osteomyelitis of the jaws following mercury poisoning is, according to Major and Bononi (1939), entirely different from the common type of pyogenic osteomyelitis and is, they feel, due to extension down the periodontal membrane from the necrotic gingiva, caused by the presence of mercury in the mouth. The most striking feature of the process is its limitation to the alveolar portions of the maxilla and mandible, rarely extending much beyond the level of the teeth apices.

The prognosis is excellent in cases of chemical osteomyelitis due to mercury poisoning, the condition running a fairly acute course and being surprisingly self-limiting.

2. **OSTEOMYELITIS ARISING FROM ARSENIC NECROSIS**
Although arseni trioxidum is now rarely used to effect devitalization of the dental pulp, Stones (1954) claims that in the past it has, with commoner usage, been the cause of necrosis. Escape of the drug sealed in a cavity either by way of the apical foramen or past the filling can cause death of a small section of alveolar bone and generally involves the loss of the tooth.

Chemical osteomyelitis following arsenic necrosis, according to Schour and Sarnat (1942), can also be an oral manifestation of an occupational disease in chemical workers, electroplaters, metal refiners, rubber mixers, lead smelters and insecticide makers.

3. OSTEOMYELITIS ARISING FROM PHOSPHORUS NECROSIS

(a) General Introduction: The necrosis once seen in workers' jaws in match factories where yellow phosphorus was used, is now almost non-existent due to the replacement of this chemical with the red, amorphous variety and also, according to Stones (1954), to the precautionary measures now taken. The outbreak of World War II brought with it the increased use of phosphorus. Due however to the knowledge of the precautions necessary to avoid phosphorus necrosis, employees in factories using the chemical had, claimed Kennon and Hallam (1944), little to fear. These writers also state that we have passed the stage of massive sequestra and their case reports are of small carious sequestra in cyst-like "cavities".

It is generally accepted that phosphorus fumes are the cause of the necrosis but, in relation to mode of
action there are two theories. Firstly, the original view was of local absorption through a lesion in the oral cavity and subsequent irritation of the periosteum by the vapour (Thoma, 1954). Secondly, it was considered that the chief effect was from inhalation of the fumes and distribution of the chemical throughout the blood stream, lowering the resistance of bones to infection. Rushton (1944) is in agreement with the latter theory, particularly in the light of the fact that:

1. phosphorus necrosis has occurred as long as two years after exposure to the chemical has ceased and,
2. the negative results of Stubenrauch's (1899) attempts to produce the lesion by plugging phosphorus into holes drilled in dogs' jaws.

(b) Clinical Features: Dull pain is associated with the early stages when a red area appears on the mucosa to be followed by sinus formation and swelling (Stones, 1954). There is, according to Wakefield (1948), little or no temperature rise indicating, he believes, that sepsis is secondary to the phosphorus necrosis. Both Wakefield (1948) and Stones (1954) claim that radiographic evidence is far behind the actual disease. When sequestra form, they are surrounded by an area of radiolucency, much wider than that usually associated with osteomyelitis and resembling, as mentioned by Kennon and Hallam (1944), a sequestrum in a dental cyst. The formation of an involucrum from perios-
teal apposition, completely surrounding the necrotic bone, is claimed by Thoma (1958).

(c) **Treatment**: The treatment of phosphorus osteomyelitis includes free drainage, antibiotic therapy to control the superimposed infection, and removal of the necrotic bone as indicated by its separation. Wakefield (1948) favours this conservative approach thus preserving the regenerative powers of the mucoperiosteum and, feels that dilute copper sulphate irrigations are helpful in checking the extension of phosphorus necrosis. Kennon and Hallam (1944) and Stones (1954) advocate, in contrast to their treatment of acute infectious osteomyelitis, that the cavity containing the sequestrum should be curetted and the resultant blood clot left undisturbed. Thoma (1958) states that, due to the unusual involucrum formation characteristic of phosphorus osteomyelitis, sequestrectomy may necessitate the cutting of a large window in this new bone.

Chemical osteomyelitis can also be caused by the application of other caustic agents to bone surfaces, and subsequent infection, a classic example being the case report of Alexander (1952). Paraformaldehyde crystals were introduced into a lower third molar socket in mistake for sulphanilamide crystals, with resultant loss of the second molar and sequestration of the bone surrounding the third molar socket.

The occurrence of chemical necrosis has in recent years
lessened, no doubt due, in the industrial field to the increased knowledge of the condition, improved safeguards and manufacturing processes, and, in the dental field, to the replacement of arsenic by instrumentation under local anaesthesia for pulp devitalization. Unfortunately accidents due to the erroneous use of caustics will probably go on while there are dentists willing to store drugs in bottles or jars which normally contain other drugs, and to place such drugs in extraction wounds or bone cavities.

4. **OSTEOMYELITIS FROM THERMAL NECROSIS**

Thermal necrosis of bone can be brought about in the mouth by two methods, namely electrocoagulation of the soft tissues and drilling into bone, both of which are avoidable using reasonable care.

(a) **Electrocoagulation:** Electrocoagulation, as used in the treatment of neoplasms of oral and associated tissues, particularly those lesions adherent to the jaw, can produce heat sufficient to kill the periosteum and adjacent bone. The soft tissue slough which follows causes further denudation and exposure of necrotic bone to infection. Sequestration, according to Thoma (1958), occurs early and generally the continuity of the jaw is maintained even though a large part of the jaw may be lost.

(b) **Effect of Drilling Into Bone:** Although there is little reference in the literature to the use of burs and drills on the jaws intraorally, it is felt that the undesirable sequelae arising from the misuse of skeletal pins,
as outlined by Anderson and Finlayson (1943), are in no way different to those produced in the mouth by abuse of rotary cutting and drilling instruments.

Thoma (1958), Anderson and Finlayson (1943), Brown, Fryer and McDowell (1949), Ivy and Curtis (1943) and Thompson (1958) all agree that fast drilling of bone, particularly if dense, will generate so much heat at the pin position that an actual localized cauterization occurs. Anderson and Finlayson (1943) use the term "aseptic thermal necrosis" to describe this coagulation and burning of the haversian canals, and state that a doughnut-like or ring sequestrum will be expressed one or more months later.

Any exposure of an area of "aseptic thermal necrosis" to infection in the mouth, as in a tooth socket or fracture line, will produce a localized osteomyelitis with subsequent sequestration.

Experimental work on the mandibles of dogs has been carried out by Thompson (1958) to determine the histological response to, and thermal changes involved in, drilling bone at various speeds. Study of the reactions following speeds ranging from 125 to 2000 R.P.M. with the Winter modification of the Roger Anderson extraoral skeletal pin, indicate that the ideal speed is 500 R.P.M. This speed, obtained by means of an electric drill, produced a minimal histological response and thermal change in the bone and yet was fast enough to prevent fragmentation and irregular pin hole
margin as seen at lower speeds.

The use of hand drills, which usually have a speed of approximately 125 R.P.M., is advocated by Ivy and Curtis (1943) who feel there is difficulty in gauging the depth and assessing the possibility of bone necrosis with electrically driven instruments.

The danger of "aseptic thermal necrosis" with subsequent osteomyelitis and sequestration, I feel is to be guarded against during:

1. removal of bone with a bur during oral surgery,
2. drilling of socket walls to promote blood supply in an effort to prevent "dry sockets" and,
3. upper border wiring of fractures.

These undesirable sequelae may be prevented or at least greatly reduced by:

1. use of slow cutting speeds (The portable electric drills in use in the Oral Surgery Department of the United Dental Hospital of Sydney have a minimum speed of approximately 400 R.P.M.).
2. use of sharp, new cutting instruments applied with firm pressure, to ensure the bur or twist drill as used in upper border wiring, cuts rather than burnishes. Constant observation of the cutting edges is necessary to insure that clogging or bluntness can be seen should they occur.
(3) use of twist (engineer's) drills which are self-clearing, mounted in a dental handpiece for making holes preparatory to the insertion of "upper border wiring" in mandibular fractures.

(4) use of constant saline spray on the bur head for cooling.

This author has seen ring-like sequestra with loops of stainless steel wire passing through them shed on several occasions, particularly where the dense external oblique ridge in the lower third molar region has been drilled. Although the "upper border wiring" is usually lost in these cases in the second or third week after operation, satisfactory union is taking place in most patients even though sequestration has occurred at the intraoral end of the fracture line, thus indicating the infection is localized in the area of the "aseptic thermal necrosis".

It must be remembered that bone is a poor conductor of heat, consequently that produced by drilling is not rapidly dissipated and may if sufficient, produce necrosis.
CHAPTER NINE

OSTEORADIONECROSIS

SYNOPSIS

1. INTRODUCTION

2. APPEARANCE AND INCIDENCE

3. EFFECTS OF IRRADIATION ON TISSUES
   (a) Skin
   (b) Mucous Membrane
   (c) Salivary Glands
   (d) Teeth
   (e) Bone

4. ETIOLOGY

5. RADIOGRAPHIC APPEARANCE

6. HISTOPATHOLOGY

7. CLINICAL FEATURES

8. PREVENTION
   (a) Preirradiation Prevention
   (b) Prevention During Irradiation
   (c) Postirradiation Prevention
9. TREATMENT

(a) Conservative Approach
(b) Radical Approach
1. **INTRODUCTION**

Osteoradionecrosis of the jaws is a frequent complication following irradiation of cancer in the oral cavity and neighbouring structures (Cutler, 1951). The condition has been called "radiation osteomyelitis" by Thoma (1954) and "radio-osteomyelitis" by Seldin and associates (1955). Although these are more accurate descriptions of the process, bone necrosis followed by infection, most authors prefer to use the term osteoradionecrosis.

The effect of irradiation on bone and teeth in experimental animals was first noticed as early as 1905, only nine years after the discovery of radio-activity. In 1922, Regaud of the Curie Institute of Paris, was first to observe cases of osteoradionecrosis of the maxillary bones occurring beneath uninjured skin during the treatment of introral cancer. He attributed the more marked effect on bone to secondary radiation arising from the calcium deposited in this tissue.

The condition may affect adults and children alike, and with the latter may result in retarded development of teeth and underdevelopment of facial bones (Cutler, 1951).

2. **APPEARANCE AND INCIDENCE**

The period of time elapsing between irradiation therapy and osteoradionecrosis is variable. There have been reports of it occurring as early as two months and as late as twelve years after therapy (Cernea and Bataille, 1947).

In a series of 1,819 patients treated with irradiation,
reported by Watson and Scarborough (1938), 235 or 13% developed osteoradionecrosis. Extractions were carried out in 94; 38 before and 56 after irradiation. On the other hand Jacobson (1948) reported a much lower incidence, 5.6% in 267 cases of lingual carcinoma.

Osteoradionecrosis rarely affects the maxilla, although in cheek lesions it receives as much irradiation as the mandible. This is due, according to Brandon and Herd (1949), to the greater vascularity of the maxilla. MacLennan (1955) found 83% of cases affected the mandible but he felt the cause was chiefly the greater number of irradiated lesions in this area, the poor blood supply being of minor importance. Gaisford and Rueckert (1956) claim that the preponderance of cases in the mandible is due to its ribbonlike shape, compact composition and presence of but a single nutrient artery on each side.

3. EFFECTS OF IRRADIATION ON TISSUES

Low-Beer (1951) states that the direct effect on the tissue cell is disruption of the nucleus and cytoplasm which leads to ultimate death of the cell. The degree of cellular change is in direct relation to the amount of radiation absorbed by the tissues. Warren (1943) and Lochman (1960) feel there is little difference in the effects produced by high, and supravoltage irradiation except a slight alteration in the timing of the reaction.

There is a marked difference in sensitivity of various tissues to irradiation; young cells are more sensitive than
mature ones and endothelial, or epithelial cells are more easily affected than connective tissue cells. Cartilage is more sensitive than bone, whereas muscle and nerve tissues are the most resistant (Macomber and associates, 1957).

(a) **Skin:** Reaction of the skin, according to Low-Beer (1951) and Shafer, Hine and Levy (1959), consists of erythema varying from simple reddening to bullous epidermolysis. This reaction subsides in four to eight weeks after treatment, leaving only a slight depigmentation of the skin. In time more marked atrophy with telangiectasis may develop, and massive doses of irradiation may produce swelling, oedema, exudation, ulceration and necrosis (Macomber and associates, 1957).

Prior to the advent of supervoltage and megavoltage irradiation therapy, the skin reaction was one of the limiting factors in treatment.

(b) **Mucous Membrane:** The changes in mucous membrane are similar to those of skin and, following regeneration in four to eight weeks, it is paler than normal with atrophy of the superficial layers and fibrosis of the submucosa. There is diminution of sensation, temporary loss of taste and the tongue loses its filiform and fungiform papillae.

(c) **Salivary Glands:** Low-Beer (1951) states that with small doses of irradiation there is an alteration in the consistency of the saliva from both parotid and submandibular glands. It becomes sticky and frothy, adheres to the mucous
membrane and loses its normal lubricating function, owing to an excess of mucus. Subsequently, secretion ceases entirely and the mucous membrane becomes shiny and dry. According to Lochman (1960), these reactions are usually transient but may persist throughout life with unpleasant secondary changes in the tissues of the oral cavity.

(d) **Teeth:** "Radiation caries", a peculiar caries like lesion at the cementoenamel junction, often occurs in patients who have received irradiation involving the oral cavity. It has been stressed by many authors that this tooth destruction occurs in all teeth, irrespective of their direct involvement in the field of irradiation. These lesions have been attributed to the alteration in $pH$ of the saliva by del Regato (1939), whereas Stafne and Bowing (1947) feel that many patients' habits of holding fruit drops or other acidic confections in their mouths to relieve the sensation of dryness could be another contributing factor.

Experimental studies carried out by Kaplan and Bruce (1953) on Syrian Hamsters showed that irradiation caused incomplete root formation, dwarfing of teeth and anodontia. Burstone (1950) and Medak, Schour and Klauber (1950) found eruption is retarded or, with increased dosage, stopped in mice and rats.

In children following irradiation involving forming tooth buds, Rushton (1947), Stafne and Bowing (1947), and Bruce and Stafne (1950) all noted delayed eruption,
variation in size of crown and root and, if the injury is extensive enough, anodontia.

(e) Bone: According to Shafer, Hine and Levy (1959), bone itself is relatively resistant to irradiation although osteoblasts are quite sensitive. If irradiation has been sufficiently intense, the normal balance between bone formation and resorption is disturbed, bone vitality is decreased and it is unable to resist infection in the normal manner.

The principal effect of irradiation on bone, according to Ewing (1926) and Shafer, Hine and Levy (1959), is the production of vascular disturbances decreasing the blood supply to the bone. This damage is usually permanent so that if infection enters, even years after irradiation, the danger of osteoradionecrosis exists. Watson and Scarborough (1938) and Niebel and Neenan (1957) suggest that bone receives, not only primary irradiation but also the more damaging secondary radiation from the calcium salts in the intercellular substance of bone. Consequently the actual amount of irradiation received by bone is much greater than the computed dose.

4. ETIOLOGY

Most authors agree that osteoradionecrosis of the jaws is produced by a combination of heavy irradiation, trauma and infection.

(a) Intensive Irradiation Therapy: The function of the periosteum and bone marrow is altered by the primary
irradiation as well as the secondary irradiation from calcium particles in the bone. The blood vessels become occluded by thrombosis or proliferating fibrosis.

(b) **Trauma:** Any type of trauma breaking the mucous membrane predisposes to infection. Extraction of teeth, trauma from dentures, gross periodontal lesions or bone involvement by the treated tumour, have all been considered responsible for invasion of microorganisms into the sub-epithelial tissues which are less resistant to infection.

(c) **Infection:** According to Niebel and Neenan (1957), entrance of organisms into the irradiated tissue appears to be the most constant single factor in initiating necrosis. These authors have never seen a purely aseptic case.

5. **RADIOGRAPHIC APPEARANCE**

Thoma (1954) states that no radiographic changes in the jaw can be detected in early osteoradionecrosis. The earliest evidence of the condition is a thickening of the periosteum, later, according to Thoma (1954) and Gaisford and Rueckert (1956), there is extensive bone destruction with possible sequestrum formation.

McIndoe and associates (1947) state that radiographically "there may be areas with a relative increase in density which may be confused with osteoplastic metastasis, or there may be areas of absorption of bone resembling osteolytic secondary processes. With secondary infection, such as occurs in the mandible, there is marked decalcific-
ation and absorption of bone which may be impossible to
distinguish from invasion of the bone by recurrence of
the neoplasm*.
6. **HISTOPATHOLOGY**
In uncomplicated irradiation necrosis, the fatty marrow
undergoes degeneration and fibrosis with a few inflam-
matory cells, polymorphonuclear leucocytes and lymphocytes
present. The trabeculae and bone cells are poorly stained
indicating bone necrosis (Stones, 1954 and Thoma, 1954).
Some osteoclastic activity is noticeable but the osteo-
blasts of the periosteum and spongiosa are usually de-
troyed so that bone regeneration is limited or completely
absent. Blood vessels show evidence of thrombosis (Thoma,
1954). It has been pointed out by McLennan (1953) that,
when there is minimal irradiation damage, the osteoblasts
not destroyed respond with formation of new coarse sclerotic
bone concurrently with the degenerative changes.
7. **CLINICAL FEATURES**
In the early stages of osteoradionecrosis, according to
Gaisford and Rueckert (1956), there may be little more than
hypersensitivity of the teeth, continuous dull tooth ache
and carieslike lesions at the cementoenamel junction of the
teeth. In the later stages, symptoms are extremely severe
and constant. There is marked trismus and a continuous,
deep boring pain which does not respond well to the usual
narcotics. Ulceration of the skin or mucosa may cause
exposure of large portions of necrotic bone with suppuration
and possibly haemorrhage (MacLennan, 1955). The formation of sequestra, compared with other forms of osteomyelitis, is extremely slow as the blood supply to the affected bone is restricted by endarteritis, making it incapable of responding to infection in the normal manner (Thoma, 1954).

Because of the trismus and pain, patients with this condition experience great difficulty in eating, consequently they are underweight and malnourished, sometimes to the point of starvation.

The lack of involucrum formation and slow sequestration are conducive to pathological fracture which, should it occur, greatly prolongs the treatment.

8. PREVENTION.

(a) Preirradiation Prevention Seldin and associates (1955) and Gaisford and Rueckert (1956) feel that many incidences of osteoradionecrosis can be prevented by proper dental management prior to irradiation. The majority of authors (Watson and Scarborough, 1938; Holland, 1948; Darland, 1949; Brandon and Herd, 1949; Lyon, 1950; Cook, 1952; Niebel and associates, 1957; Topazian, 1959 and Lochman, 1960) consider all infected and carious teeth in the mouth, plus all teeth in the path of irradiation, should be removed before treatment is commenced. Cook (1952) and Niebel and Neenan (1957) suggest that surgical removal of the teeth and alveolectomy be carried out to shorten the healing time and minimize the clot size; early organized blood clot is extremely sensitive to irradiation.
and is thus prone to breakdown. It is generally accepted that 10 to 14 days should be allowed for socket healing prior to commencement of irradiation therapy.

Elmer and associates (1959), following experiments on irradiated dogs' mandibles, expressed the though that "removal of teeth prior to irradiation of the mandible significantly decreased the incidence of osteomyelitis".

Stones (1954) adopts a slightly more conservative approach when he states that, if there is a possibility of extraction becoming necessary within two or three years after irradiation, the teeth should be removed before treatment commences. It is felt by this author, and substantiated by many case reports in the literature, that extraction even outside Stone's (1954) limit of three years is often followed by osteoradionecrosis.

Trotter (1931), Kanthak (1941), Lawrence (1946), Smith (1948), Cade (1949), and Schultz (1950) all believe that the entire mouth should be rendered edentulous prior to irradiation, whereas Quick (1941), Paterson (1948), Sarnat and Schour (1950) and Wildermuth and Cantril (1953) consider extraction prior to irradiation more hazardous than following irradiation when proper oral hygiene permits removal under more ideal conditions. These authors state there is a risk of spreading the tumour if teeth are involved and also, a delay in lifesaving treatment while sockets are allowed to heal.

When we consider that 12 patients free of cancer, died
as a direct result of jaw necrosis in the 235 cases of osteoradionecrosis reported by Watson and Scarborough (1938), it would be wise to heed Castiglione's (1957) warning ... "a handful of teeth is not worth a life."

It is felt by this author that the approach adopted by Topazian (1959) and Sleeper (1950) is the soundest. It may be summarized as follows:

(1) All infection in soft tissues should be eliminated.
(2) The mouth should be made as clean as possible.
(3) All infected or non-vital teeth should be surgically removed.
(4) All teeth in the line of irradiation should be surgically removed.
(5) All teeth periodontally involved should be surgically removed.
(6) If the parotid and submandibular glands are to be heavily irradiated, or if the mouth shows general neglect, all teeth should be removed surgically.
(7) The patient should be thoroughly instructed in maximum oral hygiene.
(8) No irradiation should be attempted for 10 to 14 days after removal of the teeth.

(b) Prevention During Irradiation: Prevention of osteoradionecrosis during treatment is confined to minim-
izing the amount of bone irradiated, protecting the bone when soft tissues are irradiated and warning the patient of the consequences of trauma to the irradiated area.

Reduction in the size of the area irradiated by using the smallest possible portal of treatment, consistent with rational therapy, has been suggested by Brandon and Herd (1949), Cutler (1951) and Topazian (1959) as a method of reducing the incidence of dental complications and osteoradionecrosis. Martin and Sugarbaker (1940) believe this method greatly reduces these unpleasant sequelae.

As a further protective measure Brandon and Herd (1949), Low-Beer (1951) and Topazian (1959) suggest the use of lead shields and individually adapted lead cones for intraoral irradiation. On the other hand Fulton (1951) states that lining applicators with lead to localize rays is of little value as, with the thickness of lining available, it only offers protection against 2% of the rays. Niebel and Neenan (1957) and Topazian (1959) state that a patient being treated with irradiation therapy should be warned of the consequences of surgical intervention without proper precautions, trauma of any type to his jaw and also the wearing of dentures. Low-Beer (1951) and Green (1960) are of the opinion that it is inadvisable to insert dentures over irradiated bone at any stage.

This author feels that more stress should be placed on warning patients of the complications to be expected after irradiation. One patient, in his experience,
developed osteoradionecrosis following the extraction of two lower molar teeth approximately 16 months after 39 consecutive days of "cobalt beam therapy" for carcinoma of the throat. Prior to his dental treatment, the patient had been asked whether he had received medical treatment or attended a hospital recently but felt that his "ray treatment" was not important enough to be included in this category.

(c) Postirradiation Prevention: The postirradiation prevention of osteoradionecrosis is a controversial issue. While all authorities agree that trauma should be avoided, there is much dissension as to the period of time that should elapse before extraction or denture insertion.

While we find that Brandon and Herd (1949), Sleeper (1950), Castigliano (1957) and Topazian (1959) consider extraction of teeth from an irradiated jaw is never to be considered an entirely safe procedure, Kanthak (1941), Darland (1949), Cutler (1951) and Stones (1954) feel that extractions can be carried out after several years. On the other hand, Wildermuth and Cantril (1953) claim that teeth can be extracted with the "proper support and caution" and have reported cases of postirradiation extractions six months after treatment with no apparent ill effects.

Niebel and associates (1957) claim that teeth can be safely removed from irradiated bone by the use of elastic bands, similar to orthodontic or fracture bands, placed over the crown of the tooth, carried to the gingival margin
and then allowed to work their way along the root taper towards the apex. The teeth being treated are ground out of occlusion and thorough prophylaxis should be given prior to application of the bands. With multirooted teeth, root canal therapy and sectioning to separate the roots may be necessary. Exfoliation of the tooth takes place primarily by bone resorption around the roots, and, to some degree, from extrusion.

Seldin and associates (1955) recommend the use of long acting local anaesthetic solutions or alcohol nerve blocks to combat dental pain, and Sleeper (1950) and Watson and Scarborough (1938) advocate pulp extirpation and root canal therapy as a means of avoiding extractions.

Cook (1952) has recorded a case of osteoradionecrosis of the mandible due to denture irritation occurring 13 years after the patient's last exposure to radium, and we find Low-Beer (1951), Niebel and Neenan (1957), Thoma (1958) and Green (1960) all suggest that dentures should never be inserted when bone has been irradiated. Castigliano (1957), on the other hand, claims that dentures may be worn after 18 months or two years.

It is felt by this author that extractions from irradiated bone are to be avoided at all costs even though the area may appear normal clinically and radiographically. When we consider the seriousness of osteoradionecrosis, it is obvious that painful teeth should be treated conservatively, pulp extirpation carried out if necessary, and if
extraction is unavoidable, consideration should be given to atraumatic removal of teeth with elastics as advocated by Niebel and associates (1957).

It is important that both general and dental health of the patient be as sound as possible to limit risk of infection. The necessity of strict oral hygiene and regular dental examinations should be impressed on the patient. With regard to dentures, it is better to dispense with them altogether, a little care in selection and preparation of food will enable an adequate diet to be maintained without the risk of initiating osteoradionecrosis.

It is suggested by Topazian (1959) that questions concerning X-ray and radium treatment be included in the routine history taking before any oral surgery procedure. This appears a very sound suggestion when we consider the seriousness of osteoradionecrosis and the increasing number of patients having malignancies, being treated and cured by these methods.

9. **TREATMENT**

The treatment of osteoradionecrosis is sharply divided into conservative and radical measures.

(a) **Conservative Approach**: Conservative treatment, which is suggested by Cook (1952), Stones (1954), Thoma (1954), Seldin and associates (1955), MacLennan (1955) and Niebel and Neenan (1957), is similar to that used in suppurative osteomyelitis except that, treatment is generally prolonged as sequestration is extremely slow in osteoradionecrosis.
Antibiotics should be used to confine the disease as much as possible and also combat associated soft tissue swellings. Adequate drainage should be maintained and strict oral hygiene enforced. The importance of a rich, easily digested diet should be stressed for, if the condition is at all extensive, mastication may be impossible. MacLennnan (1955) suggests a nasogastric tube may even be necessary.

Where pain is a major complaint, analgesics should be employed but, narcotics are to be avoided where possible as the condition is usually protracted. When drugs are ineffective against the pain, long acting local anaesthetic solutions or alcohol nerve blocks may be used or, as suggested by Niebel and Neenan (1957), severing of the nerve could be carried out.

Sequestra must be treated very conservatively, no attempt being made to remove them until there is evidence of their separation clinically and radiographically. Once bone becomes exposed, it must be irrigated frequently and, Seldin and associates (1955\(^2\)) suggest sharp free ends may be removed with rongeur forceps.

If pathological fracture should occur, simple atraumatic immobilization should be used. Union is slow but according to Seldin and associates (1955\(^2\)) usually occurs.

It is felt by this author that conservative treatment is the method of choice and waiting, until the sequestra can be removed without trauma, will ensure smaller loss of
bone and soft tissues, and consequently a minimum of deformity.

(b) **Radical Approach**: Darland (1949) advises prompt action when bone becomes exposed, either removal with rongeurs or electrocoagulation. He feels that exposed bone will not sequestrate spontaneously and, treating it with electrocoagulation, will cause its separation as a thermal sequestrum with some chance of the deeper bone healing. Should this method fail, Darland (1949) joins with Brandon and Herd (1949) La Dow (1950) Gaisford and Rueckert (1956) and Marchetta and Solomon (1958) in recommending surgical excision of all irradiated bone in the region.

Gaisford and Rueckert (1956) and Marchetta and Solomon (1958) feel that, with present day antibiotics, anaesthesia and blood replacements, the majority of patients can be prepared and carried through the necessary surgery without involving a great risk. It is felt by this author that La Dow (1950) sums up the position admirably ... "There is no panacea once this retrograde process (osteoradionecrosis) has commenced. Prevention is the only remedy".
CHAPTER TEN

CERVICO-FACIAL ACTINOMYCOSIS

SYNOPSIS

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The arrangement of this review follows the headings set out by Dr. N. E. Goldsworthy in his paper on actinomycosis, published in The Dental Journal of Australia, June, 1947 which I consider covers the subject as well as, if not better than any other publication I have reviewed.

1. GENERAL INTRODUCTION AND HISTORY

Actinomycosis, a disease which occurs frequently in cattle, horses and pigs, less often in other animals and rarely in man, was first observed as it appeared in cattle, by Leblanc in 1826. Rosh and Seldin (1948), in reporting this fact state that, at this stage the condition was classified as osteosarcoma, and it was not until 1876 that Bollinger, a pathologist at Munich, first recognized actinomycosis as a specific parasitic disease.

The micro-organism detected by Bollinger, was found while investigating the purulent discharge from a lesion in the jaw of a cow. Cope (1952) recounts that Bollinger showed the specimen to his botanical colleague Harz who, finding "a feltwork of hyphae" surrounded by a series of radiating filaments, named the organism "actinomyces" the derivation arising from two Greek words meaning "ray" and "fungus". The following year, 1877, Bollinger published an account of these findings and since then the word actinomyces has been used to denote the whole group of organisms, one of which called "Actinomyces bovis" was that found in the cow by Bollinger.

Two years later Ponfick found a similar organism in
a human patient and in 1885 Israel published a clinical study of 38 cases in man. By 1891, Wolff and Israel had succeeded in recovering the organism from an active lesion and isolating it in pure culture. It was further shown by these investigators that the organism which causes actinomycosis in man and animals was anaerobic; a fact that has gradually gained support until to-day, it is almost universally accepted. During the same year Bostroem attempted to culture the organism aerobically; little success was obtained, a few of his cultures yielding an organism which, on his description, Robinson and Ennever (1948) consider belongs to the genus streptomyces, probably a chance contaminant.

Cervico-facial actinomycosis in animals and man develops as a swelling in the region of the jaws, especially the lower. This swelling enlarges, and an abscess pointing externally if undisturbed ruptures with discharge of pus and the establishment of one or more sinuses. Local extension of the disease takes place with further sinus formation accompanied by healing and cicatrisation at the previous sites. Goldsworthy (1947) and Cope (1952) both state that untreated, the disease either regresses after several months or much less commonly ends fatally. In the pus discharged from the lesions, can often be seen small granules which on microscopic examination consist largely of filamentous micro-organisms. These can be isolated in pure culture and proven to belong to the genus Actinomyces, generally
microaerophilic in habit.

2. CLINICAL ASPECTS

(a) Nature of the Disease: Although Goldsworthy (1947) states that actinomycosis is generally regarded as a chronic disease, he does point out that such a classification tends to exclude those infections which run a subacute course. Stones (1954) and Burket (1946) describe it as a chronic inflammatory process whereas Cope (1952), Arnott and Ritchie (1949), Thoma (1954), Lucas and Kramer (1959) and Ludwig (1955) all describe both acute and chronic forms. Robinson and Ennever (1948) feel that actinomycosis may occur in many different forms and Shafer, Hine and Levy (1959) claim that the infection is usually, though not always chronic. From clinical experience the author feels that the condition does present in two separate clinical forms.

(b) Sites of Infection: The cervico-facial type is the most common form of actinomycosis, the other sites most frequently involved being the abdomen and thorax although the lesion may occur in almost any position. The percentage of cervico-facial cases range from 44%, Cope (1930) through 57%, Ziskin, Shoham and Hanford (1943), 60% Burket (1946) and Gold and Doyne (1952), 69% Dobson and Cutting (1945) to 76% Wilenius (1954).

In the cervico-facial area the majority of cases occur in the region of the lower jaw, being estimated by Goldsworthy (1947) as 50% of all cases of actinomycosis.
The rarity of this condition in the maxilla is indicated by the almost complete lack of case reports of actinomycosis in this region; Ludwig (1955) records a case, in a 35 year old female, apparently arising from a palatal swelling on an abscessed upper central. Actinomycotic lesions are not common in the tongue, occurring, according to Burkett (1946) in 3.7% of all cases of actinomycosis. Dorf - Petersen and Findborg (1954) state that less than 65 cases have been published in the literature and the most frequent location of lingual actinomycosis is the anterior third, this being due undoubtedly to the fact that the anterior part of the tongue is more prone to trauma than other parts. Figi (1926) found 12 cases in the tongue in 725 cases of cervico-facial actinomycosis at the Mayo Clinic.

(c) Clinico-Pathological Types: The acute and chronic forms described by Cope (1952), Goldsworthy (1947), Thoma (1954), Arnott and Ritchie (1949), Lucas and Kramer (1959) and Ludwig (1955) are considered.

(1) The acute form of cervico-facial actinomycosis is at first indistinguishable from any other acute abscess, causing the usual symptoms of pain, swelling, difficulty in swallowing and trismus. The abscess which forms, points rapidly on the skin surface and as claimed by Arnott and Ritchie (1949) drainage can readily be established. It is essential at this stage
that adequate bacteriological examination be carried out and effective systemic treatment commenced to prevent further spread of the disease. The condition now becomes chronic with continued slight drainage of pus from the wound. Goldsworthy (1947), states that in this acute form suppurative predominates over fibrosis to such an extent that clinically the condition is similar to other infections (cellulitis arising from carious teeth, pericoronitis, fractures, osteomyelitis etc.).

(11) The chronic form of cervico-facial actinomycosis is characterized by a slowly developing swelling, trismus due to infiltration of the masseter muscle and, once drainage has commenced, the formation of scar tissue. The infective process spreads in the subcutaneous or submucous connective tissue by direct continuity (Stones, 1954).

It is generally accepted by all writers that involvement of the bone is rare, Graessner (1929) found it was affected in only 5% of his cervico-facial cases. This complication should it occur, produces periostitis and osteomyelitis of the jaw, with possible formation of sequestra and bone loss in the latter condition. The lymphatic system is not involved unless secondary infection is present to produce lymphadenitis according to Stones (1954)
and Thoma (1954) but Cope (1952) states that although the lymphatic system is relatively immune to attack, there are several cases on record in which the fungus lodged in glands and caused their enlargement. Burkett (1946) lists actinomycosis as a cause of lymph node enlargement in the cervico-facial region, the nodes being soft and "putty-like" and sometimes matted together. The treatment of actinomycosis with lymph nodular extracts is based on the observation that regional lymph nodes are never involved in the disease (Sung, 1944 and Arlotta, 1952).

(d) Course and Duration of the Disease: MacNab (1945) considers the average duration of treated cervico-facial actinomycosis is three months with a range between six weeks and five months. This agrees with the majority of authors although Hamilton (1945) and MacGregor (1945, 1951) give minimum durations of three and four weeks respectively.

(e) Prognosis: Although actinomycotic infection of the thorax and abdomen carried, in the past, a high mortality rate, estimated by various authorities as between 50% and 90%, Cope (1938) claimed a 97% recovery rate in cervico-facial infections in the pre-antibiotic era.

The recovery of patients suffering from cervico-facial actinomycosis can now, since the advent of antibiotics and sulphonamides be confidently anticipated and a search of the literature has revealed only one death in recent times. Holmes (1958) records the death of a 37 year old male who developed a lesion on the side of the neck, which was diagnosed and treated as actinomycosis for 18 months before the patient
succumbed to a spread of the infection to the thorax and spine.

3. **THE CAUSAL ORGANISMS.**

Although there has been much confusion in the past as to the exact identity of the causal agent in actinomycosis, due to the fact that both parasitic and saprophytic actinomyces occur in normal healthy mouths, mouths of patients suffering from the infection and in the actual lesions; it is now almost generally accepted that the microaerophilic Actinomyces bovis (Wolff and Israel) is the causal organism of actinomycosis in man. Goldsworthy (1947) describes the organisms as long, filamentous and Gram-positive but staining somewhat irregularly; slow growing in culture, being heaped up in coherent colonies, adherent in a variable degree to the medium (if solid).

The organisms growing in the tissues exhibit true branching (Stones, 1954), a characteristic only possessed by higher bacteria and fungi (Lucas and Kramer, 1959). Goldsworthy (1947) states that, under ordinary conditions of laboratory technique, the distinctive character of branching filaments may be so far suppressed as to require much patience and experience to demonstrate even rudimentary budding. Consequently both in pus and cultures Actinomyces bovis commonly appears as diphtheroid rods, a small but variable proportion of which show rudimentary branching. Lucas and Kramer (1959) apparently do not agree with this description for they state that when grown on artificial culture media
this budding is generally absent.

As previously mentioned the organism is microaerophilic which according to Rosh and Seldin (1948) and Lucas and Kramer (1959) allows its proliferation in areas and lesions where other organisms such as streptococci and staphylococci have established favourable conditions by the lowering of the oxygen tension in the tissues.

Actinomyces bovis is a strict parasite and has never been isolated from any other sites than the alimentary tract and actinomycotic lesions in man and higher animals (Goldsworthy 1947, and Thoma 1954). Its distinction from the non-pathogenic aerobic actinomyces, found in soil, grain and grasses is important. These organisms are rapid growers in culture at room temperature, and the colonies are pigmented, something which does not occur with Actinomyces bovis.

4. PATHOLOGY AND HISTOPATHOLOGY.

All authorities give similar descriptions of the pathology and histopathology of actinomycosis. Although connective tissue seems particularly susceptible, no tissue appears immune from the disease which spreads by direct continuity, especially along connective tissue plains.

The reaction in the tissues is one of acute or chronic inflammation, a colony of actinomycotic organisms lying in an area of necrotic tissue infiltrated by polymorphonuclear leucocytes. Where these colonies occur in clumps and are yellowish in colour due to the adherent pus, "sulphur" granules are seen macroscopically. Around these foci of
suppuration the more chronic type of reaction, with mononuclear cells predominating, takes place and fibrous tissue is laid down. Acute conditions favour suppuration whereas chronic conditions are indicated by increased fibrosis and external scarring.

5. **GEOGRAPHICAL DISTRIBUTION.**

Although it has now been proved almost conclusively that actinomycosis is not carried to man by way of contaminated vegetable matter, there still appears the occasional article in medical and dental literature claiming the disease is more prevalent in farming districts and is exogenous. Lorenz (1958, 1959) states "Man may be infected directly by transmission from diseased animals or indirectly by chewing grass, straw or grain which has been contaminated by the fungi. Actinomycosis therefore is more often observed in people living on farms than in other classes of population".

Analysis by Lamb, Lain and Jones (1947) of the 839 cases of actinomycosis reported in America between 1920 and 1940 revealed that it was not confined to rural areas nor was it more prevalent within such groups.

6. **AGE AND SEX DISTRIBUTIONS.**

While no age group is exempt the majority of patients, according to Goldsworthy (1947) and Cope (1952) are aged between 15 and 35 years, due, they feel to the fact that this age group exhibits a high caries and resultant extraction incidence. Ziskin, Shoham and Hanford (1943) extend
this group to include people aged between 20 and 60 years and claim its rarity below the age of 10. These writers also state actinomycosis to be slightly commoner (58%) in males than in females whereas Cope (1952) and Goldsworthy (1947) state it is twice as common in men as in women although no satisfactory explanation for this fact has been advanced. Burket (1946) claims the disease is usually found in middle life, the majority of cases occurring in males due to greater exposure to infection (male agricultural workers).

7. **RELATION OF THE DISEASE IN MAN TO THAT IN ANIMALS.** Transmission of actinomycosis to man by way of straw, etc. contaminated by diseased animals is highly unlikely as Actinomyces bovis (the causal organism of actinomycosis in man) is, according to Lucas and Kramer (1959) strictly parasitic and cannot exist for any length of time outside the body. Goldsworthy (1947) accepted at the time of his writing, the theory that the organisms producing disease in man and cattle were the same but considered that there was little evidence of infection being transmitted from one to the other. Erikson (1940) on the other hand, reported the isolation of 12 human and 7 bovine strains of actinomyces which could definately be separated into two different groups. Lentze (1950) emphasized that in Europe, there was no proof that the disease was contracted through infected grasses containing the fungus and Dobson and Cutting (1945) found in 16 cases of actinomycosis, 11 of which were in
the cervico-facial region, only one patient with any contact at all with cattle, straw, etc. Lesney and Traeger (1959) reported two cases of cervico-facial actinomycosis following extractions in sailors who had been on prolonged sea duty, thereby eliminating all possibility of contact with animals or contaminated vegetable matter.

Burket (1946) on the other hand feels that the majority of actinomycotic infections occur in male agricultural workers, probably due to the greater exposure of these people to infection. Although not stated specifically, Burket infers, I feel, that the disease is thereby transmitted from animal to human. Stadnicki, Krajnik and Baranžak (1958) state that the 69 cases of cervico-facial actinomycosis they observed in the 1954 - 1958 period, were caused by Actinomyces israeli and transmitted from animals mainly cattle, horses and pigs.

8. MODE OF INFECTION.
The mode of infection has been a subject of contention since Wolff and Israel in 1891 put forward the theory that the causal organism was anaerobic, and not found outside the body. This statement followed close on Bostroem's work in 1890 from which he deduced that the organism causing disease in man was an aerobe found in grains and grasses. Thus for many years there have existed two schools of thought on the nature of the organism and the mode by which it causes infection. Although over the years evidence has accumulated from which it has become increasingly obvious that Bostroem's
theory should be rejected, publications still appear upholding his views. As outlined in a previous paragraph on Geographical distribution, Lorenz made a statement supporting Bostroem's theory, as recently as 1959. Burket (1946) claims a higher incidence in farm workers due to greater exposure to infection and although he describes both aerobic and anaerobic organisms, there is no clear cut description of pathogenicity. In fairness to Burket, it is noted that he follows with the statement, "The infection is now believed to be chiefly endogenous".

It should be remembered in this regard that, if as has been shown by many workers, Actinomyces bovis is a normal inhabitant of the oral cavity, the habit of chewing grass and straw attributed to farm workers, could easily produce the trauma necessary to introduce the already present organisms into the tissues. There in a wound with necrotic tissue presenting ideal conditions, growth of the organism, with formation of a clinical lesion could take place.

The theories of Bostroem, and Wolff and Israel, both agree that trauma occurs before infection is set up. In this regard Ziskin, Shoham and Hanford (1943) consider that trauma and the presence of the organism are not in themselves sufficient to produce actinomycosis. It would appear that this view is reasonable when we take into account that:

(a) trauma in the oral cavity is common.

(b) the organisms can be found in many normal mouths.
pain or tend or are affected there plant a

benefit immediately or the other or to the editor of course

the case listed so be found in each state

Romans
(c) cervico-facial actinomycosis is not a common disease, in fact it has been described as rare by Stones (1954).

Goldsworthy (1947) has summarized the evidence in favour of the theory of endogenous infection and his statements, a precis of which appear below, are in agreement with those of other experienced authors.

(a) The organism in a vast majority of cases is a strict parasite, never having been found outside the body.

(b) The organism is microaerophilic.

(c) There are usually small loci of diseased tissue in most mouths.

(d) Necrotic matter is always associated with these loci.

(e) The oxygen tension in these necrotic loci is lowered.

(f) Microaerophilic organisms could find suitable conditions here, if not for active growth, at least for survival.

(g) Actinomyces bovis is microaerophilic and has been found in such sites in persons who have not suffered, are not suffering and not in immediate danger of suffering from actinomycosis.

(h) Trauma is necessary for the establishment of diseases due to anaerobes like Clostridium tetani and Clostridium welchii. Trauma usually leads to necrosis, which in turn leads to the lowering of oxygen tension.
necessary before anaerobes or microaerophilics can become established in the animal body.

(i) Trauma in the mouth is by no means uncommon (inflicted by the dentist or self-inflicted).

(j) In any septic area in the mouth or alimentary canal, secondary invaders may help to lower the oxygen tension and so facilitate infection by organisms of the Actinomyces bovis type.

9. PORTALS OF ENTRY OF THE CAUSAL ORGANISM.

It is now generally accepted that Actinomyces bovis is a normal inhabitant of the oral cavity, being found in carious teeth, dento-bacterial plaques, gingival crevices, periodontal pockets, apical granulomas and tonsillar crypts. Robinson and Ennever (1948), and Goldsworthy (1947) consider that the primary invasion of the jaw is probably through ulcerated or abraded gingiva or oral mucosa or through exposed dental pulps. Wright (1905) was the first to suggest that Actinomyces bovis was a natural inhabitant of the oral cavity and intestinal tract and also claimed that foreign bodies such as straw and grass were not carriers of the fungus, but rather transmitted the organisms already present in the mouth into the tissues. Lord (1910) showed that the organisms were present in carious teeth and tonsils of normal healthy patients, and was able to produce symptoms of actinomycosis in experimental animals by inoculation with the culture thus obtained. He failed to isolate the organisms in pure culture.
Sullivan and Goldsworthy (1940) reported a comparative study of anaerobic strains of actinomyces from clinically normal mouths and from actinomycotic lesions. From this study the following results were obtained.

(a) Isolation of five microaerophilic strains of actinomyces from the exudate of actinomycotic lesions in the cervico-facial region, and six from the mouths of subjects not suffering from obvious clinical actinomycosis. Five of these strains were isolated from 100 periodontal pockets and one from 24 carious teeth.

(b) A comparative study indicated that these groups were identical.

(c) The authors agreed with other workers that Actinomyces bovis is part of the normal flora of the mouth. Emmons (1935, 1936) and Slack (1942) have also isolated this organism from normal mouths in pure culture.

(d) The acute form of the disease is a not infrequent sequel to surgical manipulation.

Ennever, Robinson and Kitchin (1951) reported the isolation of 13 different strains of actinomyces from dentobacterial plaques found at the contact points between human teeth and removable appliances. An organism which was indistinguishable morphologically from Actinomyces bovis was isolated by Gee and Sullivan (1940) in an apical granuloma located near an external sinus, and Hardwick and Newman (1955) observed a closed pulpal abscess associated with an actinomyces like organism. Crowley (1944) has found organisms of
this nature in root canals of human teeth during root canal therapy, and Villa (1957) states that actinomyces-like bodies are commonly encountered in dental pulps undergoing inflammatory changes or necrosis, particularly when they have been exposed by caries. Usually they appear as mere contaminants producing no reaction in the pulp. He reports a case, the first he has seen, where a lesion similar to that seen in the jaw has developed within the pulp.

10. THE ROLE OF TRAUMA.

Injury, in the form of extractions or fractures, is the most common predisposing factor in cervico-facial actinomycosis. Sanford and Magath (1922) felt that extraction or dental sepsis were a predisposing factor in most of their 199 cases, and Hamilton (1942) found 15 of his 25 cases apparently arising from extractions. Dobson and Cutting (1945) reported 16 cases of actinomycosis, 11 being in the cervico-facial region of which 9 were associated with extractions. In contrast to these findings and many others citing extractions as a likely predisposing cause, Henry (1940) says "It is most infrequent to find it (actinomycosis) following dental extractions".

Further suggestive evidence of the importance of trauma and the habitant of Actinomyces bovis is provided by Robinson (1944) who reports a case of actinomycosis of the subcutaneous tissues of the forearm following a human bite, and Ziskin, Shoham and Hanford (1943) who record a case of an actinomycotic lesion developing on the knuckle of a finger injured against
a tooth during an altercation.

11. PREVENTION.

There are few references in the literature dealing with the prevention of actinomycosis. Robinson and Ennever (1948), after considering the reports of actinomycosis following extractions, state that the accumulation of data has been conducted in too haphazard a manner to afford a means of appraising any one of several factors that have been considered important in the development of the disease. However, because of the ever-present possibility of infection by this route, the dental surgeon should attempt to remove as much of the debris from the oral cavity as possible before surgery. While considering prophylaxis ideal, they state that the use of hydrogen peroxide sprays to facilitate mechanical removal of debris from the gingival sulci may be an adequate measure. It is felt by the author that there is no substitute for mechanical prophylaxis and clinical observations of the decrease in post-operative pain, infection and delayed healing show its value.

Goldsworthy (1947) approaches the matter with a wider view, considering first the preventive measures as applied to the community. Any action likely to lead to the transfer of micro-organisms from one person to the other should be avoided. The hygiene of eating and drinking in hotels, restaurants, etc. where there is a possibility of using improperly washed utensils is considered. Although the organisms responsible for actinomycosis are microaerophilic,
it is possible for them to remain active long enough to be transferred from one mouth to the other in the above situations.

Secondly, Goldsworthy (1947) deals with preventive measures applicable to the individual, stressing the need for good oral hygiene, regular treatment of carious and periodontal lesions and avoidance of self-inflicted trauma with tooth brush, tooth-picks or floss-silk.

Finally he draws attention to the care necessary in dental treatment to minimize trauma and achieve as high a standard of asepsis as possible, thereby lessening the risk of infection for both dentist and patient. It should be stressed at this stage that these precautions must be applied at all times, not only to lessen the risk of infection in relation to the jaws, but also to minimize the risk of systemic infections (e.g. hepatitis following injections of contaminated local anaesthetic).

It is also felt that a reminder in adequate sterilization and fixing of bacteria to the mucous membrane surface or skin to prevent passage of organisms into the tissues should be given. Particularly to be avoided is the injection of local anaesthetic solutions into infected tissues. Lovestedt (1948) recommends the use of anaesthetic-penicillin solutions for infiltration and conduction anaesthesia to lessen the danger of post-operative infection and to enable the operator to work in an infected field. Apart from the
danger of producing resistant strains of organisms and of sensitizing the patient to penicillin, this procedure is, I feel, to be condemned also on the grounds of spreading the already present infection or in relation to actinomycosis, of introducing these organisms into an infected area which could be ideal for their proliferation if any necrosis had taken place thereby lowering the oxygen tension. Broadbent (1954) also deals with the use of penicillin combined with local anaesthetic solutions but feels its use in acutely inflamed tissues is contraindicated because of the danger of spreading the infection.

12. **DIAGNOSIS.**

Thoma (1954), in discussing the diagnosis of actinomycosis, states that it is almost entirely dependant on microscopic examination of the discharge from a fistula or of excised tissue. Goldsworthy (1947) on the other hand claims that diagnosis is a matter of weighing the available clinical evidence and then seeking for more specific information by laboratory methods. Shafer, Hine and Levy (1959) go one stage further, in saying that the diagnosis of actinomycosis is dependant not only upon the clinical findings in the patient and the demonstration of the fungi in tissue section or smear, but also upon the culture of the fungus. It is felt that this latter step is included in Goldsworthy's "laboratory methods".

The author feels that Thoma (1954) is, in his statement, overlooking the fact that most dental surgeons have, by the
time they have established drainage of a swelling, formed a reasonably accurate provisional diagnosis from a close study of x-rays and clinical evidence. Lucas and Kramer (1959) sum up the situation admirably: "The first essential in the diagnosis of actinomycosis is the suspicion that the patient may be suffering from the disease and the communication of this suspicion to the bacteriologist". It is also felt that the insistence of Shafer, Hine and Levy (1959) on "culture of the fungus" has little clinical application; the organism, a slow growing anaerobe, is often found associated with other organisms and by the time it is isolated in pure culture and identified, the condition is often cured. Consequently the diagnosis and treatment plan must be made in the manner described by Goldsworthy (1947).

It would be a grave clinical error to withhold treatment until the organism is isolated in pure culture which in the author's experience can take anything from four days to two weeks and occasionally longer.

Diagnosis, which must be made from other acute or chronic inflammatory conditions and simple or malignant tumours, is thus derived from consideration of the following:

(a) Clinical Evidence: An accurate history of the condition is of the utmost importance; not only the suspected lesion (usually external to the lower jaw) but the whole mouth must be carefully examined for conditions such as, carious teeth, fractures, recent extraction sites, pericoronitis, etc.
The pus from the external sinus or surgical incision when collected, must be examined carefully macroscopically. The importance of collecting the first few drops of pus which, as stressed by Lucas and Kramer (1959), are most likely to contain "sulphur" granules if they are present, must not be forgotten when drainage is established surgically. Close inspection of the pus obtained by rolling it around the test tube, may reveal the presence of "sulphur" granules, without which some authorities incorrectly I feel, refuse to allow a diagnosis of actinomycosis. The majority of authors (Thoma, 1954; Cope, 1952; Stones, 1954; Lucas and Kramer, 1959) state that "sulphur" granules are present in most cases but are not essential for the diagnosis of actinomycosis.

This author feels that Robinson and Ennever (1948) sum up the position admirably: "The discharged pus sometimes exhibits "sulphur" granules", and "The absence of "sulphur" granules from the pus should never be taken as positive evidence that actinomycosis does not exist. Conversely, small masses of bacteria, food debris, bone spicules, cell debris or other foreign matter in pus may appear macroscopically similar to "sulphur" granules in the absence of actinomycosis". These statements have been confirmed on many occasions in cases treated or observed by this author.

The presence of indurated tissues and persistently draining multiple fistulae as described by Burket (1946) as suggestive of cervico-facial actinomycosis has fortunately
become rare, undoubtedly due to improvement of living standards, medical facilities, patients willingness to seek treatment and the availability of effective antibiotics to combat the disease.

(b) **Bacteriological Evidence:** This must always be sought because without it the diagnosis of actinomycosis cannot be regarded as established (Goldsworthy, 1947). A "sulphur" granule, if available, is crushed between two glass slides, or a direct smear of the pus made and stained by Gram's method. The presence is then sought of Gram-positive branching filaments or "diphtheroid" rod-forms showing rudimentary branching or budding (Goldsworthy, 1947) as first evidence of the presence of actinomyces. Final identification of the organism as aerobic or micro-aerophilic is not possible until the organisms are cultured under both conditions for at least four days, preferably seven.

This author has on several occasions treated patients with infections caused by organisms of the aerobic Nocardia group, commonly found in the soil and not usually pathogenic to man (Bergey, 1957). Gruber (1952) also reports a similar case. The few organisms in this group capable of causing disease in man are, like Actinomyces bovis, a normal inhabitant of the mouth. The incidence of disease caused by these organisms is far lower than that due to Actinomyces bovis and the clinical picture generally is not similar to actinomycosis.
(c) **Histological Evidence:** In the absence of bacteriological evidence, histological evidence is valuable, but in general, it is less accessible, insofar as it involves biopsy (Goldsworthy, 1947). Thoma (1954) states that the colonies are not easily found, often it being necessary to prepare a large number of sections before they are discovered. Robinson and Ennever (1948) claim a relatively high diagnostic value in finding the colonies of actinomyces in the tissues, but Goldsworthy (1947) states that the microscopical features of the lesion are not peculiar to actinomycosis.

13. **Treatment.**

The literature presents a multitude of different treatments each claimed by its originator or adherents to be the most successful. These treatments include a wide variety of drugs, vaccines, lymph nodular extracts, x-ray therapy and surgery. A high proportion of cases of cervico-facial actinomycosis appear to recover, irrespective of the type of treatment, and it would seem that Goldsworthy is probably right when he suggests firstly, that none of the numerous drugs recommended is of value as a specific remedy, and secondly, that the cervico-facial type of the disease is almost always self-limiting.

This author has observed three cases which healed without recurrence after surgical drainage only, before the bacteriologist's report of actinomycosis was finalized.

(a) **Surgical Treatment:** Surgical treatment can be
divided into two groups:

(1) Incision of the abscess as soon as possible to promote drainage and to obtain material to verify the provisional diagnosis of actinomycosis. Free drainage is recommended by most authorities (Thoma, 1954; Stones, 1954; Burket, 1946; Arnott and Ritchie, 1949) and Goldsworthy (1947) emphasizes the need of elimination of likely factors in the aetiology of the condition (removal of carious teeth; immobilization of fractures etc.).

(2) Complete excision of the lesion is, according to Goldsworthy (1947) and Thoma (1954), a means of obtaining an effective cure but naturally only in selected cases. It is difficult to imagine complete excision of a lesion arising from an oral focus without gross tissue destruction and the danger of further break down at the surgery site. Arnott and Ritchie (1949) confine this more radical surgery to final scar elimination.

(b) Medical Treatment: Prior to the discovery of sulphonamides and antibiotics many drugs were used in the treatment of actinomycosis. Iodine in many forms, copper sulphate, thymol and salvarsan were all used, but none apparently were as satisfactory in the treatment of cervicofacial actinomycosis as surgical incision or excision of the
lesion. It must be remembered that the treatment of these cases is vastly different to that of abdominal or thoracic cases. Infections around the jaws are easily accessible whereas those in abdominal and thoracic regions are so placed that systemic treatment is usually the only method of controlling the disease. It is not then surprising that in the pre-antibiotic era cases, other than those in the cervico-facial region carried a high mortality rate.

The discovery and generalized use of antibiotics has provided a generally satisfactory method of treatment. Penicillin is accepted as the drug of choice (Tolhurst, Buckle and Williams, 1955) provided it is used in large doses for sufficient period of time. There have been reports of resistance by Actinomyces bovis to penicillin, but these have been few and streptomycin or one of the tetracycline group may be substituted. (Lucas and Kramer, 1959).

1) **Drugs:** Potassium, sodium and silver iodide, Lugol's iodine, colloidal iodine, copper sulphate, methylene blue, thymol and arsenicals have all been used. Potassium iodide was the most popular, Zitka (1951) claiming that it caused solution and resorption of the infiltrate. The dosage given was up to 24 gms. per day although it was found that the same clinical results were obtained by the use of 2 - 6 gms. per day and was better
tolerated by the patient. Thymol was used intraorally in conjunction with surgical drainage and x-ray therapy by McConnell (1944) to successfully treat two cases of cervico-facial actinomycosis. There has been little proof of the effectiveness of any of these drugs, in fact Reynolds (1922) reported that actinomyces grew well in a medium containing 2% potassium iodide. It is felt, after reading the various claims made and the combinations of treatment used, that we must agree with Goldsworthy (1947) when he suggests that none of the numerous drugs recommended is of value as a specific remedy, and that the cervico-facial type of actinomycosis is almost always self-limiting.

(2) Sulphonamides: It has been shown that at least some strains of Actinomyces bovis are susceptible to the action of sulphonamides in vitro (Goldsworthy, 1947). In comparing the action of penicillin and sulphonamides in the treatment of actinomycosis, Dobson and Cutting (1945) found that both drugs were highly effective, the sulphonamides perhaps being slightly better. It has been suggested by Lucas and Kramer (1959) that sulphadiazine combined with penicillin may prove satisfactory in the treatment of actinomycosis, but Tolhurst, Buckle
and Williams (1955) state that it is unnecessary to combine these two drugs when it is known that the infecting organism is sensitive to one of them. They further state that clinical experience has shown that the drugs are not antagonistic but it should be remembered that the sulphonamides are bacteriostatic and may interrupt the bactericidal action of penicillin.

(3) **Penicillin**: As stated earlier penicillin is considered the antibiotic of choice in the treatment of actinomycosis and, in comparison with the sulphonamides and other antibiotics, the safest and most effective drug we possess to-day. Due to the tissue reaction characteristic of actinomycosis, the treatment presents a problem, for the tissues involved are often extensively scarred, indurated, and relatively avascular. Consequently there is difficulty getting a sufficient concentration of the drug into contact with the organisms and thus, it is readily agreed by most authors that penicillin, or any drug for that matter, must be given in high doses for long periods. Penicillin penetrates tissues well, is bactericidal and can be given for long periods without side effects.
Nichols and Herrell (1948) in reporting 46 cases of actinomycosis of which 26 were in the cervico-facial region, found all organisms cultured sensitive in vitro to penicillin and this drug clinically effective in shortening the disease and treatment period to one third the time taken to cure the disease by other methods. They advocate at least 500,000 units per day for six weeks. Wilenius (1954) in recording the treatment of 22 cases of cervico-facial actinomycosis states that surgery is necessary, and penicillin, given at the rate of 4 - 6 million units per day over long periods, allows this to be more radical. Doses of one million units per day until the infection is controlled, thence smaller doses for at least 3 - 6 months were advocated by Holmes (1958). In more extensive lesions, which fortunately are rarely found these days in the cervico-facial region, even larger doses and longer treatment times have been found necessary. Fisher and Harvey (1956) giving 3 - 6 million units per day for six months or longer and Harvey, Cantrell and Fisher (1957) advocating 10 - 20 million units per day by intravenous drip for 30 - 45 days, surgical excision of the infected
tissues, packing of the wound to allow healing from the base, and then 2 - 5 million units daily for 12 - 18 months.

It must be remembered that antibiotic sensitivity tests should always be carried out even though resistance of Actinomyces bovis to penicillin is rarely seen. This author has found in cases he has observed or treated in the past four years, that 600,000 units of penicillin daily for 10 - 14 days has been sufficient, provided adequate drainage has been established and the cause of the infection, if still present, removed. In the case of a patient reporting or exhibiting a sensitivity to penicillin, 1 - 2 grams of terramycin daily for the same period has proved equally effective. Most of the cases treated would be considered mild and on completion of antibiotic therapy are kept under observation for some considerable time. There have been no cases of resistance of the organisms to penicillin and all cases have healed without induration or excessive scarring. There was one recurrence which was felt to be due to incomplete elimination of the original cause of infection and not to inadequate chemotherapy.

(4) **Other Antibiotics and Allied Drugs** Although
cases of resistance of Actinomyces bovis to penicillin are not common they have been reported; McVay, Guthrie and Sprunt (1951) stating that in vitro studies revealed many strains of Actinomyces bovis are penicillin resistant. These authors used aureomycin to successfully treat their four reported cases as did Gruber (1953) and Wright and Lowen (1950). Terramycin therapy, used successfully to treat cervico-facial actinomycosis has been recorded on many occasions, and other reports indicate that streptomycin, achromycin, isoniazid and para-amino-salicylic acid are also effective.

Although there appears to be a wide range of antibiotics to choose from in treating cervico-facial actinomycosis it is felt that, in the light of the sometimes serious side effects experienced with these drugs, penicillin is still the drug of choice provided sensitivity tests reveal its potency. In the absence or delay of antibiotic sensitivity tests, the continuation of penicillin therapy is indicated, particularly if clinical improvement is obvious.

(c) **Physical Treatment:** Ziskin, Shoham and Hanford (1943) discuss the use of x-ray radiation, radium radiation, ultra-violet and alpine lights and conclude that x-ray
radiation is the best. Goldsworthy (1947) considers any beneficial effect is almost certainly due to stimulation of the host's tissues. Burket (1946) and Arnott and Ritchie (1949) advocate its use, the latter authors in infections resistant to other forms of treatment. Zitka (1951) in discussing 176 cases of cervico-facial actinomycosis occurring over 25 years up to 1951 stated that until 1944 the best results were obtained with x-ray therapy, surgery and iodine medication. Dosage given was 150-200r daily for 3 - 4 days, then second daily until the maximum dose of 2,000r was reached. Stadnicki, Krajnik and Baranžak (1958) on the other hand in a report on 69 cases of cervico-facial actinomycosis over the period 1954 - 8 stated that x-ray therapy proved to be ineffective and unnecessary. Rosh and Seldin (1948) and Green (1960) feel that the main beneficial effect of x-ray therapy is the prevention, or more rapid dissolution of the scar tissue normally associated with actinomycosis. Levett, (1951 - 2) in discussing the prevention and treatment of hypertrophic scars with radiotherapy emphasized that the treatment is effective but large scars may need increased dosage which may cause tissue damage. The least of these effects is some slight persistent pigmentation which may be permanent and, with increased dosage, there is a risk of a few telangiectases developing. The danger of these sequelae may be lessened by spreading the dosage and lengthening the interval between doses.

(d) Treatment With Vaccines. The active immunization
by means of vaccines described by Colebrook in 1920 - 1921 is apparently of limited value according to Goldsworthy (1947). He bases this statement on the fact that there is little, if any acceptable evidence in its favour, and that there is not enough known of the antigenic nature of the organisms to allow a rational approach to this form of treatment. Ziskin, Shoham and Hanford (1943) in reporting 26 cases gave the autogenous vaccine whenever it was available, but considered the best treatment was complete excision if possible in the early stages, or at least free drainage. Lorenz (1953) writes that, recently, many dental schools in Germany have reported highly satisfactory results from autovaccination in cervico-facial actinomycosis. The autovaccine was prepared from Actinomyces israeli cultures isolated from the secretions of tissues of patients with actinomycosis. This bacterial vaccine, produced by the Hygiene Institute of the University of Cologne, caused antigenic products to be formed and liberated from the invading microorganisms, thereby bringing about the accumulation of antibodies which react specifically, in vivo and in vitro with the homologous antigens.

It would appear, with the increasing knowledge of the antigenic nature of Actinomyces bovis, thereby overcoming Goldsworthy's (1947) objection, that this form of treatment may eventually be of value.

(e) **Treatment with Lymph Nodular Extracts**: This type of treatment was first described in 1930 by Trauner who
took the view that the apparently immune attitude of the regional lymph nodes to actinomycosis might offer an element of treatment. It is suggested by Sung (1944) that lymphoid tissue contains a substance detrimental to growth of the fungus. Trauner used an alcoholic extract from the cervical glands of cattle, giving intramuscular injections in daily increasing doses according to the gravity of the condition. Sung (1944) treated two cases of cervico-facial actinomycosis successfully with an aqueous extract from goats' glands, thereby eliminating the irritant qualities of the alcoholic extract recommended by Trauner. Both cases had previously been treated unsuccessfully with local applications of potassium iodide. Adequate incision and drainage were considered important by Sung (1944) and could easily have been the reason for his successful treatment.

Arlotta (1951) also reports clinical evidence of successful treatment of cervico-facial actinomycosis with lymph nodular extract, and considers the pharmacodynamic action of the administered lymph drug may be interpreted by the enzymic factor of protease. The created leukocytosis offers an abundant medium to produce this protein-splitting enzyme in great quantities. Lymph nodular therapy, according to Arlotta (1951) is not antagonized by the use of penicillin but rather produces a synergistic result by actively supporting and increasing each others individual pharmacal effect.
CHAPTER ELEVEN

CHEMOTHERAPY WITH SULPHONAMIDES AND ANTIBIOTICS

SYNOPSIS

1. INTRODUCTION

2. HISTORY

3. DRUG SELECTION

4. SYNERGISM AND ANTAGONISM

5. PROPHYLACTIC USE OF ANTIBIOTICS

6. DRUGS
   (a) Sulphonamides
   (b) Penicillin
   (c) Streptomycin
   (d) Chloromycetin
   (e) Tetracycline Compounds

7. TREATMENT OF TOXIC EFFECTS

8. USE AND ABUSE OF ANTIBIOTICS
A chapter on Sulphonamides and Antibiotics has been included in this review because of their wide-spread use and, in the opinion of this author, often misuse, in treating infections of the jaws.

1. INTRODUCTION

Chemotherapy aims at rapid and efficient control of infection with minimum risk to the patient. The empirical use of antibiotics in treating infections of the jaws, although wide-spread, is an abuse of these drugs. According to Tolhurst, Buckle and Williams (1955) the fact that different organisms vary in their sensitivity to different drugs has made bacteriological diagnosis more important than ever before. In a seriously ill patient, chemotherapy cannot be withheld until the results of cultures are known, but every effort should be made to obtain specimens for investigation before commencing therapy so modifications can be made later if necessary. In less seriously affected patients, most dental infections can be diagnosed on clinical and/or radiographic examination, but the nature of the infecting organisms and their antibiotic sensitivity should be determined prior to the commencement of therapy.

Sensitivity tests are not highly accurate according to Tolhurst, Buckle and Williams (1955), but we find when used intelligently and interpreted with due consideration of the site and nature of the lesion, together with clinical experience, they are invaluable. In the treatment of any infection close co-operation between clinician and bacter-
2. HISTORY

In 1906, Ehrlich formulated the idea that chemical substances might be found which would be lethal to bacteria but relatively harmless to tissue, thus permitting the attack on bacteria to be carried into the body. Chemical agents were found that would kill organisms, and Lister noted that postoperative surgical complications could be impressively decreased by the use of a carbolic acid spray during surgery. Even so this agent, a protoplastic poison, itself caused severe tissue damage and extensive research failed to disclose a more selective "antiseptic" until, Ehrlich discovered "salvarsan" which proved effective in the treatment of syphilis.

The demonstration that the dye "Prostosil rubrum" had an action against streptococci paved the way for the development of sulphonamides, the first chemotherapeutic agents active against many organisms causing common suppurative infections.

The discovery of penicillin and the broad spectrum antibiotics caused an almost complete cessation in the use of sulphonamides which, unfortunately, can produce rather toxic side effects. However the development of the long-acting sulphonamide, sulphadimethoxine (Madrilobin), possessing a high degree of potency and a low order of toxicity, has led to a revival in the use of this type of drug.

It has been known for 50 years that some bacteria produce
substances which inhibit the growth of other organisms but most of these "antibiotic" substances are toxic (Kramer, 1956). In 1929, Fleming accidentally discovered that the filtrate of the mould Penicillium notatum grown in peptone broth medium, even when diluted 800 times, inhibited the growth of certain bacteria. Technical difficulties prevented the isolation of this substance, called penicillin by Fleming, until 1940 when Florey and his associates proved beyond doubt the immense value of the drug.

Although penicillin was discovered accidentally, the demonstration of its valuable properties led to a deliberate and systematic search for other antibiotics, and there are now many of proven clinical value (Kramer, 1956).

3. **DRUG SELECTION**

There are four simple rules which, according to Kramer (1956), govern the selection and use of sulphonamides and antibiotics.

(a) Unlike most antiseptics, sulphonamides and antibiotics tend to be selective in their antibacterial action, certain organisms being unaffected even by high concentrations of certain antibiotics. Therefore it is essential to select the correct drug.

(b) It is essential to bring the drug into contact with the organisms.

(c) Care must be taken to use adequate concentrations of the drug as exposure of organisms to inadequate concentration not only fails to control the infection, but also
favours the formation of drug-resistant strains.

(d) It must be remembered that sulphonamides and many of the antibiotics, do not kill bacteria but merely inhibit their growth. This allows the natural defence mechanisms of the body to destroy the organisms more easily, but this process may take considerable time. Therefore it is essential that the chosen drug be administered for an adequate period of time.

Sulphonamides and antibiotics have been grouped on the basis of their bactericidal or bacteriostatic action as follows:

Group 1: (bactericidal - cause death of bacteria) Penicillin, streptomycin, bacitracin, neomycin and polymyxin.

Group 2: (bacteriostatic - prevent multiplication of bacteria) Chloramphenicol, sulphonamides, aureomycin, terramycin, tetracycline and erythromycin.

There is no need to delay treatment until identification and antibiotic sensitivity tests have been carried out or, if it is found that no specimen is available. In such cases, selection of an antibiotic can be made on the basis of a knowledge of the organisms likely to be present and, of their probable sensitivity to the various antibiotics available. A typical example of this situation is Vincent's infection, which can usually be diagnosed on clinical appearance and symptoms. It has been found that
the organisms responsible for the infection are invariably sensitive to penicillin and, although smears and sensitivity tests are necessary for final diagnosis and treatment plan, penicillin can be administered immediately if the condition is severe enough to warrant its use.

4. **SYNERGISM AND ANTAGONISM**

Synergism is defined as the ability of two drugs to increase considerably the rate of cure of infections beyond that obtained by simple additive effects of the drugs. Antagonism is the opposite action, the ability of two drugs to decrease the rate of cure below that observed with a single drug (Small, 1955).

The antibiotics of Group 1, penicillin, streptomycin etc. are frequently synergistic with, but never antagonistic to each other, those of Group 2, the tetracyclines, chloromycetin, sulphonamides etc. are only additive in their effect and never synergistic. A combination of a Group 1 and a Group 2 antibiotic may be either indifferent, antagonistic or synergistic, depending in part on the behavior of the organisms toward the Group 1 drug.

Fortunately infections within, or associated with the oral cavity, are rarely serious enough to warrant the use of more than one antibiotic. It does however appear to be the accepted practice in general hospitals to use a combination of penicillin and streptomycin in the treatment of mandibular fractures.

5. **PROPHYLACTIC USE OF ANTIBIOTICS**
The main purpose in using antibiotics is to treat infection not to prevent it, prophylactic use of these drugs being generally unwise (Thomson, 1954). The use of an antibiotic during extractions or oral surgery in patients with valvular or congenital heart disease for the prevention of bacterial endocarditis is an accepted practice, but Thomson (1954) feels that there is little proof of its effectiveness.

Antibiotics administered systemically or applied locally in an attempt to prevent postoperative infection in simple oral surgical procedures and extractions are contraindicated. However there are circumstances when an antibiotic, given systemically in adequate doses, should be used prophylactically to prevent infection (i.e. open fractures, large cysts, antrostomies etc.).

We find various authors such as Cash (1951), Di Conza (1953) and Helmore (1958) advocating the routine use of antibiotics to prevent "dry sockets" and Rank (1950), whenever bone is involved during oral surgery. It is felt by this author that atraumatic aseptic surgery is the most important factor in the prevention of postoperative infection in oral surgery.

6. DRUGS

(a) Sulphonamides: Sulphonamides are bacteriostatic, their action being to prevent incorporation of para-aminobenzoic acid into the folic acid molecule which is essential in the internal metabolic cycle of the cell
(Dille, 1951). The organisms are thus unable to reproduce and eventually succumb to the body defence mechanisms.

Because of their undesirable toxic effects, sulphonamides have been almost completely replaced by the antibiotics. Sulphadiazine is the drug of choice among the sulphonamides (Thoma, 1953), it being the most active in vivo, due to the high concentration attained in the blood and tissues resulting from its ready absorption and slow excretion.

With sulphadiazine, toxic effects are comparatively rare but blood counts should be taken before and during treatment to guard against the possibility of blood dyscrasias developing. For each gram of the drug taken, one pint of fluid should be administered, the maximum intake being six pints per day. Urine samples should be taken and tested daily to ensure its alkalinity, these later precautions being necessary to guard against renal complications.

Local use of sulphonamides is not recommended according to Thoma (1958), as they are readily absorbed from denuded areas, exposing the patient to possible toxicity or sensitivity. In addition, sulphonamides are inactivated in the presence of pus or necrotic tissue. This author agrees with Thoma's statement above but feels that his next claim, "their use in extraction sockets is beneficial", is almost a direct contradiction.

It has been found by Cappuccio and Dobbs (1960) that
sulphadimethoxine (Madribon), a recently developed long-acting sulphonamide, is of use in dental infections. The high degree of potency, low toxicity and simple oral administration once or, at the most, twice a day, recommend this drug for consideration in the treatment of dental infections, particularly in view of the increasing emergence of antibiotic resistant strains of bacteria.

(b) **Penicillin**: Lucas and Kramer (1959) state penicillin is both bacteriostatic and bactericidal, producing its effect by interfering with the metabolism of glutamic acid, an essential factor for the growth of many organisms. Penicillin is most effective against bacteria that are actively multiplying; those in the resting stage are relatively undamaged even if sensitive to the drug. The presence of pus or blood does not reduce the activity of penicillin and it is effective against many of the pathogenic Gram-positive bacteria, a limited number of Gram-negative organisms, actinomyces bovis and the spirochaetes.

Local applications, although reported as effective by Gwinn and Grimm (1948), Northrop (1949), Long (1951), Epstein and Kauffman (1951) and Thoma (1958), are considered by Jacobs (1950), Thomson (1954), Kramer (1956), Goulding (1957) and Lucas and Kramer (1959) to be contraindicated due to the danger of producing allergic reactions, of sensitizing the patient to the antibiotic and, of hastening the production of resistant strains of organisms.

It is felt by this author that we must agree with the later group of writers when they consider local use of
penicillin most undesirable. Anaphylactic reactions have followed as simple an action as skin testing with penicillin, sucking a penicillin lozengen and ingestion of a single penicillin tablet (J.A.M.A. 170: 891 Jan. 13, 1959. Questions and Answers).

The only satisfactory method of using penicillin is by systemic administration. There has existed in the past, considerable doubt as to the efficiency of oral administration but the introduction of Penicillin V potassium (P.V.K., Lilly) appears to have provided a satisfactory "oral penicillin" which in this author's opinion, produces clinical results comparable to those of intramuscular injections.

One of the most valuable properties of penicillin is its comparative freedom from side effects (Tolhurst, Buckle and Williams, 1955). Allergic manifestations are the most important toxic effects of penicillin, the most common being urticaria, the severest, and fortunately the rarest, is anaphylactic shock which can be fatal. Consequently, penicillin should not be given to a patient who has had a previous reaction, no matter how slight, or who suffers from allergic disorders such as asthma or hay-fever.

(c) Streptomycin: Streptomycin, although still the mainstay in the treatment of tuberculosis, possesses two disadvantages which preclude its general use. It is highly toxic and resistant mutants exist which replace the original sensitive organisms and make continued treatment useless.
(Tolhurst, Buckle and Williams, 1955). For these reasons and the fact that so many other antibiotics are available, it has no application in dentistry.

(d) **Chloromycetin (chloramphenicol):** The use of chloromycetin carries with it the risk of depression of bone marrow function which may be fatal (Thompson, 1954) and, consequently should be used only if clinical observation and sensitivity tests exclude all other antibiotics.

(e) **The Tetracycline Compounds:** The tetracycline compounds, aureomycin, terramycin and achromycin, resemble each other and chloromycetin in the wide range of antimicrobial activity and are often grouped with this latter drug as the "broad spectrum antibiotics". The tetracyclines are bacteriostatic, and effective against a wide range of organisms. Routine administration is by the oral route. The dose should be taken with cold milk and supplemented with vitamin B to reduce the incidence and severity of gastro-intestinal disorders, which are the most common toxic effects following the use of these drugs. Terramycin has, in this author's experience, proved itself the most satisfactory antibiotic in cases where penicillin is contra-indicated due to patient sensitivity or resistant strains of organisms.

7. **TREATMENT OF TOXIC EFFECTS**

(a) **Sensitization:** Although sensitization is usually a consequence of previous exposure to the drug, it may occur during the first course of treatment. The sensitization is
usually manifested by a skin rash, often urticarial in form, although occasionally a serum-sickness type of reaction may occur. Very rarely acute anaphylactic reactions, invariably fatal, follow the use of penicillin and streptomycin.

According to Pillsbury and associates (1947) the most frequently encountered reaction to penicillin is urticaria, often encountered in patients who have had repeated courses of the drug. They found skin tests unreliable in predicting the occurrence of such reactions and anti-histamine compounds, particularly Benadryl 25 - 50 mgs., two or three times per day, useful in controlling the urticaria which at times may be persistent and severe.

Thomson (1952), Shuttleworth (1953), Kramer (1956), Lane (1956 and Bramley (1957) feel that the severe types of allergic reaction tend to occur in patients who have had previous reactions of some type or who have a history of allergy such as asthma or hay fever. We find Shuttleworth (1953) and Bramley (1957) emphasizing the importance of avoiding intravenous injections of penicillin although Rosenthal (1955), in recording eight fatal anaphylactic reactions, found no evidence of blood vessel penetration but the majority of cases had a history of allergic symptoms to previous penicillin injections.

Anaphylactic reactions may be recognised, according to Revzin (1958) by a sudden onset of spasms, coughing, a sudden precipitous drop in blood pressure and a weak
thready pulse. Immediate intravenous, according to some authors or subcutaneous, according to others, injection of 5 minims of 1 in 1,000 adrenalin hydrochloride should be given and followed with a further 5 minims if necessary. In case of respiratory failure, the air-way should be kept open and oxygen administered.

Spiegel (1959) recommends the use of penicillinase in allergic penicillin reactions to inactivate the circulating penicillin and relieve the acute symptoms. We find Monheim (1957) stresses the importance of careful questioning prior to injection to uncover any history of previous reaction, no matter how slight, or of allergic conditions.

(b) Direct Toxic Effects: Most simple toxic effects will cease if the causal drug is discontinued or another substituted. Gastro-intestinal symptoms such as nausea, heartburn, epigastric distress and vomiting following the use of chloromycetin and the tetracyclines can be largely avoided by the patient drinking cold milk with each dose and taking Vitamin B concurrently with the antibiotic (Tolhurst, Buckle and Williams, 1955). With Terramycin, Lane (1956) expects one case in every five to develop gastro-intestinal distress including diarrhea, vomiting and/or nausea. This author has found from clinical experience at the United Dental Hospital of Sydney, that the routine use of Vitamin B and "cold milk" has almost completely irradiated these complications.
Chloromycetin has, according to Glaser (1953), Thomson (1954), Lane (1956) and Alling and Pulaski (1959), fallen into disfavour as an antibiotic because of its potential for depressing the normal activity of the haematopoietic tissues which may lead to an aplastic anaemia.

Direct neurotoxic action on the eighth cranial nerve, causing deafness or vertigo, is likely to occur with Streptomycin if the dosage of 1 gm. per day is exceeded for long periods. This drug has, according to Kramer (1956), so many other disadvantages that its dental use is contraindicated.

(c) **Secondary Inflammation or Ulcerations Caused by Superimposed Infections from Antibiotic-resistant Organisms:**

(1) **Acquired Bacterial Resistance:** The problem of acquired resistance to antibiotics has received a large amount of publicity, Saint (1953), Lane (1956), Bramley and Clark (1955 - 6) and Thomson (1959) all recording almost unbelievable recent increases in strains of staphlococci resistant to penicillin, and to a lesser degree some of the other antibiotics. In this regard, Harris (1956) states that, during the last three years (1953 to 1956), control of infection by antibiotics has been lost in orthopaedic surgery, most surgeons having returned to "open drainage of osteomyelitis by incision of the sub-periosteum and perforation of
the cortex with drill holes. It would appear from this statement that some surgeons had allowed antibiotic therapy to replace the long established principles of treatment of bone infections. It is agreed by most authors that it is the indiscriminate use of antibiotics that has produced this alarming position. Fortunately for the patients, science is continually producing new antibiotics to replace the now useless ones. This production of new drugs plus the vast publicity each one receives, unfortunately encourages their general use and it is felt by this author that we must agree when Thomson (1959) states we should exercise restraint in the use of all antibiotics and particular care to conserve the newer drugs for the treatment of life endangering staphylococcal infections. Thomson (1959) also expresses a doubt as to how long the production of genuinely new antibiotics can keep us ahead in the "downhill race".

(2) **Fungal Overgrowth:** The overgrowth of monilia on the tongue and oral mucous membranes during antibiotic therapy is occasionally seen. Two theories exist regarding its cause, firstly
that the fungus is directly stimulated by
the antibiotic, and secondly, the generally
accepted theory, that suppression of the
bacteria normally resident in the mouth
allows overgrowth of the yeast-like organ-
isms. Although Kramer (1956) states the
condition is painful, this author has found
that the patients are usually unaware of
the condition which invariably clears
spontaneously once the antibiotic is suspen-
ded.

8. USE AND MISUSE OF ANTIBIOTICS

Degnan (1959) lists the three major faults in the abuse of
antibiotics by dental surgeons.

(a) the routine use of antibiotics for all dental
surgery.

(b) failure to take advantage of laboratory culture
and sensitivity tests when planing antibiotic therapy.

(c) use of these drugs in too small a dose and for
too short a time to achieve a therapeutic result.

To often do we see antibiotics abused in this manner
by treating minor infections and often conditions, such
as "dry socket" and "gingivitis" where they are absolutely
useless. The attitude taken by some dentists, that "a
shot of penicillin" might help and certainly will not do
any harm, is to be condemned particularly in the light
of the possible toxic effects, sensitizion of the patient
and promotion of resistant strains of bacteria.

Fortunately the dangers arising from the abuse of antibiotics are well recognized and receive a proportionate amount of attention in the dental and medical literature. It is felt by this author that Pulaski (1957) sums up the situation admirably ... "It must be emphasized again that chemotherapy is nothing but an adjunct to surgical management and should never be employed as a substitute for early operation, meticulous technique and careful preoperative and postoperative correction of physiologic and other abnormalities. The "routine" use of prophylaxis in every operative wound reflects a lack of confidence by the surgeon in his aseptic technique, a lack of understanding of the limitations of antibiotic action, or a lack of recognition of the potential effect of such practice".
CHAPTER TWELVE

Statistics and Comments on Cases of Alveolar Osteitis Treated at the United Dental Hospital of Sydney.

Synopsis

1. "Dry sockets" following extractions under local anaesthesia.

2. "Dry sockets" following extraction under general anaesthesia.


4. "Dry sockets" following pericoronitis.
1. "DRY SOCKETS" FOLLOWING EXTRACTION UNDER LOCAL ANAESTHESIA

It was found that the 400 "dry sockets" studied resulted from 59,583 extractions carried out over a period of 2 years, 3 months and 4 days in the Exodontia Department of the United Dental Hospital of Sydney. During an analysis of 5,000 consecutively extracted teeth it was found that approximately 15.44% were deciduous and, in the light of the fact that in only one case did a condition resembling "dry socket" develop, were excluded from the survey.

Hence we have 400 "dry sockets" occurring in 50,386 extractions, an incidence of approximately .79%. These 400 "dry sockets" occurred in 367 patients, 59 of whom had more than one "dry socket".

Females were affected slightly more frequently, 52.25%, than males and 72% of the cases developed in the mandible.

<table>
<thead>
<tr>
<th>Age Incidence</th>
<th>Age</th>
<th>Percentage</th>
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<tbody>
<tr>
<td>0 - 10 Years</td>
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</tr>
<tr>
<td>10 - 20 &quot;</td>
<td>10.9%</td>
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</tr>
<tr>
<td>20 - 30 &quot;</td>
<td>23.5%</td>
<td></td>
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<tr>
<td>30 - 40 &quot;</td>
<td>25.3%</td>
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<tr>
<td>40 - 50 &quot;</td>
<td>15.2%</td>
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<td>50 - 60 &quot;</td>
<td>11.5%</td>
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<tr>
<td>60 - 70 &quot;</td>
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<tr>
<td>70 - 80 &quot;</td>
<td>4.9%</td>
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<tr>
<td>Over 80 &quot;</td>
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</table>

Incidence in the Jaws: The percentage of teeth extracted from each position in the jaws was arrived at by analysis of 5,000 consecutive extractions of permanent teeth. The number of "dry sockets" for each position was
counted and the two figures combined to give a percentage incidence for each position in the maxilla or mandible.

<table>
<thead>
<tr>
<th>Teeth</th>
<th>Maxilla</th>
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<th>Mandible</th>
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<td>0.03%</td>
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<td>0.14%</td>
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</tr>
<tr>
<td>Lateral Incisor</td>
<td>0.29%</td>
<td></td>
<td>0.27%</td>
<td></td>
</tr>
<tr>
<td>Canine</td>
<td>0.96%</td>
<td></td>
<td>0.82%</td>
<td></td>
</tr>
<tr>
<td>1st. Premolar</td>
<td>0.72%</td>
<td></td>
<td>1.43%</td>
<td></td>
</tr>
<tr>
<td>2nd. Premolar</td>
<td>0.99%</td>
<td></td>
<td>1.60%</td>
<td></td>
</tr>
<tr>
<td>1st. Molar</td>
<td>0.36%</td>
<td></td>
<td>0.98%</td>
<td></td>
</tr>
<tr>
<td>2nd. Molar</td>
<td>0.54%</td>
<td></td>
<td>1.46%</td>
<td></td>
</tr>
<tr>
<td>3rd. Molar</td>
<td>0.37%</td>
<td></td>
<td>2.74%</td>
<td></td>
</tr>
</tbody>
</table>

**Time Elapsing Between Extraction and Patient Seeking Treatment.**

<table>
<thead>
<tr>
<th>Days after Extraction</th>
<th>No. of Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.5%</td>
</tr>
<tr>
<td>2</td>
<td>3.8%</td>
</tr>
<tr>
<td>3</td>
<td>9.3%</td>
</tr>
<tr>
<td>4</td>
<td>12.5%</td>
</tr>
<tr>
<td>5</td>
<td>21.5%</td>
</tr>
<tr>
<td>6</td>
<td>18.5%</td>
</tr>
<tr>
<td>7</td>
<td>18.5%</td>
</tr>
<tr>
<td>More than 7</td>
<td>15.4%</td>
</tr>
</tbody>
</table>

**Number of Daily "Dentalone" Dressings Required to Relieve Pain**

<table>
<thead>
<tr>
<th>No. of Dressing</th>
<th>No. of Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>9%</td>
</tr>
<tr>
<td>2</td>
<td>30%</td>
</tr>
<tr>
<td>3</td>
<td>41%</td>
</tr>
<tr>
<td>4</td>
<td>17%</td>
</tr>
<tr>
<td>5</td>
<td>2.75%</td>
</tr>
<tr>
<td>6</td>
<td>0.25%</td>
</tr>
</tbody>
</table>

The incidence of "dry sockets" at the United Dental Hospital of Sydney compares favourably with the figures of Krogh (1937) and Archer (1939). It is felt that this is due, partly at least, to:

(a) surgical removal of teeth appearing unsuitable radiographically for extraction due to anatomical abnormalities.

(b) surgical removal of teeth for patients who have
experienced "dry sockets" from previous extractions.

2. "DRY SOCKETS" FOLLOWING EXTRACTION UNDER GENERAL ANAESTHESIA

In a survey of 200 general anaesthesia cases over a period of approximately 2 years, it was found that, in 3161 extractions and 116 surgeries for removal of unerupted third molars, three "dry sockets" developed postoperatively. This low incidence of "dry sockets", .095%, following extraction under general anaesthesia is due, I feel, to the fact that multiple extractions are being carried out and not as some authors claim, to the absence of local anaesthetic solution in the tissues.

Postoperative clinical examination of these 200 patients revealed 25 cases of delayed healing, not complained of by the patient. These cases healed rapidly following removal of small sequestra and application of intraoral heat.

3. "DRY SOCKETS" FOLLOWING SURGICAL REMOVAL OF UNERUPTED TEETH

Over a period of two years, 524 unerupted teeth were removed in the Oral Surgery Department of the United Dental Hospital of Sydney, 109 or 20.8% of these were lower third molars. 26 cases of "dry socket" developed, 22 or 85% of which were in lower third molar wounds, and 42 cases of delayed healing were observed. Hygiene of the socket in 11 of these cases, 10 of which were lower third molar wounds, was maintained by packings of various types. The remaining 31 cases, 22 being lower third molars healed uneventfully following the application of intraoral heat.
From these figures we find a 6.6% "dry socket" incidence in all unerupted teeth removed and 20.2% in unerupted lower third molars. The average number of "Dentalone" dressings required to relieve pain was three and the treatment extended over an average period a little less than four weeks. Sockets were packed until they were self-cleansing, usually with Horsley's Bone Wax once the pain was relieved and granulation commencing. It has been found that this wax keeps sockets very clean, may be left in place over a week and is gradually expelled as the wound heals.

4. "DRY SOCKETS." FOLLOWING PERICORONITIS

70 cases of pericoronitis occurring over approximately 2 years were studied. All patients complained of pain, 67 of pain and swelling and 16 of trismus, pain and swelling. Smears were taken for bacteriological identification and antibiotic sensitivity in 55 cases; the majority revealing a mixed infection but 11 being typical of Vincent's Infection. Eight of the mixed infection were found to be resistant to penicillin.

Local treatment was used in all cases and systemic antibiotics in 26 cases, 19 penicillin and 7 terramycin. Terramycin was used in four cases because of resistant organisms and in three because of patient sensitivity. Of the 70 cases 51 or 73% showed radiographic evidence of bone destruction distal to the crown.

In 52 cases the offending tooth was removed, one was considered unnecessary and 17 patients either refused to
have the tooth removed or failed to complete treatment. Eight cases of "dry socket" developed, approximately 15.4\% and five cases, 9.6\%, of delayed healing occurred requiring some form of packing to maintain socket hygiene.

It is interesting to note that 35 or 50\% of these patients suffering from pericoronitis were Southern Europeans (mainly Italians) often with full complement of teeth and poor oral hygiene and gingival condition. 73\% of the patients were males and 63\% were aged between 20 and 30 years.
INFECTIONS OF THE JAWS

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