

CERVICO-FACIAL ACTINOMYCOSIS

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CERVICO-FACIAL ACTINOMYCOSIS

1. INTRODUCTION

Cervico-facial actinomycosis, in the human, is an infection by a fungus group which may follow any operative procedure within the mouth (Lesney, and Traeger, 1959).

Actinomycosis may occur in any site but it is generally agreed that approximately 50 per cent of cases occur in the cervico-facial region (Cappell, 1962; Monteleone, 1963; Goldsworthy, 1947; Pheils et al, 1964). The two other anatomical sites most frequently affected are the abdomen and the thorax, with an incidence of occurrence of approximately 20 per cent and 15 per cent respectively (Monteleone, 1963; Cappell, 1962).

Cervico-facial actinomycosis develops as a swelling in the vicinity of the jaws, especially the mandible. The swelling enlarges and an abscess forms and points externally. This abscess may rupture releasing a variable quantity of thick pus with the formation of one or more sinuses. Generally slow local spread of the process occurs with multiple sinus formation and fibrosis of the surrounding soft tissue (Goldsworthy, 1947).

The pus from the sinuses may show small yellowish "sulphur granules" which, on microscopic examination, are composed of mycelles or clumps of filamentous micro-organisms. These organisms may be isolated in pure culture and identified as members of the genus *Actinomyces*.

Goldsworthy (1947) considers that, with adequate treatment, the

prognosis is favourable and this author and Hunter and Westrick (1957) both report a 97 per cent recovery rate following cervicofacial actinomycosis.

2. THE CAUSAL ORGANISM.

In the past there appears to have been some difference of opinion regarding the causative organism of actinomycosis in man. However, it is now generally agreed that the micro-aerophilic *Actinomyces bovis* (Wolff-Israel) is responsible for the infection (Holmes, 1958; Bramley and Orton, 1960; Arnott and Ritchie, 1949; Arnott, 1962; Shafer, Hine and Levy, 1960; Kruger, 1959; Goldsworthy, 1947). Previously the organism *Actinomyces israeli* has been cited as the causal organism but Shafer, Hine and Levy (1960) consider *Actinomyces israeli* to be "merely a rough variant of *Actinomyces bovis*."

Goldsworthy (1947) describes *Actinomyces bovis* as a long filamentous organism which gives a positive reaction to Gram-staining. The micro-organism grows slowly on culture, forming heaped up, non-pigmented colonies on solid media. The branching filaments of the mycelium may show clubbing in vivo. These clubs, however, do not take up Gram-stain, do not occur invitro and are considered to be a reaction of the organism to the host's tissues (Holmes, 1958).

Actinomyces bovis is anaerobic or micro-aerophilic and, being a strict parasite, has never been found free in nature. The organism is considered to be a member of the normal oral flora and has been

reported by various authors as being found in carious teeth, puncture wounds of the oral mucosa, inflamed pulp tissue, apical granulomas, tonsillar crypts, and periodontal lesions (Holmes, 1958; Gee and Sullivan, 1940; Hunter and Westrick, 1957; Pheils et al, 1964; Main and MacPhee, 1964; Cran and Hannam, 1963; Villa, 1957; Ludwig, 1955; Arnott and Ritchie, 1949).

It is important to distinguish *Actinomyces bovis* from the non-pathogenic *Actinomyces* found in soils, grains and grasses. These organisms grow rapidly on culture at room temperature and the colonies are pigmented, something which does not occur with *Actinomyces bovis*.

3. MODE OF INFECTION

The importance of trauma in the aetiology of cervico-facial actinomycosis is well established. It is also generally recognised that the infection is of endogenous origin.

The current theory, according to Monteleone (1963) emphasises the importance of mixed infections and associated trauma. *Actinomyces bovis* appears to achieve a symbiotic alliance with certain endogenous bacteria, the combination showing a degree of pathogenicity not possessed by either organism alone. These other organisms, usually pyogenic cocci, may improve growth conditions for the micro-aerophilic *Actinomyces bovis*, by reducing the oxygen tension of the involved tissues. Thoma and Goldman (1960) support this theory.

Injury results in impairment of tissue vitality which affords

an ideal situation for growth of micro-aerophilic and anaerobic organisms. Cervico-facial actinomycosis has been reported following extraction of teeth, fractures of the jaw, pericoronal infections and abrasion of the mucosa (Kruger, 1959). According to Holmes (1958) extraction of lower molar teeth, especially third molars, appears to be a common initiating factor.

4. CLINICAL FEATURES

Both acute and chronic forms of cervico-facial actinomycosis are recognised by Arnott and Ritchie (1949), Bramley and Orton (1960), Goldsworthy (1947), Ludwig (1955), and Thoma and Goldman (1960).

(a) The acute form shows symptoms similar to those seen in acute pyogenic infections of the soft tissues of the cervico-facial region (Bramley and Orton, 1960). In such cases the inflammatory reaction results in a rapid, painful swelling, dysphagia, and trismus. According to Arnott and Ritchie (1949), the abscesses which form point readily and drainage can be quickly established. Careful bacteriological examination of the pus is essential to establish a diagnosis. These writers consider that once drainage is established, the acute form of the infection slowly resolves to a chronic state with continuing drainage. Goldsworthy (1947) states that, in the early stages, suppuration predominates and the lesion may be indistinguishable from other abscesses.

(b) The chronic form is often painless and is characterised by the formation of a slowly developing, indurated, purplish-red

swelling with associated trismus. Fluctuation of the swelling occurs with resultant drainage. The draining sinus frequently heals with the formation of scar tissue. The infection spreads in the submucous and subcutaneous connective tissue by direct tissue continuity with resultant new sinus formation, healing and cicatrisation.

Lymphadenitis is uncommon according to Holmes (1958), Cappell (1962), Thoma and Goldman (1960), Kruger (1959), and Sung (1944) and does not usually occur unless secondary infection occurs. The treatment of actinomycosis with lymph node extract, as reported by Sung (1944), is based on the observation that regional lymph nodes are rarely involved in actinomycosis.

Involvement of the osseous elements of the jaws is uncommon although, according to Holmes (1958) "localised decalcification, secondary to hyperaemia, often occurs". Should the micro-organisms gain access to the bone, periostitis and osteomyelitis result with possible extensive bone loss and sequestration (Nathan et al, 1963; Shafer, Hine and Levy, 1960; Goldsworthy, 1947). Holmes (1958) states that "spread to other parts of the body is the complication most to be feared since the mortality for such spread is high, even with antibiotic therapy." Cappell (1962) corroborates this statement.

Goldsworthy (1947) states that the average duration of treated cervico-facial actinomycosis is approximately three months, with a range from six weeks to five months. However he considers that the condition may progress indefinitely if not adequately treated.

5. HISTOPATHOLOGY.

The typical actinomycotic lesion in either soft tissue or bone, according to Shafer, Hine and Levy (1960) is essentially a granulomatous reaction with central abscess formation. Cappell (1962) describes the abscesses as having a multi-locular or honey-comb appearance. The lesion is surrounded by a fibrous tissue wall which is lined by granulation tissue which contains many "foam-cells" - macrophages laden with lipid material. In the centre of the granulation tissue is pus containing colonies of the ray fungus which are discharged in the pus as granules, sulphur-yellow in colour.

6. DIAGNOSIS

Goldsworthy (1947) considers that diagnosis is made by weighing the clinical evidence and seeking supportive laboratory information. Shafer, Hine and Levy (1960) expand on this view by saying that the diagnosis of actinomycosis is dependent not only on the clinical findings in the patient and the demonstration of the fungi in the tissue section or smear but also upon culture of the fungus. On the other hand Thoma and Goldman (1960) state that "diagnosis is almost entirely dependent on microscopic examination of the discharge from a fistula or of excised tissue". This reviewer considers that to divorce clinical and laboratory findings is a grave error and agrees with Lucas and Kramer (1959) that "the first essential in diagnosis of actinomycosis is the suspicion that the patient may be suffering from the disease and the communication of this suspicion to the bacteriologist".

As the organisms are slow growing, confirmation of a tentative clinical or histological diagnosis may be delayed. This reviewer considers, if the clinical features were suggestive of actinomycosis, it would be an error to withhold treatment until the organism had been isolated in pure culture and positively identified.

A history of the condition together with a careful examination of the tissues is essential in diagnosis. According to Holmes (1958) drainage from the lesion should be collected in a test tube and permitted to run down the sides of the tube. This allows easier examination of the material to detect the presence of "sulphur granules". Crowley (1944) reports that "sulphur granules" are not always present in large numbers and repeated examinations may be necessary to detect the granules. The presence of granules in the discharge from the lesion cannot be considered diagnostic of actinomycosis as, on examination it may be found that the granules are composed of tissue debris, adipose tissue or bone.

Bacteriological examination of the pus from the lesion is essential to establish a definite diagnosis (Goldsworthy, 1947; Pizer, 1960). If a "sulphur granule" is found it should be crushed between two microscope slides or a direct smear of the pus made and stained by Gram's method. The slides are examined to detect the presence of Gram-positive branching filaments which indicate the presence of Actinomyces. Final identification of the micro-organism is not possible until culture, under both anaerobic and aerobic conditions, is carried out. Differentiation must be made between

Actinomyces bovis and the aerobic Nocardia group of organisms which are normally soil dwellers. These organisms are not common pathogens in man and can readily be distinguished by their oxygen requirements (Monteleone, 1963). According to Monteleone (1963) the disease Nocardiosis has similar clinical features as Actinomycosis but responds less readily to treatment.

Histological examination of involved tissue is of value in the absence of bacteriological evidence but tissue is generally less accessible as it involves biopsy of the lesion. Thoma and Goldman (1960) state that the fungus is not readily demonstrated in tissue and a large number of sections may be necessary before it is discovered.

7. TREATMENT

The present approach to the treatment of cervico-facial actinomycosis is both medical and surgical. Prior to the development of the antibiotics, various medications were used. Various iodides, methylene blue, salvarsan, thymol and copper sulphate have all been used in the past but, with the advent of antibiotics, these preparations have fallen into disfavour.

(a) Medical Treatment.

(i) Drugs: potassium sodium and ethyl iodides, Lugols iodine or colloidal iodine, thymol and arsenical drugs have all been used in the treatment of actinomycosis. Potassium iodide, given orally in doses of 2-6 Gms. per day, was the drug most favoured. It being claimed that potassium iodide caused solution and resorption of the

of the infiltrate (Rosh and Seldin, 1948; Zitka, 1951). Holmes (1958) suggests the iodides act by liberation of iodine in the tissues but he considers them to be of doubtful value.

Although various drugs have been mentioned for the treatment of actinomycosis, very little is presented in support of the statements made regarding their mode of action. This reviewer agrees with Goldsworthy (1947) who states that none of the drugs mentioned above appear to have any specific value.

(ii) Chemotherapeutic and Antibiotic Drugs.: it is generally agreed that penicillin is the drug of choice for the treatment of actinomycosis. Tolhurst, Buckle and Williams (1963) and Spilsbury and Johnstone (1962) emphasise the need for large doses administered over a long period due to the fibrous nature of the lesions to ensure adequate contact of the antibiotic with the organisms. Tolhurst, Buckle and Williams (1963) recommend a minimum dosage of 500,000 units of crystalline penicillin given intramuscularly at six hourly intervals for a period of four weeks, or longer if indicated. Holmes (1958) considers that one million units of procaine penicillin, twice daily, should be given until the infection is under control and then daily injections of smaller doses should be maintained for at least three to six months. Monteleone (1963) states that one million units of penicillin should be given daily for at least four to six weeks. Pizer (1960) recommends a combination of penicillin, surgical intervention and potassium iodide. This author considers that antibiotics are unable to penetrate all the involved tissue due to the

fibrosis and that potassium iodide and surgical intervention enhance the action of penicillin.

Dobson and Cutting (1945), in comparing penicillin to sulphonamides in the treatment of actinomycosis, found both drugs to be highly effective, the sulphonamides being slightly more efficacious. Goldsworthy (1947) reports an in vitro sensitivity of some strains of *Actinomyces* to the sulphonamides however the use of sulphonamides in the treatment of actinomycosis is not well documented.

Achromycin, taken orally, together with irrigation of the infected area with neomycin solution is considered to be of value by Hinds and Degnan (1955). McVay et al (1951) used aureomycin with success in the treatment of their four reported cases. Lambert (1951), following unsatisfactory results using penicillin, instituted streptomycin therapy with good effect. This author recommends the administration of 1-2 Gm. per day of streptomycin until resolution of the infection is well established.

Isoniazid (Isonicotinic Acid Hydrazide), a substance which is bacteriostatic to the tubercule bacillus, was used by McVay and Sprunt (1953) and Lesney and Traeger (1959). This drug was employed because it had proved successful in the treatment of tuberculosis and some similarity exists between tuberculosis and actinomycosis. Both authors report successful treatment and McVay and Sprunt (1953) recommend that further investigation should be carried out regarding the value of this drug.

It must be emphasised that, prior to using antibiotics, sensitivity tests must be carried out to ensure the selection of the correct drug. Also, when antibiotics are used for a long period, continued sensitivity tests must be carried out to detect the emergence of resistant organisms and allow appropriate changes in therapy.

(b) Surgical Treatment.

This may be discussed in two sections:-

(i) Incision of the swelling to establish drainage and to obtain material for definitive diagnosis is recommended by many authors (Arnott and Ritchie, 1949; McVay et al, 1951; Goldsworthy 1947; Kruger, 1959; Arnott, 1962; Holmes, 1958; Bramley and Orton, 1960).

(ii) Complete excision of the lesion is suggested by Monteleone (1963), Arnott and Ritchie (1949), Arnott (1962), Ziskin et al (1943), Bernier (1959) and Thoma and Goldman (1960) as a means of effecting a cure in selected cases. In an extensive lesion it is difficult to imagine complete excision of the involved tissue without gross structural loss and resultant deformity.

(c) Physical Treatment.

Ziskin et al, writing in 1943, report that the use of X-ray irradiation was an effective method of treating actinomycosis. Arnott and Ritchie (1949) and Arnott (1962) consider that deep X-ray therapy is indicated where the infection resists the usual forms of treatment. These authors state that such physical treatment given

in small doses at frequent intervals has proved of value. Zitka (1951) recommends early small doses of 150-200r daily for 3-4 days then second daily until a total dosage of 2000r is reached. Goldsworthy (1947) states that any beneficial effect obtained from this form of treatment is almost certainly due to stimulation of the host's tissues. Rosh and Seldin (1948) consider that X-ray irradiation results in a reduction in the amount of scar tissue formed and they suggest that this is either due to a local vasodilatation caused by the irradiation or to a lethal effect on the micro-organism itself.

(d) Other Methods.

Other forms of treatment of actinomycosis are now, following the advent of antibiotics, mainly of historical interest;

(i) Treatment with Vaccines: according to Goldsworthy (1947) active immunisation by vaccines is of little value for there is little, if any, acceptable evidence in its favour. He further states that there is not enough known about the antigenic nature of the organism to permit a rational approach to this form of treatment. Ziskin et al (1943) report the use of an "antogenous vaccine", for the treatment of actinomycosis. However these authors considered the surgical approach to be the treatment of choice.

(ii) Treatment with Lymph Node Extracts: this method of treatment is based on the observation that regional lymph nodes appear to be relatively immune to actinomycosis. Sung (1944) reports the successful treatment of two long standing cases of actinomycosis with

aqueous extract of goat's lymph glands. However, the fact that Sung gave supportive treatment in the form of surgical drainage and potassium iodide therapy, cannot be overlooked. It is possible that the supportive treatment may have resulted in the successful resolution of the infection.

CHEMOTHERAPY WITH ANTIBIOTICS
AND ALLIED DRUGS

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CHEMOTHERAPY WITH ANTIBIOTICS AND ALLIED DRUGS

Following the foregoing discussion of some post-operative infections a brief review of the literature concerning chemotherapy with antibiotics and allied drugs is in order.

1. INTRODUCTION

Chemotherapy aims at rapid and efficient control of infection with a minimum of risk and discomfort to the patient.

Kramer (1956) records that, in 1906, Erlich hypothesised that chemical substances might be found which would be so much more toxic to bacteria than to the tissues that they might be introduced into the body for treatment of disease. The first drug reported with these properties was the arsenical compound "Salvarsan" which was used for the treatment of syphilis. In 1935 the in vitro use of Prontosil rubrum, a sulphonamide, was reported by Domagk. This substance was subsequently found to be successful in the treatment of haemolytic streptococcal infections in man (Tolhurst, Buckle and Williams, 1963). In 1929 Fleming isolated penicillin from the mould *Penicillium notatum* but it was not until 1940 that the drug became freely available. The realisation of the full potential of penicillin and the development of the "broad spectrum" antibiotics has revolutionised the treatment of infections.

Tolhurst, Buckle and Williams (1963) state that, as different organisms vary in their sensitivity to different drugs, the importance of bacteriological diagnosis is greater than ever before. These

authors consider that, in some cases, while it is possible to guess successfully at the best chemotherapy on clinical grounds alone, a clinical entity is not necessarily a bacteriological entity. Hence chemotherapy is more likely to be successful when the identity and sensitivity of the infecting micro-organisms have been determined.

In the seriously ill patient, as early treatment is of paramount importance, Tolhurst, Buckle and Williams (1963) believe that chemotherapy should not be withheld until results of cultures and sensitivity tests are known. If possible, specimens for investigation should be obtained prior to initiating therapy so that modifications can be made later if necessary. In the less seriously ill patient it is often desirable to establish the diagnosis before giving chemotherapy. These authors consider that properly controlled sensitivity tests yield valuable information, however the results of these investigations must be properly integrated with the clinical nature of the lesion to establish an accurate diagnosis and to formulate the correct treatment plan.

2. PRINCIPLES RELATING TO USE OF CHEMOTHERAPEUTIC DRUGS.

Four basic rules govern the use of chemotherapeutic drugs (Kramer, 1956).

(a) As antibiotics and sulphonamides are selective in their anti-bacterial activity it is essential that, for efficient therapy, choice is based on sensitivity tests.

(b) To be effective chemotherapeutic substances must come into contact with the infecting micro-organisms.

(c) Adequate concentration of the selected drug must be used. If the bacteria are exposed to drugs in concentrations less than is required for effective action, the emergence of resistant strains of organism is possible with a consequent reduction of the chances of successful treatment.

(d) The treatment must be continued until the infecting micro-organisms have been destroyed. Premature termination of therapy may lead to exacerbations of the infection and also the possibility of development of drug-resistant bacteria.

Frequently, in acute infections, it is unwise to postpone antibiotic therapy until the results of sensitivity tests are known. In such cases, selection of an antibiotic can be made on the basis of a knowledge of the likely infective organisms and their expected sensitivity to the drug of choice. Often a smear from a specimen of exudate taken by aspiration or incision may be of value in the initial selection of a chemotherapeutic agent (Deichmann, 1955). Further, when this method of selection of antibiotics is employed, material should be made available for culture and sensitivity tests so that modification can be made later to the selection of drugs, if necessary (Tolhurst, Buckle and Williams, 1963; Kramer, 1956).

3. MODE OF ACTION.

Antibiotics and allied drugs are described as having two modes

of activity:-

(a) Bacteriostatic, which implies inhibition of bacterial growth. Such drugs prevent multiplication of the organisms and, if the concentration of the drug is adequate, sensitive micro-organisms are ultimately destroyed. However the ultimate action of the bacteriostatic drug is dependent on the patient's defence mechanisms.

(b) Bactericidal, which is a direct progressive lethal action on the bacteria.

Tolhurst, Buckle and Williams (1963) state that both activities may be exhibited by sulphonamides and antibiotics depending on the concentration of the drug and the sensitivity of the organism. However, in the levels usually obtainable in the blood, a drug generally exhibits either one or the other property.

The chemotherapeutic drugs have been divided according to their mode of activity into two basic groups:-

GROUP 1. (bactericidal) penicillin, streptomycin, bacitracin, neomycin, vanomycin, methicillin, ampicillin, cloxacillin.

GROUP 2. (bacteriostatic) the sulphonamides, the tetracyclines, erythromycin, chloramphenicol.

4. SYNERGISM AND ANTAGONISM

Saint (1953) reports that when two antibiotic substances are given together three possible effects may result:-

(a) The combination may have an additive effect - such an effect may be obtained by simply increasing the dosage of one or other drug.

(b) There may be interference to the action of one drug by the other - an antagonistic effect.

(c) The total antibacterial activity may be increased beyond that which would be obtained by simple additive effect - a synergistic effect.

The Group 1 (bacteriocidal) antibiotics, when combined with one another, are frequently synergistic. On the other hand the Group 2 (bacteriostatic) drugs have merely an additive effect and are never synergistic. Combinations of the two groups may be synergistic, antagonistic or indifferent in effect. Tolhurst, Buckle and Williams (1963) point out that uniformly synergistic or antagonistic pairs of drugs do not exist either within each group or in mixtures of a member of each group. The effect obtained is dependent on the micro-organism and may vary with different strains of the same organism.

Lucas and Kramer (1959) and Anderson (1964) state that combinations of drugs may be considered for therapeutic use when;

(i) A mixed infection is present and no one drug would be effective against all the organisms present.

(ii) Resistant variants are shown to emerge rapidly.

(iii) Prolonged therapy is required for treatment of a chronic infection.

Fortunately infections of the oral cavity and associated tissues are rarely serious enough to warrant the use of combined antibiotic therapy.

5. PROPHYLACTIC USE OF ANTIBIOTICS

The routine use of antibiotics as a prophylactic in oral surgery is to be deplored. The use of these drugs, either parenterally or topically, following simple oral surgical procedures in an attempt to prevent post-operative infection is unwarranted, as;

(a) infection may not occur

(b) if an infection does develop, it may be resistant to the antibiotics used.

(c) the patient is exposed to the risks that are involved in the use of antibiotics, these are; sensitisation to the drug, emergence of resistant strains of the organism, toxic effects and superinfection (e.g. moniliasis).

However prophylactic antibiotic therapy seems desirable when surgical interference with infected tissue is undertaken and when large areas of tissue are exposed to potentially pathogenic organisms (e.g. following radical antrostomies, enucleation of large cysts, etc.). In such cases their use is dependent upon the judgment of the surgeon.

Prophylactic administration of antibiotics to patients with rheumatic or congenital disease of the heart is now accepted practice. This procedure is justified in an attempt to prevent a bacteraemia which may occur following oral surgical procedures and result in bacterial endocarditis (Tolhurst, Buckle and Williams, 1963; Lucas and Kramer, 1959; Johnson, 1960). On the other hand Thomson (1954) considers that little proof has been offered in support of the efficacy of this prophylactic procedure and he reports that cases of bacterial

endocarditis have been recorded following dental extraction performed under antibiotic cover. It is however the policy of both the National Heart Foundation of Australia (1963) and the American Heart Association (1964) to recommend the administration of prophylactic antibiotic therapy in such cases.

6. CHEMOTHERAPEUTIC DRUGS.

(a) Sulphonamides: the antibacterial action of the sulphonamides is dependent on the chemical resemblance of the para-amino benzene group to para-amino-benzoic acid, an essential metabolite for many bacteria. This similarity results in the drug being incorporated in place of para-amino-benzoic acid with resultant inhibition of bacterial multiplication. This bacteriostatic action is aided by the patient's natural defence mechanisms.

Lucas and Kramer (1959) report that sulphonamides are active against most Gram-positive and Gram-negative cocci and the Gram-negative *Bacillus coli*. Tolhurst, Buckle and Williams (1963) state that the use of sulphonamide drugs over a long period of time has led to the emergence of resistant strains. This reviewer has noted, in the treatment of oral infections, that many organisms found in smears demonstrate resistance in vitro to the sulphonamide drugs. It is important to note that sulphonamides are inactivated by pus and necrotic tissue and are thus not indicated for the treatment of closed abscesses however Tolhurst, Buckle and Williams (1963) consider that these drugs are of value in checking spread of infection

due to the high tissue concentration obtained.

Thoma (1963) and Glaser (1953) state that, because of their undesirable toxic effects and the increasing resistance demonstrated by bacteria, the sulphonamides have generally been replaced by antibiotics. Thoma (1963) considers that sulphadiazine is the sulphonamide of choice as its rapid absorption and slow excretion result in high blood and tissue concentration. Also toxic reactions to sulphadiazine are uncommon.

Sulphonamides are routinely administered orally. To avoid renal complications care must be taken to make the urine alkaline and it is suggested that one pint of fluid for each gram of sulphonamide should be administered (Tolhurst, Buckle and Williams, 1963).

The most common toxic effects of the sulphonamides are headache, mental depression and general malaise. However these complications are not considered dangerous and therapy need not be interrupted. Renal complications may occur (Thoma, 1963) and these are first evidenced by haematuria and renal pain. If these symptoms present or the patient shows dermal reaction to the drug, treatment should be suspended. Rarely, serious blood dyscrasias may occur so that if prolonged therapy is undertaken, regular blood counts should be carried out. Most toxic effects are due to sensitisation and it is important to inquire whether the patient is aware of previous sensitivity reaction which may have resulted from the previous use of any sulphonamides.

(b) Penicillin: penicillin acts by interfering with the metabolism of glutamic acid, an essential growth factor of many bacteria. According to Lucas and Kramer (1959), Thoma (1963) and Northrop (1949), penicillin has both bacteriostatic and bactericidal activity while other authors consider it to have principally a bactericidal action (Tolhurst, Buckle and Williams, 1963; Accepted Dental Remedies, 1963).

Penicillin is most active against organisms undergoing multiplication, those organisms in the resting state being relatively unaffected even if they are sensitive. One valuable property possessed by penicillin is its continued activity in the presence of blood, serum and pus (Northrop, 1949; Tolhurst, Buckle and Williams, 1963).

Penicillin acts against many pathogenic Gram-positive bacteria, actinomyces, spirochaetes and a limited number of Gram-negative organisms. However certain strains of many common organisms e.g., *Staphylococcus aureus*, have developed resistance to penicillin. Organisms previously susceptible may develop resistance after continued exposure to the drug, while some form enzymes, collectively known as penicillinases, which destroy penicillin. This emphasises the importance of isolating the infecting organism and testing its sensitivity.

Systemic administration of penicillin is by either injection or the oral route. The most satisfactory method of administration is by injection of crystalline penicillin where rapid high blood

concentration is required, but this form of the drug is rapidly excreted and, when an effective level is desired over a long period, procaine penicillin should be given intra-muscularly. In recent years phenoxymethyl penicillin, or penicillin V has been synthesised. This form of the drug is capable of resisting gastric acidity and hence may be given orally. The oral administration of penicillin V has been reported by Lucas and Kramer (1959), Tolhurst, Buckle and Williams (1963), and Blumenthal et al (1957) to produce effective blood levels of the drug.

Topical use of penicillin in healing wounds has been reported by Scrivener and Shantz (1947) and Epstein and Kauffman (1951). This reviewer agrees with Lucas and Kramer (1959), Kramer (1956) and Thoma (1963) that topical application of penicillin is contra-indicated due to the possibility of inducing sensitisation and the emergence of resistant strains of bacteria. Kharitanov and Artemova (1961), Bramley (1957) and Cook (1948) report sensitivity reactions following the placing of penicillin in extraction wounds and the use of penicillin lozenges for gingival infection.

Lucas and Kramer (1959) state that penicillin is almost free from toxic effects and that large doses may be given without danger to the patient. However sensitisation to the drug may occur and, with the widespread use of the drug today, allergic reactions are more frequently encountered. The most common allergic response is a mild urticarial reaction, however severe serum-sickness type of delayed reaction and varying dermal reactions may occur. Rarely, a severe anaphylactoid reaction may occur and may prove fatal.

Penicillin should never be given to a patient who has had a previous reaction to the drug. It has been recommended that the drug should not be administered to patients who suffer from allergic disorders such as asthma or hay fever.

Antihistamine drugs are generally administered for treatment of urticarial reactions. Thoma (1963) suggests the use of diphenhydramine hydrochloride (Benadryl), 25 mg. given four hourly or tripeleannamine hydrochloride (Pyribenzamine), 25 mg. given thrice daily, for the treatment of dermal reactions to penicillin.

Anaphylactoid reactions are characterised by sudden loss of vasomotor tone, muscular spasm, respiratory difficulty, cyanosis, coughing and the presence of a weak, thready pulse (Thoma, 1963). Often associated dermal changes occur. Sara (1963) recommends immediate intravenous administration of 1 in 10,000 adrenalin when anaphylaxis occurs. He states that 1 ml. should be injected every thirty seconds until recovery is apparent. If venipuncture is not possible 5 ml. of the adrenalin should be given by deep intramuscular injection. Other forms of resuscitation (closed chest cardiac massage, artificial respiration) should also be carried out if necessary.

Spiegel (1959) and Minno and Davis (1957) report the use of penicillinase in the treatment of allergic penicillin reactions. These authors state that the enzyme inactivates the circulating penicillin and is valuable in relieving symptoms. It must be noted that penicillinase is slow acting and its use is not indicated in

emergency treatment. The latter authors consider that penicillinase may be used in association with antihistamines in the treatment of allergic reactions.

(c) Erythromycin: this antibiotic substance is one whose therapeutic indications resemble penicillin. Erythromycin is grouped with the bacteriostatic group of chemotherapeutic drugs.

Erythromycin is active against Gram-positive and Gram-negative cocci and Gram-positive bacilli. Tolhurst, Buckle and Williams (1963) state that erythromycin is of value for treatment of infections due to organisms resistant to penicillin. However resistant organisms may emerge following its use as a result of mutation or of selective growth of resistant strains. Erythromycin is routinely administered by mouth however, when an immediate high blood concentration is required, the drug may be given intravenously.

Following oral administration of erythromycin abdominal cramps, nausea, vomiting and, rarely, diarrhoea may occur. These gastrointestinal disturbances appear to be related to the dosage. Skin rashes and black hairy tongue have been reported occasionally.

Following an evaluation of 278 cases where erythromycin was administered for treatment of infection, Grimm and Albertson (1954) consider that this drug is of value in dental practice. These authors report that few side effects resulted from use of the drug.

(d) Streptomycin: Tolhurst, Buckle and Williams (1963) report that two factors militate against the use of streptomycin;

(i) High toxicity, this strictly limits dosage for which reason

it should not be used if some other less toxic drug is suitable.

(ii) Highly resistant mutants emerge which replace the original sensitive organisms and make continued treatment valueless.

The complications involved in the use of streptomycin make it unsuitable for use in dental practice (Lucas and Kramer, 1959).

(e) Chloramphenicol (Chloromycetin): Chloramphenicol has been shown to produce severe bone marrow damage which, in some cases, has resulted in death. In consideration of this fact this drug should not be used in infections where other drugs are likely to be effective, therefore the drug is not suited for general use in dental practice.

(f) Tetracyclines: the principal tetracycline compounds are chlortetracycline (aureomycin), oxytetracycline (Terramycin) and the parent compound tetracycline (Achromycin).

These drugs resemble each other and chloramphenicol in antibacterial activity and collectively, with chloramphenicol, are often referred to as the "broad spectrum antibiotics". Each of these drugs is active against most of the Gram-positive and Gram-negative bacteria and has some activity against some rickettsiae and certain viruses.

Tetracyclines are routine given orally, the duration of the therapy depending on the severity of the infection. It is suggested by Tolhurst, Buckle and Williams (1963) that the minimum period of treatment should be not less than five days.

Following oral administration of the tetracyclines the most

common toxic effect is gastro-intestinal disturbance such as nausea, vomiting and diarrhoea (Goulding, 1957; Schaffer, 1953; Harvey, 1964). Lucas and Kramer (1959) suggest that if the tetracyclines are taken with milk the gastro-intestinal disturbances are minimised, however Tolhurst, Buckle and Williams (1963) state that milk may interfere with absorption of the drug.

As the tetracycline group of drugs is effective against most organisms, the normal balance of flora of the gastro-intestinal tract may be disturbed permitting superinfection by the fungus group of organisms (e.g. *Candida albicans*).

The reviewer considers that, although antibiotics and allied drugs are invaluable in the treatment of infection, their use must be based on sound principles and they should not be used in place of other accepted forms of treatment.

The clinician must bear in mind that the indiscriminate use of antibiotics may result in sensitisation of the patient to the drug or emergence of resistant strains of organisms. This may preclude the use of a drug which may be required at a future date as a life-saving measure.

SOFT TISSUE REACTION TO NECROTIC FRAGMENTS OF ALVEOLUS.

1. INTRODUCTION
2. AETIOLOGY
3. CLINICAL FEATURES
4. FATE OF NECROTIC ALVEOLAR BONE
5. DIAGNOSIS
6. TREATMENT.

1. INTRODUCTION

Following extraction of a tooth or other surgical procedures involving the alveolar bone, small pieces of alveolus may be left without nutrient supply. As a result these fragments may undergo necrosis and may act as irritants in the healing wound, and subsequently result in the exuberant outgrowth of friable granulation tissue from the region (Archer, 1961). This tissue growth may cause concern to the patient, possibly due to the purulent appearance of its surface or its haemorrhagic tendency. According to Glickman et al (1947), its presence may retard healing.

2. AETIOLOGY

Fracture of portion of the superficial alveolus may occur during extraction of a tooth (Berlove, 1959). If the circulation to the fragment remains intact there is a favourable prognosis for its continued vitality. However, if the periosteal attachment is lost, the bone will become necrotic and will stimulate a localised inflammatory reaction in the healing extraction socket (Glickman et al, 1947).

Radden (1959) reports that necrotic fragments of bone were found in surgical wounds following removal of bone by means of burs. This finding is supported by Simpson (Jan. 1961) who also found that these fragments caused little reaction if they lay between the muco-periosteal flap and uncut bone. However he notes that, if the fragments approximated the incision, a marked inflammatory response frequently occurred.

In an experimental study concerning healing following the use of the surgical chisel, Simpson (Mar. 1961) found that, as the chisel produced larger bony fragments, these were more likely to be removed during debridement. He also found that, if fragments were left in the wound, they resulted in a less severe inflammatory response than when a bur was used, however he does not give any explanation for the difference in degree of inflammatory response.

3. CLINICAL FEATURES

The presence of necrotic bone fragments in a healing extraction socket or a surgical wound may result in an excessive overgrowth of granulation tissue from the region. Archer (1961) describes this mass as "irregular 'mulberry-like' reddish-blue granulation tissue" and notes that pus can often be freely expressed from its cut surface. A similar description is given by Berlove (1959), Thoma (1963) and Hayward (1957). Berlove (1959) notes that the tissue is frequently haemorrhagic and that the patient may experience post-operative haemorrhage from the site.

Moderate pain may often be present at the wound however frequently the principal complaint is that of the appearance of the wound as the patient is concerned about the possibility of infection of the site (see Illus. page 178).

4. FATE OF NECROTIC BONE

Radden (1959) states that pieces of detached bone lying free

SOFT TISSUE REACTION
TO
NECROTIC ALVEOLUS



Sixteen days following extraction of five upper anterior teeth the patient returned concerned about the appearance of the extraction sockets. She was experiencing slight discomfort but was more worried with the possibility of infection of the area.

No discreet sequestra could be detected by gentle probing and radiographs of the sockets did not reveal any abnormality. The patient was instructed to use intra-oral heat to the region. When seen ten days later the unhealthy granulation tissue was no longer visible and the sockets appeared to be healing normally.

in the blood clot rarely survive and frequently undergo necrosis. Both Smith (1958) and Radden (1959), in experimental studies, found that the small necrotic fragments became surrounded by epithelium which appeared to proliferate, from the wound surface, in the direction of the inflammatory exudate around the bony spicules. The proliferating epithelium enclosed the fragments and appeared to be the principal factor in "exteriorising" the bone (Smith, 1958), leading to shedding of the sequestra.

The other method of elimination of necrotic alveolar bone is by osteoclastic action. This often occurs when the fragments are deeply placed in the wound. This process, according to Glickman et al (1947), may result in localised abscess formation which may even occur under "a well-formed, healed epithelial surface."

5. DIAGNOSIS

The clinical appearance of the wound would lead the oral surgeon to suspect the presence of sequestra. The wound may contain an excessive growth of granulation tissue which bleeds easily and has a purulent surface. Small pieces of sequestra, which appear irregular and rough in texture and yellowish-grey in colour (Weinman and Sicher, 1955), may be visible in the tissue.

Radiographs of the wound often reveal the presence of sequestra which, according to Thoma (1963), may be recognised by their radiopacity and their demarcation from the surrounding bone. However, frequently no distinct sequestra may be demonstrated radiographically in the wound.

6. TREATMENT

Treatment is directed towards removal of the irritant namely the sequestrum. Frequently careful exploration of the friable granulation tissue filling the wound may reveal the presence of superficial sequestra which may be gently lifted from the socket.

If, however, the bony fragment is more deeply placed or is not readily removed, two approaches may be adopted:-

(a) Conservative treatment:- Localised intra-oral heat may be used to hasten the healing process and to encourage spontaneous shedding of the sequestra.

(b) Surgical treatment:- Thorough debridement of the wound may be carried out to remove the bone fragments.

Following removal of the irritant, healing is usually rapid and uneventful.

ORO-ANTRAL FISTULA

1. INTRODUCTION

2. AETIOLOGY

3. CLINICAL FEATURES

4. TREATMENT

(a) Closure with a buccal muco-
periosteal flap.

(b) Closure with a palatal flap.

(c) Closure with a metal plate.

1. INTRODUCTION

A communication between the maxillary sinus and the oral cavity may occur as a result of operative procedures in the maxillary premolar-molar region. This complication most frequently occurs during extraction of one of the maxillary molar teeth (Wallner, 1952) and is usually the result of the anatomical form of the maxillary sinus.

When such a communication occurs during a surgical procedure an immediate attempt at closure is frequently successful. However infection of the antrum or local factors may lead to the formation of an oro-antral fistula which usually necessitates closure by a plastic operation to correct the defect.

2. AETIOLOGY

Communication between the oral cavity and the maxillary sinus may result from:

(a) Removal of part of the floor of the maxillary sinus during tooth extraction or other surgical procedures (Archer, 1961). According to Balogh (1963) this is most likely to occur during the extraction of the upper second premolar tooth and the upper first and second molar teeth as the root apices of these teeth frequently approximate the floor of the maxillary sinus. This intimate relationship is also noted by Bourgoyne (1948) and Fickling (1957).

(b) Destruction of the floor of the maxillary sinus by chronic infection at the apex of a maxillary tooth. The extraction of the involved tooth may then result in a communication between the mouth

and the maxillary sinus (Balogh, 1963). Fickling (1957) however considers that most openings into the antrum are due to an anatomic relationship rather than the presence of bony pathology.

(c) Perforation of the bony antral floor or epithelial lining by the incorrect use of curettes or elevators (Fickling, 1957; Archer, 1961).

(d) Forcing a fractured or retained root fragment into the maxillary sinus while attempting to remove it (Archer, 1961).

When an oro-antral communication occurs during an oral surgical procedure the following authors Fickling (1957), Wallner (1952), Thoma (1963), Breakstone (1957), Bourgoyne (1948), Archer (1961) and Berlove (1959) recommend that an immediate attempt should be made to achieve closure by approximating the tissue margins of the wounds with sutures. Archer (1961) and Wallner (1952) recommend that antibiotic therapy should be commenced together with the use of nose drops to shrink the nasal mucosa to ensure adequate drainage of the sinus. The patient should be instructed to avoid blowing his nose or sneezing as these actions may result in pressure on the healing tissues.

Both Archer (1961) and Wallner (1952) consider that a nasal window should be made to facilitate drainage if there is any sign of sinusitis, either at operation or developing later as a result of antral involvement.

It has been the experience of the reviewer that, if no antral infection is present, the potential for uneventful closure of the opening is very good.

If the maxillary sinus is infected drainage may occur through the oro-antral opening with the formation of an oro-antral fistula (Cash, 1951; Fickling, 1957; Breakstone, 1957; Thoma, 1963; Wallner, 1952). Thoma (1963) considers that any local factor which tends to keep the opening patent e.g. the placement of a drain or gauze pack, may result in the formation of a fistulous tract.

3. CLINICAL FEATURES

When an oro-antral fistula is present the patient most frequently complains of passage of air and fluids through the opening. Examination with a pliable silver probe will reveal the presence of a communication between the mouth and the maxillary sinus.

When an oro-antral fistula is present it is possible that the maxillary sinus may be infected. The infection is usually chronic in form as drainage occurs freely through the oro-antral fistula. However acute exacerbations of the infection are possible (Shafer, Hine and Levy, 1960).

A patient with acute maxillary sinusitis usually experiences severe pain and swelling overlying the sinus. The associated soft tissues are tender and frequently the remaining posterior teeth on the affected side are sensitive to percussion. The patient may also experience a foul post-nasal discharge, (Shafer, Hine and Levy, 1960).

Chronic maxillary sinusitis may be asymptomatic. Sometimes vague pain is present and there may be a feeling of fullness of the affected side. Also there may be mild discharge of purulent material into the nose.

In an inflammatory condition of the maxillary sinus, thickening of the mucosa may occur with metaplasia of the ciliated epithelium to stratified squamous epithelium (Schram, 1962). "Antral polyps", hyperplastic granulation tissue with infiltration of inflammatory cells, may develop and often one of these "polyps" may extrude through the oro-antral fistula into the oral cavity (Breakstone, 1957). Radiographic examination of an infected sinus reveals increased radio-opacity due to the hyperplastic tissue and inflammatory products.

4. TREATMENT

Moose (1962) states that an early attempt at closure of an oro-antral fistula is contra-indicated and should be delayed until healthy marginal tissue is present.

This reviewer considers that no attempt should be made to effect closure of an oro-antral fistula for some months as, if the antrum is not infected, spontaneous closure or reduction in diameter of the defect may occur.

Fickling (1957) suggests that light cauterisation of the epithelialised tract may result in closure of a very small oro-antral fistula. The reviewer, using copper sulphate as a chemical cautery, has found this procedure to be efficacious in treatment of small communications.

If maxillary sinusitis is present it is generally agreed that an attempt to close an oro-antral fistula must be preceded by

treatment of the antral infection (Fickling, 1957; Wallner, 1952; Moose, 1962; Burch, 1949).

Breakstone (1957), Thoma (1963) and Archer (1961) consider that, with antibiotic cover, curettage of the infected antral membrane should be carried out by means of a Caldwell-Lue approach. However, Fickling (1957) does not favour antrostomy as he considers the associated trauma leads to complications. He therefore uses pre-operative antral lavages to control antral infection.

Wallner (1952) suggests making a nasal window to allow aeration and drainage of the sinus and states that this procedure will "result in the end of nasal suppuration".

Following treatment of the antral infection, if present, closure of the oro-antral fistula may be attempted. Several methods of closure of the fistula have been presented in the literature. The procedures most often suggested are:-

(a) Closure with a buccal muco-periosteal flap.

This operative technique is advocated by Herd (1950), Cooley (1954), Schram (1962), Moose (1962) and Wallner (1952). The procedure as outlined by Moose (1962) is representative of the other authors and is briefly as follows:-

- (i) The fistulous tract is excised.
- (ii) The palatal muco-periosteum is elevated to provide some tissue mobility.
- (iii) A broad based buccal flap is raised and elevated deep into the sulcus.

(iv) The margins of the flaps are approximated by sliding the buccal flap over the defect.

(v) The tissues are then sutured in position using mattress sutures.

It is important, when this technique is employed, that the tissues must be free of tension otherwise the activity of the cheek musculature would tend to tear the tissues apart before healing could occur. Herd (1950) and Wallner (1952) state that added tissue mobility can be obtained by incising the periosteum of the buccal flap in the region of the muco-buccal fold.

One criticism of this method is that it may result in obliteration of the buccal sulcus which may complicate denture construction at a later date.

(b) Closure with a palatal flap.

Rotation of a palatal pedicle flap to cover an oro-antral fistula is suggested by Breakstone (1957), Thoma (1963), Kruger, (1959), Burch (1949), and Arnott (1963). This technique is as follows:-

- (i) A palatal muco-periosteal flap is designed so that it includes the greater palatine artery and is of sufficient length and breadth to provide adequate coverage of the fistula. Kruger (1959) suggests the use of a template made from a study model to aid flap design.
- (ii) Definite incision of the palatal tissues is made according to the flap design but the flap is not elevated.
- (iii) A small V-shaped section of tissue is removed at the

disto-lingual margin of the fistula to facilitate rotation of the flap.

- (iv) The anterior 2 mm. of the flap is denuded of epithelium.
- (v) The epithelial lining of the fistula is excised.
- (vi) The buccal muco-periosteum adjacent to the fistula is elevated.
- (vii) The palatal flap is elevated and rotated to cover the osseous opening. The end of the flap being inserted under the buccal tissues.
- (viii) The flap is sutured using stainless steel sutures (Arnott, 1963; Tholen, 1945).
- (ix) The donor area is protected with a Tinc. Benz. Co. gauze dressing.

Both Breakstone (1957) and Burch (1949) suggest the use of an acrylic splint to support and protect the palatal flap during the healing process.

(c) Closure with a metal plate.

Tantalum plate is used by Budge (1952) to effect closure of a chronic oro-antral fistula. Following excision of the epithelialised fistulous tract, the buccal and palatal tissues are reflected to allow good exposure of the bony defect. A piece of 32 gauge tantalum plate is cut and contoured to overlap the margins of the opening. Holes are drilled in the buccal and lingual extensions of the plate to allow periosteal reattachment to the bone thus holding the plate firm. The tissue flaps are approximated and sutured, healing occurring by first

intention. The metal plate is not removed.

Crolius (1956) and Steiner (1960) recommend the use of 24 carat 36 gauge gold plate in the treatment of an oro-antral fistula. The technique used is as follows:- following excision and curettage of the fistulous tract, the buccal and palatal tissues are elevated to allow good access to the bony opening. A piece of gold plate is cut to overlap the defect by 2-3 mm. and burnished to conform to the bony contour. The tissues are sutured over the gold plate using silk sutures however no attempt is made to appose the tissues and during healing the gold plate remains visible. The sutures are removed after one week. Crolius (1956) reports that healing occurs within 3-4 weeks and usually by this time the gold plate is quite loose and is easily removed at that time. Steiner (1960) found, however, that healing takes approximately eight weeks. Following removal of the gold plate both authors report the presence of a bridge of healthy tissue covering the defect and note that, following removal of the gold plate, the tissue flaps heal readily. Steiner (1960) considers that the gold plate acts either as a periosteal irritant and thus stimulates repair or as a mechanical barrier which permits the normal regenerative processes to take place.

Following operative closure of an oro-antral fistula the formation of a nasal window to facilitate antral drainage is suggested by Kruger (1959), Crolius (1956), Wallner (1952), Archer (1961) and Tholen (1945).

If a surgical approach to close an oro-antral fistula is either

unsuccessful or not indicated for the patient, consideration may be given to covering the opening by means of a denture. Such an appliance, with adequate extension in the region of the defect, would seal the opening and prevent passage of fluids and air between the oral cavity and the maxillary sinus.

NERVE INVOLVEMENT

1. SENSORY NERVE INVOLVEMENT.

- (i) Introduction
- (ii) Aetiology
- (iii) Nerves involved
- (iv) Symptoms
- (v) Incidence of Inferior Alveolar Nerve Involvement.
- (vi) Duration of Inferior Alveolar Nerve Involvement.
- (vii) Treatment

2. MOTOR NERVE INVOLVEMENT.

- (i) Introduction
- (ii) Permanent Facial Paralysis
- (iii) Transient Facial Paralysis.

It is possible that, during any oral surgery procedure, interference may occur with the nerve supply of a region. To facilitate the review of such involvements and their possible sequelae this section is divided into two parts - one embracing sensory nerve damage, the other motor nerve damage.

1. SENSORY NERVE INVOLVEMENT

(i) Introduction.

Following oral surgery a patient may experience anaesthesia or paraesthesia of a part of the oral cavity, or associated structures, due to trauma of a sensory nerve during the operative procedure. This altered sensation may persist for as little as a few hours or may persist indefinitely, depending on the degree of damage sustained by the nerve.

Anaesthesia, or loss of sensation, is experienced when the nerve trunk innervating the area is either traumatised or severed. Paraesthesia occurs due to lesser trauma of the nerve and is defined as "a perverted sensation: tingling crawling of the skin which occurs in peripheral neuritis" (New Gould Med. Dict., 1956). According to Simpson (1958) this altered sensation may be described by the patient as a tingling feeling, a burning sensation, aching, hyperaesthesia or as actual pain.

Paraesthesia may follow anaesthesia of the affected area and may be the first symptom of which the patient complains as, according to Howe and Poyton (1960), anaesthesia is well tolerated but the irregular

sensation that occurs in paraesthesia is more likely to cause distress.

(ii) Aetiology.

Trauma to the nerve is the aetiological factor in both post-operative anaesthesia and paraesthesia. This trauma may occur in the following ways:-

(a) Needle penetration of the nerve bundle during injection is suggested by Schram (1962), Thoma (1963), Archer (1952), and Cogswell (1942). Schram (1962) considers that trauma is more likely to occur when injection is made directly into a foramen as the nerve is unable to move away from the needle point. Monheim (1961) states that needle trauma probably causes haemorrhage into the neural sheath and the resultant pressure causes disturbed sensation.

(b) Nerves may be severed by incision while raising a muco-periosteal flap. This is thought by Cogswell (1942) to be likely when making an incision in the region of the mental foramen. Simpson (1958) suggests that the surgical removal of hypertrophy tissue overlying the premolar region of a resorbed ridge may result in severance of the mental nerve.

(c) During extraction of a tooth or elevation of a root fragment, compression of a sensory nerve may occur with resultant trauma to the nerve and paraesthesia (Holland, 1948; Thoma, 1963). This is most likely to occur during removal of lower third molar teeth.

(d) In deep bone surgery the extent of a lesion may necessitate unavoidable damage or severance of a nerve (Cogswell, 1942).

(e) Careless use of a flap retractor may permit undue pressure

to be applied to the nerve with resultant abnormal sensation.

(iii) Nerves Involved.

The nerves most frequently involved are the inferior alveolar nerve and its terminal branch, the mental nerve. Other nerves less commonly affected are the lingual nerve, the infra-orbital nerve, the anterior superior alveolar nerve and the nasopalatine nerve.

(a) Inferior Alveolar Nerve: This structure may be involved during the removal of lower premolar and molar teeth or roots, especially third molar teeth, which often lie in close relation to the mandibular canal. Cases are reported in the literature by Austin (1947), Repass (1946), Beauchamp (1947) and Jackson (1951) where the inferior alveolar nerve either grooved or passed through the roots of mandibular third molar teeth. In such cases some damage to the nerve during extraction is inevitable.

Cysts or tumours of the mandible may involve the inferior alveolar nerve and, according to Thoma and Goldman (1960), injury to the nerve may occur during surgical treatment of these lesions.

Where severance of the inferior alveolar nerve has occurred Thoma (1963) and Cogswell (1942) consider that, if the nerve ends are approximated, repair is possible. However, even if the nerve ends are approximated satisfactorily, the distal portion of the nerve may undergo Wallerian degeneration, innervation being re-established by an outgrowth from the proximal stump (Schram, 1962).

It is stated by Holland (1948) and De Bats and Phillips (1944) that return to normal sensation is likely as the mandibular canal provides a favourable situation for repair.

(b) Mental Nerve: Injury to this nerve may be caused by removal of unerupted teeth or roots in the region of the mental foramen. Simpson (1958) considers that, in this region, trauma is frequently unavoidable even though every effort is made to prevent it. Holland (1948) states that careless flap design in this region is a factor in mental nerve involvement and he considers that the potential for regeneration of this nerve is less than for the inferior alveolar nerve due to the mobility of the tissues.

(c) Lingual Nerve: Trauma may occur during an inferior alveolar nerve block injection (Archer, 1952; DeBats and Phillips, 1944). Extraction of lower third molar teeth may also cause damage to the lingual nerve which frequently approximates the bone overlying the roots of these teeth (Archer, 1952; DeBats and Phillips, 1944).

Archer (1961) suggests that the lingual nerve may be involved during removal of salivary calculus from the submaxillary gland or during marsupialisation of a ranula. This author also states that, if the nerve is severed, the resulting anaesthesia may be permanent.

(d) Infra-Orbital Nerve and Anterior Superior Alveolar Nerve:

These two nerves may be damaged during a Caldwell-Lue approach to the maxillary sinus (Kruger, 1959) while DeBats and Phillips (1944) consider that the infra-orbital nerve may be involved during exodontia. This latter possibility seems unlikely to the reviewer.

(e) Naso-palatine Nerve: It is an interesting observation that no mention is found in the literature of altered sensation due to

severance of the naso-palatine nerve as it leaves the incisive fossa. Archer (1961) states that union readily occurs between the severed ends due to suturing of the flap. However, even if approximation was ideal, a period of altered sensation would be expected.

(iv) Symptoms of Sensory Nerve Involvement: Following sensory nerve involvement a patient may complain of either anaesthesia or paraesthesia of the area supplied and this will correspond to the anatomical distribution of the nerve.

(a) Inferior Alveolar and Mental Nerves: According to Simpson (1958) the principal indication of injury to either of these nerves is either labial anaesthesia or paraesthesia. Intra-orally the inner surface of the lip and the adjacent labial mucosa of the alveolar process are similarly affected although the patient is frequently unaware of this.

If the inferior alveolar nerve is injured proximal to the point where it divides into mental and incisal branches, the symptoms are the same as for mental nerve involvement except that the teeth anterior to the injury may show altered sensation (Simpson, 1958).

(b) Lingual Nerve: Damage to this nerve results in either anaesthesia or paraesthesia of the anterior two-thirds of the tongue, the mucous membrane of the floor of the mouth and the lingual mucoperiosteal tissue of the affected side. Archer (1952) points out that the chorda tympani nerve, which lies in a common sheath with the lingual nerve, may be traumatised with that nerve resulting in altered taste sensation.

(c) Infra-Orbital Nerve and Anterior Superior Alveolar Nerve:

When the infra-orbital nerve is damaged the patient may complain of numbness of the upper lip, the soft tissues of the cheek and the side of the nose.

Interference with the anterior superior alveolar nerve may cause altered sensation in the incisor and canine teeth (Kruger, 1959).

(v) Incidence of Involvement of Inferior Alveolar Nerve.

The studies reported in the literature emphasise the fact that the inferior alveolar nerve is the sensory nerve most often involved during oral surgery procedures. The available literature appears to be solely concerned with the incidence and duration of involvement of this nerve.

In a series of 1335 cases of removal of lower third molar teeth Howe and Poyton (1960) found an incidence of 5.2 percent of impairment of labial sensation. This figure compares favourably with the incidence of 5.2 percent reported by Frank (1959) in a smaller series of extractions.

No figures were found by this reviewer concerning the incidence of sensory nerve involvement following surgery in other areas of the mouth.

(vi) Duration of Anaesthesia and Paraesthesia of Inferior Alveolar Nerve.

Howe and Poyton (1960) investigating impairment of labial sensation, report the following results:

In 70 cases of impaired sensation:

18.6 per cent were symptom free in 7 days

64.3 per cent were symptom free in 8 weeks

81.5 per cent were symptom free in 12 weeks

90.0 per cent were symptom free in 16 weeks

2 cases returned to normal after 72 weeks while 5 cases had permanent altered sensation.

Frank (1959), in reporting on the return of normal sensation in 16 patients found:

1 patient recovered in less than 1 month

4 patients recovered in 1 - 3 months

6 patients recovered in 3 - 6 months

1 patient recovered in 8 months.

2 patients still had abnormal labial sensation after 18 months.

In 50 cases of paraesthesia following oral surgery Simpson (1958) found that 80 per cent had recovered symptom-free within six months. He draws the following conclusions from his studies:-

(a) Not less than 50 per cent of patients can be expected to recover within eleven weeks.

(b) The possibility of spontaneous recovery after six months is slight.

(vii) Treatment.

According to Archer (1952) there is no specific treatment that will aid in nerve regeneration. However it is apparent from the literature that, if a sensory nerve is severed, the ends should be

approximated as closely as possible to aid regeneration.

From the foregoing it can be seen that in some oral surgical procedures some sensory nerve damage is inevitable. In the experience of the reviewer, altered sensation has most often been seen following removal of retained lower premolar roots. In such cases where nerve damage is anticipated the oral surgeon must, prior to surgery, warn the patient that impairment of sensation may occur and that, if such impairment does occur, the altered sensation may persist for an indefinite period post-operatively.

2. MOTOR NERVE INVOLVEMENT.

(i) Introduction.

The seventh cranial nerve is the motor nerve most likely to be involved during oral surgery procedures. When this nerve is damaged a facial paralysis results, the extent of which depends on the number of fibres involved.

When the facial nerve is involved, one or more of the following signs may be present - inability to close the eye, wrinkle the forehead or elevate the lip. The eyeball may roll upwards revealing the sclera of the eye (Bell's Sign).

(ii) Permanent Facial Paralysis.

Fibres of the facial nerve may be damaged during incision for drainage (DeBats and Phillips, 1944) or during operative procedures in the region of the temporo-mandibular joint (Archer, 1961).

Severance of the facial nerve or one of its branches is a

particularly disturbing complication because of the resulting facial disfigurement. DeBats and Phillips (1944) suggest approximation of the severed nerve ends but consider that muscle atrophy is likely to occur before regeneration could take place. Archer (1961) considers that, when facial paralysis has occurred, some restoration of activity may be achieved by the plastic surgeon using muscle and fascial implants.

(iii) Temporary Facial Paralysis.

Temporary facial paralysis may occur following inferior alveolar nerve block injection (Archer, 1961; DeBats and Phillips, 1944; Schram, 1962). Cases of transitory Bell's Palsy immediately following inferior alveolar nerve block injection are reported by Anwandter (1944) and Droter (1959) while Stoy and Gregg (1951) report a case that occurred five weeks following extraction of a lower molar tooth. In each of these cases the characteristic signs of facial paralysis were observed. Droter (1959) observed, that after one hour, tonus of the facial muscles started to return and total recovery occurred within a few hours. However, Stoy and Gregg (1951) report that, in the case they observed, restoration of normal activity took six weeks.

According to Archer (1961), Schram (1962) and Droter (1959) this phenomenon is due to a direct action of the local anaesthetic solution on the facial nerve and the resulting paralysis lasts as long as the anaesthesia (Stoy and Gregg, 1951). This may occur when injection for the inferior alveolar nerve is made too deep and the

needle enters the substance of the parotid gland (Droter, 1959). However, Anwandter (1944), following a clinical study, disagrees with this theory as he found that frequently adequate anaesthesia of the inferior alveolar nerve was obtained and that there was a low percentage of paralysis reported following intentional deep injection.

Stoy and Gregg (1951) and Anwandter (1944) consider that the injection of local anaesthetic solution may cause a vascular reflex, acting via the sympathetic plexus of the external carotid artery, resulting in an ischaemic paralysis in the region of the stylo-mastoid foramen. This causes impaired activity of the facial nerve. The duration of the transient paralysis, according to Stoy and Gregg (1951), is dependent on the degree of damage caused by the ischaemia.



OSTEORADIONECROSIS

1. INTRODUCTION
2. IRRADIATION EFFECT ON ORAL AND PARAORAL TISSUES
 - (a) Skin
 - (b) Mucous Membrane
 - (c) Salivary Glands
 - (d) Teeth
 - (e) Bone
3. AETIOLOGY
4. CLINICAL FEATURES
5. RADIOGRAPHIC FEATURES
6. PREVENTION
 - (a) Pre-Irradiation
 - (b) During Irradiation
 - (c) After Irradiation.
7. TREATMENT
 - (a) Conservative Approach
 - (b) Radical Approach.

1. INTRODUCTION

Radiation, in therapeutic doses, destroys or impairs the vitality of bone. The bone thus affected loses its normal defence barriers to infection and its normal reparative properties after infection or trauma (Topazian, 1959).

Osteoradionecrosis of the jaws is a frequent complication following irradiation of neoplasms in the oral cavity and neighbouring structures (Cutler, 1951).

The condition is also referred to as "radiation osteomyelitis" (Thoma and Goldman, 1960) and "radio osteomyelitis" (MacLennan, 1955). However most authors use the term osteoradionecrosis which, according to Shafer, Hine and Levy (1960), "is that pathologic process which sometimes follows heavy irradiation of bone and is characterised by a chronic, painful infection and necrosis accompanied by late sequestration...".

2. IRRADIATION EFFECT ON ORAL AND PARA-ORAL TISSUES.

Radiation acting on a cell causes ionisation resulting in disruption of the nucleus and cytoplasm. According to Low-Beer (1951), the degree of damage is proportional to the amount of absorbed radiation. Frequently, in the treatment of neoplastic tissue, some degree of injury to normal tissue occurs (Colby et al, 1961). There is a range of radiosensitivity of different types of living cells. Generally immature or poorly differentiated cells are more easily injured than differentiated cells of the same type. All cells show

increased vulnerability to radiation injury during mitosis (Shafer, Hine and Levy, 1960).

(a) Skin: Colby et al (1961) state that there is no immediate visible indication of damage to the skin, however, within a period of two weeks, evidence of injury is apparent. The skin reaction, according to Low-Beer (1951) and Shafer, Hine and Levy (1960), may vary from a mild erythema to a bullous epidermylysis. After four to eight weeks following irradiation this early reaction subsides and later secondary changes are seen due to alteration in the vascular bed. As a result of subcutaneous changes the epithelium may become thin and atrophic and telangiectasis of the superficial blood vessels may occur (Shafer, Hine and Levy, 1960). Also, as reported by Colby et al (1961), there is depigmentation of the irradiated area.

(b) Mucous Membrane: the mucosal changes are similar to those seen in the skin but the erythema may occur earlier than in the analogous dermal reaction (Shafer, Hine and Levy, 1960). Low-Beer (1951) describes the reaction as initially an erythematous change followed by the formation of a yellowish-white fibrinous membrane which subsides after four to eight weeks. Regeneration of the mucosa occurs but the tissue appears paler than normal due to superficial atrophy and fibrosis of the submucosal tissue.

On the tongue, there may be a loss of the fungiform and filiform papillae together with a diminution in sensation and a temporary loss of taste (Low-Beer, 1951).

(c) Salivary Glands: Xerostomia is one of the earliest effects of irradiation about the head and neck. The salivary glands react functionally to irradiation and, following large doses, oedema may occur within a few hours. With smaller doses of irradiation, an alteration in consistency may occur and the saliva loses its normal lubricating function due to an excess of mucus (Low-Beer, 1951).

The altered secretion of the salivary glands may be a permanent sequela of irradiation or a gradual return to normal salivation may occur (Shafer, Hine and Levy, 1960; Low-Beer, 1951).

(d) Teeth: Erupted teeth, in the line of irradiation, are frequently affected following irradiation of neoplasms of the head and neck. Injury to the tooth manifests as a caries-like destruction of the tooth substance which has been called "radiation caries" by many authors (Shafer, Hine and Levy, 1960; Marchetta and Solomon, 1958; Castigliano, 1961; La Dow, 1950). Cutler (1951) describes the lesion as similar to dental caries but notes that it is most frequently seen at the cervical region of a tooth. Frequently the tooth may become brittle and coronal fracture is not uncommon. Stafne and Bowing (1947) consider that these lesions may result from the alteration in quantity and quality of the salivary secretion. These authors found that patients often sucked sweets to relieve the xerostomia and consider that this may influence the occurrence of radiation caries. This theory is supported by Thoma and Goldman (1960).

Irradiation during the development and eruption of teeth can

cause disturbance to the normal pattern of tooth development.

Stafne and Bowing (1947), Bruce and Stafne (1950), Ennis and Berry (1959) and Shafer, Hine and Levy (1960) all report that irradiation of the developing tooth germ may cause arrest of odontogenesis with resultant anodontia or dwarfing of the crown and roots of the teeth.

(e) Bone: According to Shafer, Hine and Levy (1960), bone itself is relatively resistant to irradiation although the osteoblasts are quite sensitive. If the irradiation is sufficiently intense, the normal balance between bone formation and bone resorption is disturbed and general bone vitality is decreased. The greatest clinical significance of bone which has been irradiated is its inability to react in the normal manner to infection. This is due mainly to the damage of the vascular bed as a result of the irradiation (Ennis and Berry, 1959; Low-Beer, 1951; Topazian, 1959). Because of this damage, the bone is liable to infection even years following irradiation. Niebel and Neenan (1957) consider that, due to the high calcium content of bone, irradiation of that tissue results in the production of a large amount of secondary radiation which thus increases the dosage absorbed by the bone.

3. AETIOLOGY

The mandible is more frequently affected by osteoradionecrosis than the maxilla and this is thought to be due to the difference in blood supply between the two bones (Shafer, Hine and Levy, 1960; Gaisford and Rueckert, 1956; Topazian, 1959; Castigliano, 1961).

It is generally agreed that osteoradionecrosis is caused by a triad of factors: irradiation, trauma and infection.

(a) Irradiation: The exposure of bone to therapeutic doses of irradiation results in destruction of the osteogenic cells of the periosteum and endosteum. Also there is an increase in the amount of fatty bone marrow together with "an extensive obliterative sclerosis of the nutrient vessels" (Ennis and Berry, 1959). These degenerative changes result in reduced vitality and increased susceptibility to infection.

(b) Trauma: Trauma to the mucous membrane overlying irradiated bone may open pathways leading to infection of the bone. Extraction of teeth, periodontal disease, trauma from coarse foods and denture irritation have all been cited as aetiological factors in osteoradionecrosis (Topazian, 1959; Cook 1952, 1953; Ennis and Berry, 1959; Castigliano, 1961; La Dow, 1950).

(c) Infection: Once micro-organisms gain access to the bone there is relatively diffuse spread of the infection. Little localisation occurs and a considerable amount of bone, periosteum, and overlying mucos may be involved (Shafer, Hine and Levy, 1960). Niebel and Neenan (1957) in a study of 19 patients found that infection was present in all cases. Necrosis due to irradiation alone is rare according to Stafne and Bowing (1947).

4. CLINICAL FEATURES

Pain is the principal clinical feature of osteoradionecrosis. Gaisford and Rueckert (1956) report that, initially the pain may be little more than a hypersensitivity of teeth. In the later stages the pain becomes very severe and frequently there is also marked trismus.

Abscess formation in the soft tissues may occur and later draining sinuses may form (Topazian, 1959). Ulceration of the mucous membrane may lead to oral exposure of necrotic bone with subsequent delayed sequestration (Seldin et al, 1955). Sequestration in osteoradionecrosis is very slow as the blood supply in the affected bone is restricted by endarteritis, making it impossible to respond normally to an infection (Thoma and Goldman, 1960).

The combination of severe pain and trismus make eating very difficult and MacLennan (1955) and Gaisford and Rueckert (1956) state that loss of weight is a common clinical feature of the condition.

Pathological fracture is not an uncommon complication of osteoradionecrosis (Seldin et al, 1955) and, should it occur, greatly prolongs treatment (Thoma and Goldman, 1960).

5. RADIOGRAPHIC FEATURES.

According to Thoma and Goldman (1960) and Gaisford and Rueckert (1956) no early radiographic change can be detected in osteoradionecrosis.

The earliest change demonstrated is a thickening of the periosteum. As the infection progresses areas of bony destruction appear with or without sequestrum formation. Thoma and Goldman (1960) consider that the osteolytic changes may be confused with secondary involvement of the area by malignant disease and state that in such cases differential diagnosis may be difficult.

6. PREVENTION.

(a) Pre-Irradiation Prevention.

The majority of authors recommend extraction of all teeth in the path of irradiation as well as all other teeth which are infected or involved by caries or periodontal disease (Ennis and Berry, 1959; Niebel and Neenan, 1957; Topazian, 1959; Seldin et al, 1955; Cook, 1952, 1953; Castigliano, 1961; Daland, 1949; Thoma and Goldman, 1960; MacLennan, 1955; Brandon and Herd, 1949; Lyon, 1950; La Dow, 1950; Low-Beer, 1951). At the time of extraction of these teeth, Cook (1952, 1953), Niebel and Neenan (1957) and Topazian (1959) consider that an alveolectomy should also be carried out to reduce the size of the blood clot, to promote healing and to ensure soft tissue coverage of the sockets. It is generally agreed that ten to fourteen days should elapse following removal of the teeth before irradiation is commenced.

Marchetta and Solomon (1958) consider that all teeth should be extracted prior to irradiation while Topazian (1959) recommends this only if the salivary glands are to be irradiated and the patient's

oral hygiene is poor.

On the other hand Gaisford and Rueckert (1956) consider that prophylactic exodontia is not warranted as, in their study, osteoradionecrosis occurred in a large percentage of patients who had teeth removed prior to irradiation. These authors do not state the time period allowed to elapse, following exodontia, before irradiation was commenced. After consideration of the number of cases of osteoradionecrosis resulting from extraction of teeth this reviewer cannot support this view. Castigliano (1961) emphasises that osteoradionecrosis can prove fatal and this reviewer agrees with him that "... a handful of teeth is not worth a life".

Cook (1952,1953) states that before irradiation is commenced the patient should be given strict oral hygiene instruction and warned of the possible sequelae of trauma to the irradiated area.

(b) Prevention During Irradiation:

During irradiation of a lesion of the head or surrounding tissues, care should be taken to minimise the amount of bone irradiated. Castigliano (1961) states that the smallest amount of irradiation consistent with effective treatment should be used to avoid needless exposure of the tissues. It is also suggested by Brandon and Herd (1949), Cutler (1951) and Topazian (1959) that the area irradiated may be further minimised by using the smallest possible portals of treatment.

Another protective measure considered of value by Topazian (1959),

Brandon and Herd (1949) and Castigliano (1961) is the employment of lead shields and cones in order to limit the spread of irradiation.

During the period of irradiation Topazian (1959) considers that the patient should be again informed of the possible complications that may arise from trauma to the irradiated region.

(c) Prevention After Irradiation.

Following irradiation of the jaws prevention of osteoradionecrosis is achieved by meticulous avoidance of all trauma to the tissues. In the literature, there is some controversy as to the period of time that should elapse before teeth may be extracted or dentures worn without risk.

Colby et al (1961), Topazian (1959), Castigliano (1961), and Brandon and Herd (1949) all consider that extraction of teeth following irradiation of the jaws, can never be considered an entirely safe procedure. On the other hand, Cutler (1951), Ennis and Berry (1959) and Daland (1949), state that extractions can be carried out after "several years". While a number of authors consider that exodontia following irradiation is hazardous, it is generally agreed that, if exodontia becomes essential, it should be carried out with antibiotic cover. It is suggested by Topazian (1959) that the dentist should consult with the radiotherapist prior to undertaking treatment of these patients. Niebel and Neenan (1957) suggest the use of rubber bands for extracting teeth following irradiation of the supporting bone. This reviewer considers that, while this method may have merit when used on single rooted teeth, difficulties occur

with multi-rooted teeth. Also the rubber band is likely to cause irritation to the gingival tissues and the resultant gingivitis may lead to infection of the alveolar bone and osteoradionecrosis.

Topazian (1959) considers that all remaining teeth, following irradiation, should be treated conservatively rather than being extracted. This reviewer commends this conservative approach.

Cook (1952, 1953) reports two cases of osteoradionecrosis due to denture trauma and he considers that dentures should not be worn following irradiation. This view is supported by Low-Beer (1951) and Niebel and Neenan (1957). Nevertheless Castigliano (1961) and Topazian (1959) both consider that dentures can be worn safely eighteen to twenty-four months after irradiation.

Strict oral hygiene must be maintained by the patient to prevent dental caries and periodontal disease. Seldin et al (1958) suggest the prescription of suitable mouthwashes to aid in alleviating xerostomia when present, but they do not specify any particular mouthwash. Topazian (1959) emphasises the need for regular dental examinations in order to avoid the need for extraction of teeth.

7. TREATMENT

Osteoradionecrosis may be treated either conservatively or radically.

(a) Conservative approach: this method of treatment is

advocated by Cutler (1951), MacLennan (1957), Niebel and Neenan (1957), Castigliano (1961), Seldin et al (1955) and Thoma and Goldman (1960).

Pain is the major consideration in the treatment of osteoradionecrosis. Analgesic drugs, rather than narcotics, should be prescribed due to the protracted nature of the condition. La Dow (1950) considers that the dosage may have to be increased at night as reclining frequently tends to increase the pain.

When analgesics prove ineffective Castigliano (1961), MacLennan (1957), and Seldin et al (1955) suggest the use of long acting local anaesthetic drugs or alcohol block injections. Niebel and Neenan (1957) also suggest that severance of sensory nerve trunks may be performed.

Antibiotic therapy should be instituted in the early stages in an endeavour to confine the infection and to control acute exacerbations. Strict oral hygiene must also be enforced to aid in control of the infection and to reduce foeter oris which accompanies the suppurative process.

Sequestration is delayed in osteoradionecrosis and sequestrectomy must be deferred until definite demarcation of the sequestra from the vital bone has occurred (Castigliano, 1961; La Dow, 1950; Thoma and Goldman, 1960). Seldin et al (1955) recommend that the exposed sequestrum may be smoothed with rongeur forceps until separation has occurred.

Should pathological fracture occur, Seldin et al (1955) consider

that treatment should be conservative and state that union usually occurs although healing is slow.

Thoma and Goldman (1960) emphasise the need for adequate nutrition. MacLennan (1957) suggests the use of a naso-gastric tube if the patient is unable to take food per oris.

(b) Radical Approach: as the sequestration of bone is delayed in osteoradionecrosis Daland (1949) feels that exposed bone should either be removed with rongeur forceps or treated by electro-coagulation. The latter method results in the necrosis of the exposed bone and its separation as a heat sequestrum, with some chance that the deeper bone will become covered by the soft tissues.

Brandon and Herd (1949), Marchetta and Solomon (1958), La Dow (1950) and Gaisford and Rueckert (1956) consider that surgical removal of the involved bone and the associated necrotic tissue is the method of choice. However this reviewer believes that this radical approach may result in excessive loss of bone with resultant deformity and interference with function.

From the foregoing it can be seen that either method of treatment is far from satisfactory and the key to the problem lies in prevention. Careful consideration must be given to the advisability of retaining teeth before irradiation is commenced and, following irradiation, every possible care must be taken to avoid trauma to the involved area. Also the patient must be warned of the complications to be expected from traumatic procedures to the region exposed to the irradiation.

POST-OPERATIVE FRACTURE

1. INTRODUCTION.
2. CASE REPORTS.
3. TREATMENT.

1. INTRODUCTION

Fracture of the mandible, as a post-operative complication of oral surgery, is poorly documented in the literature. Thus it is proposed, in this review, to present two case reports as examples of post-operative fractures of the mandible. Such fractures may occur without the presence of any complicating pathological condition and are primarily due to weakening of the jaw as a result of the surgical procedure.

2. CASE REPORTS.

Case (1). A male patient, aged 67 years, presented for construction of full upper and lower dentures. He appeared well nourished and had no significant medical history. A radiographic examination of the jaws revealed the presence of two retained roots in the right mandible and a vertically placed, unerupted lower left third molar tooth. It was decided that removal of both the retained roots and the unerupted tooth should be carried out prior to construction of dentures.

On January 31st, 1964, the two retained roots were removed and healing was uneventful. On February 6th the unerupted lower left third molar tooth was removed and, during surgery, the inferior dental bundle was involved. Four days following surgery a radiograph of the mandible was taken (page 217 fig. 1). Analgesics were prescribed for pain and the patient was instructed to use intra-oral heat to the surgery site. On February 13th the wound appeared to be healing satisfactorily and the patient was free of pain. As there was still

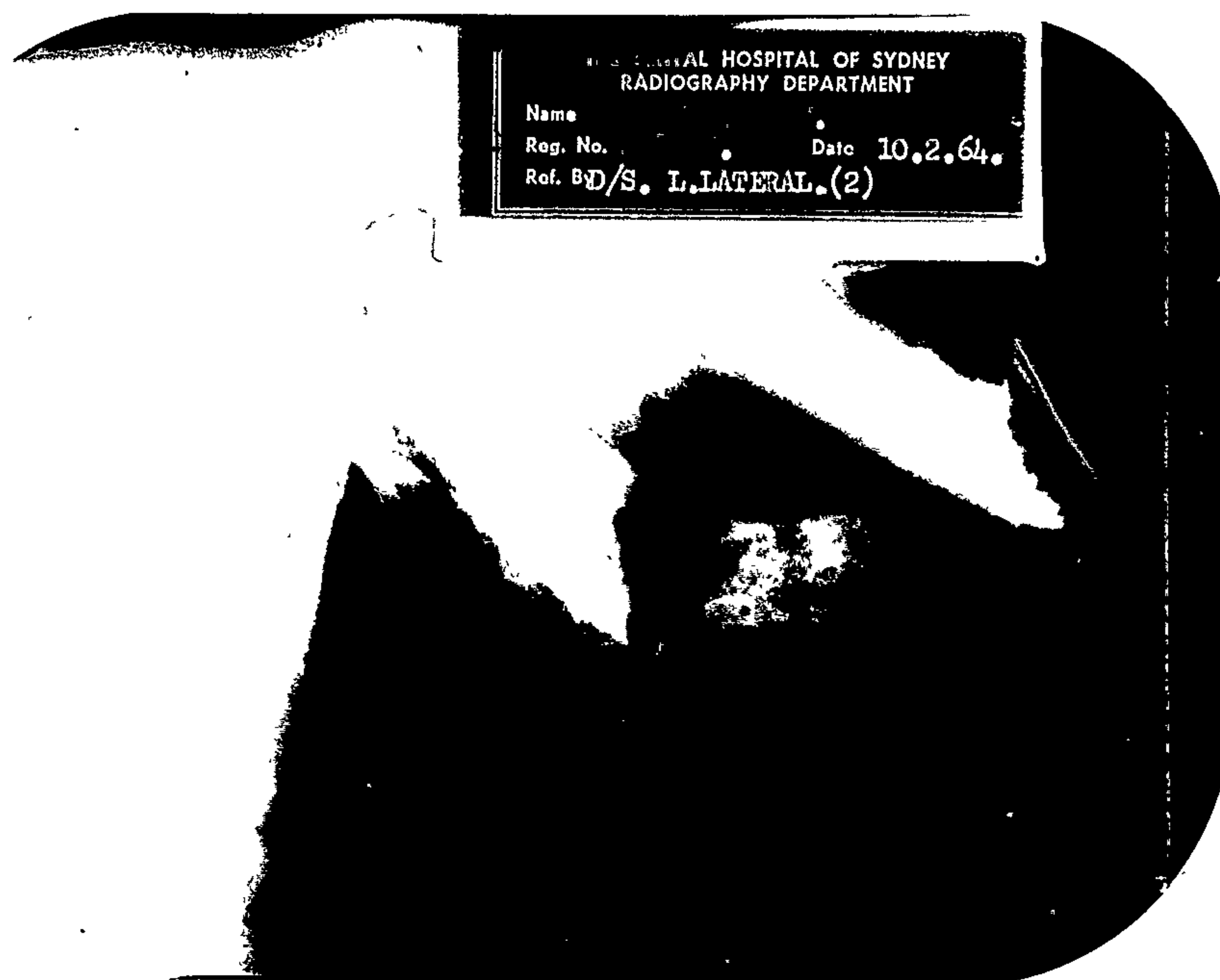


FIG. 1. Radiograph taken four days following surgical removal of an unerupted lower left third molar tooth.

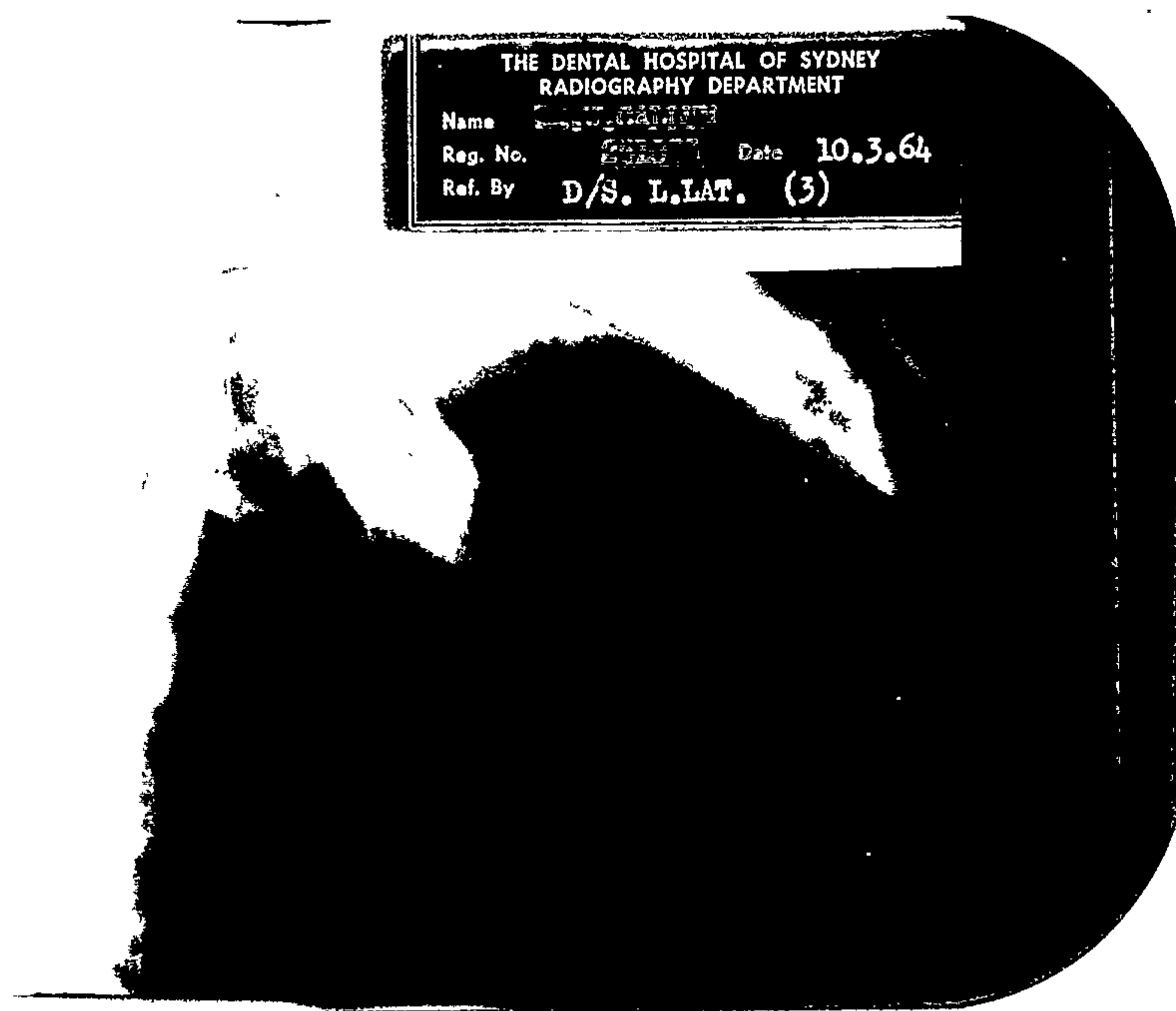


FIG. 2. Radiograph of above case taken five weeks post-operatively showing fracture of the mandible through the surgery site.

slight external swelling, the use of intra-oral heat was continued. The patient was seen on February 19th when it was found that some breakdown of the surgery site had occurred, however the patient was experiencing no pain and had a normal temperature. The socket was packed with iodoform gauze which, two days later, was removed and bone wax substituted. This wax was changed at weekly intervals. On March 10th (five weeks following surgery) the patient attended complaining of severe pain at the surgery site. A radiograph of the region (page 217 fig. 2) revealed a fracture through the socket of the lower left third molar tooth.

Impressions and bite registration were immediately recorded for construction of Gunning Splints which were inserted the following day. The patient was seen regularly and remained comfortable and free of pain. On April 13th the union at the fracture site was found to be clinically firm and radiographically appeared satisfactory. Dentures were then constructed for the patient who was last seen by the author on July 23rd, 1964, when clinically and radiographically the fracture site was satisfactory.

It is considered that probably a number of factors acting together resulted in the fracture described above. Firstly, the mandible was weakened by the surgical removal of the impacted tooth. Secondly, a further weakening may have occurred during the healing process. Re-organisation of the bone in the lower left third molar region would result in some resorption of the older, more dense bone adjacent to the socket with replacement by young, less calcified bone. This process may have been aggravated by the fact that there was some

breakdown of the wound with partial loss of the clot.

The patient was unaware of any traumatic incident that could have resulted in the fracture of the mandible. However, if the mandible was sufficiently weakened as a result of the surgical procedure, forces that would normally be withstood by the jaws could result in fracture post-operatively.

Miner (1939) reports a similar case in which "pathologic fracture" of the mandible occurred eight weeks following removal of a lower right third molar.

Case (2). A 58 year old female patient attended requesting full dentures. The patient was slightly built and appeared older than her chronological age. It was noted that the patient was a heavy smoker. The patient had a history of gastrectomy and cerebral haemorrhage with resultant partial paralysis of the right extremities. She was receiving treatment for pernicious anaemia and renal infection.

A radiographic survey of the jaws revealed a large dentigerous cyst in the lower right molar region which occupied the full thickness of the body of the mandible. Associated with this cyst was an unerupted molar tooth, the crown of which was involved in the cyst, however the roots appeared to be firmly placed in bone.

The patient's physician was consulted and, following haematological and urine analyses, it was decided that the cyst and the unerupted tooth should be removed surgically using an intra-oral approach. The possibility of post-operative fracture was recognised and prior to surgery Gunning splints were constructed.

On March 25th, 1964, the dentigerous cyst and the associated molar tooth were removed. During surgery the neuro-vascular bundle

was involved. The cavity was packed with iodoform gauze and closed with merselene sutures. The patient was given broad spectrum antibiotics by her own physician. On March 28th the gauze packing was changed following irrigation of the cavity. On March 31st most of the post-operative oedema had subsided and the previously constructed Gunning splints were inserted.

On April 3rd it was noted that the patient had developed black hairy tongue and that there were some areas suggestive of candidiasis. The patient reported that the broad spectrum antibiotics had been discontinued two days previously. Radiographs taken of the lower right molar region revealed that a fracture had occurred. The Gunning splints were adjusted to provide increased support distally. Penicillin therapy was commenced as a prophylactic measure. On April 7th bacteriological examination showed that organisms found in the cyst cavity were resistant to penicillin so oral terramycin was commenced. Nystatin tablets were given to treat the candidiasis. Following dietary analysis parenteral vitamin B complex was given. The cavity was repacked with iodoform gauze.

Terramycin, nystatin and parenteral vitamin B were continued together with regular changing of the cavity dressings until the 20th April when antibiotic therapy was suspended. It was noted that the cavity was granulating well at this stage and packing with bone wax was continued until May 7th when the cavity had healed and union at the fracture site was clinically firm.

The patient has been followed carefully and progress x-rays reveal that no bony union has occurred. However clinically there is firm fibrous union and the patient is able to wear full dentures

satisfactorily,

Although splints had been prepared prior to surgery fracture still occurred and it was found necessary to modify the splints to provide added distal support. It is felt that the cystic lesion of the mandible had produced sufficient bony destruction to result in fracture and that, even without surgical intervention, fracture was imminent.

Two other interesting facts emerge from this case report; (a) the appearance of penicillin resistant organisms and need for continued antibiotic sensitivity tests and (b) the possibility of monilial infection following use of broad-spectrum antibiotics.

3. TREATMENT OF POST-OPERATIVE FRACTURE.

A review of the various methods of treatment of fracture is beyond the scope of this review. However the principles of treatment that apply to traumatic fractures are appropriate for use in such cases, that is, the effecting of reduction and immobilisation together with any necessary supportive treatment. The method of fixation, according to Bourgoyne (1945) depends on the amount of bone loss and he considers that, if excessive bone loss has occurred, a bone graft may be necessary.

BIBLIOGRAPHY

ACCEPTED DENTAL REMEDIES, 1963.

Chicago, American Dent. Assoc. 1963.

ALLING, C.C., and Kerr, D.A. Trauma as a factor causing delayed repair of dental extraction sites.

J. Oral Surg. 15:3-10, Jan. 1957.

AMERICAN HEART ASSOCIATION and American Dental Association.

Management of dental problems in patients with cardiovascular disease.

J.A.D.A. 68:333-342, March 1964.

AMLER, M.H., Johnson, P.L., and Salman, J. Histological and histochemical investigation of human alveolar socket healing in undisturbed extraction wounds.

J.A.D.A. 61:32-44, July 1960.

ANDERSON, K. Rational antibiotic therapy in clinical practice.

Med. J. Aust. 1:372-374, March 7, 1964.

ANDERSON, M.F. A report on the use of crystalline trypsin in the treatment of alveolar osteitis.

Oral Surg., Oral Med. and Oral Path. 10:1166-1168, Nov. 1957.

ANTIOCH, R.J. Haemorrhage in oral surgery.

Oral Surg., Oral Med. and Oral Path. 12:265-276, March 1959.

ANWANDTER, G. The paralysis of the facial nerve after mandibular anaesthetic injection.

Am. J. Orth. and Oral Surg. (Oral Surg.) 30:782-788, Dec. 1944.

ARCHER, W.H. An analysis of 226 cases of alveolalgia.

J.D. Res. 18:256-257, June, 1939.

ARCHER, W.H. Needle trauma to nerve tissue during injection.

Oral Surg., Oral Med. and Oral Path. 5:1291-1294, Dec. 1952.

ARCHER, W.H. Oral Surgery 3rd Ed.

Philadelphia - W.B. Saunders Co. 1961.

ARNOTT, A.J. The management of some diseases of the oral cavity and jaws.

Aust. D.J. 7:11-16, Feb. 1962.

ARNOTT, A.J. Closure of an oro-antral fistula.

Lecture to 4th Year students, University of Sydney, 1963.

- ARNOTT, A.J. and Ritchie, C.H. Cervico-facial actinomycosis.
Oral Surg. Oral Med. and Oral Path. 2:252-257, Feb. 1949.
- ARTZ, C.P. Protection of the surgical wound with a new plastic film.
J.A.M.A. 174:1865-1868, Dec. 3, 1960.
- ATTERBURY, R.A. and Vazirani, S.J. Removal of sutures following oral surgery.
Oral Surg., Oral Med. and Oral Path. 14:658-661, June 1961.
- AUSTIN, L.T. Perforation of roots of impacted lower third molar by contents of mandibular canal.
Am. J. Orth. and Oral Surg. 33:623-624, 1947.
- BALOGH, K. Prevention of maxillary sinusitis after tooth extraction.
D. Digest, 69:260-262, June 1963.
- BARAB, B.B. Sulphonamides in the prophylaxis of dry socket.
D.J. Aust. 12:617-620, Oct. 1940.
- BARLING, E.V. Surgery lectures to 4th year students.
University of Sydney, 1963.
- BEAUCHAMP, W.S. Severance of mandibular nerve during extraction.
Brit. D.J. 83:259-260, Dec. 19, 1947.
- BELL, R.H. The clinical importance of the surgical anatomic spaces of the head and neck.
Oral Surg., Oral Med. and Oral Path. 11:339-354, April 1958.
- BENZER, P. A preliminary report on the use of hyaluronidase in the treatment of traumatic swellings.
Oral Surg., Oral Med. and Oral Path. 4:1515-1522, Dec. 1951.
- BENZER, P. and Schaffer, A.B. The use of hyaluronidase in the treatment of traumatic swellings.
Oral Surg., Oral Med. and Oral Path. 5:1315-1341, Dec. 1952, also in J.D. Res. 31:467, Aug. 1952.
- BERGAN, R.D., Gores, R.J. and Wakim, K.G. Fluid balance after multiple dental extractions and alveoplasty.
Oral Surg., Oral Med. and Oral Path. 12:929-940, Aug. 1959.
- BERLOVE, I.J. Dental-Medical emergencies and complications.
Chicago. The Year Book Publishers, 1959.

BERMAN, S.J. and Wright, I.S. Dental surgery during continuous anticoagulant therapy.
J.A.D.A. 62:172-180, Feb. 1961.

BERNIER, J.L. The management of oral disease, 2nd Ed.
St. Louis. The C.V. Mosby Co. 1959.

BLAINE, G. Experimental observations on absorbable alginate products in surgery.
Ann. Surg. 125:102-114, Jan. 1947.

BLUMENTHAL, E.E., Catania, A.F. and Kringsten, G.J. Clinical evaluation of penicillin V in oral surgery.
N.Y. State D.J. 23:260-265, June-July 1957.

BORDEN, S.M. Histological study of healing following detachment of tissue as is commonly carried out in the vertical incision for the surgical removal of teeth.
J. Canad. D.A. 14:510-515, Oct. 1948.

BOURGOYNE, J.R. Fixation of pathologic fractures of the mandible.
Am. J. Orth. and Oral Surg. (Oral Surg) 31:492-500, Aug. 1945.

BOURGOYNE, J.R. Oral contributions to the diseased antrum.
Oral Surg. Oral Med. and Oral Path. 1:706-718, Aug. 1948.

BOYNE, P.J. and Kruger, G.O. Topical implantation of oxytetracycline cones in extraction sockets.
J.A.D.A. 64:224-235, Feb. 1962.

BRAMLEY, P. Severe reactions to penicillin.
Brit.D.J. 102:314-316, April 16, 1957.

BRAMLEY, P. and Orton, H.S. Cervico-facial actinomycosis.
Brit.D.J. 109:235-238, Sept. 20, 1960.

BRANDON, S.A. and Herd, R.M. Report of case and management of radiation necrosis of the mandible.
Oral Surg., Oral Med. and Oral Path. 2:987-992, Aug. 1949.

BRANSBURY-ZACHARY, G.M. The sub-masseteric space.
Brit. D.J. 84:10-13, Jan. 2, 1948.

BRASH, J.C. Cunningham's Textbook of Anatomy, 9th Ed.
London. Oxford University Press. 1953.

BREAKSTONE, G.E. The maxillary sinus. Common difficulties and complications.
N.Y. State D.J. 23:473-479, Dec. 1957.

- BRENMAN, H.S. Oral analgesics: preoperative vs. postoperative use.
J.A.D.A. 67:23-27, July 1963.
- BRUCE, K.W. and Stafne, E.C. The effect of irradiation on the dental system as demonstrated by the roentgenogram.
J.A.D.A. 41:684-689, Dec. 1950.
- BUCHANAN, G. Infections of the jaws.
M.D.S. Thesis. Uni. Syd. 1961.
- BUDGE, C.T. Closure of antro-oral opening by the use of tantalum plate.
J.Oral Surg. 10:32-33, Jan. 1952.
- BURCH, R.J. Repair of antro-oral fistula: case report.
J. Oral Surg. 7:72-74, Jan. 1949.
- BURCH, R.J. and Morris, C.R. Response to surgical trauma.
J. Oral Surg. Anes. and Hosp.D. Serv. 20:240-249, May 1962.
- CAHN, L.R. Oral infections: the evaluation of their treatment.
Brit. D.J. 98:245-250, April 5, 1955.
- CANNON, P.R. The importance of proteins in resistance to infection.
J.A.M.A. 128:360-362, June 2, 1945.
- CANTAROW, A., and Shepartz, B. Biochemistry 2nd ed.
Philadelphia. W.B. Saunders Co. 1957.
- CAPPELL, D.F. Muir's Textbook of Pathology 7th Ed.
London. Edward Arnold. 1962.
- CASH, H.R. A consideration of the problems concerned with the management of patients requiring surgery.
Aust. J.D. 55:407-424, Dec. 1951.
- CASTIGLIANO, S.G. Oral Cancer.
in Burket, L.W. Oral Medicine 4th Ed.
Philadelphia. J.B. Lippincott Co. 1961.
- CHAPUT, A. Oral haemorrhage.
Int. D.J. 14:1-17, March 1964.
- CHERASKIN, E. The control of bleeding.
J.A.D.A. 58: 17-28, April 1959.
- CHRISTOPHER, F.R. A histological study of bone healing in relation to extraction of teeth.
Northwestern Univ. Bul. 43:5, Dec. 7, 1942.

CLARK, H.B. Practical Oral Surgery.
Philadelphia. Lea and Febiger, 1955.

COGSWELL, W.W. Surgical problems involving the mandibular nerve.
J.A.D.A. 29:964-969, June, 1942.

COHEN, L. Dental haemorrhage.
Oral Surg., Oral Med. and Oral Path. 11:704-706, July 1958.

COLBY, R.A., Kerr, D.A. and Robinson, H.B.G. Color Atlas of Oral Pathology 2nd Ed.
Philadelphia. J.B. Lippincott Co. 1961.

COLLINS, D.A. The etiology and nature of dental pain.
D.Clin. N. Am. p 25-36, March 1958.

COMMONWEALTH DEPARTMENT OF HEALTH. Notes on special diets for use in hospitals.
Canberra 1961.

COOK, T.J. Stomatitis following the use of penicillin traches.
Oral Surg., Oral Med. and Oral Path. 1:265-266, Mar. 1948.

COOK, T.J. Late radiation necrosis of the jaw bones.
J. Oral Surg. 10:118-137, April 1952.

COOK, T.J. Late radiation necrosis of the jaw bones (abstract)
N.Y. State D.J. 19:86, Feb. 1953.

COOK, T.J. Osteomyelitis and osteoradionecrosis.
Oral Surg., Oral Med. and Oral Path. 16:257-260, March 1963.

COOLEY, D.O. Closure of oro-antral openings.
J. Oral Surg. 12:28-30, Jan. 1954.

COSTICH, E.R., Youngblood, P.J. and Walden, J.M. A study of the effects of high speed rotary instruments on bone repair in dogs.
Oral Surg., Oral Med. and Oral Path. 17:563-571, May 1964.

COUNCIL ON DRUGS. Buccal and intramuscular use of streptobinase-streptodornase (Varidase).
J.A.M.A. 172: 701-702, Feb. 13, 1960.

CRAN, J.A. and Hannam, A.G. Cervico-facial actinomycosis.
Aust. D.J. 8:106-108, April 1963.

CROKER, C.C. Haemorrhage.
D.J. Aust. 24:45-52, April 1952.

- CROLIUS, W.E. The use of gold plate for the closure of oro-antral fistulas.
Oral Surg., Oral Med. and Oral Path. 9:836-838, Aug. 1956.
- CROWLEY, M.C. Actinomyces in the normal mouth and in infectious processes.
Am. J. Orth. and Oral Surg. (Oral Surg) 30: 680-682, Nov. 1944.
- CUTLER, M. The problem of extraction in relation to osteoradionecrosis complicating radiotherapy for intra-oral cancer.
Oral Surg., Oral Med. and Oral Path. 4:1077-1090, Sept. 1951.
- DALAND, E.M. Radiation necrosis of the jaw.
Radiology, 52:205, Feb. 1949.
abs. in Oral Surg., Oral Med. and Oral Path. 2:1347, Oct. 1949.
- DALITSCH, W.W. Haemorrhage: causes and management.
J.A.D.A. 28: 230-233, Feb. 1941.
- DAVIS, W.H., Hubbell, A.O., Bogart, W.E. and Graves, V.M.
Extraction wound healing: clinical observations.
J. Oral Surg. 13: 244-247, July 1955.
- De BATS, M.L. and Phillips, W.H. Nerve injuries incident to dental surgery.
J. Oral Surg. 2: 13-18, Jan. 1944.
- DEDOLPH, T.H. and Clark, H.B. A histological study of muco-periosteal flap healing.
J. Oral Surg. 16: 367-376, Sept. 1958.
- DEICHMANN, W.B. Review of antibiotics.
J. Oral Surg. 13:214-231, July 1955.
- DEVINE, J.C. The use of hyaluronidase to prevent post-operative complications.
D. Digest. 60: 23-24, Jan. 1954.
- DILLE, J.M. The nature of pain and the action of analgesics.
Oral Surg., Oral Med. and Oral Path. 4:321-336, March 1951.
- DINGMAN, R.O., and Hayward, J.R. Oral surgery in general practice.
J.A.D.A. 35:607-626, Nov. 1947.
- DOBSON, L. and Cutting, W.C. Penicillin and sulphonamides in the therapy of actinomycosis.
J.A.M.A. 128: 856-863, July 21, 1945.

- DORNER, A.F. and Morgan, D. Ludwig's angina treated with penicillin: Report of a case.
J. Oral Surg. 3:232-234, July, 1945.
- DOUGLAS, B.L. Clinical observations on the use of absorbable hemostatic bone wax in dental and oral surgery.
Oral Surg., Oral Med. and Oral Path. 6:1195-1198, Oct. 1953.
- DROTER, J.A. Transitory Bell's phenomenon after mandibular injection: case report.
J. Oral Surg. Anes. & Hosp. D. Serv. 17:57-58, July 1959.
- DURBECK, W.E. Mandibular osteomyelitis: its diagnosis and treatment.
J. Oral. Surg. 4:33-39, Jan. 1946.
- EDWARDS, R.W. Origin of the term dry socket.
J.A.D.A. 66:215-216, Feb. 1963.
- ELWELL, K.R. A new approach to the treatment of dry socket.
J.A.D.A. 31:615-621, May 1944.
- ENNIS, L.M. and Berry, H.M. Dental Roentgenology 5th Ed.
Philadelphia. Lea and Febiger. 1959.
- EPSTEIN, S. and Kauffman, R.R. Penicillin tablets in oral surgery procedures.
J. Oral Surg. 9:115-118, April 1951.
- ESKIN, L.C. Anatomical and surgical considerations in acute neck infections of oral origin.
D. Digest 64:261-264, June 1958.
- EWING, J.W. Technics in the control of operative and post-operative haemorrhage.
J. Oral Surg. 5:315-318, Oct. 1947.
- FAILLO, P.S. Proteolytic enzyme treatment for the necrotic alveolar socket.
Oral Surg., Oral Med. and Oral Path. 1:608-613, July 1948.
- FICKLING, B.W. Oral surgery involving the maxillary sinus.
Ann. Royal Coll. Surg. 20:13-35, Jan. 1957.
- FLEMING, W.E. Chronic osteitis and osteomyelitis of the jaws.
D.J. Aust. 26:143-146, Aug. 1954.
- FLEUCHAS, P.T. Effect of hyaluronidase on swelling and trismus after removal of impacted mandibular third molar teeth.
Oral Surg., Oral Med. and Oral Path. 9:493-500, May 1956.

FRANDSEN, A.M. and Pindborg, J.J. The influence of sulphonamide cones upon socket healing in the rat.
Arch. Oral Biol. 5:98-104, 1961.

FRANK, V.H. Paresthesia: evaluation of 16 cases.
J. Oral Surg. Anes. and Hosp. D. Serv. 17:27-33, Nov. 1959.

FRY, W.K. and Goldman, V. A new treatment of "dry socket".
Brit. D.J. 104:55-56, Jan. 21, 1958.

GAISFORD, J. and Rueckert, F. Osteoradionecrosis of the mandible.
J. Plastic and Reconstructive Surg. 18:436-447, Dec. 1956.

GEARY, J.R. and Frantz, V.K. New absorbable haemostatic bone wax.
Ann. Surg. 132:1128-1137, Dec. 1950.

GEE, E.J. and Sullivan, H.R. Actinomyces within an apical granuloma.
D.J. Aust. 12:213-214, April 1940.

GEORGIADIS, N., Mitchell, T., Lemler, J. and Heid, J.
Use of a new improved hemostatic sponge in oral surgery.
J. Oral Surg. Anes. and Hosp. D. Serv. 19:215-218, May 1961.

GLASER, R.J. General principles of antibiotic therapy.
J.A.D.A. 46:129-134, Feb. 1953.

GLICKMAN, I., Pruzansky, S. and Ostrach, M. The healing of extraction wounds in the presence of retained root remnants and bone fragments.
Am. J. Orth. and Oral Surg. 33:263-283, April, 1947.

GOLD, L. Retarded Healing of wounds.
D. Clinics of N. America p. 533-546, July 1957.
Philadelphia. W.B. Saunders Co. 1957.

GOLDSWORTHY, N.E. Actinomycosis: with special reference to the cervico-facial type.
D.J. Aust. 19:225-241, June 1947.

GOULDING, R. Antibiotics in theory and practice.
Brit. D.J. 103:379-387, Dec. 3, 1957.

GOULDING, R. Handbook of Dental Pharmacology and Therapeutics.
London. W.H. Heinmann Medical Books Ltd. 1960.

GRIMM, D.H. and Albertson, G.L. Clinical investigation in the use of erythromycin for infection of the oral cavity.
Oral Surg., Oral Med. and Oral Path. 7:858-864, August 1954.

GUFSTAFSON, G. and Wallenius, K. Effect of local application of trypsin on post-extraction alveolar osteitis.

Oral Surg., Oral Med. and Oral Path. 14:280-286, March 1961.

GURALNICK, W.C. Absorbable gelatin sponge and thrombin in oral surgery.

Am. J. Orth. and Oral Surg. (Oral Surg.) 32:792-794, 1946.

GURALNICK, W.C. and Berg, L. Gelfoam in oral surgery.

Oral Surg., Oral Med. and Oral Path. 1:632-639, July 1948.

GWINN, C.D. and Grimm, D.H. Healing of extraction wounds after use of penicillin and sulphathiazole. Comparative clinical study.

J.A.D.A. 37:526-529, Nov. 1948.

GWINN, C.D., Grimm, D.H. and Ferber, E.W. Oral use of fibrin foam and thrombin in the prevention and treatment of post-operative haemorrhage.

J.A.D.A. 35:7-11, July 1947.

GWINN, C.D., Grimm, D.H. and Ferber, E.W. Oral use of absorbable oxidised cellulose in the prevention and treatment of post-operative haemorrhage.

J.A.D.A. 36:155-159, Feb. 1948.

GWINN, C.D., Grimm, D.H. and Ferber, E.W. Oral use of absorbable gelatin sponge in the prevention and treatment of post-operative haemorrhage.

J.A.D.A. 36:397-401, May 1948.

HANSON, E.H. Alveolitis sicca dolorosa (dry socket): frequency of occurrence and treatment with trypsin.

J. Oral Surg. Anes. and Hosp. D. Serv. 18:409-415, Sept. 1960.

HARANG, H.L. The prevention of dry sockets in extraction of teeth.

Oral Surg., Oral Med. and Oral Path. 1:601-607, July 1948.

HARDING, R.L. The use of protein in oral and maxillo-facial surgery.

J. Oral Surg. 6:235-244, July 1948.

HARVEY, P. Drug reactions involving the alimentary tract.

Med. J. Aust. 230-233, Feb. 15, 1964.

HARVEY, S.C. The healing of the wound as a biologic phenomenon.

Surgery. 25:655-671, May 1949.

HAYWARD, J.R. Exodontic problems.

J. Oral Surg. 11:208-215, July 1953.

- HAYWARD, J.R. Suppuration from slow healing sockets. "Queries and Comments".
J. Oral Surg. 15:83, Jan. 1957.
- HELMORE, F.E. Some phases of pre-operative and post-operative treatment.
Aust. D.J. 3:76-84, April 1958.
- HENKEL, G.H. The role and applicability of hyaluronidase in clinical dentistry.
Oral Surg., Oral Med. and Oral Path. 9:463-492, May 1956.
- HENNESSY, T. External heat and cold therapy in oral surgery.
Am. J. Orth. and Oral Surg. (Oral Surg.) 27:472-474, Sept. 1941.
- HENNY, F.A. and Barron, J. Tube feeding in oral surgery.
J. Oral Surg. 12:238-240, July 1954.
- HERD, R.M. Technique for closure of antro-oral opening.
Oral Surg., Oral Med. and Oral Path. 3:1385-1387, Nov. 1950.
- HERRELL, W.E. and Nichols, D.R. Penicillin in the treatment of cellulitis of the mouth.
Am. J. Orth. and Oral Surg. (Oral Surg.) 30:1-7, Jan. 1944.
- HINDS, E.C. Control of post-operative edema.
J. Oral Surg. 16:109-118, March 1958.
- HINDS, E.C. and Degnan, E.J. The use of achromycin and neomycin in the treatment of actinomycosis.
Oral Surg., Oral Med. and Oral Path. 8:1034-1038, Oct. 1955.
- HINDS, E.C. and Keats, A.S. Analgesia and Sedation.
Prac. D. Monographs p. 10-19, March 1960.
- HOLLAND, D.J. Pre-operative and post-operative care in oral surgery.
J. Oral Surg. 5:298-304, Oct. 1947.
- HOLLAND, D.J. Complications in exodontia and oral surgery.
Oral Surg., Oral Med. and Oral Path. 1:758-778, Aug. 1948.
- HOLLAND, M.R. and Jurgens, P.E. The effectiveness of an anti-histamine to reduce pain following oral surgical procedures.
Oral Surg., Oral Med. and Oral Path. 11:138-140, Feb. 1958.
- HOLLAND, M.R. and Tam, J.C. The use of pure crystalline penicillin G. tablets in extraction wounds.
Oral Surg., Oral Med. and Oral Path. 7:145-148, Feb. 1954.

- HOLMES, P.E.B. Cervico-facial actinomycosis in relation to dental treatment.
Brit.D.J. 104:314-317, May 6, 1958.
- HOWE, G.L. The use of pre-extraction radiographs in the management of difficult extractions.
D. Pract. and D. Record, 10:196-201, May 1960.
- HOWE, G.L. and Poyton, N.G. Prevention of damage to the inferior dental nerve during extraction of mandibular third molars.
Brit.D.J. 109:355-363, Nov. 1, 1960.
- HOWELL, J.T. and Monto, R.W. Recognition and management of bleeding disorders in oral surgery.
J. Oral Surg. 11:129-136, April 1953.
- HUEBSCH, R.F. Clinical and histological study of alveolar osteitis.
J. Oral Surg. 16:473-482, Nov. 1958.
- HUEBSCH, R.F., Coleman, R.D., Frandsen, A.M. and Becks, H. The healing process following molar extraction (1) normal male rats (Long-Evans strain).
Oral Surg., Oral Med. and Oral Path. 5:864-876, Aug 1952.
- HUNTER, G.C. and Westrick, C.M. Cervico-facial by actinomycosis.
Oral Surg., Oral Med. and Oral Path. 10:793-800, Aug. 1957.
- HYSON, J.M. Use of buccal streptobinase in the treatment of infection and oedema.
Oral Surg., Oral Med. and Oral Path. 13:812-818, July 1960.
- IVY, R.H. and Curtis, C.L. Fractures of the Jaws, 2nd Ed.
Philadelphia. Lea and Febiger, 1938.
- JACKSON, M. Relationship of impacted lower second molar to inferior dental canal.
Brit. D.J. 41:294, Dec. 4, 1951.
- JACOBS, B.J. and Rafel, S.S. Absorbable haemostatic agents.
Oral Surg., Oral Med. and Oral Path. 3:356-376, March 1950.
- JENKINS, H.P. et al. Present status of gelatin sponge for the control of haemorrhage.
J.A.M.A. 132:614-619, Nov. 16, 1946.
- JOHNSON, A. Antibiotics: preventive use in medicine and surgery.
Med. J. Aust. 2:792-793, Nov. 12, 1960.
- JOHNSON and Johnson. Surgicel. (pamphlet)
New Brunswick, New Jersey, U.S.A. 1961.

JOYCE, C.R.B. Sedatives and analgesics in dental practice.
D. Pract. and D. Rec. 12:111-115, Dec. 1961.

KAYNE, C.B. Sulphanilamide and urea in the treatment of osteomyelitis
J.A.D.A. 28:1251-1254, Aug. 1941.

KEESLING, G.R. and Hinds, E.C. Clinical evaluation of anti-histamines
in oral surgery using the double unknown technic.
J. Oral Surg. 15:279-282, Oct. 1957.

KEESLING, R. and Keats, A.S. The efficiency of a placebo and several
other oral analgesics in the treatment of pain in alveolar osteitis.
Oral Surg., Oral Med. and Oral Path. 11:736-741, July 1958.

KEMP, H.R. Post-operative treatment and complications in relation to
oral surgical procedure.
D.J. Aust. 24:189-197, Oct. 1952.

KHARITANOV, V.K. and Artemova, G.V. Case of anaphylactic shock
associated with penicillin therapy in stomatological practice.
Stomat. Moscow, 40:92-93, Jul.-Aug. 1961,
in D. Abs. 7:359, June 1962.

KRAMER, R.H. Antibiotic therapy in dental practice.
Brit. D.J. 100:69-80, Feb. 7, 1956.

KRASHEN, A.S. A preliminary report on the use of sulphonamide in
the average extraction case.
J.A.D.A. 27:1199-1204, Aug. 1940.

KRAUSE, J.D. Controlled study on the removal of mandibular third
molars; effect of an enzyme mixture on sequelae.
J.A.D.A. 63:804-809, Dec. 1961.

KROGH, H.W. Incidence of dry socket.
J.A.D.A. and D. Cos. 24:1829-1836, Nov. 1937.

KROGH, H.W. Prevention of dry sockets.
J.D. Res. 27:3-8, Jan. 1948.

KRUGER, B.J. A comparison of three modern haemostatic agents commonly
used by the general practitioner.
D.J. Aust. 26:228-233, Dec. 1954.

KRUGER, G.O. Textbook of Oral Surgery.
St. Louis. The C.V. Mosby Co. 1959.

KURE, A.F. and Rudy, H.N. The use of Premarin intravenous in oral
surgery.
Oral Surg., Oral Med. and Oral Path. 17:434-442, April 1964.

- La DOW, C.S. Osteoradionecrosis of the jaw.
Oral Surg., Oral Med. and Oral Path. 3:582-590, May 1950.
- LAMBERT, A. Traitment de l'actinomycose par la Streptomycine.
Semaine d. hop. 27:1477, 1951.
abs. in Oral Surg., Oral Med. and Oral Path. 5:911, Aug. 1952.
- LANCET (Editorial). Relief of post-operative pain.
Lancet II: 188-189, July 25, 1964.
- LESNEY, T.A. and Traeger, K.A. Cervico-facial actinomycosis - a post-extraction complication.
J. Oral Surg. Anes. and Hosp. D. Serv. 17:51-59, Jan. 1959.
- LIEBOWITZ, D. and Ritter, H. Anaphylactic reaction to chymotrypsin.
J.A.M.A. 172:159-160, Jan. 9, 1960.
- LOSCH, P.K. The use of fibrin foam with thrombin in dental operations.
J. Oral Surg. 3:177-185, July 1945.
- LOW-BEER, B.V.A. Radiation therapy and dental medicine.
Oral Surg., Oral Med. and Oral Path. 4:739-761, June 1951.
- LUCAS, R.B. and Kramer, R.H. Bacteriology for Dental Students, 2nd Ed.
London. J. and A. Churchill Ltd. 1959.
- LUDWIG, T.G. Actinomycosis originating in the maxillary region.
Oral Surg., Oral Med. and Oral Path. 8:877-880, Aug. 1955.
- LYON, L.Z. Radiation necrosis of the mandible: report of a case.
J. Oral Surg. 8:159-161, April 1950.
- MACALISTER, A.D. Acute swellings of the face and jaws.
N.Z. Dent. J. 53:11-16, Jan. 1957.
- MACALISTER, A.D. A report on the use of buccally administered streptobinase in oral surgery.
Aust. D.J. 6:187-191, Aug. 1961.
- MACCHIA, A.F. Anti-histamines and their value in allaying post-operative oedema in the removal of impacted teeth.
Oral Surg., Oral Med. and Oral Path. 10:122-128, Feb. 1957.
- MAC LENNAN, W.D. Some aspects of the problem of radionecrosis of the jaws.
Proc. Roy. Soc. Med. 48:1017, Dec. 1955.
Abs. in Oral Surg., Oral Med. and Oral Path. 10:109, Jan. 1957.

- MAIN, J.P. and MacPhee, I.T. Actinomycosis of the maxilla in relation to a periodontal abscess.
Oral Surg., Oral Med. and Oral Path. 17:299-304, March 1964.
- MANGOS, J.F. The healing of extraction wounds.
N.Z. Dent. J. 37:5-22, Jan. 1941.
- MARCHETTA, F.C. and Solomon, H.A. Osteoradionecrosis of the mandible.
J. Oral Surg. 16:69-72, Jan. 1958.
- MARLETTE, R.H. Incision and drainage in the treatment of cellulitis arising from dental origin.
Oral Surg., Oral Med. and Oral Path. 7:474-476, May 1954.
- MASLANSKY, M.M. and Maslansky, L. Injectable anti-histamines - an aid in oral surgery.
N.Y. State D.J. 19:274-278, June-July 1953.
- MC VAY, L.V., Guthrie, F. and Sprunt, D.H. Aureomycin in the treatment of actinomycosis.
New England J. Med. 245:91, 1951.
- MC VAY, L.V. and Sprunt, D.H. Treatment of actinomycosis with isoniazid.
J.A.M.A. 153:95-98, Sept. 12, 1953.
- MILLER, J.M. and others. Clinical experience with streptobinase administered buccally.
J.A.M.A. 166:478-481, Feb. 1, 1958.
- MILLHON, J.A. and others. An evaluation of sulpha drugs and other dressings in dry sockets in lower third molars.
J.A.D.A. 30:1839-1844, Dec. 1943.
- MINER, C.L. Pathologic fracture of the mandible.
J.A.D.A. 26:165-166, Jan. 1939.
- MINNO, A.M. and Davis, G.M. Penicillinase in the treatment of penicillin reactions.
J.A.M.A. 165: 222-224, Sept. 21, 1957.
- MITCHELL, D.F. Products of human blood fractionation and their uses in dentistry.
J.A.D.A. 33:1287-1291, Oct. 1946.
- MOLT, F.F. Diet as a factor in healing.
J.A.D.A. 23:1442-1446, Aug. 1936.
- MONHEIM, L.M. The therapeutic management of pain.
D. Clin. N. America. Philadelphia, W.B. Saunders Co. 1959.

MONHEIM, L.M. Local Anaesthesia and Pain Control in Dental Practice. 2nd Ed.
St. Louis. C.V. Mosby Co. 1961.

MONTELEONE, L. Actinomycosis.
J. Oral Surg. Anes. and Hosp. D. Serv. 21:313-318, July 1963.

MOODY, J.E.H. Osteomyelitis of the jaws and its treatment.
Aust. D.J. 3:85-96, April 1958.

MOORE, E.E., Bishop, J.G., Matthews, J.L. and Dorman, H.L.
Effect of temperature change on mandibular blood flow.
J.D. Res. 40:988-992, Oct. 1961.

MOOSE, S.M. The rational therapeutic use of thermal agents with special reference to heat and cold.
J.A.D.A. 24:185-196, Feb. 1937.

MOOSE, S.M. Surgical procedure for closing large oro-antral openings.
J. Oral Surg. Anes. and Hosp. D. Serv. 20:198-201, May 1962.

MOSS, R.L. Control of post-operative oedema with promethazine.
Oral Surg., Oral Med. and Oral Path. 11:464-469, May 1958.

MOURFIELD, W.R. and Barron, J.M. Clinical evaluation of erythromycin dental cones in oral surgery.
Oral Surg., Oral Med. and Oral Path. 11:584-586, June, 1958.

MUHLER, J.C. Textbook of Biochemistry for Students of Dentistry.
St. Louis. C.V. Mosby Co. 1959.

NATHAN, A.S. The use of oxidised cotton in dental haemostasis.
J. Oral Surg. 6:37-41, Jan. 1948.

NATHAN, M.H. and others. Osseous actinomycosis of the head and neck.
Am. J. Roengenol. 87:1048, June 1962.
Abs. in Oral Surg., Oral Med. and Oral Path. 16:639, May 1963.

NATHANSON, N.R. Control of bleeding with Premarin intravenous in oral surgery.
Oral Surg., Oral Med. and Oral Path. 14:396-400, April 1961.

NATIONAL HEART FOUNDATION OF AUSTRALIA. Pamphlet "How to protect a dental patient from bacterial endocarditis".
1963.

NAYLOR, M.N. and Moore, J.R. Post-extraction haemorrhage.
Brit. D.J. 112:349-353, May 2, 1962.

NEW GOULD MEDICAL DICTIONARY.

New York. McGraw-Hill Book Co. 1956.

NIEBEL, H.H. and Neenan, E.W. Dental aspects of osteoradionecrosis.
Oral Surg., Oral Med. and Oral Path. 10:1011-1024, Oct. 1957.

NIZEL, A.E. Diet in oral surgery.
Oral Surg., Oral Med. and Oral Path. 14:539-546, May 1961.

NORTHROP, P.M. Penicillin in oral surgery.
J. Oral Surg. 7:39-55, Jan. 1949.

OLECH, E. Value of implantation of certain chemotherapeutic agents
in sockets of impacted low third molars.
J.A.D.A. 46:154-159, Feb. 1953.

OSTERLON, J.P. Surgical prevention of dry sockets.
J.A.D.A. 32:1109-1111, Sept. 1945.

PETERS, R.A., Coward, K.H. Krebs, H.A. and others. Vitamin C
requirements of human adults: experimental study of vitamin C
deprivation in man.
Lancet. 1:255, 1948.

PHEILS, M.T., Reid, D.J. and Ross, C.F. Abdominal actinomycosis.
Brit. J. Surg. 51:345-350, May 1964.

PIZER, M.E. Cervico-facial actinomycosis of the mandible.
Oral Surg., Oral Med. and Oral Path. 13:775-780, July 1960.

PONDOLIN, M. Cold and heat therapy in dental inflammations.
J.A.D.A. 23:61-65, Jan. 1936.

POSSOFF, A. External thermal applications in post-extraction therapy.
J.A.D.A. 50:147-156, Feb. 1955.

QUINLEY, J.F., Royer, R.G. and Gores, R.J. Dry socket after mandibular
odontectomy and use of soluble tetracycline hydrochloride.
Oral Surg., Oral Med. and Oral Path. 13:38-42, Jan. 1960.

RADDEN, H.G. Local factors in healing of the alveolar tissues.
Ann. Royal Col. Surg. 24:366, June 1959.

RANKOW, R.M. Surgical management of acute infections of the oral and
neck regions.
N.Y. State D.J. 23:5-8, Jan. 1957.

REPASS, F.G. Impacted right and left mandibular third molars with
inferior dental nerve passing through mesial root.
J. Oral Surg. 4:243-244, July 1946.

RICKER, O. L. Haemorrhagic conditions of importance in oral surgery.
J. Oral Surg. 7:129-144, April 1949.

RITCHIE, C.H. Maxillary conduction anaesthesia.
D.J. Aust. 24:60-62, April 1952.

RITCHIE, C. Oral Surgery for the general practitioner.
D.J. Aust. 26:114-118, June 1954.

ROOTS, M. Dietitian United Dent. Hosp. Sydney.
Personal communication 1964.

ROSE, K.D. Anaphylactic reaction to aqueous chymotrypsin injection.
J.A.M.A. 173:796-797, June 18, 1960.

ROSH, R. and Seldin, H.M. Actinomycosis: treatment of 5 cases.
J. Oral Surg. 6:93-105, April 1948.

ROSS, R. and White, C.P. Evaluation of hydrocortisone in prevention of post-operative complications in oral surgery; a preliminary report.
J. Oral Surg. 16:220-226, May 1958.

RUBELMAN, P.A., Reback, J.F. and Loveman, C.E. Post-extraction cellulitis caused by *Salmonella choleraesius*.
J. Oral Surg. Anes. and Hosp. D. Serv. 19:254-255, May 1964.

RUD, J., Baggesen, H. and Moller, J.F. Effect of sulpha cones and suturing on the incidence of pain after the removal of impacted lower third molars.
J. Oral Surg. Anes. and Hosp. D. Serv. 21:219-226, May 1963.

RUSHTON, M.A., and Cooke, B.E.D. Oral Histopathology 1st Ed.
Edinburgh. E. & S. Livingstone Ltd. 1963.

RUSSELL, L.T. A comparison in methods of prevention of local alveolar osteitis.
Am. J. Orth. and Oral Surg. (Oral Surg.) 30:95-97, Feb. 1944.

SAINT, E.G. Antibiotic problems.
Aust. J. Dent. 57:191-198, Aug. 1953.

SANDY, C.E. An investigation of some recently reported adjuncts to oral surgery.
Aust. J. Dent. 52:381-385, Nov. 1948.

SARA, C. Emergencies in dental practice.
Aust. D.J. 8:408-414, Oct. 1963.

SCHAFER, J. A clinical study of terramycin in dentistry.
Oral Surg., Oral Med. and Oral Path. 6:965-973, Aug. 1953.

- SCHMITT, J., Ingram, R.C. and Harpole, H.J. Haematoma following dental extraction in a patient receiving unreported anti-coagulant therapy.
Oral Surg., Oral Med. and Oral Path. 13:791-794, July 1960.
- SCHNEIDER, P. Post-operative oedema and its reduction by trypsin.
J. Oral Surg. Anes. and Hosp. D. Serv. 17:49-56, May 1959.
- SCHNEIDER, P. Evaluation of Orenzyme for prevention of post-operative oedema.
N.Y. State D.J. 27:83-86, Feb. 1961.
- SCHRAM, W.R. A Manual of Oral Surgery.
Philadelphia. W.B. Saunders Co. 1962.
- SCOPP, I.W. and Fredrics, H. Dental extractions in patients undergoing anticoagulant therapy.
Oral Surg., Oral Med. and Oral Path. 11:470-474, May 1958.
- SCOPP, I.W. Fredrics, H. and Anderson, J.D. An evaluation of carboxymethyl cellulose as an absorbable coagulant in oral surgical procedures.
Oral Surg., Oral Med. and Oral Path. 14:1009-1013, Aug. 1961.
- SCRIVENER, C.A. and Schantz, C.W. Penicillin: new methods for its use in dentistry.
J.A.D.A. 35: 644-647, Nov. 1947.
- SEEVERS, M.H. Pharmacology of the newer synthetic analgesics.
J. Oral Surg. 7:30-38, Jan. 1949.
- SELDIN, H.M., Rakower, W. and Selman, A.J. Radio-osteomyelitis of the jaw.
J. Oral Surg. 13:112-119, April 1955.
- SHAFER, W.G., Hine, M.K. and Levy, B.B. A Textbook of Oral Pathology.
Philadelphia. W.B. Saunders Co. 1960.
- SHAPIRO, H.H., Sleeper, E.L. and Guralnick, W.C. Spread of infection of dental origin.
Oral Surg., Oral Med. and Oral Path. 3:1406-1430, Nov. 1950.
- SHATTAN, B.M. Carboxymethyl cellulose in post-extraction sockets.
N.Y. State D.J. 27:335-336, Aug. 1961.
- SHATTAN, B. and Kutscher, A.H. Effect of chlorophyll ointment on post-extraction healing.
J. Oral Surg. 10:324, Oct. 1952.

SHIRA, R.B. Control of post-operative haemorrhage.
J. Oral Surg. 16:144-159, March 1958.

SHIRA, R.B., Hall, R.J. and Guernsey, L.H. Minor oral surgery during prolonged anticoagulant therapy.
J. Oral Surg. Anes. and Hosp. D. Serv. 20:93-99, March 1962.

SHUTTEE, T.S. Hyaluronidase in relief of post-operative trismus, swelling and pain.
Oral Surg., Oral Med. and Oral Path. 15:114-120, Jan. 1962.

SICHER, H. Oral Anatomy, 3rd Ed.
St. Louis. The C.V. Mosby Co. 1960.

SILVERMAN, L.M. An investigation of gelatin sponge with thrombin and penicillin in the treatment of oral surgical wounds.
Oral Surg., Oral Med. and Oral Path. 2: 260-288, Feb. 1949.

SILVERMAN, R.E. The use of anti-histamines in oral surgery: a preliminary report.
J. Oral Surg. 11:231-237, July 1953.

SILVERMAN, R.E. Further clinical observations on the use of anti-histamines in oral surgery.
J. Oral Surg. 12: 310-313, Oct. 1954.

SIMPSON, H.E. Injuries to the inferior dental and mental nerves.
J. Oral Surg. 16:300-305, July 1958.

SIMPSON, H.E. Experimental investigation into the healing of extraction wounds in *Macacus rhesus* monkeys.
J. Oral Surg. Anes. and Hosp. D. Serv. 18:391-399, Sept. 1960.

SIMPSON, H.E. Effects of suturing extraction wounds in *Macacus rhesus* monkeys.
J. Oral Surg. 18:461-464, Nov. 1960.

SIMPSON, H.E. Healing of surgical extraction wounds in *Macacus rhesus* monkeys: I the effect of burs.
J. Oral Surg. Anes. and Hosp. D. Serv. 19:3-9, Jan. 1961.

SIMPSON, H.E. Healing of surgical extraction wounds in *Macacus rhesus* monkeys: II the effect of chisels.
J. Oral Surg. Anes. and Hosp. D. Serv. 19:126-129, March 1961.

SMITH, A. Placebos.
Editorial. J.A.M.A. 159:780, Oct. 22, 1955.

SMITH, R.L. The role of epithelium in the healing of experimental extraction wounds.

J.D. Res. 187-194, April 1958.

SNYDER, B.S. Effect of anti-histamine agents on inflammatory response after surgical trauma.

J. Oral Surg. Anes. and Hosp. D. Serv. 18:319-328, July 1960.

SOLNITZKY, O. The fascial components of the head and neck in relation to dental infections.

Bull. Georgetown Univ. Med. Centre 7:86-95, 1954.

SOWDRAY, J.H. An assessment of the value of lyophilised chymotrypsin in the reduction of post-operative swelling following the removal of impacted wisdom teeth.

Brit. D.J. 110:130-133, Feb. 21, 1961.

SPIEGEL, L.H. Penicillinase treatment of allergic penicillin reaction.

J. Oral Surg. Anes. and Hosp. D. Serv. 17:27-29, May 1959.

SPIES, T. (Editor). Nutrition and disease (reprint)

Post-grad. Med. 17: March 1955.

SPILSBURY, B.W. and Johnstone, F.R.C. The clinical course of actinomycotic infections.

Canad. J. Surg. 5:33, Jan. 1962.

abs. in Oral Surg., Oral Med. and Oral Path. 16: 639, May 1963.

STAFNE, E.C. and Bowing, H.H. The teeth and their supporting structures in patients treated by irradiation.

Am. J. Orth. and Oral Surg. (Oral Surg.) 33: 567-581, 1947.

STEG, R.F. Osteomyelitis of the mandible: report of a case.

Oral Surg., Oral Med. and Oral Path. 16:914-918, Aug. 1963.

STEINER, M. Oro-antral closure with gold plate: report of case.

J. Oral Surg. Anes. and Hosp. D. Serv. 18: 514-516, Nov. 1960.

STICKEL, F.R. and Clark, H.B. Prophylactic use of tetracycline after removal of impacted teeth.

J. Oral Surg. Anes. and Hosp. D. Serv. 19:149-152, March 1961.

STOY, P.J. and Gregg, G. Bell's Palsy following local anaesthesia.

Brit. D.J. 41:292-293, Dec. 4, 1951.

STUTEVILLE, O.H. New treatment of osteomyelitis.

J. Oral Surg. 8:301-313, Oct. 1950.

SUNG, R.R. Actinomycosis treated with crude lymph gland extract.
J. Oral Surg. 2:328-332, Oct. 1944.

SUTHERLAND, K.J.G. The use of cellulosic acid (absorbable gauze) in dental practice.
Aust. J. Dent. 52:250-252, July 1948.

SUTHERLAND, K.J.G. A scientific evaluation of vitamin K. therapy as a routine haemostatic measure.
Aust. D.J. 1:30-32, Feb. 1956.

SZMYD, L. A clinical evaluation of an anti-histamine preparation in oral surgery.
Oral Surg., Oral Med. and Oral Path. 9:928-931, Sept. 1956.

TAINTER, M.L. and Thronndson, A.H. Haemostatic effects of administration of calcium, viosterol, stypose and ceathyn by mouth.
J.A.D.A. and D. Cosmos. 25:638-646, April 1938.

TAINTER, M.L., Thronndson, A.H. and Richardson, A.P. Alleged haemostatic action of gelatin, coagulen, fibrinogen and histidine administered by mouth.
J.A.D.A. 28:420-427, March 1939.

TAM, J.C. and Clark, H.B. Use of chlorophyll ointment in impacted third molar wounds.
J. Oral Surg. 9:237-239, July 1951.

THOLEN, E.F. Closure of oral antral fistula.
J. Oral Surg. 3:255-257, July, 1945.

THOMA, K.H. The cause and treatment of haemorrhage.
J. Oral Surg. 6:185-203, July 1948.

THOMA, K.H. Oral Surgery Vols. 1 and 2, 4th Ed.
St. Louis. C.V. Mosby Co. 1963.

THOMA, K.H. and Goldman, H.W. Oral Pathology 5th Ed.
St. Louis. C.V. Mosby Co. 1960.

THOMPSON, E.C. and Morris, C.R. Control of post-operative pain.
J. Oral Surg. 16:126-130, March 1958.

THOMSON, E. Antibiotics - a review.
D.J. Aust. 26:167-175, Oct. 1954.

TOLHURST, J.C., Buckle, G. and Williams, S.W. Chemotherapy with Antibiotics and Allied Drugs 2nd Ed.
National Health and Medical Research Council. Spec. Report Series No. 6, 1963.

TOPAZIAN, D.S. Prevention of osteoradionecrosis of the jaws.
Oral Surg., Oral Med. and Oral Path. 12:530-538, May 1959.

VERBIC, R.L. Local implantation of aureomycin in extraction wounds:
a preliminary study.
J.A.D.A. 46:160-163, Feb. 1953.

VERSNEL, J.C. Healing of extraction wounds after introduction of
haemostatics and antibiotics.
J.A.D.A. 46:146-153, Feb. 1953.

VILLA, V.G. Pulp abscess associated with actinomyces.
Oral Surg., Oral Med. and Oral Path. 10:207-209, Feb. 1957.

WAGGENER, D.T. and others. A study of the relationship between
oral sepsis and post-surgical complications (abstract of a report).
J.D. Res. 40: 743, July-Aug. 1961.

WAITE, D.E. Infections of dental aetiology in the mandibular and
maxillo-facial region.
J. Oral Surg. Anes. and Hosp. D. Serv. 18:312-318, July 1960.

WALKER, D.G. Nutrition in oral surgery and its relationship to wound
healing and infection.
Oral Surg., Oral Med. and Oral Path. 7:797-812, Aug. 1954.

WALLACE, D.A. Analgesic drugs of value in the control of dental pain.
D. Clin. N. Am. 65-76, March 1958. Philadelphia. W.B. Saunders Co. 1958.

WALLNER, L.J. Treatment of oro-antral fistula.
Arch. Otolaryng. 55:666-670, June 1952.

WEINMANN, J.P. Bone formation and bone resorption.
Oral Surg., Oral Med. and Oral Path. 8:1074-1078, Oct. 1955.

WEINMANN, J.P. and Sicher, H. Bone and Bones 2nd Ed.
St. Louis. C.V. Mosby Co. 1955.

WHITFIELD, A.J. Use of thiamine chloride in the prevention of pain
from dry sockets.
J.A.D.A. 28:450-452, March 1941.

WHITTINGTON, P.B. Control of bleeding with intravenous estrogens.
J.A.D.A. 53:595, Nov. 1956.

WOLFER, J.A., Farmer, C.J., Carroll, W.W. and Manshardt, D.O.
An experimental study in wound healing in vitamin C depleted human
subjects.
Surg. Gyn. Obs. 84:1-15, Jan. 1947.

YACKEL, E.C. and Kenyon, W.O. Oxidation of cellulose by nitrous oxide.

J. Amer. Chem. Soc. 64:121, 1942.

cited by Croker (1952) Kruger (1954)

YOUNG, H.E. and Kingsburn, B.C. Hyaluronidase for prevention of edema after removal of impacted mandibular third molars.

J.A.D.A. 50:418-421, April 1955.

ZIFFER, A.M. and others. Profound bleeding after dental extractions during dicumorol therapy.

New England J. Med. 256: 351-353 Feb. 21, 1957.

also cited by Behrman and Wright (1961) and Scopp and Fredrics (1958).

ZISKIN, D.E. and others. Actinomycosis.

Am. J. Orth. and Oral Surg. 29:193-201, April 1943.

ZITKA, E. Klinische und Therapeutische Erfahrungen Bei Cervicofacialer Aktinomykose (Clinical and therapeutic experience in cases of cervicofacial actinomycosis.)

Ztschr. f. Stomatol. 48: 67, 1951.

abs. in Oral Surg., Oral Med. and Oral Path. 4:1342, Oct. 1951.