

ECCHYMOSIS FOLLOWING SURGICAL REMOVAL
OF LOWER LEFT THIRD MOLAR TOOTH



The patient, a healthy 76 year old male, had a partly-erupted, carious lower left third molar tooth removed on the 19th February, 1964. The wound was closed with four catgut sutures and an ice pack was applied for thirty minutes post-operatively.

Twenty-four hours later a moderate haematoma was noted at the surgery site and the patient was instructed to use intra-oral heat to promote resolution. Five days following surgery the surgery site was found to have broken down and there was marked ecchymosis as shown in the photograph.

The discolouration faded after eighteen days.

Over a period of ten to fourteen days, due to the release and breakdown of the blood pigments, the discoloured area changes from a deep purple colour to greenish-yellow finally fading away (Shira, 1958; Thoma, 1963; Dingman and Hayward, 1947; Kruger, 1959).

Ecchymosis may occur in patients with a bleeding diathesis. Shira (1958) states that it is most frequently seen in females who have a history of easy bruising and in elderly patients who have decreased capillary resistance and poor tissue tone. Dingman and Hayward (1947) and Thoma (1963) consider that, if the soft tissues have been firmly approximated following surgery, in the event of any haemorrhage blood may diffuse into the sub-epithelial tissues. All authors agree that ecchymosis may result from rough handling of the soft tissues causing possible capillary damage and extravasation of blood into the tissues.

Dingman and Hayward (1947) state that ecchymosis may be minimised by the immediate post-operative application of an ice pack and a pressure bandage to the soft tissues adjacent to the surgery site. These authors consider that, once ecchymosis has occurred, nothing can be done to aid resorption of the blood pigments. On the other hand Thoma (1963) and Archer (1961) report that the use of heat applications to the discoloured area results in more rapid resorption of the blood pigments.

(b) Haematoma is an effusion of blood into the tissues resulting in the formation of a tumour-like mass of blood. It is an occasional complication of oral surgical procedures which Berlove (1959)

and Monheim (1961) consider to be, as a rule, a minor occurrence as resolution generally occurs without treatment.

Haematoma most commonly results from the piercing of a blood vessel by the needle during injection with subsequent extravasation of blood into the tissues (Archer, 1961; Berlove, 1959). Monheim (1961) states that haematoma is most commonly associated with the zygomatic and infra-orbital nerve block injections. Ritchie (1952) considers that a haematoma may develop following a maxillary block injection. This may occur as a result of penetration of the pterygoid plexus of veins by the needle as it passed into the pterygo-palatine fossa. Other causes of haematoma are failure of the surgeon to effect total haemostasis during surgery and contusion or other trauma to the mucous membrane or skin of the face (Kruger, 1959; Croker, 1952; Berlove, 1959).

Haematoma formation is generally a self-limiting occurrence due to the build up of local pressure which prevents further extravasation of blood. A haematoma may develop either rapidly or slowly and, in the latter case, marked ecchymosis is a common associated complication due to diffusion of the blood through the sub-epithelial tissues.

When a haematoma is observed to be forming, for example, following injection, external pressure together with an ice pack should immediately be applied to the affected area (Ritchie, 1952). Prompt treatment may arrest bleeding and avoid formation of a large collection of blood. Archer (1961) recommends that ice packs should be

maintained over the involved area for the first twenty four hours following surgery. He considers that this procedure tends to minimise extravasation of blood. Both Archer (1961) and Berlove (1959) consider that, after twenty four hours, the application of heat to the region hastens resorption of accumulated blood. However Kruger (1959) states that a haematoma should be incised and drained as he feels that, if such a collection of blood became infected, management may prove difficult due to the lack of circulation in the coagulated blood.

Henkel (1956) reports the use of hyaluronidase for the treatment of haematoma. He found that prompt injection of hyaluronidase into the area of a haematoma, together with application of intra-oral or extra-oral pressure, resulted in more rapid resolution of the effused blood. The increased rate of resolution reported by Henkel is dependent on the activity of the hyaluronidase as a "spreading factor", allowing diffusion of the blood into the sub-epithelial tissues. It seems possible that such spread may firstly, result in prolonged bleeding as increase in local pressure is prevented and secondly, cause the formation of large ecchymotic areas which may be more alarming to the patient than the original haematoma.

6. CONTROL OF HAEMORRHAGE

(a) Local haemostatic measures:

(i) Direct pressure applied to a bleeding wound is frequently sufficient to control mild haemorrhage. Pressure, usually applied by

means of pads of sterile gauze, causes some compression of the traumatised vessels thus preventing egress of blood and allowing coagulation to occur. Continual application of pressure for ten to twenty minutes should be a routine post-surgical procedure in order to maintain the haemostasis effected during surgery.

Packing of a wound with sterile gauze may, in some instances, be required to effect haemostasis. This procedure may be necessary, for example, to control persistent bleeding from a large bony cavity where haemorrhage may occur from a number of vessels. It is common practice to use gauze impregnated with medicaments such as petrolatum, with or without iodoform, or Whitehead's Varnish. The major disadvantage of this method is that the gauze must subsequently be removed so disturbing the blood clot and perhaps initiating further bleeding.

In the case of bleeding from bone the vessel may be closed by crushing the bone with rongeur forceps or burnishing with a blunt instrument. This results in compression of the damaged vessel and allows coagulation to occur. Sterile bone wax is also of value in the control of haemorrhage. It has a twofold action - firstly, it occludes the bleeding vessel and, secondly, the carbolic acid content of the wax causes coagulation of the blood protein forming a mechanical plug which further occludes the vessel (Ricker, 1949; Kemp, 1952; Thoma, 1948, 1963; Shira, 1958). Naylor and Moore (1962) suggest that arterial haemorrhage from bone may be controlled by gently

driving a chisel into the adjacent bone to compress the vessel between the walls of its canal.

Direct pressure to bleeding vessels in soft tissue may be applied by means of artery (Spencer-Wells) forceps. The vessels should be grasped with the forceps with as little as possible of the surrounding tissues. As a rule bleeding from smaller vessels may be controlled simply by twisting the forceps and leaving for a few minutes. Sometimes, however, such bleeding cannot be controlled by this method and the blood vessels need to be tied off (Thoma, 1963).

(ii) Sutures and ligatures are valuable local measures for effecting haemostasis. Excessive capillary bleeding can frequently be controlled by passing a suture deep to the wound to apply pressure to the deeper vessels supplying the area. Closure of a wound using sutures results in apposition of the soft tissues and, in certain sites, holds the tissues against the bone. Both actions assist haemostasis by impeding the escape of blood.

Ligatures are used to arrest haemorrhage by occluding the bleeding vessel. Vessels that require to be tied off should first be grasped with a pair of artery forceps at the bleeding point. In doing this care must be taken to avoid trauma to the adjoining tissues by ensuring that only the vessel itself is grasped in the forceps. Otherwise, if other structures are involved, sloughing of the tissues may occur.

In cases of haemorrhage which cannot be controlled by local methods consideration should be given to ligating the artery supplying

the region. In extreme cases, ligation of the external carotid artery may be necessary.

(iii) Thermal applications are of value in control of haemorrhage in some cases.

The application of cold causes vasoconstriction of the superficial blood vessels and may be used to aid haemostasis (Shira, 1958; Thoma, 1963; Kemp, 1952; Cheraskin, 1959). However it must be remembered that the vascular response is transient, thus cold applications should only be used as an adjunct to other haemostatic measures.

Hot normal saline packs applied to an open wound or bleeding from bone may be used in order to accelerate the coagulation of blood. The normal saline should be heated to 120° F and applied, with pressure to the bleeding area (Barling, 1963). The use of thermocautery will be discussed subsequently.

(iv) Absorbable haemostatic materials are agents which, when placed in contact with a bleeding area, effect haemostasis by promoting coagulation. Also, some, by virtue of their physical structure, provide a framework for blood clot formation. Following blood coagulation these haemostatic agents are absorbed, the rate of absorption depending on the nature and the quantity of the material used.

1. Topical thrombin application is an effective haemostatic measure which results in immediate coagulation by direct action on

on fibrinogen. To be effective the thrombin, used either as a powder or in a reconstituted form, must be placed in direct contact with the bleeding surface. The powder may be dusted over the wound while the thrombin solution is generally used in combination with one of the absorbable sponges (Howell and Monto, 1953; Shira, 1958; Thoma, 1963).

All authors emphasise that the haemostatic effect of topical thrombin is increased if pressure is maintained on the area following its application. Of course pressure, while ensuring intimate contact of the thrombin with the wound surface, would itself aid in control of bleeding.

It must be pointed out that thrombin is intended for topical application only and under no circumstances should it be used parenterally. If thrombin is injected intravenously, or otherwise forced into a vein, intravascular thrombosis will occur, which may cause serious or even fatal embolism (Accepted Dental Remedies, 1963).

Howell and Monto (1953) consider that failure to effect haemostasis with topical thrombin application is generally due to too large a bleeding vessel, oozing from beneath an imperfect clot, or the use of too little thrombin.

2. Oxidised cellulose (Trade name: OXYCEL; Parke Davis Co.) is formed by the oxidation of cellulose by the action of nitrogen dioxide (NO_2). This process was developed by Yackel and Kenyon (1942). The reaction produces, from ordinary gauze, a solid organic acid which maintains its original shape and has a tensile strength

approximately two-thirds that of the original fabric (Kruger, 1954; Gwinn et al, Feb. 1948). Oxidised cellulose must be sterilised by chemical means as it is heat labile and decomposes above 80°C.

On contact with blood, oxidised cellulose forms a sticky coagulum which is dark brown to black in colour. The haemostatic effect is generally considered to be due to at least two factors. Firstly, the acidic nature of the oxidised cellulose, which causes coagulation on contact with blood, secondly, swelling of the material that occurs as it becomes soaked with blood, thus causing pressure on the bleeding vessels (Kruger, 1954; Gwinn et al, Feb. 1948; Croker, 1952). On the other hand Accepted Dental Remedies (1963) and Sutherland (1948) believe the haemostatic activity to be principally due to the formation of a coagulum of complex salts of oxidised cellulose and haemoglobin. The haemostatic activity of oxidised cellulose is not enhanced by the addition of other haemostatic agents such as thrombin, due to the acidity of the material (pH 4).

Oxidised cellulose is of value in controlling haemorrhage from capillaries, arterioles and venules. A layer of the gauze should be placed on the bleeding surface and held in position with firm pressure until haemostasis is effected. In some cases, where more extensive haemorrhage is present, a second or third layer of oxidised cellulose may be required to control all bleeding. Where profuse local bleeding is encountered oxidised cellulose may be

firmly packed into the wound and held under firm pressure for five to ten minutes. In cases of post-operative haemorrhage from extraction sockets and surgical wounds, the reviewer has found oxidised cellulose sutured into the wound to be a most efficacious method of effecting haemostasis.

Nathan (1948) states that generally, following the insertion of oxidised cellulose into an extraction wound, the sockets appeared to be enlarged due to expansion of the soft tissue. This author also noted that adjacent soft tissues become engorged, however he reports that rapid healing usually followed. On the other hand Versnel (1953), in an experimental study, found that after the use of oxidised cellulose in extraction sockets, healing is delayed by as much as two weeks. Jacobs and Rafel (1950) and Versnel (1953) consider that epithelialisation of extraction wounds is delayed when oxidised cellulose is used to effect haemostasis. It is for this reason that Accepted Dental Remedies (1963) recommend that oxidised cellulose should not be used in superficial wounds. However, Helmore (1958) and Gwinn et al (Feb. 1948) believe that, clinically no delay in healing occurs subsequent to the use of oxidised cellulose.

Oxidised cellulose is completely absorbed from the site of implantation when left in the body tissues. The rate of absorption being related, among other things, to the amount of material placed in the wound (Gwinn et al, Feb. 1948). Jacobs and Rafel (1950)

in experimental animals, found that oxidised cellulose is absorbed slower than the other absorbable haemostatic agents used, total absorption taking as long as twenty six days.

Geary and Frantz (1950) report the use of an absorbable haemostatic bone wax composed of oxidised cellulose and polyethylene glycols. These authors found the combination to be efficient in the control of haemorrhage in neuro-surgical cases and suggest that its use might be extended to other surgical branches. Douglas (1953) reports that the use of absorbable haemostatic bone wax of similar formula to that used by Geary and Frantz (1950), resulted in prevention of post-operative haemorrhage in patients with a history of abnormal bleeding following dental extractions.

One disadvantage of oxidised cellulose is that once the material comes in contact with blood, it becomes sticky and tends to adhere to the surgeon's gloves and instruments.

3. Carboxymethyl cellulose (Trade name: SURGICEL, Johnson and Johnson) is prepared from sodium carboxymethyl cellulose which is converted to the free acid compound, namely, modified carboxymethyl cellulose. This product is further purified and is supplied either as a knitted fabric or as compressed fibre cones which are suitable for insertion into extraction sockets (Scopp et al, 1961).

The material possesses uniform molecular weight as a result of its chemical purity and uniformity of physical structure. These properties, according to Georgiade et al (1961), result in a predictable rate of absorption of the material when implanted into the

tissues. Carboxymethyl cellulose is less friable than oxidised cellulose and has less tendency to adhere to instruments and gloves after contact with blood.

Carboxymethyl cellulose, like oxidised cellulose is a heat labile substance and therefore cannot be autoclaved or boiled. The material, as supplied, is sterilised by chemical means (Johnson and Johnson, 1961).

When exposed to blood, carboxymethyl cellulose is converted into a dark brown gelatinous mass which appears to form, in effect, an artificial blood clot within the openings of the bleeding vessels and the surrounding area (Johnson and Johnson, 1961). The haemostatic action is thought to be due to the marked affinity of the material for haemoglobin and also to its acidity which causes blood coagulation. Also, when carboxymethyl cellulose becomes soaked with blood, some swelling of the material occurs causing pressure on the bleeding vessels (Shattan, 1961; Scopp et al, 1961; Accepted Dental Remedies, 1963). Like oxidised cellulose, the haemostatic effect of carboxymethyl cellulose cannot be increased by the addition of thrombin solution as the activity of the thrombin is destroyed by the acidity of the carboxymethyl cellulose.

Georgiade et al (1961), using experimental animals, found that no tissue reaction to carboxymethyl cellulose occurred and histologically, absorption occurred within twenty one days following implantation into the tissues. Following the favourable results obtained with animals, these authors used the material as a

haemostatic agent in 1036 surgical patients. The operative procedures ranged from extraction of teeth to radical neck dissection. In all cases no untoward reaction was noted post-operatively and haemostasis was satisfactory. Shattan (1961) in a study involving 19 normal patients, reports that carboxymethyl cellulose used as a haemostatic, was completely absorbed with no tissue reaction to the material being seen. According to Accepted Dental Remedies (1963) and Johnson and Johnson (1961), carboxymethyl cellulose does not interfere with epithelialisation and, therefore, may be used as a surface dressing in the treatment of superficial wounds.

The method of use of carboxymethyl cellulose is identical to that of oxidised cellulose. That is, simply the placing of a layer of the material over the bleeding surface followed by the application of firm pressure.

4. Gelatin Sponge is an absorbable haemostatic material that is produced by foaming a purified gelatin solution which is subsequently air dried and sterilised. A tough, white, porous sponge is formed which may be cut to any shape or size without fragmenting. Gelatin sponge absorbs fluid readily and is capable of absorbing up to forty-five times its own weight of blood (Thoma, 1963; Gwinn et al, May, 1948; Kruger, 1954; Accepted Dental Remedies, 1963).

Gelatin sponge has been found to possess some direct haemostatic activity and it is considered that this may occur as a result of disruption of the platelets or due to the fact that the sponge

provides a framework in which coagulation can occur. Also, when gelatin sponge absorbs blood some swelling of the material occurs, resulting in pressure on the bleeding tissues which may aid haemostasis (Gwinn et al, May 1948; Accepted Dental Remedies, 1963; Jenkins et al, 1946).

Gelatin foam may be used as a vehicle for thrombin solution as the two substances are compatible (Goulding, 1960). Kruger (1954) considers that the use of gelatin foam and thrombin is unnecessary for most oral surgical procedures. Gwinn et al (May 1948) and Guralnick and Berg (1948) consider that the combination is only necessary in persistent cases.

Silverman (1949) reports that complete absorption of gelatin sponge occurs within twenty-five to thirty days post-operatively. This author also noted that no delay occurred in the healing process. This finding is supported by Jacobs and Rafel (1950) who noted that, histologically, fragmentation of the gelatin sponge occurred after four days with complete absorption after twenty-six days. Epithelialisation of extraction wounds is retarded following the use of gelatin sponge according to Jacobs and Rafel (1950), who found that approximately twenty-six days were required for complete epithelial covering to occur. On the other hand, Goulding (1960) states that the material causes "no hinderance to epithelialisation".

Ricker (1949) suggests that, as gelatin is used as a bacterial culture medium, the introduction of gelatin sponge into a wound may create an environment that favours bacterial growth.

Absorbable gelatin sponge is applied directly to the bleeding surface in amounts sufficient to cover the area. It may be applied in the dry form or may first be saturated with sterile normal saline or thrombin solution. Goulding (1960) notes that gelatin sponge should only be relied on to check capillary oozing and should not be used in an attempt to control overt bleeding.

5. Fibrin foam is an absorbable haemostatic processed from human blood. It is formed by clotting a solution of fibrin and foaming the fibrin thus formed. The product is then dried in the frozen state and then heated for three hours. This results in a fine, white sponge of firm texture which is water insoluble (Crocker, 1952; Goulding, 1960).

For use as a haemostatic the material is cut to a suitable size and soaked in thrombin solution and then applied to the bleeding area. The resulting blood clot forms in the interstices of the foam, forming a firm blood clot. Goulding (1960) considers that fibrin foam probably constitutes more of a mechanical framework for blood coagulation rather than supplying a specific coagulation factor. The effectiveness of fibrin foam used in conjunction with thrombin solution is reported by Losch (1945), Mitchell (1946) and Gwinn et al (1947). Ewing (1947) states that one disadvantage of the material is that to be efficient as a haemostatic agent, fibrin foam should be placed directly over the bleeding point.

Gwinn et al (1947) and Jacobs and Rafel (1950) report normal healing following the use of fibrin foam in extraction sockets.

The latter authors also found the implanted fibrin foam to be indistinguishable from the organising blood clot after four days.

Crocker (1952) points out that while fibrin foam is a satisfactory absorbable haemostatic agent, it is made from human blood and thus difficulty in supply may occur.

6. Alginates: the commercial alginate preparations used as absorbable haemostatic agents are composed of sodium and calcium alginate, derivatives of alginic acid. Alginic acid itself is unstable, the substance used for the manufacture of haemostatic material is sodium alginate.

The sodium salt of alginic acid is water soluble and insoluble salts can be readily prepared by double decomposition of sodium alginate and salts of a bivalent metal. Such a reaction occurs when an aqueous solution of sodium alginate is acted upon by ionic calcium, the product being insoluble calcium alginate.

Clinically it was found that when sodium alginate came in contact with blood, an "artificial clot" was formed by reaction with the calcium ions present (Kruger, 1954; Crocker, 1952).

For haemostasis, use can be made of a mesh or weave of calcium alginate alone or a combination of calcium and sodium alginates used in powder or liquid form. By altering the ratio of sodium alginate to calcium alginate products may be made with varying coagulation rates.

Blaine (1947), using rabbits, found that complete absorption of calcium alginate occurred in every case. He noted that the rate

of absorption is proportional to the bulk of the alginate used and its physical form. Sandy (1948), in a clinical study, reports that satisfactory haemostasis was achieved using absorbable alginate and that subsequent healing was uneventful. On the other hand, Kruger (1954), also in a clinical study, found alginate to be a poor haemostatic agent. Following its use, an increase in post-operative pain was noted and the extraction sockets showed delayed healing.

(v) Adrenaline is a powerful vasoconstrictor drug that is effective in controlling haemorrhage from small blood vessels when used as a topical application to the bleeding site. However the value of this drug is limited by its transitory effect and, unless a firm blood clot is established during the period of vasoconstriction, haemorrhage may recur.

In cases where local anaesthesia is required to effect haemostasis, the presence of adrenaline in the local anaesthetic solution may aid in the control of haemorrhage by causing vasoconstriction of the bleeding vessels. The vasoconstricting action of the adrenaline is, as mentioned above, only transitory and cannot be relied upon to maintain haemostasis.

(vi) Thermocautery is of value for controlling haemorrhage from small blood vessels. The action of the thermocautery is to simply coagulate the tissues by heat resulting in the formation of a mechanical plug which effects haemostasis. It must be noted, however, that necrotic tissue, or eschar, may predispose to secondary infection or may slough away from the adjoining tissues causing renewed

haemorrhage (Crocker, 1952; Howell and Monto, 1953; Cheraskin, 1959).

(vii) Chemical escharotic agents have been used in the past for the control of haemorrhage. Drugs commonly utilised included silver nitrate, phenol, chromic acid, trichloroacetic acid, ferric subsulphate (Monsel's solution), tincture of ferric chloride and tannic acid (Thoma, 1948; Crocker, 1952; Helmore, 1958; Cheraskin, 1959; Accepted Dental Remedies, 1963). These chemical substances effect haemostasis by coagulating tissue and blood proteins thus forming a mechanical barrier to the escape of blood. The tissue destruction which results from their use may lead to delay in healing and secondary haemorrhage. Hence these substances have generally been replaced by agents which are more effective and have a more desirable mode of action.

(b) Systemic Haemostatic Measures: much has been written on the value of certain drugs which, when administered orally, would exert a favourable influence on the coagulating mechanism. Among the substances recommended are calcium gluconate, calcium lactate, vitamin D, gelatin, coagulen, histidine, fibrinogen and vitamin K (Helmore, 1958; Sutherland, 1956; Shira, 1958; Tainter and Thronson, 1938; Tainter et al, 1939).

Although calcium is necessary for coagulation to occur, it is now known that the blood calcium level never falls to a level at which there is insufficient calcium for the process of blood clotting. Hence the administration of calcium is of no value in promoting

haemostasis (Helmore, 1958; Croker, 1952; Sutherland, 1956; Shira, 1958). Vitamin K, a substance essential for synthesis of prothrombin by the liver, is often prescribed when a haemorrhage occurs (Cheraskin, 1959; Shira, 1958; Sutherland, 1956). Hypoprothrombinaemia, due to vitamin K deficiency, may occur due to inadequate intake of vitamin K, failure of absorption or failure of the liver to utilise the available vitamin K. This may occur in hepatic disorders such as hepatitis or cirrhosis or may result from therapy with certain anticoagulant drugs. Any patient with a bleeding disorder due to hypothrombinaemia presents a complicated medical problem and consultation with the patient's physician prior to undertaking treatment is advisable. Unless one of the foregoing conditions is present the administration of vitamin K is without foundation (Cheraskin, 1959; Shira, 1958; Helmore, 1958). Support for this statement is presented by Sutherland (1956) who found, in a clinical study involving 232 patients, that the routine use of vitamin K is of no value. The other haemostatic agents mentioned above, that is, vitamin D, gelatin, coagulen, histidine and fibrinogen have been shown by Tainter and Thronson (1938) and Tainter, Thronson and Richardson (1939) to be ineffective and therefore their use is unjustified.

"Premarin intravenous" (Ayerst Laboratories), a conjugated oestrogenic preparation, is reported by Nathanson (1961) to be efficient in preventing post-operative haemorrhage in patients who

had experienced prolonged bleeding after previous oral surgical procedures. Nathanson (1961) states that intravenous injection of Premarin did not cause any significant changes in post-operative bleeding or clotting times. The first dental use of this agent was reported by Whittington (1956) who found that one 20 mg. injection controlled profuse post-extraction haemorrhage in a patient who failed to respond to other forms of therapy. Kure and Rudy (1964) state that the mechanism of action of Premarin as a haemostatic agent is not fully understood. It is thought to act intravascularly to enhance the normal coagulation mechanism by increasing the concentration of prothrombin and factor V and extravascularly by increasing the strength and integrity of the vascular bed by altering the amount and nature of the ground substance surrounding the blood vessels. Thoma (1963) considers Premarin to be of value in the treatment of excessive capillary oozing or haemorrhage from bone. Kure and Rudy (1964) in a controlled clinical study found that Premarin intravenous was of value in minimising blood loss during surgery and preventing post-extraction haemorrhage, ecchymosis and haematoma formation. Nathanson (1961) is so impressed with the results obtained in his study that he considers they (the results)... "demonstrate the value of injecting Premarin intravenous as a routine prophylactic measure for haemostasis." The present reviewer considers that such routine use of a drug is irrational. It is believed that drugs should only be administered when examination indicates a need for specific therapy.

When haemorrhage has been profuse and of long standing blood transfusion may be necessary to replace the lost blood. Also transfusion of whole blood or specific purified blood fractions may be of value in the control of haemorrhage due to a coagulation defect. In any case where transfusion is indicated hospitalisation and medical consultation become necessary.

7. MANAGEMENT OF POST-OPERATIVE HAEMORRHAGE.

When a patient presents with post-operative haemorrhage a rapid assessment of the general appearance and condition of the patient should be made and the duration of bleeding determined. Naylor and Moore (1962) suggest that the patient should be questioned regarding the duration of the bleeding rather than the amount of bleeding as, due to dilution with saliva, the patient's estimation of the latter is frequently unreliable.

The oral cavity is then examined using a good light together with an aspirator to remove loose blood clots and saliva so that clear vision of the bleeding area is possible. Local anaesthesia of the area should then be induced, partly in order to make it possible to work efficiently without causing pain to the patient and partly because infiltration into the area may itself reduce the flow of blood by the vasoconstricting action of the adrenaline in the local anaesthetic solution. Once anaesthesia is obtained, large blood clots extending from the wound should be removed to permit visualisation of the bleeding area.

The origin of the haemorrhage and the type of bleeding should be determined at this stage. Arterial bleeding is characterised by spurting of bright red blood, venous bleeding is manifest by a constant flow of darker blood while in capillary haemorrhage a continued oozing of blood occurs. Haemorrhage may arise from bone or from the overlying soft tissues and it is this factor which determines the plan of treatment.

When haemorrhage is occurring from the soft tissues a careful examination of the surgical site should be made to detect the presence of any irritating factors that may predispose to haemorrhage. Any bone splinters, sharp bony margins or vascular granulation tissue should be removed (Chaput, 1964; Shira, 1958; Cohen, 1958).

When bleeding from a large vessel is detected it should be controlled by grasping with a pair of artery forceps and a ligature applied. Generally, however, soft tissue haemorrhage appears as a steady flow of blood from a traumatised vessel or capillary oozing and in such cases pressure may effect haemostasis. Direct pressure to the bleeding area should be applied by means of a sterile gauze pad for fifteen to twenty minutes. In persistent cases a strip of one of the absorbable haemostatic materials may control bleeding when placed in contact with the bleeding surface. Sutures may also be used with effect in controlling haemorrhage from soft tissues. They should be so placed as to draw the tissues against the underlying bone thus causing pressure on the bleeding vessels.

If haemorrhage is occurring from bone, all the blood clot in

the surgical site should be removed to allow observation of the bleeding point. Bleeding from bone can often be controlled by crushing the bone with a pair of rongeur forceps or by burnishing with a blunt instrument. The application of bone wax may also be of value. Generally bleeding from bone appears as a continuous oozing or a brisk flow and, in most cases, packing of the wound effects haemostasis. The absorbable haemostatic materials are indicated in such cases and their use usually controls bleeding. In cases of persistent haemorrhage from bone, packing with plain or iodoform gauze is necessary. This allows firm pressure to be applied to the bleeding site and its effect may be enhanced by suturing the soft tissues across the wound to keep the packing in position. As a temporary measure to maintain pressure on the wound and to keep the gauze in position, a piece of compound may be moulded to form an appliance which the patient can hold in place between the teeth. The gauze should be left in position for three to four days and then carefully removed. The disadvantage of packing a wound with gauze is that haemorrhage may recur following removal of the gauze. However Shira (1958) considers that this is generally mild compared to the initial haemorrhage and seldom presents a problem in management.

Once haemostasis is effected by local means, a pressure dressing should be placed over the wound and firm pressure maintained for at least twenty minutes. The patient should then be given strict instructions to ensure that the wound is not disturbed.

Rinsing of the mouth should be forbidden for at least twenty-four hours, after which time gentle rinsing with warm saline solution may be carried out. Home rest should be advised, preferably with the patient lying down with the head elevated. The patient should be warned against physical exertion and the use of stimulants which may cause a rise in blood pressure and initiate haemorrhage. A soft diet should be prescribed, the patient being warned to avoid hot or coarse foods which may cause further haemorrhage. When haemorrhage has been severe or prolonged the patient should also be advised to increase his fluid intake to aid in maintenance of the normal fluid balance. The patient should also be told that local pressure applied to a bleeding wound will control any further haemorrhage but that if haemorrhage becomes severe or is persistent the oral surgeon should be contacted at once.

If bleeding cannot be controlled by local measures, the presence of a bleeding disorder should be suspected. In such cases the patient should be referred immediately for haematological investigation. If any abnormality is detected, medical consultation should be sought to plan further treatment. In cases of post-operative haemorrhage where excessive blood loss has occurred replacement of the lost blood by transfusion may be indicated. When this situation occurs hospitalisation and medical consultation become necessary.

Preventive measures taken prior to surgery can do much to avoid post-operative haemorrhage. Each dental surgeon should be aware of the need for taking a full case history of the patient and have a full

appreciation of the clinical signs and symptoms that may indicate a haemorrhagic tendency.

DRY SOCKET

1. INTRODUCTION
2. INCIDENCE OF DRY SOCKET
3. CLINICAL FEATURES
4. AETIOLOGY
5. BACTERIOLOGY
6. HISTOPATHOLOGY
7. PREVENTION
8. DIAGNOSIS
9. TREATMENT.

DRY SOCKET

1. INTRODUCTION.

The term "dry socket" has been attributed by Edwards (1963) to an American dentist James Young Crawford who, in 1896, used the term to describe a socket that was devoid of blood clot and associated with severe pain.

Since then many terms have been used to describe the condition, some in more common usage being alveolar osteitis, alveolalgia, alveolitis, localised osteomyelitis, post extraction alveoladgia and post-operative osteitis.

A dry socket is defined by Krogh (1948) as being "a socket in which the blood clot disintegrates with the production of a foul odour without pus formation and accompanied by a severe neuralgia of the fifth cranial nerve persisting over a period of several days".

Although a great many articles have been written on the subject, no single aetiological factor has been determined nor is there complete agreement on methods of treatment. However most authors concur that the primary object of treatment is to control the pain which is the most distressing symptom and to pave the way for optimal healing of the wound.

2. INCIDENCE OF DRY SOCKET.

Dry socket has been studied by several authors both in statistical surveys of the problem and also during the course of investigation into methods of treatment.

Perhaps the most comprehensive statistical report is that presented by Buchanan (1961) who found that, in a series of 50,386 extractions, the incidence of dry socket was 0.79 per cent. Archer (1939) after studying 23,886 extraction cases, found the incidence of dry socket to be 0.9, per cent, while both Krogh (1937) - (6,403 extractions) - and Gufstafson and Wallenius (1961) - (5,679 extractions), in a smaller series of cases, report an incidence of 1.2 per cent. Hanson (1960), in less than 1,000 extractions, reports an incidence of 2.0 per cent.

Buchanan (1961), and Krogh (1937), report an equal incidence of dry sockets in male and female patients.

Archer (1939) notes that 79 per cent of all dry sockets occur in the mandible and Buchanan (1961) agrees with this finding. The latter author reports that 72 per cent of the cases in his survey occurred in the lower jaw.

Both Krogh (1937) and Buchanan (1961) analyse the incidence of occurrence for each tooth situation in the jaws and present the following results:

(i) Krogh (1937) Percentage Incidence of Dry Socket Occurrence
(Erupted Teeth)

Socket	Central	Lateral	Canine	1st Prem.	2nd Prem.	1st Molar	2nd Molar	3rd Molar
Maxilla	0%	0%	0%	0%	0.8%	0.2%	0%	0.4%
Mandible	0%	0%	0.6%	2.2%	2.2%	4.2%	5.0%	3.5%

After Krogh (1937)

(ii) Buchanan (1961) Percentage Incidence of Dry Socket Occurrence.

Socket	Central	Lateral	Canine	1st Prem.	2nd Prem.	1st Molar	2nd Molar	3rd Molar
Maxilla	0.03%	0.29%	0.96%	0.72%	0.99%	0.36%	0.54%	0.37%
Mandible	0.14%	0.27%	0.82%	1.43%	1.6%	0.98%	1.46%	2.74%

3. CLINICAL FEATURES.

Characteristically the patient with a dry socket returns three or four days following extraction of a tooth complaining of severe pain. This period of time may, however, be as short as 24 hours or may be in excess of ten days.

According to Gold (1957) the pain is described as severe and throbbing in nature. It may radiate from the affected socket and be referred to other regions supplied by the trigeminal nerve. It is well known that patients with dry sockets in the lower premolar-molar region frequently complain of earache. The pain accompanying dry socket is often severe enough to prevent sleeping and may not be relieved by mild analgesics.

The patient may complain of a bad taste in the mouth (Gold, 1957) and frequently foetor oris is present (Thoma, 1963). Examination of the socket reveals either total absence of a blood clot or a partly degenerated clot which no longer fills the socket and is unevenly attached to the socket walls (Rud et al, 1963). The remnants of the clot are readily removed by irrigation and an exploring instrument may be passed into the bony cavity without experiencing the resistance that is encountered in a normal healing socket. If this instrument

DRY SOCKET

This photograph illustrates the characteristic appearance of dry socket. The patient returned five days following extraction of four upper anterior teeth complaining of severe pain from the sockets.

is permitted to touch the socket walls the severity of the pain is increased.

Thoma (1963) notes that the opening of the socket may be obscured by a flap of gingival tissue or by necrotic granulation tissue.

4. AETIOLOGY.

The literature reflects the indecision that exists among authors regarding the aetiology of dry socket. This reviewer considers that dry socket is the result, not of any one particular factor, but rather a number of factors acting conjointly.

It has been shown on page 11 that normal healing is dependent on organisation of the blood clot which forms in the socket. Hence, any factors which tend to prevent or delay organisation of the clot must be regarded as aetiological factors in dry socket.

The principal factors presented in the literature are:-

(a) Physical status of the patient: advancing age, the presence of chronic disease or nutritional deficiency may, either directly or indirectly, influence healing of an extraction wound (Gold, 1957).

This view is supported by Thoma (1963), Harang (1948) and Molt (1936).

(b) Pre-existing local factors: sclerosis of the socket wall is advanced by a number of authors as an important factor. The presence of a condensing osteitis, due to either low grade infection or trauma, results in the formation of sclerotic bone with a reduced blood supply to the socket (Osterloh, 1945). This prevents ready nourishment of the clot which subsequently may break down exposing the bony walls of the socket (Archer, 1961).

(c) Infiltration with vaso-constricting drugs: Archer (1961)

considers that use of too much or too high a concentration of vaso-constricting drugs may prevent blood clot formation in the socket by reducing vascularity and allowing subsequent ingress of bacteria-laden saliva. It has been suggested that this bacterial contamination may predispose to breakdown of the clot. This view is supported by Fry and Goldman (1958) and Elwell (1944) but Krogh (1937) and Osterloh (1945) found that the use of local anaesthetic solutions containing vaso-constrictor drugs was not a significant factor in the aetiology of dry socket.

(d) Mechanical loss of the blood clot: Disruption of the blood

clot by the patient is suggested by Thoma (1963), Gold (1957) and Elwell (1944) to be significant though Buchanan (1961) feels that it is of minor importance. The patient may disturb the clot by excessive rinsing, expectorating and even disturbing the clot with the tongue and fingers.

(e) Infection of the clot by oral organisms: Cash (1951)

believes that dry socket occurs when the blood clot becomes infected and is subsequently lost on the second or third day following extraction. This opinion is supported by Rud et al (1963) who, in their study, utilised local implantation of antibiotic cones in an attempt to prevent bacterial destruction of the clot.

Russel (1944) employs adhesive tin foil to cover fresh extraction wounds in order to prevent contamination of the clot by the saliva.



He claims that, by this method, the incidence of dry sockets in his patients was reduced from 14 per cent to 3 per cent. However, even this lower figure of 3 per cent compares unfavourably with other authors. Buchanan (1961) and Krogh (1937), in an unselected series, found an incidence of less than 1 per cent.

Many authors consider infection of the clot to be an important consideration. This is indicated by the numerous studies reported in which antibiotic drugs have been used both locally and systemically in an attempt to prevent infection of the clot. The results of these studies are reviewed on page 105.

(f) Trauma during extraction: this is the factor quoted by almost every author and the general consensus is that, although a complex of factors may act, trauma is probably the major cause of dry socket.

Gold (1957) considers that the traumatic removal of a tooth may result in crushing of the bony walls of the socket and an increase in the inflammatory reaction. This may cause reduced support of the clot and thus precipitate its breakdown.

Burnishing of the socket walls during a difficult extraction may cause necrosis of the remnants of the periodontal membrane and the superficial lamina dura. This devitalised tissue is thus unable to contribute to the support of the clot or the healing process and may even prove a liability to healing as the necrotic material must be subsequently removed by phagocytic cells. Alling and Kerr (1957)

have shown, in experimental animals, that burnishing of the socket walls produces a condition similar to the dry socket seen in man. Archer (1939) found in his series of dry sockets 56 per cent were predicated by traumatic removal.

It is the clinical experience of most dental surgeons that the predisposition to dry socket formation is greater following difficult extractions than when removal is relatively uncomplicated.

(g) Loss of the periodontal membrane: following luxation of a tooth, remnants of the periodontal membrane are left attached to the socket wall and, according to Shafer, Hine and Levy (1960), Huebsch (1958) and Hayward (1953), these remnants play an important role in the subsequent organisation of the blood clot.

If, during extraction of a tooth, the periodontal membrane is damaged by the applied forces, the remaining portions of the membrane are unable to contribute to the regenerative process. Huebsch (1958), and Alling and Kerr (1957) consider that this predisposes to formation of a dry socket.

However Radden (1959) and Mangos (1941), in their papers on the healing of extraction wounds, found that the periodontal membrane plays only a minor part in the healing process. Radden (1959) suggests that the degenerating periodontal membrane provides a nidus for bacterial growth which may lead to dry socket formation.

5. BACTERIOLOGY.

Infection of the blood clot, either primary or secondary, appears to be an important consideration in its breakdown. No single organism has been cited by authors as being responsible for clot degeneration and it appears that almost all micro-organisms found in the oral flora have been linked with dry socket.

Helmore (1958) feels that two types of bacteria, namely *Streptococcus haemolyticus* and *Bacillus fusiformis*, are always found in dry sockets, while Thoma (1963) considers that there is always a mixed infection with a predominance of spirochaetes and fusiform bacilli. This finding is supported by Holland (1948). Nevertheless, Archer (1939) found either "diplostreptococci or streptococci" in 80 per cent of dry sockets and thus considers these organisms to be important in the aetiology of dry socket.

All organisms mentioned are those which are able to lyse blood or are capable of growth in anaerobic and micro-aerophilic situations.

6. HISTOPATHOLOGY.

Cash (1951) describes the overall histopathologic appearance of a dry socket as "an osteitis of the lamina dura".

The socket wall is composed of necrotic bone beneath which is a layer of inflammatory cells and phagocytes. There is evidence of intense osteoclastic activity beneath the necrotic layer. Christopher (1942) notes that the osteoclasts "cut windows" in the socket wall so

that healing takes place by granulation from the base and sides of the socket. Weinman and Sicher (1955) consider that healing is delayed if the granulation tissue is exposed to the insults of the oral cavity. The reviewer, in the treatment of dry sockets, has noted that granulation in open sockets is often slow but, when the socket is protected with a soft bland dressing rapid granulation occurs. According to Faillo (1948) the necrotic socket wall may be shed as a sequestrum or may be completely resorbed by the osteoclasts. This has been observed by the reviewer who, on one occasion, has removed a "ring sequestrum" from a healing lower premolar socket. This sequestrum was approximately $\frac{1}{2}$ cm. in length and 1 m.m. thick.

7. PREVENTION.

(a) Surgical Measures. It is generally agreed that trauma is a major contributing factor in the formation of dry sockets. Surgical removal of a tooth reduces trauma and should be employed in any situation where the probability of a dry socket exists.

Harang (1948) feels that the surgical approach allows atraumatic removal of a tooth and permits careful debridement of the socket. Suturing of the muco-periosteal flap affords greater protection to the clot by reducing its exposed area. This also reduces the size of the clot and allows more rapid organisation (Cash, 1951; Radden, 1959). This method of treatment is considered by the reviewer to afford the most satisfactory method of preventing dry sockets.

Removal of sclerotic socket walls is advocated by Osterloh (1945) to provide increased nourishment to the blood clot. Huebsch (1958) suggests perforation of the socket wall and he has found increased rates of healing when this method has been used. However, according to Buchanan (1961) this procedure is hazardous as -

"it carries with it the dangers of introducing infection into the cancellous bone if strict asepsis is not observed, thermal necrosis with sequestration if burs are used, and excessive haemorrhage".

(b) Local Measures. Many authors have suggested that the incidence of dry sockets could be reduced by the insertion of certain agents into the sockets immediately following extraction. The object of this procedure is to maintain the blood clot and consequently most of these agents used have been anti-bacterial substances.

Millhon et al (1943) recommend reducing the size of the blood clot by the use of removable dressings and thus allowing more rapid organisation. This procedure involves the changing of dressings and it is possible that the clot may be disturbed when the dressings are removed. Guralnick (1946) suggests the use of gelatin sponge as a means of reducing the "dead space" in the socket. He maintains this absorbable haemostatic material is well tolerated by the tissues and does not delay healing or repair.

The use of cones of sulphanilamide or sulphathiazole, or a combination of the two drugs, has been reported by various writers, but with markedly differing results. Millhon et al (1943),

Archer (1939), Krogh (1948), Barab (1940), Davis et al (1955), Gwinn and Grim (1948) and Rud et al (1963), all record reduction in the incidence of dry sockets using sulphonamides. The majority of these authors report no adverse reaction to the drugs.

Local use of sulphonamides was found by Krashen (1940) to result in increased local irritation, increased post-operative pain and delayed healing. Versnel (1953), in an experimental study, found that implanted sulphonamides caused breakdown of the clot and retarded healing by a period of up to three weeks. However Frandsen and Pindborg (1961) found no significant difference in the rate of healing following implantation of sulphanilamide/sulphathiazole cones. Olech (1953), in a clinical study involving implanted sulphonamides and penicillin cones, found there was no justification for the use of chemotherapeutic agents in extraction wounds.

Scrivener and Schantz (1947) and Epstein and Kauffman (1951) both report good results following the use of penicillin cones in extraction sockets, while Holland and Tam (1954) report that the use of penicillin implants did not significantly reduce the incidence of dry socket. Versnel (1953) found that penicillin produced the least delay in healing and was rapidly absorbed from the socket. This finding is supported by Rud et al (1963).

Reduction in post-operative complications following the insertion of cones of erythromycin into extraction sockets is reported by Mourfield and Barron (1958).

Achromycin was used by Stickel and Clark (1961) and Quinley et al (1960) who both report a significant reduction in the incidence of dry sockets. However Quinley et al (1960) found some cases of foreign body reactions after several weeks. Verbie (1953) records the effectiveness of aureomycin in reducing the number of dry sockets in his study and he found no adverse reaction to the drug. Terramycin was used by Davis et al (1955) who found a reduction in dry sockets and no untoward reaction to the antibiotic, but Boyne and Kruger (1962), also using Terramycin, noted a delay in the rate of healing together with large particles of unresorbed tetracycline in the wound.

This author feels that the implantation of antibiotic substances in extraction wounds is not justified for the following reasons:

- (i) Local application of antibiotics may result in the emergence of resistant strains of bacteria.
- (ii) Allergic or toxic reactions are possible following absorption of the drug.
- (iii) Local irritation and foreign body reactions may occur with subsequent delay in healing.
- (iv) There is conflicting opinion in the literature regarding the efficacy of this prophylactic procedure.

Tam and Clark (1951) used chlorophyll ointment in lower third molar wounds in an endeavour to promote healing but in 33 1/3 per cent of cases breakdown of the site occurred. These authors concluded

that chlorophyl ointment was unsuitable for prevention of dry socket. However Shattan and Kutscher (1952) in a similar study found that chlorophyl ointment caused no difference in healing as compared to a control group. It may be significant that the latter investigators, unlike the former, did not suture the medicament into the wound.

(c) Systemic Measures. Thoma (1963) considers that antibiotics may be used parenterally in the prevention of dry socket and he recommends the use of broad spectrum antibiotics. Cash (1951) states that parenteral penicillin is also of value.

This author feels that the routine prophylactic use of antibiotics is not in the best interest of the patient for the following reasons:-

(i) There is a risk of sensitising the patient to the drug used so that it becomes dangerous to use the drug at any future occasion, even though the need may be great.

(ii) Hypersensitivity reaction to the drug is possible and such a reaction could prove fatal.

(iii) Resistant strains of micro-organisms may develop.

(iv) There exists an uncertainty as to the value of this method of preventing dry sockets.

The use of intra-muscular injection of thiamine chloride is suggested by Whitfield (1941) as a prophylactic measure and Molt (1936) found that vitamin C supplement resulted in satisfactory healing at extraction sites.

The value of anti-histamines in the reduction of dry sockets is reported by Silverman (1953) who claims a reduction of 60 per cent but as no statistical data are given, the significance of this report cannot be assessed.

8. DIAGNOSIS.

Diagnosis of a dry socket can be readily established following a clinical examination of the patient. The affected socket reveals either a bony cavity or may contain a necrotic blood clot in the process of breaking down. An instrument can be passed readily into the socket and, if the instrument touches the socket walls, the intensity of the pain is increased. The patient reports severe pain, often inability to sleep due to this pain, and may complain of a bad taste in the mouth.

A radiograph of the socket is of value in eliminating the possibility of a retained root fragment, the presence of fractured alveolus or a foreign body.

9. TREATMENT.

Treatment of an established dry socket is partly symptomatic and partly to carry out a gentle debridement of the socket and to establish a suitable environment for healing. The offending socket should be thoroughly irrigated with warm normal saline to remove any debris and necrotic blood clot. Then an obtundent dressing should

be inserted into the cavity. Gauze soaked in eugenol is suggested by Schram (1962), Gold (1957) and Archer (1961). The use of a surgical pack, composed of zinc oxide-eugenol paste, incorporated in gauze or cotton wool, is stated to be an effective method of treatment. This method has been used satisfactorily by the author who has found it of value in shallow sockets which would otherwise fail to retain a dressing. "Dentalone" (Parke Davis Co.), a saturated solution of chloretone in a liquid composed of oils of cloves, cassia and wintergreen, is preferred by Kemp (1952), Cash (1951) and Buchanan (1961). In the hands of this author this preparation has proved to be very efficient in the relief of even severe pain. It must be noted, however, that, although Dentalone is an efficacious medicament, its prolonged use may result in delayed healing (photograph, page 111). Once the pain is relieved, usually after three or four daily dressings a bland dressing should be substituted to act as a mechanical barrier to contamination from the mouth in order to keep the socket clean and encourage healing. Kruger (1959) considers that the socket should be dressed until the walls are covered with granulation tissue.

Helmore (1958) feels that systemic antibiotic therapy may be indicated in the early painful stages but this reviewer feels that; as dry socket is basically a localised osteitis, the use of these drugs is unwarranted.

Surgical interference with a dry socket is a hazardous procedure

EFFECT OF OVERMEDICATION
IN THE
TREATMENT OF DRY SOCKET



A 47 year old healthy female patient was referred for treatment of a lower right third molar socket.

The patient stated that $2\frac{1}{2}$ months previously she had an unerupted lower right third molar tooth removed and subsequently developed a "localised osteomyelitis". She was given antibiotic therapy at the time and local treatment consisting of second daily dressing of the socket with "Dentalone". The dressings were continued up until the time of referral.

On examination an open, unhealed socket in the lower right third molar region was seen. There was no sign of granulation tissue, the base of the socket being covered with necrotic tissue. The appearance was consistent with that of chemical irritation. No radiographic evidence of osteomyelitis was seen.

Treatment consisted of lightly packing the socket with a non-irritant dressing, Iodoform gauze, which was changed weekly. Complete healing of the socket took thirteen weeks.

as any attempt to curette or perforate the socket walls to initiate healing may well cause spread of infection and result in osteomyelitis (Helmore, 1958; Hayward, 1953; Buchanan, 1961).

"Biosone G.A. Dental Paste" (Biorex Laboratories Ltd.) is claimed by Fry and Goldman (1958) to rapidly relieve pain and stimulate healing. This reviewer has found Biosone paste to be unsatisfactory in the control of pain, most patients experiencing little or no relief. This observation is supported by Buchanan (1961).

An absorbable haemostatic bone wax, composed of carbowax, polyethylene glycol and oxidised cellulose, was employed by Douglas (1953). Having obtained local anaesthesia of the socket, Douglas dresses the cavity with a topical anaesthetic solution and then places the bone wax in position. He claims relief of pain on recovery of sensation in most cases. However, it is conceivable that this is due to the use of the topical anaesthetic liquid used prior to packing with bone wax.

In severe cases of dry socket where pain is prolonged or healing delayed, in the lower molar-premolar region, Holland (1948) suggests the use of an alcohol block injection to relieve pain. This reviewer feels that this is a radical approach to a problem which can be treated effectively by more conservative means.

Faillo (1948), Anderson (1957) and Hanson (1960) all report decrease in pain and shortened healing time following the use of proteolytic enzymes to cleanse dry sockets. However, according to Anderson (1957), pain is often not completely relieved following this

"physiologic cleansing" of the socket and he considers that some form of sedative dressing should be placed in the socket.

Anderson (1957), using trypsin, found a 39 per cent faster rate of healing when compared to the usual methods of treatment. Gufstaffson and Wallenius (1961), using trypsin alone, found there was no significant reduction in the duration of pain and some patients complained of a burning sensation following its application while others exhibited erosion of the tongue, lips and mucosa.

The painful dry socket is usually treated by the insertion of an obtundent dressing however the healing of the socket is slow and little else can be done other than relieving the subjective symptoms and establishing a suitable site for healing.

CELLULITIS

1. INTRODUCTION
2. ANATOMY OF THE FASCIAL SPACES
3. AETIOLOGY
4. BACTERIOLOGY
5. CLINICAL FEATURES
6. DIAGNOSIS
7. TREATMENT
8. LUDWIG'S ANGINA

1. INTRODUCTION

Cellulitis is defined by Shafer, Hine and Levy (1960) as "a diffuse inflammation of soft tissues which is not circumscribed or confined to one area but which, in contra-distinction to an abscess, tends to spread through tissue spaces and along fascial planes".

Characteristically, as facial cellulitis persists, the infection frequently tends to become localised with resultant facial abscess formation. When this occurs the abscess may become fluctuant and spontaneous drainage may occur. On the other hand, if localisation and suppuration do not occur a brawny inflammatory infiltration of the subcutaneous tissue may develop which is known as a phlegmon. According to Thoma (1963) and Thoma and Goldman (1960), a phlegmon has a tendency to be progressive, whereas an abscess is a localised collection of pus walled off from the surrounding tissues.

Rankow (1957) states that the advent of antibiotic therapy has revolutionised treatment of cellulitis. However, when abscess formation has occurred, drainage either spontaneous or surgical, is essential for resolution of infection (Marlette, 1954; Rankow, 1957).

2. ANATOMY OF THE FASCIAL SPACES

In his textbook of oral anatomy Sicher (1960) states that oral infection most frequently spreads by tissue continuity, the mode of

propagation of the infection being determined by the distribution of the loose connective tissue which lies between the fascial planes of denser connective tissue. The fascial planes are continuous layers of connective tissue which envelop, separate and support internal structures and these planes are bound together by the loose connective tissue. These areas of loose connective tissue form potential spaces between the layers of fascia. These potential spaces may, by destruction of the loose connective tissue by invading organisms, become actual spaces (Rankow, 1957; Bell, 1958; Shapiro et al, 1950; Waite, 1960; Eskin, 1958; Kruger, 1959).

All authors emphasise that an understanding of the anatomy of these potential fascial spaces is essential because of the propensity for their involvement by spread of oral infection. An excellent discussion of the fascial spaces of the head and neck is reported by Solnitzky (1954) and the following review of this topic is based on his paper.

(a) The Cervical Fasciae: the cervical fasciae are continuous layers of connective tissue consisting of superficial and deep layers.

(i) The superficial fascia is a continuous sheet extending from the head and neck into the region of the thorax, shoulders and axillae. In its deeper layers it contains the platysma muscle, while more superficially it contains the muscles of facial expression.

(ii) The deep cervical fasciae underlie the superficial fascia and are composed of superficial, middle and deep layers.

The superficial layer of deep fascia completely encircles the

neck and continues upward on the face where it sheaths the parotid gland and masseter muscle, attaches to the zygoma and continues superiorly as the outer layer of the temporal fascia.

The middle layer of deep cervical fascia is subdivided into three layers, a sternohyoid-omohyoid layer and a sternothyroid-thyrohyoid layer, each of which invests these respective muscles and the visceral layer (also known as the buccopharyngeal or pretracheal layer) which surrounds the thyroid gland, trachea and oesophagus.

The middle layer of deep cervical fascia, according to Bell (1958) and Shapiro et al (1950), is also known as the pretracheal fascia. A lateral extension of the pretracheal fascia fuses to the carotid neuro-vascular bundle, often referred to as the carotid sheath, which contains the carotid artery, the internal jugular vein and the vagus nerve.

The deepest layer of the deep cervical fascia is further subdivided as follows. The alar layer posteriorly and laterally joins with the middle cervical fascia in forming the carotid neurovascular bundle. The prevertebral fascia lies anterior to the bodies of the vertebrae while the scalenus fascia covers the scalenus muscles (Bell, 1958; Shapiro et al, 1950; Solnitzky, 1954).

(b) The Masticator Space: this space includes the subperiosteal region of the mandible and a fascial sheath containing the ramus of the mandible and the muscles of mastication. This space is formed by the superficial and middle fascial layers of the deep cervical fascia. The superficial layer passes upwards external to the

masseter muscle but internal to the parotid gland, Stenson's duct, the seventh nerve and the superficial temporal artery and vein.

The fascia passes upward over the zygomatic bone, attaches to it, and then continues upward over the temporal muscle, affixing itself to the periosteum of the temporal bone. The middle layer of deep cervical fascia passes medially to the ramus, enclosing the internal and external pterygoid muscles and attaches to the base of the temporal bone. The two layers fuse anteriorly along the anterior border of the masseter and temporal muscles and posteriorly along the posterior border of the ramus and temporal muscle.

The masticator space is bounded inferiorly by its firm periosteal attachment, laterally by the masseter muscle, medially by the medial border of the internal pterygoid muscle, anteriorly by the anterior borders of the masseter, pterygoid and temporal muscles and the anterior border of the ramus. Superiorly the space is continuous with the superficial and deep temporal spaces (Bell, 1958; Solnitzky, 1954; Waite, 1960).

The masticator space is continuous with the space of the body of the mandible which is a muscular fascial space extending from the symphysis to the third molar region. This space is formed by the continuation of the superficial and middle layers of deep cervical fascia with the free muco-periosteum overlying the alveolar portion of the mandible (Shafer, Hine and Levy, 1960; Shapiro et al, 1950; Bell, 1958; Waite, 1960).

A sub-masseteric space is described by Thoma (1963), Brash (1953), Bransbury-Zachary (1948) and Shafer, Hine and Levy (1960). This space is situated between the masseter muscle and the lateral surface of the ramus of the mandible. The masseter, according to these authors, at its attachment to the ramus is divided into three heads of insertion with a resultant narrow space lying between the muscular heads. The posterior boundary of the sub-masseteric space is the parotid gland and anteriorly it adjoins the retro-molar fossa.

Infections of the masticator space are usually due to extension of infection from the mandibular molar teeth, sepsis following injection into the region and trauma to the mandible. However infection of the masticator space may take place by direct extension from the space of the body of the mandible, sub-masseteric space, parotid space or lateral pharyngeal space (Solnitzky, 1954).

(c) The Temporal Spaces: these two fascial spaces are in relation to the temporal muscle and are named according to their anatomical situation.

The superficial temporal space lies between the temporal muscle medially and the superficial temporal aponeurosis laterally. The deep temporal space lies medial to the temporal muscle and is bounded by the temporal bone. The temporal spaces communicate with the masticator space and, below the level of the zygomatic arch, the temporal spaces also communicate directly with the infratemporal and pterygo-palatine fossae (Bell, 1958; Solnitzky, 1954).

Infection of the temporal spaces are usually secondary to

primary involvement of the masticator, infra-temporal and pterygo-palatine spaces.

(d) The Submandibular Spaces: there are three submandibular spaces which are in anatomic continuity with each other as well as with the corresponding space of the other side.

(i) The Submaxillary space is described by Shapiro et al (1950) as that region of the neck containing the submaxillary gland. This space lies medial to the mandible and below the posterior portion of the mylohyoid muscle. The hyoglossus and digastric muscles form the medial boundary and laterally the space is demarcated by the superficial fascia and skin.

An infection of this space, as well as spreading to involve the other submandibular spaces, may extend to the lateral pharyngeal space, the carotid neurovascular bundle, the cranial fossa or even the mediastinum (Shafer, Hine and Levy, 1960).

(ii) The sublingual space is that area bounded superiorly by the mucosa of the floor of the mouth, inferiorly by the mylohyoid muscle, anteriorly and laterally by the body of the mandible, posteriorly by the hyoid bone and medially by the median raphe (Shapiro et al, 1950).

(ii) The submental space is described by Shafer, Hine and Levy (1960) as extending from the anterior border of the submaxillary space to the midline and being limited in depth by the mylohyoid muscle.

(e) The Parapharyngeal Space: this is divided into:-

(i) The Lateral Pharyngeal Space (pharyngo-maxillary space) which is described by Waite (1960) and Bell (1958) as a "visceral vascular space" rather than a muscular fascial space. The difference being that the fascia of a "visceral vascular space" covers viscera instead of muscle and permits more rapid dissemination of infection. The lateral pharyngeal space is bounded medially by the superior constrictor muscle of the pharynx, anteriorly by the fascia covering the masticator space, posterolaterally by the parotid space and posteriorly by the styloid muscle and carotid neurovascular bundle. Superiorly the lateral pharyngeal space is limited by the base of the skull and is divided from the neck by the capsule of the submaxillary gland. Inferiorly the lateral pharyngeal space communicates with the mediastinum by way of the prevertebral fascia (Bell, 1958; Shapiro et al, 1950).

(ii) The Retropharyngeal Space lies between the posterior wall of the pharynx and the prevertebral fascia, the pharynx forming the anterior boundary of the space, the posterior limit being established by the prevertebral fascia. Since the prevertebral fascia extends inferiorly to the posterior mediastinum, direct extension of infection may occur by this route (Shafer, Hine and Levy, 1960; Shapiro et al, 1950).

The parapharyngeal space may be involved by the spread of infection from the masticator space in infections of dental origin. The complications of involvement of this region are particularly

serious especially if infection of the retropharyngeal space occurs. These complications include respiratory obstruction, thrombosis of the internal jugular vein and erosion of the internal carotid artery (Solnitzky, 1954).

(f) The Parotid Space: the parotid space is a compartment formed by splitting of the superficial layer of the deep cervical fascia. It contains the parotid gland as well as the intra and extra-glandular parotid lymph nodes. The internal layer of the fibrous capsule is thin and often incomplete superiorly where direct communication with the lateral pharyngeal space may occur (Solnitzky, 1954).

Waite (1960) and Solnitzky (1954) state that this space is not often involved by infections of dental origin.

(g) The Pterygopalatine and Infratemporal Spaces: the pterygopalatine (or pterygomaxillary) space lies posterior to the maxillary sinus, below the apex of the orbit, lateral to the muscular plate of the pterygoid process of the sphenoid bone and deep to the temporomandibular joint. The pterygopalatine space communicates with the infratemporal space through the pterygomaxillary fissure (Solnitzky, 1954).

The infratemporal space lies posterior to the ramus of the mandible below the level of the zygomatic arch medially, it is bounded by the medial pterygoid plate and the lateral wall of the pharynx and the pharyngo-tympanic tube covered by the tensor palati muscle.

Posteriorly, the space is limited by the parotid gland and the space extends into the cheek superficial to the buccinator muscle.

Superiorly, the roof of the infratemporal fossa is formed by the infratemporal surface of the greater wing of the pphenoid. Inferiorly, the infratemporal space is continuous with the region deep to the body of the mandible which, above the mylohyoid line forms part of the wall of the mouth and, below the mylohyoid line, constitutes part of the submandibular region (Solnitzky, 1954; Shafer, Hine and Levy, 1960).

Infections of both these spaces are considered to be rare and when infection does occur, is most often due to non-sterile injection technique or spread of infection from maxillary molar teeth.

All authors emphasise that the aforementioned spaces of the head and neck are contiguous, thus infection may pass readily from one space to another.

3. AETIOLOGY

When micro-organisms invade tissue the infection may remain localised if the body defence mechanisms are capable of limiting the infection. If the bacterial infection is overwhelming, the bacteria are highly virulent or the patient has lowered resistance, the infection may progress through the surrounding tissues to areas remote from the site of entry.

Cellulitis of the face and neck may result from:-

- (a) extension of dental infection.
- (b) bacterial contamination during oral surgical procedures.
- (c) injection with a contaminated needle.
- (d) injection of a contaminated solution.
- (e) injection through an infected area

(Herrell and Nichols, 1944; Thoma, 1963; Shapiro et al, 1950; Bell, 1958; Waite, 1960). Spread of infection in osteomyelitis is considered by Shafer, Hine and Levy (1960), to be a possible aetiological factor in cellulitis.

From the foregoing discussion it is apparent that infection may pass readily from one fascial space to another thus involving tissues perhaps far distant to the site of original infection (Bell, 1958; Shapiro et al, 1950; Archer, 1961; Sicher, 1960).

4. BACTERIOLOGY

Cellulitis is considered by Shafer, Hine and Levy (1960), to be caused by micro-organisms that produce significant amounts of hyaluronidase and fibrolyns. These substances act to breakdown or dissolve, respectively, hyaluronic acid, an intercellular cementing substance, and fibrin, a product of the inflammatory reaction.

It is generally agreed that cellulitis results from infection by a wide variety of organisms. Streptococci, staphylococci, Gram-negative bacilli, spirochaetes and anaerobic fusiform bacilli

all being cited in the literature as causal organisms. However Tolhurst, Buckle and Williams (1963), Thoma and Goldman (1960), Thoma (1963) and Shafer, Hine and Levy (1960) consider the organisms most frequently responsible for cellulitis of the face and neck are the streptococci (Thoma (1963) mentions specifically the alpha and beta haemolytic streptococci and the non-haemolytic gamma streptococci). Staphylococcus aureus and albus are considered by these authors and Rankow (1957) to also be common causative organisms. Rubelman et al (1961) report a case of cellulitis following tooth extraction in which the infecting organism was Salmonella choleraesuis, an organism normally found in the intestine. The author states that this is the first reported case of cellulitis resulting from infection by this organism.

5. CLINICAL FEATURES

The patient with cellulitis usually experiences general malaise, anorexia and has an elevated temperature, which Berlove (1959) states may be as high as 106° F. In severe cases the pulse is feeble, rapid and irregular, and an increase in respiratory rate is common.

Marked oedema and inflammation of the involved soft tissues occurs, and the swelling, which has a firm brawny appearance, is very tender to palpation. The skin or mucosa overlying the swelling is erythematous and is sometimes even purplish in colour if the superficial tissue spaces are involved. Burch and Morris (1962) point out that, if the infection involves the deeper tissue planes,

the overlying tissues may be of normal appearance. Movement of the mandible and the head is often painful and regional lymphadenitis is a common finding. Archer (1961) notes that the patient may experience dysphagia and difficulty in breathing. In the event of the latter complication developing, the patient must be carefully observed to detect respiratory obstruction due to the swelling and in some cases a tracheotomy may become necessary to maintain the airway (Rankow, 1957; Thoma, 1963).

Kruger (1959) states that laboratory investigations carried out at the acute stage of cellulitis may show an increased white blood cell level which, in severe cases, may be accompanied by an altered differential count. Also an increased blood sedimentation rate may be noted which is indicative, among other things, of an infectious process.

Thoma (1963) considers that the progress of the condition depends on the causal organism, its virulence, the resistance of the patient and the region involved. Frequently resolution may occur due to the natural defences of the patient or the use of specific antibiotic therapy. In other cases Kruger (1959) and Shafer, Hine and Levy (1960) state that, as the infection persists, localisation occurs with abscess formation. An abscess, in some cases, may develop very slowly with relatively mild associated symptoms. Such cases may take ten days or more for localisation to occur while, in other cases, development is very rapid and the abscess is ready for drainage a day or two after onset of the infection (Thoma, 1963).

When abscess formation does take place a point of fluctuance develops in the swelling. The overlying skin or mucosa becomes reddened and shiny due to the proximity of the abscess to the surface (Thoma, 1963). When fluctuation occurs spontaneous drainage may result if surgical incision is not performed. Eskin (1958) records that marked cicatrisation of the tissues may result from spontaneous drainage and emphasises the importance of prompt surgical intervention once abscess formation becomes apparent.

If localisation of cellulitis does not occur, a brawny inflammatory infiltration of the tissues may develop. This spread of infection is termed a phlegmon (Thoma, 1963; Thoma and Goldman, 1960). The skin is bluish-red in colour due to tissue cyanosis and, in the fascial spaces, there is an accumulation of a brownish, foul smelling exudate. Thoma and Goldman (1960) state that the course of a phlegmon varies with the infecting organism.

The proximity of the fascial spaces to the skull and other fascial planes makes possible the spread of infection to other sites with serious consequences. Such spread may occur to the mediastinum via the pre-vertebral fascia, to the carotid sheath or to the cranial vault. The resulting infection of these regions may develop rapidly and may even prove fatal (Thoma and Goldman, 1960; Shafer, Hine and Levy, 1960).

6. DIAGNOSIS

Diagnosis of cellulitis can be made by considering the patient's history and by a careful clinical examination. Radiographs should be taken to eliminate the possibility of suppurative osteomyelitis although, in the early stages of acute suppurative osteomyelitis, no radiographic change can be detected (Durbeck, 1946).

A haematological examination may reveal a leukocytosis and, in severe cases, may also show an alteration in the differential white cell count. Kruger (1959) reports that often in cellulitis an increased blood sedimentation rate may be observed and this is indicative of infection, among other conditions. Thoma (1963) and Kruger (1959) consider that in all severe cases where the patient shows symptoms of toxæmia, a blood culture should be carried out. These authors emphasise that blood cultures should be made prior to administration of antibiotics.

7. TREATMENT.

The advent of antibiotics and allied drugs has revolutionised management of infections and, if prompt treatment is given, the infection can often be aborted before abscess formation occurs (Rankow, 1957).

Identification of the infecting organism prior to selection of antibiotics is desirable. However, as time is an important factor in the treatment of cellulitis, treatment is frequently empirical. The choice of chemotherapeutic agents must perforce be

based on a knowledge of the likely infecting organism and its anticipated antibiotic sensitivity.

When material is available for bacteriological examination, sensitivity tests should be carried out so that modification can be made to the selection of drugs later, if necessary, (Kramer, 1956; Tolhurst, Buckle and Williams, 1963).

Shapiro et al, writing in 1950, state that oral infections are most often caused by penicillin-sensitive organisms and, as empirical treatment is necessary in the early stages of cellulitis, penicillin is the drug of choice. This reviewer has found penicillin to be of value in the treatment of post-operative cellulitis. Archer (1961) advocates the initial use of penicillin for the treatment of cellulitis but suggests that, if no response to treatment is noted within forty eight hours, a broad spectrum antibiotic should be substituted. Kruger (1959) suggests that, in severe or fulminating infections, a blood culture should be performed which may indicate a specific drug to use in therapy. Shapiro et al (1950) consider that the administration of the antibiotic should be continued until the patient has been afebrile for forty eight hours.

General supportive treatment of the patient is essential. Care must be taken to avoid dehydration and fluids should be forced if necessary (Archer, 1961; Waite, 1960; Rankow, 1957). If the patient is unable to swallow consideration must be given to intravenous administration of fluids to achieve an adequate fluid balance (Kruger, 1959).

As a result of pain, trismus and dysphagia the patient is frequently unable to take adequate nourishment and a soft or liquid diet should be prescribed by the oral surgeon. Archer (1961) considers that vitamin supplements should be given as pyrexia increases the patient's vitamin requirements.

It is generally agreed that moderate heat is of value in aiding localisation of the infectious process and reduction of pain and discomfort. In the early stages of cellulitis intra-oral heat in the form of hot saline mouthwashes should be used. If abscess formation occurs and fluctuation becomes apparent, hot moist dressings or poultices are indicated to hasten fluctuation (Kruger, 1959; Shapiro et al, 1950; Rankow, 1957; Archer, 1961).

Respiratory obstruction, as a result of the inflammatory process, may result in asphyxia. This may have an insidious onset and once respiratory difficulty is noticed tracheotomy should be considered (Rankow, 1957).

Once localisation and fluctuation have occurred surgical drainage should be carried out. The optimum time for drainage may be difficult to determine. A point of fluctuation is characterised by the development of a shiny, erythematous area which, on palpation, is slightly softer than the surrounding tissues. Finger pressure applied to one side of the softened area conveys a fluid movement which may be felt by a finger placed on the other side of the area. When the presence of pus is diagnosed surgical incision and drainage of the abscess should be carried out.

Hilton's method of drainage is the procedure of choice. An incision is made just through either the skin or the mucosa overlying the point of fluctuation. Through the incision a pair of closed artery forceps are introduced into the wound and forced through the soft tissues until an abscess cavity is encountered. The forceps are then opened in several directions within the cavity to ensure that adequate drainage of the involved space occurs. A rubber drain should then be introduced into the abscess cavity to ensure that the drainage established is maintained (Kruger, 1959; Thoma, 1963).

If incision is made before suppuration has occurred, a drain should be inserted deep into the involved tissues. Rankow (1957) considers that following this form of treatment drainage occurs within one or two days.

While the oral surgeon should generally await localisation and fluctuation to occur before instituting surgical treatment, Archer (1961) states that, if localisation does not occur within five to seven days, despite antibiotic therapy, surgical exploration of the involved tissues should be carried out. Archer (1961) considers that delay in surgical treatment may result in further dissemination of the infection which may have serious results.

Once drainage is established a bacteriological culture should be made to determine the antibiotic sensitivity of the organisms so that a change may be made in the antibiotic therapy if necessary.

8. LUDWIG'S ANGINA.

Ludwig's angina is a severe, extensive cellulitis which characteristically involves all the submandibular spaces and shows a tendency to involve the cervical tissues (Sicher, 1960; Dorner and Morgan, 1945; Shafer, Hine and Levy, 1960). It is a rapidly progressive infection and, if unchecked may prove fatal.

Extension of infection from a mandibular molar tooth is considered to be the most common aetiological factor and Kruger (1959) notes that the condition is usually observed following tooth extraction. However, Ludwig's angina may result from extension of infection from other fascial planes, pericoronar infection, osteomyelitis or infection as a result of operative procedures.

The patient shows all the symptoms of acute cellulitis and has a brawny, boardlike swelling of the floor of the mouth and the submandibular tissues. There is no evidence of localisation of the infection and, as a result of the swelling, the tongue is elevated. Because of the marked oedema, there is considerable danger of mechanical interference with the patency of the airway. The oral surgeon must be constantly alert to the possibility of this complication arising and, if respiratory obstruction occurs, a tracheotomy may become necessary to prevent asphyxiation (Rankow, 1957; Kruger, 1959; Thoma and Goldman, 1960).

No specific organism has been associated with Ludwig's angina, a mixed infection usually being present (Shafer, Hine and Levy, 1960; Thoma and Goldman, 1960).

The patient with Ludwig's angina is seriously ill and should be hospitalised. Initially, treatment consists of intensive antibiotic therapy and general supportive care. It is of particular importance to note that, due to the position of the tongue, the patient is frequently unable to swallow and is thus incapable of taking nutrients. In such cases intravenous fluids should be administered. If improvement does not occur readily with antibiotic therapy, surgical intervention is essential to release the tension in the tissues and to provide drainage (Kruger, 1959).

Thoma and Goldman (1960) consider that, in these days of antibiotic therapy, prompt definitive treatment leads to a favourable prognosis.

With the advent of antibiotics true Ludwig's angina has become a rare post-operative complication. However, once the condition is established, the same surgical principles of the pre-antibiotic era, that is surgical incision to release tissue tension and to provide drainage, still apply in the treatment of this infection.

SUPPURATIVE OSTEOMYELITIS

1. INTRODUCTION
2. AETIOLOGY
3. CLINICAL FEATURES
4. BACTERIOLOGY
5. HISTOPATHOLOGY
6. RADIOGRAPHIC APPEARANCE
7. DIAGNOSIS
8. TREATMENT.

1. INTRODUCTION

Osteomyelitis is defined by Thoma (1963) as an inflammation of bone generally as a result of pyogenic organisms and involving the cancellous bone, the bone marrow, the cortex and the periosteum. On the other hand, Weinman and Sicher (1955) and Fleming (1954) consider that the terminology should be applied according to the site of the inflammation: osteomyelitis - inflammation of the soft tissues within the bone; periostitis - inflammation of the periosteum. When all the elements of the bone are involved in the inflammatory reaction these authors consider the term osteitis should be applied. Cappell (1962) points out that, although these different terms are applied to inflammation of bone according to the site, they should not be taken as indicating separate affections as one may readily lead to the other and frequently they are present simultaneously. We find in the literature that the term osteomyelitis as defined by Thoma (1963) is almost universally accepted and thus it will be used in this review.

2. AETIOLOGY.

Osteomyelitis of the jaws, according to Thoma and Goldman (1960), is most commonly due to odontogenic infection. Considering the frequency of dental sepsis, the incidence of osteomyelitis is low. Disruption of the natural defence barriers walling off such infections

is considered by these authors to be an important aetiological factor in post-operative infection of the bone.

Osteomyelitis following an oral surgical procedure may occur as a result of injudicious treatment of a suppurative lesion or of a non-sterile technique which permits pathogenic organisms to gain access to previously unaffected tissue. Rushton and Cooke (1963) consider that the removal of an unerupted or partly erupted tooth in the presence of an acute pericoronal infection may result in osteomyelitis. Surgical interference with an acutely inflamed socket is considered by Helmore (1958) and Hayward (1953) to be hazardous, as any attempt to curette or perforate the socket walls in an attempt to promote healing may cause spread of the infection and lead to osteomyelitis. Injection of local anaesthetic solution into infected tissue may result in periostitis which, in turn may extend to the bone. Kruger (1959) warns that, without strict asepsis, intra-osseous injection may lead to infection of bone.

Thoma and Goldman (1960) report that many authors consider extraction of an acutely infected tooth may lead to osteomyelitis. On the other hand Thoma and Goldman (1960) and Shapiro et al (1950) consider that often the simplest surgical method of establishing drainage in such cases is to extract the tooth. This can generally be accomplished without undue trauma as the tooth is, as a rule, loose as a result of partial destruction of the periodontal fibres. These authors consider that osteomyelitis following extraction is generally

due to delayed extraction, application of external heat and failure to maintain the drainage established.

Poor surgical technique in the extraction of teeth may produce non-vital fragments of alveolar bone which may form small localised areas of osteomyelitis (Thoma and Goldman, 1960; Clark, 1955). Such areas present little difficulty in treatment but the possibility of extension of the infection to the adjacent alveolar bone cannot be overlooked.

It is generally accepted that osteomyelitis occurs as a result of infection of the bone by pathogenic micro-organisms. Some authors also point out that there are often predisposing factors that are important in the aetiology of the condition (Thoma and Goldman, 1960; Thoma, 1963; Kruger, 1959; Archer, 1961). Patients with systemic disease, those recovering from infectious conditions or those with low nutritional status are considered to be more prone to infection. An example of this is given by Steg (1963) who reports a chronic case of osteomyelitis in an alcoholic patient.

3. CLINICAL FEATURES

Acute, sub-acute and chronic suppurative osteomyelitis are described in the literature. However Thoma and Goldman (1960) consider that there is no clear demarcation between these clinical divisions.

Osteomyelitis of the maxilla is considered to be uncommon and,

when it does occur, the condition is usually well localised. As a result of the bony architecture of the mandible and the nature of its vascular supply, the lower jaw is more commonly involved by osteomyelitis and large sequestra may result (Rushton and Cooke, 1963; Bernier, 1959; Kruger, 1959; Thoma and Goldman, 1959).

(a) Acute Suppurative Osteomyelitis: the patient initially experiences severe pain which is spasmodic at first but is replaced by a constant dull, boring ache. The patient's temperature is typically elevated, a one to three degree rise being usual (Durbeck, 1946) and regional lymphadenitis is present. Archer (1961) states that a differential blood count taken at this stage frequently shows a high leukocyte level which is indicative of the acute infection. The patient may also appear toxic and experience general malaise.

Erythema and swelling of the overlying skin or mucosa does not occur until the periosteum is involved by the infection. Trismus may be present and, in cases of mandibular osteomyelitis, anaesthesia or paraesthesia of the lip may occur due to involvement of the inferior alveolar nerve (Thoma and Goldman, 1960).

The teeth involved in the infected area may loosen and become tender to percussion and as a result the patient often has difficulty in mastication. As the infection progresses the gingival tissues become deep red and oedematous because of the periostitis and often pus may exude from around the necks of the teeth.

The swelling may become fluctuant and spontaneous drainage,

either intra-orally or extra-orally, may occur. Shafer, Hine and Levy (1960) state that, unless acute suppurative osteomyelitis is properly treated, facial cellulitis may develop due to spread of the infection in the soft tissues.

Thoma and Goldman (1960) state that once drainage occurs the acute phase rapidly subsides and a chronic state is established. This chronic state is maintained until sequestration of the necrotic bone has occurred and, if during this period, the draining sinuses become blocked, sub-acute or acute exacerbations may occur.

(b) Chronic Suppurative Osteomyelitis: Shafer, Hine and Levy (1960) state that chronic suppurative osteomyelitis may develop after the acute phase of the condition has subsided or occasionally may occur without a preceding acute phase.

Generally the symptoms of chronic suppurative osteomyelitis are milder than the acute form. Pain is less severe, the temperature is only slightly elevated and a moderate leukocytosis occurs. Those teeth which were loose during the acute phase may become firm again and symptoms of inferior alveolar nerve involvement may disappear.

Periodically, if there is interference with drainage, acute and sub-acute exacerbations may occur and these may present all the clinical features of acute suppurative osteomyelitis.

It is recognised by most authors that pathologic fracture of the mandible may result from osteomyelitis. As a preventive measure,

when the infection is extensive, immobilisation or support of the jaws must be considered.

4. BACTERIOLOGY.

The most common infecting micro-organism in suppurative osteomyelitis is *Staphylococcus aureus* (according to Shafer, Hine and Levy, 1960; Archer, 1961; Durbeck, 1946; Kruger, 1959; Weinmann and Sicher, 1955 and Lucas and Kramer 1959). These authors also consider that osteomyelitis often results from infection by *Staphylococcus albus*, various types of streptococci and, in some instances, mixed organisms. Cappell (1962) and Thoma and Goldman (1960) consider that the most common infections in osteomyelitis are due to *Staphylococcus aureus* and *Streptococcus haemolyticus*.

When drainage from the lesion occurs, either spontaneously or by incision, cultures should be made to establish the identity of the infecting organism and its antibiotic sensitivity (Arnott, 1962; Tolhurst, Buckle and Williams, 1963; Cook, 1963; Bernier, 1959; Thoma and Goldman, 1960).

5. HISTOPATHOLOGY

The infection most commonly commences in the cancellous bone and spreads through the Haversian system to involve the periosteum. However, following periostitis, infection from the periosteum may gain access to the medullary spaces by the reverse route.

The micro-organisms proliferate in the cancellous spaces and cause a local inflammatory reaction. The medullary spaces become filled with inflammatory exudate which is chiefly composed of polymorphonuclear leukocytes and occasionally lymphocytes and plasma cells. Later pus is formed and the osteoblasts bordering the adjoining bony trabeculae are displaced and, depending on the duration of the process, the trabeculae lose their viability and undergo slow resorption (Shafer, Hine and Levy, 1960).

The inflammatory reaction causes thrombosis of the nutrient vessels which results in necrosis of the bone normally supplied by them. The necrotic bone becomes separated from its neighbouring vital bone by osteoclastic action. The sequestra thus formed may be exfoliated or resorbed by continued osteoclastic action (Rushton and Cooke, 1963).

6. RADIOGRAPHIC APPEARANCE

Durbeck (1946) states that no change in bony structure is seen for seven to ten days. After this period the trabeculae which are normally distinct lose their sharp outline and appear "fuzzy" due to diffuse lytic changes occurring in the bone (Shafer, Hine and Levy, 1960). As the condition progresses the radiographic appearance of the bone takes on a diffuse moth-eaten pattern with the development of radiolucent areas.

Thoma and Goldman (1960), Durbeck (1946) and Kruger (1959)

report that as sequestra form they appear more radio-opaque than the surrounding bone. These authors consider that the necrotic bone becomes more highly calcified with a resultant increase in radio-density. Once chronicity is established radiographs should be taken at regular intervals to observe the extent of the lesion and to detect separation of sequestra.

Durbeck (1946) considers that the radiographic appearance is similar to that seen in malignant disease of bone and emphasises the need for accurate differential diagnosis. One diagnostic feature is that sequestration is uncommon in malignancy except when coincidental infection occurs or following radiotherapy.

7. DIAGNOSIS

Diagnosis of acute suppurative osteomyelitis is made following review of the patient's history and a thorough clinical examination. Radiographs taken in the first week of acute osteomyelitis are non-contributory as discussed previously. Durbeck (1946) states that a blood count taken in the acute stages of the condition will reveal 15,000-20,000 leukocytes per cu.mm. with 85-95 per cent polymorphonuclear neutrophils. Blood culture may or may not be positive and Durbeck (1946) considers that repeated positive cultures are indicative of an unfavourable prognosis.

Chronic suppurative osteomyelitis may be diagnosed by considering the history and the clinical appearance of the patient together with

radiographs of the jaws. Durbeck (1946) considers that some radiographic similarity exists between bone loss in osteomyelitis and in malignant disease and notes that care must be taken in differential diagnosis.

8, TREATMENT

(a) Systemic Treatment. Tolhurst, Buckle and Williams (1963) and Cahn (1955) report that, if antibiotic treatment is given sufficiently early in acute suppurative osteomyelitis, it is possible to abort the infection without the need for surgical intervention. The former authors consider that one million units of crystalline penicillin given six hourly is an effective method of treatment but point out that many penicillin resistant strains of the common infecting organism, *Staphylococcus aureus*, have developed.

All authors report the use of antibiotics to control the infection and to prevent complications following surgical intervention to establish drainage or to remove necrotic tissue. When drainage has occurred sensitivity tests should be carried out to determine the appropriate antibiotic. With closed lesions therapy must perforce be empirical and this reviewer considers that, as penicillin resistant infecting organisms have been reported by some authors, thought should be given to the use of broad spectrum antibiotics.

Arnott (1962) and Thoma and Goldman (1960) recommend that the patient should be given a nutritious, high-calorie diet together with vitamin supplements. If the extent of the infection makes pathologic

fracture a possibility then an appropriate soft diet should be prescribed. Archer (1961) considers that, during the acute phase of suppurative osteomyelitis, the patient should be hospitalised. He also recommends that an adequate fluid intake and output should be maintained and suggests that intra-venous fluids may be required.

(b) Local Treatment: when acute suppurative osteomyelitis is not controlled by antibiotic therapy alone surgical intervention is frequently necessary. Where fluctuance of the swelling occurs incision and drainage should be carried out. This results in rapid relief of the patient's symptoms. Drainage must be maintained until sequestration of the necrotic bone occurs (Archer, 1961; Moody, 1958; Thoma, 1963). Thoma (1963) states that incision should be performed as soon as fluctuation is evident but prefers to carry out surgery at least three days following commencement of antibiotic therapy. This author states that drainage of the swelling lessens the amount of absorption of toxic products and prevents spread of the infection in the bone, allowing localisation to occur.

Once the acute phase of the condition has subsided, a chronic state is established. While there is unanimity regarding treatment of the acute stage of osteomyelitis, there are two diverse schools of thought regarding treatment of the chronic state.

(i) Conservative treatment: conservative treatment is recommended by a number of writers. This entails maintaining drainage from the infected area, supportive antibiotic therapy to

control the infection and prevent complications and the surgical removal of sequestra when clear demarcation from the surrounding bone has occurred (Shafer, Hine and Levy, 1960; Kayne, 1941; Archer, 1961; Bernier, 1959; Arnett, 1962; Cook, 1963). Thoma (1963) and Stuteville (1950) consider this approach to be of merit where limited necrosis has occurred but feel that, in extensive lesions, the resultant defect produced by sequestrectomy predisposes to further infection. These authors advocate a more radical approach in such cases.

(ii) Radical treatment: this form of treatment is favoured by Durbeck (1946), Stuteville (1950), Moody (1958), and Thoma (1963). These authors recommend wide exposure of the infected bone, removal of sequestra, curettage of the granulation tissue and saucerisation of the resultant bone defect to permit obliteration of the cavity. Antibiotic therapy is continued for two to three weeks post-operatively and most authors report that healing by primary intention usually occurs. If primary closure cannot be effected Thoma (1963) suggests the use of petrolatum gauze to pack the cavity to allow granulation from the base of the site to occur.

Stuteville (1950) notes that, when the entire thickness of the mandible is involved or where sufficient bone is removed to produce deformity, the necrotic bone and granulation tissue can be removed and a metal plate applied to the bone fragments to maintain their relation and to support bone chips from the iliac crest.