POST-OPERATIVE TREATMENT

AND COMPLICATIONS IN ORAL SURGERY

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This critical review of the literature concerning post-operative treatment and complications in oral surgery is submitted in support of candidature for the degree of Master of Dental Surgery.

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"Healing is a matter of time,
but it is sometimes also a matter of opportunity".

Hippocrates.
Precepts, Chapter I.

INDEX

	page
INTRODUCTION	1
HEALING OF ORAL WOUNDS	4
PRINCIPLES OF NORMAL POST-OPERATIVE TREATMENT	13
POST-OPERATIVE HAEMORRHAGE	58
DRY SOCKET	94
CELLULITIS	114
SUPPURATIVE OSTEOMYELITIS	134
CERVICO-FACIAL ACTINOMYCOSIS	146
CHEMOTHERAPY WITH ANTIBIOTICS AND ALLIED DRUGS	160
SOFT TISSUE REACTION TO NECROTIC ALVEOLUS	175
ORO-ANTRAL FISTULA	181
NERVE INVOLVEMENT	191
OSTEO-RADIO NECROSIS	202
POST-OPERATIVE FRACTURE	215
BIBLIOGRAPHY	222

INTRODUCTION.

Post-operative treatment is an essential part of the surgical procedure to maintain continuity of treatment and to detect any deviation from the normal post-operative course and institute the appropriate treatment. This phase of treatment is as important as the actual surgery itself. Kemp (1952) states that post-operative treatment in relation to oral surgical procedure should be simple and uncomplicated, provided that the operation is well planned and skilfully performed. In order to place this review in the proper perspective one must first consider the entire surgical procedure. Holland (1948) stresses that a balance must exist between the pre-operative planning and preparation, the actual surgical operation and the post-operative treatment.

The pre-operative planning should include the following steps:-

(a) A careful evaluation of the patient. This involves the taking of a thorough case history, both medical and dental, to evaluate the patient's physical status.

(b) A clinical examination of the oral cavity and associated tissues to enable the operator to assess the problem at hand and allow him to decide if laboratory investigations need to be employed.

(c) A comprehensive radiographic survey of the region involved. This is essential to the planning of any operation and enables the oral surgeon to assess the extent of a bony lesion or, in the case of a soft tissue lesion, if any bony involvement has occurred. Radiographs may disclose unsuspected abnormalities and may give an indication of

the degree of difficulty that might be encountered in the ensuing surgery (Howe, 1960).

It is at this stage that laboratory tests indicated during ~~the first~~ the first stages of examination are carried out. These are used to obtain a further assessment of the patient's physical condition or to aid in making a final diagnosis.

When all diagnostic aids have been utilised, the information is correlated, a final diagnosis established and a treatment plan formulated.

Adequate pre-operative preparation of the patient is essential to place him in the best possible condition to withstand the procedure and hasten recovery. The following may be necessary:-

(a) Systemic Measures: this may involve consultation with the patient's medical advisor and may include control of systemic disease or deficiencies that may otherwise influence the proposed treatment plan.

(b) Local Measures: this involves the general hygiene of the oral cavity. It has been shown that patients with poor oral health have a higher incidence of post-operative complications (Waggener et al, 1961). All cases of neglected oral hygiene should receive a thorough prophylaxis and instruction in future care of the mouth.

Adequate pre-surgical planning by the oral surgeon is essential so that he is in a position to deal with any untoward complication that may occur during the operation. Ritchie (1954) points out that

the oral surgeon must be aware of his own limitations and should not undertake any procedure he is not competent to perform.

Careful surgical technique reduces the incidence of post-operative complications and simplifies the post-operative course. During the operation trauma should be reduced to a minimum, retraction of soft tissues should be carefully carried out, debridement of the wound should be thorough and care should be paid to repositioning and suturing of the flap (Helmore, 1958).

In any facet of oral surgery the welfare of the patient is the prime consideration; and the oral surgeon must act so as to avoid needless discomfort and inconvenience to the patient (Cash, 1951).

This review will discuss normal post-operative treatment and healing in relation to oral surgical procedures, together with some post-operative complications, under the ensuing chapter headings.

HEALING OF ORAL WOUNDS

1. INTRODUCTION
2. SOFT TISSUE REPAIR
   - (a) Primary Healing
   - (b) Secondary Healing
   - (c) Mucoperiosteal Flap Healing
3. HEALING OF EXTRACTION WOUNDS.

HEALING OF ORAL WOUNDS

1. INTRODUCTION

Repair of tissue is generally considered to be a phase of the inflammatory reaction as it cannot be entirely separated from the preceding vascular and cellular phenomena occurring in response to an injury. Following wounding or destruction of tissue, repair occurs by tissue proliferation which,according to Cappell (1962), is initiated by the breach of continuity of the tissues. Under normal circumstances all the cells of the body have a relation to the surrounding cells and this relation acts as a restraint on cell proliferation; if this relation be disturbed, for example by an incision, the cells in the neighbourhood of the injury proliferate to restore the tissue continuity. Once this is effected proliferation ceases.

Shafer, Hine and Levy (1960) state that the healing of all tissues after injury follows an essentially similar pattern. However they note that oral wounds, by virtue of their anatomical situation, may show some modification in the normal healing reaction. These reparative phenomena and their alterations will be reviewed in the subsequent sections of this subject.

2. SOFT TISSUE REPAIR

(a) Healing by first intention (primary healing): occurs when close apposition of the tissues of the wound is achieved. This is

the form of healing one might expect to follow an aseptic surgical procedure where the pliability of the tissues is such that the wound may be drawn together and sutured (Shafer, Hine and Levy, 1960).

When haemostasis has been achieved and the wound margins are fixed in apposition, the adjacent surfaces are "glued together with a thin layer of coagulum" (Cappell, 1962). Within a short time a small number of leukocytes emigrate from the adjacent vessels into the clot. Mitotic division of the connective tissue cells in the region occurs and the new fibroblasts migrate across and into the thin layer of coagulum which undergoes absorption by the action of these cells and of leukocytes. Endothelial budding occurs and capillaries grow across the wound but often little vascularisation is necessary. The fibroblasts produce collogen fibrils which parallel the surface of the wound and these fibrils bring about the permanent union. Epithelialisation occurs rapidly, the epithelial cells growing over the line of incision from the two sides of the wound restoring the continuity of the epithelial covering (Shafer, Hine and Levy, 1960; Cappell, 1962; Harvey, 1949; Kemp, 1952).

Cappell (1962) states that the repair process is completed in five to six days. Harvey (1949) reports that the tensile strength of the wound does not reach a maximum until twelve to fourteen days being preceeded by a "lag phase". The lag phase is approximately four days during which the tensile strength is no greater than that provided by the sutures and the agglutination of the two surfaces by the fibrin formed in the blood clot. This author also points out

that the lag phase in wound healing can be increased by trauma to the wound during surgery, tight sutures, haematoma formation and excessive use of suture material which may exert a foreign body effect.

(b) Healing by second intention (healing by granulation): occurs when there has been loss of tissue and the edges of the wound cannot be approximated or when failure of primary healing occurs. Secondary healing is often described as healing by granulation, the term granulation tissue being originally applied to the appearance of the deep-red tissue seen in the floor of a healing ulcer (Cappell, 1962). This type of wound may result, for example, following removal of a lesion of the hard palate or a large lesion of the alveolar ridge where the surrounding tissues are not able to be mobilised and approximated by sutures.

In the floor of the wound an active formation of new capillaries occurs and these new vessels grow upwards as vascular loops at right angles to the surface. At the same time fibroblasts, arranged parallel to the capillaries, grow upwards. Large numbers of polymorphonuclear leukocytes accumulate on the surface of the wound where they play an important part in checking bacterial growth and thus allowing healing to progress. The growth of the vascular connective tissue is directed towards filling the defect and, when this has gone on for some time, the fibroblasts in the deep layers arrange themselves parallel to the surface and collagen fibrils appear between them. As these fibrils mature their contraction results in reduction of the surface area of the wound.

Epithelial covering of the wound occurs by proliferation and growth of the marginal epithelium over the young vascular connective tissue. When the epithelial covering is complete the layer of polymorphs is replaced by plasma cells and lymphocytes which gradually disappear. The layer of epithelium thus formed increases in depth and the normal structural layers of the epithelium become demarcated.

Once epithelialisation is complete the growth of granulation tissue ceases and devascularisation of the young tissue occurs with the formation of dense, relatively avascular connective tissue - white scar tissue. This process of devascularisation is gradual and eventually the only evidence of a wound may be a small depressed area of mucosa (Cappell, 1962; Shafer, Hine and Levy, 1960).

(c) Mucoperiosteal Flap Healing: a large proportion of oral surgical procedures are initiated by the reflection of a mucoperiosteal flap in order to gain access to the underlying bone. This flap, on completion of the operation is returned to its relation with the adjoining soft tissues to provide a "soft tissue blanket for coverage of bone" (Dedolph and Clark, 1958).

While a complete discussion of flap design is beyond the scope of this review, it must be emphasised that the principles of design should be followed to ensure reattachment and healing of the reflected mucoperiosteum. The basic principles of flap design are as follows:-

(i) The flap should give wide access to the surgical site.

(ii) The flap should have a maximal blood supply.

(iii) The flap should be of such a design as to minimise trauma during retraction of the reflected tissues.

(iv) The flap should be supported by a plateau of undisturbed bone on returning it to its former position.

(v) The flap should be designed to facilitate repositioning and suturing.

Radden (1959) using experimental animals, found that reattachment of the periosteum occurs by deposition of a thin layer of bone on the cortical surface of the alveolus. He also reports that inclusion of the crevicular epithelium in the flap design resulted in a reduction in the height of the epithelial attachment on healing. Radden (1959) considers that the attachment is unaffected if incision for reflection of a mucoperiosteal flap avoids the crevicular epithelium by 3 mm. In a study using both experimental animals and human material Borden (1948) found that healing of the periodontal tissues occurs by granulation with subsequent fibrosis at the site of separation. He concludes that reattachment of soft tissues to the tooth does occur, although he considers that some decrease in the depth of the gingival crevice occurs.

On the other hand Dedolph and Clark (1958) in a histological study of human specimens found that, after three weeks, the attachment of periodontal membrane fibres and other connective tissue elements was restored. The epithelial attachment was complete and the appearance was indistinguishable from that of the control sections. These authors, in contra-distinction to Radden (1959), state that "there appears to be no reason for refraining from stripping up buccal and labial mucoperiosteum to enhance surgical exposure and access whenever it is required".

3. HEALING OF EXTRACTION WOUNDS

Many careful scientific studies have been carried out, both on the experimental animal and in the human, dealing with the healing process in extraction wounds. Simpson (Sept. 1960; Nov. 1960; Jan. 1961; Mar. 1961) and Radden (1959) with Macacus Rhesus monkeys, Huebsch et al (1952) with Long-Evans strain of male rats, and Mangos (1941) and Amler et al (1960) with human material, have all presented well illustrated histological studies concerning extraction socket healing. The healing process in experimental animals is histologically identical with that occurring in humans, however Mangos (1941) points out that, in the human, healing of the epithelium takes twice as long and bone takes three times as long as healing in the dog. The steps in healing of an extraction socket may be summarised as follows (Shafer, Hine and Levy, 1960; Mangos, 1941; Radden, 1959; Weinmann and Sicher, 1955);

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

(b) Within twenty four hours vasodilatation and engorgement of the blood vessels of the periodontal membrane occurs. The surface of the clot is covered with a thick layer of fibrin and may also have a layer of leukocytes present. The unsupported gingival tissue collapses into the wound and is considered to aid in maintaining the position of the clot.

(c) On the third day proliferation of the fibroblasts from

connective tissue cells in the periodontal membrane is evident and these cells begin to grow into the clot around the entire periphery. The epithelium at the periphery exhibits mild mitotic activity at this time and osteoclastic activity at the alveolar crest may be seen.

(d) Five days following extraction the blood clot shows evidence of organisation by young connective tissue. Much of the remaining periodontal membrane shows signs of degeneration and, as yet, no evidence of bone formation is seen.

(e) After seven days organisation of the clot is becoming marked. New delicate capillaries have penetrated to the centre of the clot and fibroblasts are seen throughout the fibrin meshwork. The degenerating periodontal membrane remnants are no longer recognisable and the socket wall appears slightly frayed with some evidence of osteoid tissue extending outward from the deeper parts of the socket wall. Osteoclastic action is now marked at the alveolar crest and extensive, though not complete, epithelialisation is seen.

(f) The ten day wound shows osteoblastic and osteoclastic activity. Osteoid tissue is seen extending from all parts of the socket wall especially in the apical third.

(g) Fourteen days after extraction the socket shows a continuation of the healing process. The clot is almost completely vascularised and replaced by connective tissue. Young bone is forming throughout the socket especially in the region of the apical third. This new bone is formed by osteoblasts which are derived from the multi-potential of the connective tissue cells which assume an

osteogenic function (Weinmann, 1955). The socket outline is indistinct, the original cortical bone of the socket being remodelled by the osteoclasts. Total epithelial coverage of the wound is seen at this time.

(h) Three weeks after extraction the socket is almost completely filled with young bone and active osteoblastic and osteoclastic activity in the socket occurs for as long as fifteen weeks following extraction. As a result of osteoclastic action and the nature of the healing process the alveolar crest comes to assume a rounded contour and lies below the level of the adjacent bone.

Simpson (Jan. 1961; Mar. 1961), Radden (1959) and Shafer, Hine and Levy (1960) state that surgical removal of teeth, during which the buccal plate of alveolar bone is removed, always results in increased loss in alveolar bone height. This produces a smaller ridge which may lead to difficulties in the construction of a prosthetic appliance.

Simpson (Jan. 1961; Mar. 1961), using monkeys, studied the difference in healing rate following bone removal using surgical burs and chisels. He found no significant difference in the healing process for either method.

Costich et al (1964), using water-cooled,ultra-speed rotary instruments for bone instrumentation on dogs, found that the initial repair process was faster than that seen with other forms of rotary instruments. However he reports that, after eight weeks, no difference in the degree of repair was noticed.

PRINCIPLES OF NORMAL POST-OPERATIVE TREATMENT

1. INTRODUCTION

2. NORMAL POST-OPERATIVE TREATMENT

3. POST-OPERATIVE DIET

4. CONTROL OF POST-OPERATIVE PAIN

5. CONTROL OF POST-OPERATIVE OEDEMA.

POST-OPERATIVE TREATMENT

1. INTRODUCTION

Post-operative treatment is an integral part of every oral surgical procedure and its importance has been stressed in a preceding section. This phase of treatment is essential to ensure the continued well-being of the patient, to maintain the continuity of treatment, to minimise complications by early detection and treatment and to restore normal function as rapidly as possible (Holland, 1947; Ritchie, 1954). If the surgical procedure has been well planned and carefully performed, the post-operative course is generally uneventful. However, each patient should be examined regularly following all surgical procedures to ensure satisfactory healing occurs (Kemp, 1952; Helmore, 1958; Ritchie, 1954).

2. NORMAL POST-OPERATIVE TREATMENT

Hayward (1953) states that the patient should be seen forty-eight hours post-operatively at which time both pain and traumatic oedema should be subsiding. Cash (1951) believes the patient is better resting on the first post-operative day and he considers that this results in less post-operative pain and oedema. On the other hand, Ritchie (1954) states that the patient should be examined twenty-four hours following surgery. This reviewer considers that, following many oral surgical procedures, the patient may be safely recalled forty-eight hours post-operatively. However, in any case where complications are anticipated the patient should be examined the next

day. The time of the post-operative examination is therefore left to the discretion of the surgeon.

When a patient is seen following an oral surgical procedure his general appearance should be noted and, following this, he should be questioned as to the amount of discomfort resulting from the surgery, the presence and degree of pain and whether any haemorrhage has occurred. Such information will give an indication as to whether anything untoward has occurred and also has a favourable psychological effect in that it shows the patient the oral surgeon is concerned with his welfare. Next the patient's temperature should be taken. If the temperature is elevated the pulse and respiration rates may also be checked.

The surgical wound should then be copiously irrigated with warm, normal saline solution to remove any tissue slough and debris. Irrigation of the wound is carried out in order to remove those factors, viz., necrotic tissue and food debris, which, if allowed to remain around the wound, may predispose to infection. The mouth should be gently rinsed as often, due to impairment of function, the patient neglects general oral hygiene. These preventive measures are considered by Artz (1960) to be more important than treating an infected wound itself. Kemp (1952) states that a wound healing by first intention usually requires little post-operative treatment other than mechanical cleansing, using cotton wool moistened with saline to wipe the wound surface. Deep wounds, such as cystic cavities, should be carefully irrigated using an aqua syringe or a Higginson's

APPEARANCE OF A WOUND TWENTY FOUR HOURS FOLLOWING SURGERY

On 11-8-64 a 60 year old male patient attended complaining of pain and swelling in the upper left anterior region. Clinical and radiographic examination revealed the presence of an infected cyst overlying the apices of the upper left central lateral and canine teeth. These teeth were all non-vital. These teeth were extracted and good drainage from the cyst resulted.

Following antibiotic sensitivity tests, penicillin therapy was commenced and on 13-8-64 the cyst was surgically removed.

The photograph shows the appearance of the wound 24 hours following surgery.

syringe, care being taken to irrigate all parts of the wound to remove any tissue slough or foreign material.

A careful examination should then be carried out, particular notice being taken of the amount of swelling, both intra-oral and extra-oral, the appearance of the wound and whether regional lymphadenopathy is present. Oedema, to a greater or lesser degree, is a normal post-operative occurrence and can often be related to the amount of trauma occurring during the operative procedure. A traumatic swelling generally is soft to palpation, reaches its fullest extent within forty-eight hours after which it commences to subside, and is not associated with regional lymphadenitis or elevated temperature. If on examination, the swelling is tender with associated palpable lymph glands, increased pain and elevated temperature, infection should be suspected and the appropriate therapy instituted as described in an ensuing section.

Dressings, which are employed in some operations to protect the healing wound, to cover bare bone, to prevent lodgement of food in deep wounds and also to control pain, should not be allowed to become contaminated by the oral fluids. If such contamination does occur infection of the wound is possible with resultant delay in healing. However it must be remembered that, if dressings are changed too frequently disturbance to the regenerating tissues may occur leading to delayed repair of the wound. When dressing wounds an aseptic procedure should be adopted. Kemp (1952) suggests that sterile instruments and dressings should be arranged on a sterile, covered

tray to aid in maintaining asepsis.

Non-absorbable sutures in small intra-oral wounds may be removed after two to five days following surgery provided that the edges of the wound are in apposition and are not under tension (Atterbury and Vazarini, 1961; Thoma, 1963; Dingman and Hayward, 1947). Dermal sutures are usually removed five to ten days post-operatively and are rarely left longer than ten days. Atterbury and Vazarini (1961) consider that, on the face where scarring is to be minimised, skin sutures may be removed after three to four days provided that dehiscence of the wound does not occur. These authors state that, if a wound is under tension and the sutures are mechanically holding the tissues in apposition, the sutures should be left a further two to three days. If sutures are left too long, however, they may act as foreign bodies, cut through the tissues and increase scarring.

When sutures are removed care must be taken not to draw through the tissues that part of the suture which has been exposed in the oral cavity. This may instroduce infection into the tissues and cause localised abscess formation (Thoma, 1963). The following procedure for suture removal is suggested by Thoma (1963) and Atterbury and Vazarini (1961):

(a) Using cross-ribbed dissecting forceps, the suture is grasped just beyond the knot.

(b) The suture is lifted with light tension to draw it slightly out of the tissues.

(c) The tissue on one side is depressed by the scissors with the blades separated.

(d) The suture is then cut. The cut being made on the moist buried portion of the suture material.

(e) The maintained tension draws the rest of the suture up out of the wound thus avoiding pulling part of the exposed material through the tissue.

Atterbury and Vazarini (1961) consider that, once sutures are removed, the suture holes act both as drains for any fluid in the wound and also as portals of entry for bacteria. For the latter reason these authors suggest that the wound should be treated with an antiseptic solution following suture removal, however they do not specify any particular antiseptic.

3. POST-OPERATIVE DIET.

The nutritional state of the patient markedly affects the rate of wound repair. It is well known that delay in healing may occur in a patient who is deficient in any of a large number of dietary factors. Also, decreased antibody production leading to increased susceptibility to infection and decreased resistance to established disease may result from dietary deficiencies (Nizel, 1961; Wolfer et al, 1947; Peters et al, 1948; Cannon, 1945).

Deficiencies may occur due to dietary insufficiency, reduced intake, malabsorption, failure in transportation or utilisation, or to increased demand or destruction of the essential factors (Walker, 1954; Nizel, 1961). Spies (1955) states that deficiency states are

particularly prevalent in the following three groups:-

(a) The indigent, and those with erroneous dietary habits or idiosyncrasies.

(b) Persons with organic disease. For example, in persons with diseases of the alimentary tract or liver and in persons with diabetes mellitus.

(c) Persons addicted to alcohol.

When a dietary deficiency is suspected, dietary analysis should be carried out and if any nutritional factors are found to be deficient, dietetic advice should be given to establish an optimal nutritional status prior to surgery. According to Walker (1954) and Nizel (1961) the corrected dietary regime should be followed for at least one week before surgery is undertaken. Thoma (1963) states that, in cases where a patient is likely to be unable to maintain adequate food intake post-operatively, a diet rich in protein, carbohydrate and essential minerals and vitamins should be prescribed pre-operatively to establish a "body store of essential nutrients".

Following oral surgery, intake of food may be impaired for varying periods as a result of soreness of the surgery site and possibly associated oedema and trismus. To maintain a satisfactory nutritional intake a specific post-operative diet should be prescribed for the patient. This should be formulated after considering the nutritional requirements of the patient, his physical state and the degree of impairment to normal function.

A brief review of specific nutritional factors is necessary to

obtain an understanding of their influence on healing.

(a) Protein: is one of the most important compounds which may influence the rate of wound healing (Shafer, Hine and Levy, 1960). Many clinical studies have indicated that hypoproteinaemia results in delay in the appearance of fibroblasts in the wound together with a reduced rate of mitotic activity (Walker, 1954; Harding, 1948; Shafer, Hine and Levy, 1960). Walker (1954) states that prolonged protein deficiency predisposes to infection due to "reduced tissue vitality". High protein intake results in increased fibroblastic activity and thus an increased rate of healing (Harding, 1948; Shafer, Hine and Levy, 1960).

(b) Carbohydrate: is an essential nutrient to provide energy for muscular activity. Carbohydrate has a protein saving action in that it is used preferentially for energy production, thus preventing catabolism of the protein stores of the body. Thus sufficient carbohydrate must be provided in the diet to supply the caloric requirements of the individual which vary according to the patient's degree of activity. The average requirement for an active patient is 2,000-3,000 calories per day, while a patient requiring bed rest should have an intake of 1,500 calories per day (Ivy and Curtis, 1938; Roots, 1964). These average requirements vary according to the patient's age, sex and body surface. In infection, the demand may be increased (Walker, 1954).

(c) Vitamins: are compounds, required only in trace amounts,

which are necessary for the transformation of energy from one compound to another and for the regulation of body metabolism (Muhler, 1959; Cantarow and Shepartz, 1957). Vitamins are generally stored in the body and these stores are drawn upon to prevent a deficiency state developing. A period of reduced food intake may reduce the vitamins stored and the demand for vitamins is increased following oral surgery. This demand is more marked when infection occurs (Archer, 1961; Thoma, 1963; Walker, 1954).

Vitamin A is essential for maintaining the normal activity of the epithelial tissues. Thus a deficiency in this substance may result in altered epithelial activity in the healing wound.

Vitamin B - complex group: while there are many vitamins in this group, the following are those most frequently mentioned in the healing of wounds: Almost every tissue in the body is dependent upon adequate amounts of thiamine. Thiamine is unstable in the tissues and thus little storage in the body occurs so there remains a continuous daily need for the vitamin (Muhler, 1959). Riboflavin deficiency may cause a delay in formation of granulation tissue and inepithelialisation of healing wounds. This predisposes to secondary infection and often scarring occurs following healing (Muhler, 1959; Walker, 1954). Walker,(1954) states that pyridoxine aids protein metabolism and deficiency can result in disturbed collogen production. Niacin, formerly known as nicotinic acid, is required for normal function of various enzyme systems especially

those concerned with the metabolism of carbohydrates and in this respect is similar to thiamine. Niacin has a joint function with riboflavin in promoting cellular respiration (Muhler, 1959; Cantarow and Shepartz, 1957).

Vitamin C: Deficiency of Vitamin C produces a scrobutic state which has been shown in experimental animals to result, among other things, in failure of collogen formation by the connective tissue cells (Shafer, Hine and Levy, 1960; Muhler, 1959; Cantarow and Shepartz, 1957). Wolfer (1947) and Peters et al (1948) in clinical studies, found that hypovitaminosis C caused delayed wound healing and increased susceptibility to wound infection. It is for these reasons that Archer (1961) suggests the routine prescription of ascorbic acid, plus other vitamins, following oral surgery. This practice however, is not rational as the Vitamin C intake in the average diet is adequate and increased amounts are only excreted.

Other vitamins, while not playing a major part in wound healing, are essential for normal tissue metabolism and must be provided in the post-operative diet.

The maintenance of correct post-operative water balance is of importance and care must be taken to prevent dehydration. Clinically, dehydration is manifest as dry parched lips, dry mouth, increased pulse and respiratory rates and anorexia (Thoma, 1963). Excessive post-operative vomiting, diarrhoea, or marked blood loss may lead to dehydration which, in turn, results in disturbed body metabolism and delayed wound healing. Following oral surgical procedures the

patient should be encouraged to consume increased quantities of fluid to avoid becoming dehydrated (Bergan et al, 1959; Thoma, 1963; Kruger, 1959; Walker, 1954). Febrile patients and those who have had marked blood loss require forced fluid therapy to maintain the normal fluid balance.

Post-operative a liquid diet may be required and this should be as varied and as nourishing as solid diets. A homogeniser or similar food mixer is of value in preparing semi-liquid diets as large amounts of meat, fish and vegetables can be macerated and added to stock (Commonwealth Department of Health, 1961). Nizel (1961) suggests that ice cream, junket, milk shake, egg flip and fruit juices are suitable nutritious foods for the first post-operative days.

As tenderness of the wound improves the patient may commence a soft or non-chewing diet. This may include such foods as minced meat, mashed or shredded vegetables and stewed fruits. As the wound heals the patient will tend to eat more solid foods until the normal dietary habits are restored.

Henny and Barrow (1954) consider that, following extensive oral surgical procedures, food intake by mouth may be inadequate and also that this method of feeding is not conducive to rapid wound healing as disturbance to the wound may occur. These authors prefer tube feeding or liqefied foods to ensure adequate nutrition and to promote rapid healing. Such method of feeding would, of course, be impracticable unless the patient was hospitalised.

In extreme cases where nutrition by the above methods is not possible, intravenous feeding may become necessary but, according to Thoma (1963) parenteral therapy cannot take the place of normal ingestion of food and normal feeding should be instituted as soon as possible.

4. CONTROL OF POST-OPERATIVE PAIN.

(a) Introduction.

Pain is a natural sequel to oral surgical procedures and its control is one of the principal factors in the post-operative care of the patient (Kemp, 1952; Thompson and Morris, 1958; Cash, 1951). According to Monheim (1959), in the majority of cases, the oral surgeon can predict the need for post-operative pain control and, in cases where pain is anticipated provision should be made for medication to ensure adequate relief.

Pre-operative preparation of the patient should include an explanation of the degree and duration of the post-operative pain that may be anticipated. The patient should be made to understand that some pain is a normal response to operative procedures. It is reported in the Lancet (1964) that the post-operative requirements for analgesic drugs can be reduced when the above pre-operative explanation is carried out. This approach to patient education can only be commended as it leads to a better understanding of the post-operative course on the part of the patient and also to a better surgeon-patient relationship. Both of these factors facilitate treatment.

During the surgical procedure post-operative pain can be reduced by careful handling of the tissues and by paying meticulous attention to debridement and closure of the wound. It is not uncommon for a patient to experience pain because of failure to reduce or smooth a bony margin or sharp bony prominence during surgery (Thompson and Morris, 1958; Berlove, 1959).

(b) Pain Perception and Pain Reaction.

Pain is defined by Monheim (1961) as "An unpleasant sensation created by a noxious stimulus which is mediated along specific nerve pathways to the central nervous system where it is interpreted as such".

The transmission of the impulse, which is initiated by the stimulus, and the reaction to the impulse demonstrate the dual aspects of pain viz. (i) pain perception and (ii) pain reaction.

(i) Pain Perception: is the physio-anatomical process by which pain is received and transmitted by the neural structures from the pain receptors through the conductive and perceptive mechanisms.

Pain perceptors, or nociceptors, are non-myelinated nerve fibres which are found throughout the body. These form separate pathways for transmission of impulses to the central nervous system. Each pathway is considered to be a unit in itself and a group of these units forms an afferent nerve trunk.

Pain stimulus may be electrical, thermal, chemical or mechanical in nature and must be of sufficient intensity to excite the free nerve endings. Once this minimum or threshold intensity is reached an impulse passes along the nerve trunk to the central

nervous system. The threshold intensity may vary under differing conditions as well as from tissue to tissue. The impulse that results from the stimulus is self-propagating along the nerve fibre and, unless otherwise blocked, will continue along the entire course of the nerve without loss of speed or intensity (Monheim, 1961; Dille, 1951; Collins, 1958).

Pain stimuli are mediated through the thalamus and, it is thought that a pain centre probably exists in this region of the brain (Dille, 1951). It is at this point that the salicylates and the aniline derivatives are thought to act. From the thalamus pathways to the cerebral cortex transmit impulses which result in perception of pain and localisation to its place of origin. It is at this level that the narcotic group of drugs are believed to achieve their function in pain control.

(ii) Pain Reaction: "is the patient's manifestation of his perception of an unpleasant experience" (Monheim, 1961). This involves the integration and appreciation of pain within the central nervous system and is a complex psycho-physiological process based largely on the past experience and the present attitude of the patient. This aspect of pain may differ from person to person and, in the same individual, may show daily variation.

Pain perception has been shown to be uniform for most persons. Thus pain reaction is dependent upon the pain reaction threshold, that is, the patient's ability to tolerate pain (Collins, 1958; Monheim,1961).

The reaction to painful stimuli is subject to wide variation.

The following factors have been shown to be significant.

(a) Emotional status: patients who are emotionally unstable have a low pain reaction threshold even though the level of pain perception is still within the range of stable individuals.

(b) Fatigue: lowers the patient's pain reaction threshold.

(c) Age: older persons tend to tolerate pain better than younger individuals, a factor which is probably related to past experience. However in advanced age or senility the pain reaction may be altered.

(d) Racial characteristics: Monheim (1961) notes that no apparent difference in pain reaction exists between Caucasians and Negroes. This reviewer has noted, clinically, a lower pain reaction threshold in patients from Southern European countries.

(e) Sex: males are reported to tolerate pain better than females.

(f) Fear and apprehension: lower the pain threshold. Patients who fear dental pain may exaggerate a painful experience (Collins, 1958; Monheim, 1961; Thompson and Morris, 1958).

(c) Drugs for the Control of Post-Operative Pain.

Analgesic drugs relieve pain by raising the pain threshold, thus modifying pain perception. In theory these drugs may act on any part of the perceptive mechanism, however their action is generally thought to be specific on the central nervous system (Dille, 1951). Ideally analgesic drugs should relieve pain without interfering with cerebral cortical functions and the gastro-intestinal, respiratory and other body systems. However none of the analgesics available to-day are entirely free from side effects. Thus, as the majority of

oral surgical patients are ambulatory and desire to carry out their normal everyday activities, the possible side effects of these analgesic drugs must be considered before they are prescribed (Thompson and Morris, 1958; Monheim, 1959;1961).

Before consideration is given to specific analgesic drugs, brief mention of the "placebo effect" should be made. Since clinical studies of pain relief must rely on the subjective estimate of the patient, the psychological effect noted subsequent to the administration of placebo preparations may seriously influence the results (Accepted Dental Remedies, 1963). Keesling and Keats (1958), Hinds and Keats (1960) and Smith (1955) consider the response to a placebo is linked with the patient's desire for pain relief coupled with the suggestion of relief in the giving of medication. Hinds and Keats (1960) report that an average response of 35 per cent occurs to placebo preparations. These authors found however, that the response was inconsistent and they consider this to be related to, among other things, the patient's physical environment, the rapport that exists between the patient and the dentist, together with the natural variation in intensity of pain. In a study of dental out-patients, Keesling and Keats (1958) found that 61 per cent of patients experiencing pain from dry socket obtained relief from placebo preparations. It must be noted that patients in such a study have free access to proprietary analgesics which, if used, could markedly influence the results obtained. Brenman (1963) following a double-blind study of patients undergoing periodontal surgery, reports

numerous responses to placebo medication. According to Accepted Dental Remedies (1963) some 30 per cent of the benefit credited to analgesic drugs may be attributed to the placebo effect. This emphasises the difficulty that occurs in accurately evaluating the efficacy of analgesic drugs.

A. Mild Analgesic Drugs: are employed for the relief of low intensity pain, their value for the control of severe pain being limited. These drugs are thought to act at the level of the thalamus by raising the pain threshold (Dille, 1951).

(i) The Salicylates are derivatives of salicylic acid which itself is highly irritating to the gastric mucosa and is not suitable for systemic administration. These drugs are thought to have a depressive action on the thalamus thus blocking pain impulses before they reach the cerebral cortex. As well as their analgesic action, the salicylate group have an antipyretic effect. This is only manifest in febrile patients, there being no lowering of body temperature if it is normal (Dille, 1951; Goulding, 1960).

The salicylates have few side effects. They are non-habit forming, have no sedative effect and have been shown to have no circulatory or respiratory depressive action when used in therapeutic doses. Contact with the oral mucosa may result in irritation and sloughing and, prolonged use may cause irritation of the gastric mucosa (Monheim, 1959; 1961; Wallace, 1958). Occasionally allergy to the salicylates may occur (Wallace, 1958; Goulding, 1960; Accepted Dental Remedies, 1963), however, according to Dille (1951), this is rare. Prolonged salicylate therapy is reported to produce a decrease

in blood prothrombin with a subsequent disturbance to the blood coagulation mechanism (Kemp, 1952; Goulding, 1960; Accepted Dental Remedies, 1963; Hinds and Keats, 1960; Wallace, 1958).

Salicylates are administered orally and are rapidly absorbed from the gastro-intestinal tract. The drugs are partially hydrolysed in the gastro-intestinal tract itself and about 35 per cent are destroyed in the tissues. A further 20 per cent is excreted in the urine. Average adult dosage is 0.3 - 0.6 Gm. (5-10 grains) which may be taken three or four hourly as required. It is important to note that increased dosages above approximately 0.6 Gm. (10 grains) do not increase the analgesic effect and may result in salicylate intoxication (Hinds and Keats, 1960; Dille, 1951).

Salicylism, or salicylate intoxication, is manifested by tinnitis, headache, vertigo and visual changes, along with nausea and vomiting. Following excessively large doses stupor and coma, followed by convulsions and death from respiratory failure may occur.

Acetylsalycilic acid (aspirin) is the most commonly used salycilate preparation. It is a white, slightly water soluble, odourless powder. It is the least irritating drug of the salycilate group and is least likely to cause toxic effects.

The salycilates are frequently combined with other drugs to increase their efficacy. The drugs most commonly used in such combinations are phenacetin, caffeine and codeine.

(ii) <u>Aniline Derivatives</u>: the most commonly used drugs of this group are acetophenetidin (phenacetin) and acetanilid. These drugs

differ chemically from the salicylates but, pharmacologically, they have essentially similar actions. Like the salicylates, the aniline derivatives are non-addicting drugs. However Wallace (1958) reports that phenacetin habituation may occur. This group of drugs are thought to control pain by their effect on the thalamus and do not interfere with cortical function. The antipyretic activity of these drugs is more marked than the salicylates and Dille (1951) considers that this action may be at the expense of their analgesic potency.

These drugs are administered orally and are rapidly absorbed from the gastro-intestinal tract. Prolonged use may result in the appearance of toxic symptoms, cyanosis may occur due to formation of methhaemoglobin or sulphaemoglobin (Accepted Dental Remedies, 1963; Monheim, 1961;1959; Goulding, 1960). Joyce (1961) states that the aniline derivatives may produce renal damage when taken over long periods, and it is for this reason he considers an alternative drug should be used if possible.

Acetophenetidin (phenacetin) is a white, rather insoluble powder which is administered orally in doses of 0.3 Gm. (5 grains). This drug is not as toxic as acetanilid. Phenacetin is frequently used in combination with other drugs, the most common mixture being the proprietary A.P.C. powders and tablets containing aspirin, phenacetin and caffeine.

Acetanilid (antifebrin) is a white, relatively insoluble powder which is given in 0.3 Gm. doses. If it is used conservatively it is a safe analgesic although Monheim (1961) states that it should not be used with patients suffering from anaemia, renal or hepatic disease.

B. The Narcotics.

The narcotic group of drugs have the property of raising the pain threshold and depressing the pain reaction to a greater degree than any other drugs. The narcotics, in addition to their excellent analgesic action, have a depressant effect on the cerebral cortex. This cortical effect may, in some cases, preclude their use for ambulant patients as it may interfere with the patient's judgement and muscle co-ordination (Monheim, 1959;1961; Dille, 1951). Morphine and codeine, which occur naturally as opium alkaloids, and the synthetic drugs, pethidine and methadone, are the most commonly used drugs of the narcotic group.

The value of the narcotic drugs for general use is further limited due to their many possible side effects. Nausea, vomiting and constipation are the most frequent side effects seen,while respiratory depression may follow larger doses. The addiction liability for all compounds of this group, particularly morphine, is high and no narcotic should be administered if an analgesic of less harmful potential will result in relief of pain (Accepted Dental Remedies, 1963; Monheim, 1961). From the foregoing it can be seen that the drugs of this group must be prescribed with discretion as undesirable sequelae may follow their incautious use. However the potential dangers associated with the use of these drugs should not preclude their administration where a patient is experiencing severe pain.

(1) Naturally Occurring Alkaloids

Morphine is a bitter, white, water soluble powder which is

commonly used as the sulphate salt. This drug has a potent analgesic effect which is thought to be due to a depressive action on the cerebral cortex, raising the threshold to afferent pain stimuli (Dille, 1951).

The systemic effects of morphine are greatly dependent on the dosage used. The smallest doses producing therapeutic effect result in the relief of pain and frequently lead to a state of euphoria. Increased dosages of morphine may lead to a degree of hypnosis and respiratory depression which may prove fatal.

Morphine may be administered by intravenous, intramuscular or subcutaneous injection. The average adult dose being 8-20 mg. (1/6 - 1/3 grain) administered four to six hourly. A dose of 16 mg. of morphine raises the pain threshold by 50-75 per cent and, when administered intravenously, the maximum effect is achieved within twenty minutes. Following intramuscular injection of morphine the maximum effect is noted forty-five minutes to one hour later.

Miosis, or pupillary constriction is a typical sign that morphine has been administered and, in some patients, slight respiratory depression may also be noted. Gastro-intestinal disturbances such as nausea, vomiting and constipation are not uncommon side effects following use of this drug. However no effect on the heart or the vasomotor centre occurs (Accepted Dental Remedies, 1963; Monheim, 1961; Thoma, 1963; Dille, 1951; Goulding, 1960).

Goulding (1960) warns that morphine should not be administered to patients who suffer from asthma or severe chronic respiratory disease

as the depressant action of the drug may result in respiratory difficulty.

The dangers of morphine are generally well recognised but can scarcely be overemphasised. The repeated use of the drug can rapidly lead to alarming side effects, morphine tolerance and addiction.

The specific antidote for morphine intoxication is Nalorphinine Hydrochloride which is administered by intravenous injection of doses of 5-10 mg. up to a total of 40 mg. (Goulding, 1960; Thoma, 1963; Accepted Dental Remedies, 1963).

Codeine (methyl morphine) is an opium derivative and is commonly used as the phosphate or sulphate salt which is water soluble to about 3 per cent. It is similar both chemically and pharmacologically to morphine but is less potent. Its ability to relieve pain is about one-sixth that of morphine however, its undesirable side effects are proportionally less than morphine and its potential for addiction is not as high.

Codeine is well tolerated when given orally and, if required, it can also be given by intramuscular injection. The average adult dose of 15-60 mg ($\frac{1}{4}$-1 grain) raises the pain threshold by 50 per cent. According to Seevers (1949), and Dille (1951) if 60 mg. of codeine salt is not effective in relieving pain, increased dosage is generally ineffective and, if administered may result in excitement and restlessness.

Following administration of codeine in therapeutic doses euphoria may be slight or entirely absent and nausea, vomiting and

other gastro-intestinal disturbances are also absent or minimal.

Codeine can be administered either alone or in combination with acetylsalicylic acid, acetophetidin, caffeine or other drugs for the control of most dental pain. Monheim (1961) considers that, as codeine is generally effective in relieving most dental pain, the use of more potent analgesics is rarely necessary.

Dihydromorphinone is a naturally occurring alkaloid which chemically is closely related to morphine. The potency of dihydromorphinone is greater than that of morphine and the average dose is 2.5 mg. given orally. The onset of action of this drug is rapid and consequently its duration is less. It has a less marked effect on the gastro-intestinal tract but has a marked depressive action on the respiratory system. Thus it is not suitable for use in dental practice for the relief of post-operative pain (Dille, 1951; Seevers, 1949).

Metopon is methyldihydromorphinone and is therefore related to dihydromorphinone. The average dose is 5 mg. This drug may be given orally and has been shown to have less gastro-intestinal effects and does not depress respiration as markedly as morphine. This drug is, at present, used mainly for control of chronic pain (Dille, 1951; Monheim, 1961; Seevers, 1949).

(ii) Synthetic Compounds.

The most commonly used synthetic potent analgesic drugs are as follows:

Pethidine (Meperidine) is a drug which, chemically, has no relation to morphine or the other opiates. It is a slightly bitter, white, odourless powder which is water soluble. This drug may be administered either orally or by intramuscular injection, the average adult dosage being 50-100 mg. given four hourly.

As an analgesic drug pethidine has a potency weight-for-weight about one tenth that of morphine. The drug in therapeutic doses does not cause significant respiratory depression but, sometimes, may cause nausea and vomiting. It is far less hypnotic than morphine and, to some extent, less euphoric. However, although a synthetic compound, pethidine is capable of producing addiction but its addictive potential is of a lower order than that of morphine (Goulding, 1960; Monheim, 1961; Kemp, 1952; Dille, 1951; Seevers, 1949; Thoma, 1963).

Methadone (Methadon) is chemically unrelated to morphine and has a somewhat different pharmacologic action. Methadone is a powerful analgesic drug which is considered to be as effective, if not more so, than morphine. Unlike the latter drug, the therapeutic administration of methadone results in minimal sedative or hypnotic effects.

Methadone may be given orally or by intramuscular or subcutaneous injection in 5-15 mg. doses. The dose should not be repeated more than four hourly and no more than three doses should be administered in any twenty-four hour period (Kemp, 1952; Accepted Dental Remedies, 1963). Following administration of this drug vertigo, nausea, vomiting and constipation may occur. Euphoria is rarely experienced and although addiction may occur, the addiction potential is significantly less than for morphine (Accepted Dental Remedies, 1963; Kemp, 1952; Dille, 1951;

Seevers, 1949).

The selection of analgesic drugs for the relief of post-operative pain should be based upon a knowledge of the action and the side effects of these drugs together with an understanding of the patient and his individual needs. This reviewer agrees with Helmore (1958) who considers it is wise to select the "simpler drugs" for the relief of pain. The narcotic drugs should not be employed unless really necessary and, if they are used, their possible side effects, including addiction, must be considered.

5. CONTROL OF POST-OPERATIVE OEDEMA

(a) Introduction.

Following surgical intervention inflammatory oedema occurs as a result of the trauma sustained by the tissues. As oral surgical procedures mostly involve bone as well as soft tissues, considerable manipulation of the soft tissues is often necessary to obtain adequate access to the surgery site. This may cause an increase in the inflammatory response. In the facial region the problem of post-operative oedema is more pronounced as its presence is very noticeable. Also, because the involved tissues contain much loose areolar tissue, the swelling is likely to be more extensive due to wide diffusion of the inflammatory exudate (Hinds, 1958; Schneider, 1959). If the oedema involves the soft tissues adjacent to the molar regions trismus is often an accompanying complication due to interference with normal muscle function (Henkel, 1956; Thoma, 1963).

(b) The Inflammatory Response.

In order to understand the rationale of the methods used for the control of post-operative oedema, a brief review of the principles of the inflammatory reaction is necessary.

Inflammation is a defensive tissue reaction to injury which can be initiated by bacterial, physical or chemical agents. The inflammatory response is a mobilisation of the body defences. It brings to the area leukocytes and tissue fluid. The function of the leukocytes is principally antibacterial while the inflammatory exudate dilutes the irritant, brings antibodies to the area and aids in localisation by producing a fibrin meshwork. The cardinal signs of inflammation are well known. They are:

Redness - due to increased amount of blood in the inflamed area.

Heat - as a result of increased vascularity.

Swelling - caused by increased amount of blood and accumulated serous exudate and cellular elements.

Pain - due to irritation of sensory nerves by toxic substances and tissue tension resulting from the swelling.

Following injury a momentary vasoconstriction of the small arteries and capillaries occurs which is followed by dilatation of these vessels leading to a slowing of the circulation and eventually stasis. An increase in vessel permeability also occurs, leading to increased exudation of plasma and emigration of white blood cells through the vessel walls into the tissue spaces. This phenomenon is known as diapedesis.

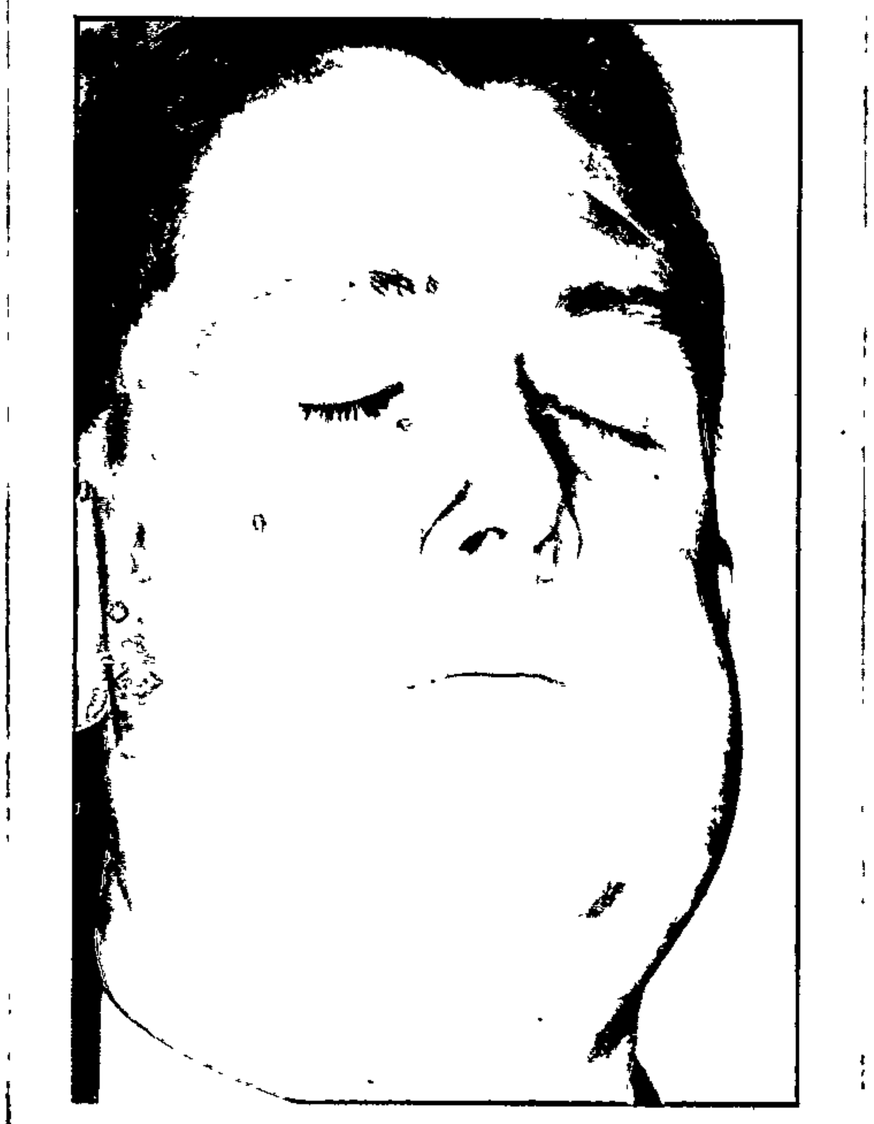
If the irritation is maintained, exudation continues and the tissue spaces become filled with exudate, which constitutes the inflammatory oedema. Later the exudate forms a fibrin reticulum which causes obstruction of the lymphatic channels, the patency of which is essential for resorption of the exudate (Archer, 1961; Cappell, 1962; Henkel, 1956).

Post-operative oedema, primarily due to operative trauma, is basically composed of inflammatory exudate, little emigration of leukocytes occurring.

Resorption of the inflammatory exudate usually commences forty-eight hours following surgery unless infection supervenes (Henkel, 1956; Burch and Morris, 1962). Resolution is characterised by a retrogression of the various inflammatory phenomena. Stasis passes off and restoration of the circulation occurs. The exudate is resorbed through the lymphatic channels and the fibrin is digested by the leukocytes (Cappell, 1962). If resorption fails to occur or the fibrin meshwork formed is very dense, organisation of the exudate may take place by an ingrowth of fibroblasts and capillaries (Henkel, 1956).

There are many factors which influence the degree of post-operative inflammatory reaction, all of which are not fully understood. Hinds (1956) states the two most important factors are the degree of operative trauma and the presence or absence of infection. It is also generally recognised that, all other factors being equal, there is an individual reaction to trauma which is not entirely predictable.

POST-OPERATIVE OEDEMA



This photograph shows the degree of post-operative oedema experienced by this 24 year old patient forty-eight hours following the surgical removal of an impacted lower left third molar tooth. This patient previously experienced a corresponding amount of oedema subsequent to the surgical removal of a similarly impacted lower right third molar tooth.

(c) The Management of Post-operative Oedema.

The various methods used for the control of post-operative oedema are directed towards reducing the amount of exudation and promoting and maintaining tissue permeability.

(i) The use of heat and cold; this is the most widely employed method for the control of post-operative oedema. Archer (1961) and Holland (1947) state that the intra-oral and extra-oral accessibility of the jaws, the vascularity of the soft tissues of the face and the ease of application of either heat or cold, together with the desire to prevent post-operative oedema of the tissues, have contributed to the universality of this method of treatment.

The rationale for the use of cold depends upon the fact that its application to the tissues results in constriction of the capillaries. This in turn reduces the blood and lymphatic flow leading to a reduction of exudate which constitutes the inflammatory oedema (Moore et al, 1961; Berlove, 1959; Thoma, 1963; Archer, 1961; Holland, 1947; Hennessy, 1941).

However vasoconstriction deprives the tissues of the products of the inflammatory reaction thus the continuous use of cold will impede healing and may lead to cellular damage or tissue death due to decreased nutrition (Archer, 1961; Berlove, 1959; Podolin, 1936; Kruger, 1959). Thus application of cold should be brief and intermittent. Berlove (1959) suggests that cold should be applied for twenty to forty-five minutes and then discontinued for one to one and a half hours before being re-applied. The immediate post-

operative use of cold is indicated before inflammatory oedema occurs.

According to Holland (1947), Thoma (1963), Archer (1961) and Berlove (1959) the use of cold, either as ice packs or cold compresses, as well as controlling oedema has some analgesic effect. The application of cold, by causing vasoconstriction is also of value in controlling haemorrhage which, otherwise, may contribute to swelling (Kemp, 1952; Cheraskin, 1959; Shira, 1958). Ritchie (1954) considers that the application of an ice pack is of value in limiting haematoma formation. However, as the vascular response is transient, cold is only of value as an adjunct to other haemostatic measures.

The penetration of heat into the soft tissues is slight, approximately 1 mm, the heat being dissipated by circulatory reflexes (Holland, 1947; Archer, 1961). Archer (1961) reports that luminous sources of heat such as sunlight, tungsten and carbon filament lamps have a greater depth of penetration than heat applied by means of poultices or hot compresses.

Local heat causes vasodilatation of the capillaries, increases the peripheral blood and lymphatic flow and accelerates local tissue metabolism (Moore et al, 1961; Holland, 1947; Hennessy, 1941; Archer, 1961; Moose, 1937). As capillary dilatation occurs the area of the capillary wall available for fluid exchange is increased. Hence more blood and tissue fluid are brought to the region, augmenting the local defence mechanisms. Hennessy (1941) states that increased vessel permeability allows greater diapedesis to occur.

When local circulation is increased, an increase in oedema formation also occurs. However heat also dilates the lymphatic channels

and accelerates lymphatic drainage. The enhanced leukocyte activity together with increased lymphatic drainage is important in the control of infection. Archer (1961) states that if "infection is markedly pathogenic and is inclined towards formation of pus and localisation, either intra-orally or extra-orally, heat will hasten the process". The converse is also true that, if the tendency is toward resolution, this will be hastened by heat.

It is generally agreed that moderate heat has a soothing effect and its use results in increased comfort to the patient (Kruger, 1959; Moose, 1937; Berlove, 1959; Podolin, 1936). Possoff (1955) following a controlled clinical study, states that patients showed a marked preference for heat after the first twenty-four hours and he suggests that this may be due to the anti-spasmodic effect of heat on the facial muscles in the traumatised area. According to Hinds and Keats (1960) the value of thermal applications may be due to distraction or dilution of pain stimulation by other types of stimuli from the same area.

Intra-orally heat may be applied by either in the form of irrigations or mouthwashes of hot normal saline solution or as hot saline packs. Extra-oral heat may be applied by the use of hot foments. Thoma (1963) recommends that, prior to applying extra-oral heat, the skin should be protected with petrolatum or some other lubricant. Archer (1961) and Thoma (1963) consider that the saline should be at 120-125^{0}F and the applications of heat should be carried out every two hours.

Heat should be used twenty-four hours following surgery to aid in the resolution of traumatic oedema, to increase comfort and to increase local tissue metabolism and thus hasten healing. In infectious conditions the application of heat may aid in localisation of the infection and, once drainage occurs, the use of heat is of value in maintaining free drainage.

Possoff (1955), in a controlled study, investigated the value of heat and cold applications following extraction. He concluded:

(1) Moderate thermal applications cause little change in mucosal temperature.

(2) Little difference in pain, tenderness and general comfort result from the use of heat and cold during the first twenty-four hours.

(3) There was a noticeable preference for application of heat after twenty-four hours.

(4) No demonstrable difference in the rate of healing occurred in the region of the control, applied heat or applied cold.

(5) No difference was seen in swelling following the post-operative use of thermal applications.

However it is the general consensus of opinion that the use of heat and cold has definite application as a post-operative measure for the reduction of post-operative oedema (Archer, 1961; Thoma, 1963; Podolin, 1936; Kruger, 1959; Moose, 1937; Berlove, 1959; Holland, 1947; Hennessy, 1941; Macalister, 1957).

(ii) <u>The use of anti-histamine drugs</u>: the rationale for the use of anti-histamine drugs for the control of post-operative oedema

is based on the hypothesis that histamine is the activator of the vascular phenomenon in the inflammatory reaction. According to Silverman (1953;1954), histamine, which is thought to be released from the tissues following trauma, causes vasodilatation of the arterioles and capillaries and increases vessel permeability. This permits transudation of fluid into the tissue spaces which results in the formation of inflammatory oedema. Anti-histamines are considered to specifically block the action of histamine and are thought to be effective in the control of post-operative oedema when administered post-operatively (Silverman, 1953;1954).

Silverman (1953;1954) reports that the post-operative use of Pyronil and Co-Pyronil (100 mg. given eight hourly) resulted in a reduction of post-operative pain, swelling and trismus. This author also reports that healing by first intention occurred more often, following their post-operative administration. Moss (1958) studied the effect of giving Promethazine orally twenty-four hours pre-operatively and for forty-eight hours post-operatively on twelve patients requiring various oral surgical procedures. He reports that the use of Promethazine resulted in decreased post-operative oedema and considers that healing appeared to be more rapid. In a study involving thirty patients Maccia (1957) found that the use of anti-histamines subsequent to the removal of impacted upper third molars resulted in reduction of post-operative pain, oedema and trismus. Maslansky and Maslansky (1953) report that the use of injectable anti-histamines significantly reduced post-operative swelling and

trismus in two hundred cases in which both local and general anaesthesia were used. These authors found that the number of patients with moderate to marked post-operative oedema was reduced from 74 per cent to 10 per cent.

However, in 1956, Szmyd published the results of a double blind study involving five hundred patients selected at random from an oral surgical clinic. An attempt was made to include in the group a large number of extractions of impacted teeth and mandibular third molars. Szmyd (1956) reports that "no significant effect of anti-histamine drugs (Pyronil, Histadyl and Clopane hydrochloride), as compared to the placebo preparation, on the degree of post-operative oedema and trismus was noted". Keesling and Hinds (1957) in a double blind study, found that anti-histamines were not effective in reducing post-operative pain or oedema. Following a controlled clinical study, Snyder (1960) found that no significant alteration in the post-operative course resulted from the use of anti-histamines. Similar findings are reported by Holland and Jurgens (1958) and Hinds (1958).

It would appear, at present, that the anti-histamines have little value in the control of post-operative oedema.

(iii) <u>The use of enzymes:</u>

<u>Hyaluronidase:</u> Benzer (1951) records that, in 1929, Duran-Reynals isolated an enzyme which promotes diffusion of fluids through the tissues. This substance, now recognised as hyaluronidase, acts on the intercellular substance of connective tissue, hyaluronic acid, a

complex mucopolysaccharide. Thus hyaluronidase decreases the interfibrillar and intercellular viscosity and increases the permeability of connective tissue. Any increase in permeability results in more rapid resolution of inflammatory oedema by allowing diffusion and, hence absorption of the inflammatory exudate. Following depolymerisation with hyaluronidase, the normal hyaluronic acid barrier is partly restored within twenty-four hours and fully restored in forty-eight hours (Young and Kingsbury, 1955; Devine, 1954).

Hyaluronidase is a purified enzyme which is naturally present in certain human and animal tissues. The most commonly used form of hyaluronidase is prepared from bovine testicular extract and occurs as a white, water soluble powder. In the dry state the enzyme is stable at room temperature and, if kept refrigerated, the reconstituted form retains its activity for two weeks (Accepted Dental Remedies, 1963; Thoma, 1963). The activity of hyaluronidase is expressed in turbidity reducing units (T.R.U.) (A T.R.U. is defined by Young and Kingsbury (1955) as "the amount of hyaluronidase which will reduce the turbidity produced by 0.2 mg. of potassium hyaluronate in acidified horse serum to that produced by 0.1 mg. under assay conditions). As hyaluronidase is a protein substance, sensitisation is possible (Accepted Dental Remedies, 1963; Hinds, 1958), but, according to Thoma (1963), Henkel (1956) and Young and Kingsbury (1955), hypersensitivity is rare and toxicity of the enzyme is minimal. Benzer and Shaffer (1952) report that administration of up to 200,000 times the maximum proposed therapeutic dosage to experimental animals

resulted in no signs of systemic or local reaction.

Accidental intravascular injection of hyaluronidase is harmless mostly because of a hyaluronidase inhibitor in the blood (Henkel, 1956; Benzer and Shaffer, 1952; Thoma, 1963).

The prophylactic use of hyaluronidase to promote or maintain the tissue permeability of early oedema formation and prevent post-operative swelling has been suggested by some authors. Devine, in a study without controls, injected 150 T.R.U. of hyaluronidase into the region of the mandibular foramen immediately after mandibular block injection. He found that this measure prevented pain, oedema and trismus following removal of impacted lower third molar teeth. In a controlled study, Young and Kingsbury (1955) injected 150 T.R.U. of hyaluronidase into the anterior border of the masseter muscle of patients undergoing removal of bilaterally impacted mandibular third molar teeth under general anaesthesia. These authors found the degree of post-operative swelling and trismus to be markedly reduced and report that no local or systemic effects were seen following administration of the enzyme. Similar results are reported by Macalister (1957) and Shuttee (1962) who both used hyaluronidase immediately following surgery. Fleuchas (1956) combined hyaluronidase with local anaesthetic solution to determine the value of the enzyme in controlling oedema. In patients requiring bilateral removal of lower right third molar teeth Fleuchas used the combination for one procedure and standard local anaesthetic solution for the other. This author found less swelling and trismus on the experimental

side, however he reports that, although the difference could be measured, it was not markedly apparent.

The prophylactic use of hyaluronidase is contra-indicated because, if used too soon, it may interfere with the normal inflammatory response. Also, according to Henkel (1956) and Accepted Dental Remedies (1963), the premature use of hyaluronidase may result in post-operative haemorrhage and widespread ecchymosis. Local haemorrhage into the tissues is normally self-limiting and ceases as pressure in the tissues rises with continued extravasation of blood. However if the permeability of the tissues is increased prolonged haemorrhage may occur.

Henkel (1956) states that "a reasonable time" should be allowed to elapse before hyaluronidase is administered. Benzer and Shaffer (1952) consider that it is advisable to wait at least twenty-four hours after surgery as, by that time, the initial inflammatory response has taken place and haemorrhage is controlled. Most other authors do not specify the optimal time for administration of the enzyme.

Following the use of hyaluronidase in the treatment of a variety of traumatic swellings, Benzer (1951) reports that the use of the enzyme resulted in both objective and subjective relief of symptoms. This author also noted, clinically, a reduction in the healing time. Hyaluronidase was used by Benzer and Shaffer (1952) for the treatment of oedema and trismus resulting from operative procedures and injuries. These authors found a reduction in trismus and swelling following the

use of the enzyme. Similar results are reported by Henkel (1956) which support the findings reported above.

The technique for the use of hyaluronidase is as follows. After preparation of the skin or mucous membrane, a few drops of local anaesthetic solution are deposited in the superficial tissues to prevent discomfort when the large needle for injection of the enzyme is inserted. The enzyme, supplied as a powder, is then reconstituted with sterile normal saline solution and is drawn up into a Luer-Lok syringe with a 22-24 gauge needle. The usual dosage of hyaluronidase is 150-300 T.R.U. in 3-4 cc. of saline, which volume is sufficient to cause sufficient increase in interstitial pressure to enhance diffusion of the enzyme. The needle is then inserted into the centre of the swelling and, following aspiration, the solution is slowly injected. After withdrawal of the needle intra-oral and extra-oral pressure, by means of gauze pads and a pressure bandage respectively, is applied for thirty minutes. This increases the local tissue pressure and aids diffusion of the enzyme (Thoma, 1963; Henkel, 1956).

The use of hyaluronidase in infected tissue is contra-indicated due to the possibility of spreading the infection (Accepted Dental Remedies, 1963). However Thoma (1963) considers that hyaluronidase may be safely injected into infected tissues provided that antibiotic therapy is also instituted. This latter view is shared by Henkel (1956) who considers that hyaluronidase, in association with antibiotic therapy, is beneficial in the treatment

of some infections because rapid dispersion of the antibiotic in the infected tissue may be anticipated.

Proteolytic Enzymes: trypsin and chymotrypsin are the two members of this group which have received most attention. These enzymes are obtained from animal pancreatic tissue and are capable of digesting a wide variety of proteins. However they act only on necrotic tissue, having no effect on living cells. The rationale for the use of these drugs is not fully understood. According to Hinds (1958) the administration of these substances is thought to activate a series of enzymatic reactions which serve to dissolve fibrin. Thus these enzymes may be of value for dissolution of the fibrin produced by coagulation of the inflammatory exudate.

Intramuscular injection of trypsin was used in a double blind study by Schneider (1959) to assess its value in reducing post-operative oedema. He found that the parenteral use of trypsin resulted in reduced post-operative swelling and considers the enzyme to be a valuable adjunct to oral surgery. In a similar study Schneider (1961) evaluated the efficacy of enteric coated trypsin tablets. He found that this method of administration also resulted in a reduction of the degree of post-operative swelling and pain. Schneider concluded that no difference in the results occurred with the different modes of administration. On the other hand Krause (1961),following a clinical study of sixty patients requiring bilateral extraction of lower third molar teeth, found that while enteric coated trypsin was effective in limiting post-operative oedema, this method

of administration was not as effective as intramuscular injection of the enzyme.

The use of chymotrypsin for the management of post-operative oedema has been reported by Accepted Dental Remedies (1963) and Sowdray (1961). In a clinical study to evaluate the efficacy of chymotrypsin Sowdray (1961) found that administration by intramuscular injection was of no value in the prevention of post-operative pain and swelling.

The antigenic nature of these protein materials cannot be overlooked. Both Liebowitz and Ritter (1960) and Rose (1960) report an anaphylactoid reaction following the injection of chymotrypsin for the control of oedema.

According to Accepted Dental Remedies (1963) the proteolytic enzymes are only of value for debridement of wounds by topical application, the parenteral use of these substances being contra-indicated due to the possibility of inducing both local inflammation at the site of injection and pyrogenic or hypersensitivity reactions. The proteolytic enzymes are thought to be inhibited by enzyme inhibitors in the plasma (Accepted Dental Remedies, 1963).

It can be seen that, although some clinical evidence indicates that the proteolytic enzymes may be of value in the control of post-operative oedema, further study of the mode of action of these substances and their clinical applications is indicated before their efficacy can be properly assessed.

Plasminogen activators: streptokinase, an enzyme elaborated by haemolytic streptococci, is the only plasminogen activator which has had extensive clinical trials. The mode of action of this substance is complex and is based upon the ability of streptokinase to accelerate the formation of the fibrinolytic enzyme plasmin, by catalysing the reaction of plasminogen to plasmin. Plasmin causes lysis of fibrin in the inflammatory exudate thus reducing the viscosity of oedema fluid, dissolving the fibrin clots in the vascular and lymphatic channels and leading to resorption of the oedema fluid (Miller et al, 1958; Accepted Dental Remedies, 1963; Macalister, 1961).

Varidase buccal (Lederle Lab.), a concentrated partially purified preparation containing 10,000 units of streptokinase and at least 2500 units of streptodornase, has been used in several clinical studies. The tablets are placed in the buccal sulcus or under the tongue and allowed to dissolve slowly so that absorption can take place from the mucosa (Hyson, 1960; Thoma, 1963). The swallowed enzyme is reported by Thoma (1963) to be destroyed by gastric acidity.

Hyson (1960), in a double blind study using Varidase and a placebo in combination with achromycin, found the combination to be of value in reducing post-operative oedema. This author also reports absence of pain and tenderness of the surgery site following the use of the two drugs. In a study involving two hundred and

fifty patients requiring removal of impacted and unerupted mandibular third molar teeth, Macalister (1961) found that Varidase buccal, given pre- and post-operatively, resulted in reduction of both post-operative pain and trismus. Miller et al (1958) found, in fifty-seven patients undergoing various surgical procedures, the use of the streptokinase-streptodornase combination resulted in less post-operative swelling in 88 per cent of cases. This author also considers that, as streptokinase causes dissolution of the inflammatory barrier allowing bacterial spread to occur, antibacterial drugs should also be used. The Council on Drugs (J.A.M.A. 1960) consider that, although clinical observations tend to support the fact that plasminogen activators are of value in the control of inflammatory oedema, the evidence presented is not conclusive and further substantiating investigations are required.

(iv) <u>The use of corticosteroids:</u> For the sake of completeness brief mention must be made of the corticosteroids which are considered to be true anti-inflammatory agents.

The corticosteroids have a broad scope of physiological action and are concerned with, among other things, the regulation of water and electrolyte balance, control of neuro-muscular activity, metabolism of carbohydrate, fat and protein, regulation of the cellular components of blood and lymphoid tissue, the regulation of secretory mechanisms and are significant in anti-inflammatory and anti-allergic reactions (Hinds, 1958; Ross and White, 1958; Accepted Dental Remedies, 1963).

Three basic groups of corticosteroids occur naturally and they are grouped according to their principal activity. The mineral corticosteroids are primarily concerned with the maintenance of electrolyte balance. The glucocorticoids are most active in carbohydrate protein and fat metabolism and have anti-inflammatory properties, this group includes cortisone and hydrocortisone. The third group comprise the low potency sex hormones (Ross and White, 1958).

Since 1949 some of the glucocorticoids have been used for treatment of arthritis and since then have been employed for the palliation of allergic and inflammatory conditions (Hinds, 1958; Ross and White, 1958; Accepted Dental Remedies, 1963).

Cortisone and hydrocortisone act at a cellular level and are thought to suppress the entire inflammatory reaction, including wound repair. Because the use of these drugs may mask the early signs of inflammation, infection may go unnoticed until it becomes widespread. It is for this reason, together with the delay in wound healing and the possible systemic effects that may follow the administration of the corticosteroids, that Hinds (1958) and Accepted Dental Remedies (1963) consider the use of these drugs for the control of traumatic oedema to be unwise.

Ross and White (1958) consider that in normal patients requiring oral surgical treatment the small dosage and the short period of administration of these substances preclude side effects. In a clinical study of patients undergoing removal of lower third molar teeth these authors found that hydrocortisone capsules,

administered both pre-and post-operatively, resulted in a reduction of post-operative oedema and trismus. They also noted that, clinically, no delay in healing occurred.

From the foregoing review it appears that the immediate post-operative use of cold and, later, the use of moderate heat has some value in the control of post-operative oedema. The anti-histamines appear to have little merit in reducing swelling. The enzymes, particularly hyaluronidase, seem to have some benefit, but the general consensus is that further investigations need to be carried out to accurately determine their place in post-operative treatment. The use of corticosteroids is contra-indicated due to the possible unfavourable sequelae that may follow their use. In summary, it can be stated that none of the aforementioned agents are a substitute for careful surgical technique and judgement in the control of post-operative oedema.

POST-OPERATIVE HAEMORRHAGE

1. INTRODUCTION
2. PHYSIOLOGY OF HAEMOSTASIS
3. CLASSIFICATION OF HAEMORRHAGE
4. CAUSES OF ABNORMAL HAEMORRHAGE
5. ECCHYMOSIS AND HAEMATOMA
6. CONTROL OF HAEMORRHAGE
7. MANAGEMENT OF POST-OPERATIVE HAEMORRHAGE.

1. INTRODUCTION

Haemorrhage associated with minor oral surgical procedures generally ceases spontaneously due to the normal haemostatic mechanisms. However, a disturbance in the normal mechanism due to hereditary, systemic or local factors may result in post-operative haemorrhage.

A careful evaluation of the patient for bleeding tendencies should be a part of all preliminary examinations. Such evaluation necessitates questioning the patient regarding previous bleeding episodes, familial bleeding tendencies and the taking of any medications, which may influence the normal haemostatic mechanisms (Archer, 1961; Chaput, 1964; Antioch, 1959; Shira, 1958; Cheraskin, 1959; Naylor and Moore, 1962; Thoma, 1948; 1963; Dalitsch, 1941; Kemp, 1952; Helmore, 1958).

A patient with a history of haemorrhage should be referred for haematological investigation. Such investigation may include full and differential blood count, haemoglobin estimation, platelet count, coagulation time, bleeding time, capillary fragility tests and any other special tests considered necessary by the oral surgeon. If any marked abnormality is detected in the results obtained, consultation with the patient's physician is advisable prior to undertaking any oral surgical procedures.

Post-operative haemorrhage can be minimised, if not completely prevented, by taking meticulous care during the surgical procedure. Trauma to the tissues during surgery increases the danger of

post-operative haemorrhage as sloughing or infection of the tissues is likely to occur. At each stage of the surgical procedure haemorrhage should be carefully controlled. Once the surgical procedure is completed, the operative site should be carefully inspected to ensure that satisfactory haemostasis has been achieved. Sharp bone margins and bone splinters should be removed since they cause irritation and may predispose toward secondary haemorrhage. Pathologic soft tissue should also be removed at the time of surgery since for the most part, this tissue is highly vascular and is conducive to continued haemorrhage if allowed to remain (Shira, 1958; Naylor and Moore, 1962; Cohen, 1958; Howell and Monto, 1953). Sutures should be carefully placed to ensure correct approximation of the soft tissues and to effect haemostasis. Care must be paid to avoid too much tension on sutures as this may cause them to tear through the tissues or cause local tissue necrosis and sloughing which may result in post-operative haemorrhage.

Once surgery is completed the patient must be given thorough post-operative instructions regarding care of the wound. Haemorrhage may result from mechanical interference with the blood clot at the surgery site e.g., too vigorous rinsing, disturbing clot with tongue or fingers or trauma from coarse foods (Naylor and Moore, 1962).

Continued post-operative treatment is essential until satisfactory healing of the wound has occurred. For example, if infection of the surgical wound occurs and is not promptly and adequately treated, post-operative haemorrhage, together with other complications, may result.

In this review primary attention will be given to post-operative haemorrhage of local origin in patients who do not suffer from bleeding disorders and to the methods most commonly employed to effect haemostasis in such cases.

2. PHYSIOLOGY OF HAEMOSTASIS.

There are two mechanisms which effect the arrest of bleeding, namely, the behaviour of the blood vessels and the formation of the blood clot. A defect in either of these may result in an abnormal bleeding tendency.

(a) Response of the blood vessels: following injury to a blood vessel, a reflex vasoconstriction occurs. This is most marked in the smaller blood vessels and, normally, haemorrhage from arterioles, capillaries and venules is controlled by this mechanism in less than a minute, although coagulation may take longer than ten minutes. Naylor and Moore (1962) state that some doubt exists as to whether the capillaries themselves undergo active contraction or whether, because the arterioles supplying them have contracted, they merely collapse, allowing their walls to adhere to one another.

(b) Coagulation of blood: in the circulating blood are certain factors which, when acted upon by certain local enzymes, react together to form a meshwork of fibrin; the basis of the blood clot. There are many complex factors involved in the coagulation of blood and, while a detailed discussion of these factors is beyond the scope of this review, an understanding of the principles of blood coagulation

is essential for the treatment of post-operative haemorrhage.

The following schematic diagram of the steps in blood coagulation sets out the generally accepted stages of the mechanism.

Stage I.

Anti-Haemophiliac Globulin + platelet Factor —Christmas Factor→ Intermediate Product

Stage II.

Intermediate Product + Labile Factor V —Stable FactorVII→ Thromboplastin

Stage III.

Prothrombin —Thromboplastin→ Thrombin

Stage IV.

Fibrinogen —Thrombin→ Fibrin

(after Naylor and Moore, 1962)

The process is initiated at Stage I by the platelet factor which is released from the platelets, tissue fluid and injured tissues. Since all other factors are normally present in the serum the chain of reaction, once initiated, proceeds until the clot is formed. Calcium ions, probably acting as an enzyme activator, appear to be essential for several of the reactions.

The formed clot is a reticulum of fibrin strands, the interstices of which contain serum, blood cells and platelets. The clot seals the breach in the blood vessel and thus controls haemorrhage. The absence of one or more of the factors in the coagulation mechanism may be responsible for a defect in clotting and may cause post-operative haemorrhage.

Shira (1958) points out that while the above mechanisms achieve haemostasis this can only be regarded as "temporary haemostasis". "Permanent haemostasis" being achieved only after organisation of the blood clot.

3. CLASSIFICATION OF HAEMORRHAGE

Various classifications of haemorrhage are presented in the literature. Those most frequently used are as follows:

(a) Classification based on time of occurrence in relation to the time of surgery.

(i) Primary haemorrhage occurs as a direct result of the operative procedure. This type of bleeding is initiated by the severance of a blood vessel. In a small blood vessel primary haemorrhage frequently ceases spontaneously due to vasoconstriction. Primary haemorrhage should be controlled during the operative procedure.

(ii) Intermediate or reactionary haemorrhage occurs a few hours following surgery. Post-operative bleeding in this period is due to a failure of maintenance of temporary haemostasis effected by vasoconstriction and coagulation. Intermediate haemorrhage may result from the following:-

(1) premature removal of pressure dressings allowing the blood pressure to dislodge the clot which no longer has the support of the dressing.

(2) increase in blood pressure. During the surgical procedure, especially if a general anaesthetic is used, haemostasis

may be aided by a fall in blood pressure. As the patient recovers the blood pressure returns to the normal level and may cause post-operative haemorrhage. Elevation in blood pressure may also result from physical exertion or the use of stimulants.

(3) mechanical disruption of the clot by too vigorous rinsing or trauma from hard foods may result in haemorrhage.

(4) vaso-dilatation of constricted blood vessels following the use of vaso-constrictor drugs may result in post-operative haemorrhage (Shira, 1958; Croker, 1952; Archer, 1961).

(iii) Secondary haemorrhage occurs after twenty four hours. Generally secondary haemorrhage is caused by infection of the surgical site which results in lysis of the blood clot by bacterial enzymes or erosion of blood vessel walls, with subsequent haemorrhage (Archer, 1961; Croker, 1952). Helmore (1958), Ricker (1949) and Thoma (1963) note that secondary haemorrhage may result from sloughing of the soft tissues. Sloughing may occur due to trauma of the tissues during the surgical procedure, infection or poor suturing of the soft tissues. Sutures that are too tight or too closely placed may result in tissue necrosis and sloughing of the wound.

(b) Classification based on the type of blood vessel.

(i) Arterial haemorrhage is characterised by spurting of bright red blood.

(ii) Venous haemorrhage manifests as a slow continuous flow of darker blood.

(iii) Capillary haemorrhage appears as a continual oozing of

blood. Often in this form of haemorrhage it is difficult to locate individual bleeding points.

(c) Classification based on the site of bleeding.

Archer (1961) states that in the oral cavity bleeding occurs either from (i) bone or (ii) soft tissues. This affords another form of classification of haemorrhage.

4. CAUSES OF ABNORMAL BLEEDING.

The causes of prolonged haemorrhage following oral surgical procedures may be due to either general or local factors.

(a) General causes: brief mention must be made of some of the general factors that might lead to excessive post-operative haemorrhage. The general causes of abnormal bleeding may be subdivided into defects of the coagulation and of the vascular haemostatic mechanisms.

(i) Defects in the coagulation mechanism: disorders of coagulation in this category include haemophilia, Christmas disease and the group of conditions referred to as the thrombocytopoenic purpurae. In all these conditions thromboplastin production is deficient thus retarding or preventing the conversion of prothrombin tothrombin and, further, the completion of the coagulation reaction. Haemophilia and Christmas disease are hereditary conditions while two forms of purpura, idiopathic and secondary, are recognised. The latter form may occur secondarily in such conditions as pernicious and aplastic anaemia, leukemia, carcinoma, radiation damage and toxaemia following certain infections (Naylor and Moore, 1962; Shafer,

Hine and Levy, 1960; Thoma and Goldman, 1960; Shira, 1958).

Prothrombin deficiencies may lead to abnormal post-operative haemorrhage. This may occur in advanced hepatic disorders, Vitamin K deficiency and in those patients taking large quantities of salicylates and certain anticoagulant drugs (commonly used prothrombin depressing drugs Dicoumarol, Phenindione (Dindevan), and Ethyl Biscoumacetate (Tromexan)). Heparin, a drug generally used for short-term anticoagulant therapy, causes inhibition of thrombin formation. Many authors consider that oral surgery on patients receiving anticoagulant therapy is contra-indicated due to the problem of excessive haemorrhage (Naylor and Moore, 1962; Scopp and Fredrics, 1958; Ziffer et al, 1957; Schmitt et al, 1960; Thoma, 1963). On the other hand Shira et al (1962) and Berman and Wright (1961) state that minor oral surgical procedures can be safely carried out provided that care is paid to avoid unnecessary trauma and strict attention is given to haemostasis. This reviewer considers that, when a patient is receiving anticoagulant therapy, consultation with the patient's physician is advisable prior to surgery being performed.

(ii) <u>Defects in the vascular mechanism:</u> haemorrhage may occur due to congenital abnormalities of the vessels or to increased permeability or fragility of the vessel walls. Hereditary haemorrhagic telangiectasis and von Willebrand's disease are two familial conditions in which a vascular abnormality is manifest (Naylor and Moore, 1962; Shafer, Hine and Levy, 1960). Certain infectious conditions, such as scarlet and typhoid fever, produce

toxins which may exert a deleterious effect on the vascular endothelium resulting in an increased haemorrhagic tendency (Cheraskin, 1959). Advancing age leads to an increase in capillary fragility and thus post-operative haemorrhage may occur in elderly patients. This increased tendency for post-operative haemorrhage often manifests as ecchymosis which is not an uncommon complication in older persons (Shira, 1958).

Nutritional factors play an important part in the maintenance of endothelial integrity (Cheraskin, 1959). For example, deficiencies in ascorbic acid result in faulty synthesis of the cementing substance of the capillaries which causes an increased permeability of the vascular walls (Shira, 1958; Cheraskin, 1959). Any abnormality in the vascular structure may lead to prolonged bleeding following oral surgical procedures.

(b) Local Causes: in practice, haemorrhage due to local factors is much more commonly encountered than haemorrhage due to the bleeding diatheses.

Operative trauma is a well recognised aetiological factor. Laceration and contusion of the gingival tissues may result in necrosis and sloughing of the mucous membrane, leading to haemorrhage from the underlying blood vessels (Naylor and Moore, 1962).

Berlove (1959) considers that over-vigorous curettage of bone during surgery may open up deep blood vessels which may cause excessive post-operative haemorrhage. If a healing surgical wound is traumatised, for example, by coarse food, disturbance of the

regenerating tissues may initiate post-operative bleeding.

Calculus deposits, food debris and dental caries cause inflammation of the supporting structures of the teeth. This in turn leads to an increased bleeding tendency of these tissues as a result of the local inflammation. Likewise, apical extension of an infection causes inflammatory changes of the periapical tissues with resultant increase in vascularity. Injury to these inflamed, vascular tissues may result in profuse haemorrhage. Infection of the surgical wound is another cause of post-operative haemorrhage. The bacteria may produce toxins which cause lysis of the blood clot or may cause ulceration of vessel walls with subsequent secondary haemorrhage (Berlove, 1959; Naylor and Moore, 1962).

5. ECCHYMOSIS AND HAEMATOMA.

(a) Ecchymosis is not an uncommon complication of oral surgical procedures and, while not serious, frequently causes alarm to the patient because of the accompanying discolouration of the tissues. Ecchymosis results from a continued post-operative subcutaneous or submucosal extravasation of blood which is finally arrested as pressure builds up in the surrounding tissues. The accumulated blood then undergoes coagulation and this produces discolouration of the overlying soft tissues. In the face, such ecchymotic areas may spread to become dispersed in the cervical region by way of the fascial planes (see photograph page 69.).