C J Mieny, U Mennen: Principles of Surgical Patient Care - Volume II

Chapter 6: Plastic Surgery

Chapter 6.1: Techniques of Excision and Debridement

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Introduction

It has been repeatedly shown over the years that dead tissue provides an excellent media for bacterial proliferation. This chapter is based on that premise.

Definitions

Debridement

This term has a French origin. It means removal of foreign material and devitalised or contaminated tissue from or adjacent to a traumatic or infected lesion until surrounding healthy tissue is exposed.

Excision

Simply means removal by cutting.

Another related term is *e'pluchage* which means removal of the contused and contaminated tissue of a wound.

There are only two main areas which demand the doctor to debride a lesion: viz a traumatic wound including burns, and sepsis.

Occasionally fungating wounds from malignancy have been excised - just short of amputation.

The major problem has been to define with precision what dead, devitalized tissue looks like, particularly the skin. The major guiding principle is that living tissue bleeds when cut, while dead tissue does not.

This should only be a guiding principle, for not all non-bleeding tissue is dead, neither will all bleeding tissue necessarily survive. To illustrate, the presence of a pulse and sensation with damage to all venous drainage systems will in time lead to death and gangrene to that part.

Various attempts have been made to determine the viability of skin after trauma. Intravenous fluorescein injection, thermography and radioactive isotopes and dye clearance have been employed.

Classification

Broadly, debridement/excision can be classified into two main groups:

Primary Debridement

Done ideally within the first 12 hours after the incident. Delayed primary debridement has been done outside this initial period where the wound was cleaned and packed. An old septic finger done for the first time (say one week old) would constitute a primary debridement.

Secondary Debridement

(The "second", "third" or even "fourth" look.) Here there has been an initial debridement. Because of the difficulty in removing all dead tissue the first time, a "revisit" to the lesion is made to make sure all dead tissue is removed.

The general and ultimate aim of a debridement would be to achieve a clean, healthy wound that will heal and restore structural normality as best as possible without complications.

Debridement Following Trauma

General anaesthesia is ideal, although local anaesthetic has been used. However, there is always the fear that during injection bacterial dissemination may occur. Local infiltration may also not be adequate for larger areas.

In general, tourniquets are not used. They tend to lull the surgeon into a false sense of security regarding haemorrhage. It is also difficult to see which tissues are dead and which are viable.

Clean the wound with running water and soap. Though this is a non-physiological solution. The alternative is mild detergent i.e., iodine or hibitane in water on surrounding skin, or better still, ringer's lactate in the wound. This process is carried on till the wound is literally clean. Scrubbing wounds with brushes should be discouraged. Foreign material like soil, gras, etc. are mechanically removed and picked out of the wound.

The presence of grease and tar near or in a wound can be removed by ether or acetone. This should be done carefully without damaging the tissue.

After the surgical preparation is completed, the wound should be meticulously examined and explored to determine the extent of the damage. For this purpose wider incisions may have to be made joining or extending from the wound. Oozing is controlled by pressure and brisque haemorrhage by fine ligatures. Care is taken not to compromise the already damaged tissue further. Once the extent of the damage has been determined, excision follows.

Skin

The anatomy of the blood supply to the skin is relevant at this stage. The major layers of skin and underlying tissues are the epidermis, dermis, subcutaneous tissue, fascia, muscle and bone.

Blood vessels supplying the skin are the:

- direct cutaneous system
- the musculocutaneous perforators and
- fasciocutaneous system.

These arterioles give branches to form a network sandwiching the dermis as the deep dermal plexus and the superficial plexus. The deep plexus supply branches to the fat cells and hair follicles below as well as the sweat glands. From the above it can be understood that if the skin is degloved with the subcutaneous tissue all its source of blood supply is disrupted, and chances of recovery are zero.

Degloving

This can be obvious or "hidden". Obvious degloving implies skin that has literally been ripped off from the underlying tissues. It is usually from proximal to distal, i.e., from midforearm to fingers in the whole circumference of the limb. This skin might be brought separately with the patient or it may still be hanging by loose bits and pieces of tissue. This skin is usually dead, and avascular and it often serves no purpose to reattach it. However, it might be defatted and used as a full thickness skin graft, if it is deemed clean enough.

"Hidden" Degloving

This is commonly seen when a turning wheel of a vehicle rolls over a limb, say a leg. It detaches the skin off the underlying tissue circumferentially. Though the skin may not be broken, it is usually completely devitalized. This skin is also doomed to necrose. Excision oif the skin should continue till normal bleeding skin edges are reached. A sharp knife and fine toothed forceps are used. Cutting with scissors should be avoided for fear of its crushing effect to the edge. Primary split skin graft can then be done or this can be done at a later stage.

Burns affect large areas of skin. This dead skin usually does not bleed, and should be excised.

Avulsed skin flaps that are distally based are likely to fail. If the wound is clean, these can be sutured back and, during the second look stage, assessed for viability.

Sometimes long skin flaps may carry major vessels along them. In this case they should be reattached as survival chances are better here. Special mention should be made about foot sole skin. No other skin seems to be adapted to carry weight. Therefore conservatism should be the rule here whenever possible. All other skin replacements to the foot are too fragile and lack sensation.

Where the possibility of suturing exists, skin excision should leave edges perpendicular to each other to facilitate good approximation and healing later.

When excising skin, only up to 3 mm of normal skin should be removed. If ever in doubt it is better to leave alone and review at the second look stage, usually after 36 to 48 hours.

Puncture Wound

This should be excised and thoroughly cleaned. An elliptical excision is the usual one made as it facilitates suturing. Deeper tissue should be explored as serious injuries have often been discovered when handling them was no longer optimar, i.e., a puncture wound on the wrist front may have a lacerated *palmaris longus,* median nerve and one or more of the superficial and deep flexor tendons.

Fat

Devascularized fat rots quickly. It should therefore be washed out or excised because it forms a good medium for bacterial sepsis.

Fascia

If devitalized, it should be removed to within 5 mm of normal tissue. Fascia that i stense and encasing a tight compartment should be incised (fasciotomy). This is done even if there is no wound. The presence of impending or full-blown compartment syndrome is indication enough for a fasciotomy.

Haematoma

This represents a space occupied by dead tissue and should be removed. After evacuation the feeding vessels may be seen and it should be tied off. Otherwise, the space is laid open to heal by secondary intention.

Tendons

Handle with care. Avoid crushing. In clean wounds these are sutured. Never leave exposed for they perish fast. In non-optimal conditions, clean, tag, and cover with skin or muscle.

Nerves

Conditions are frequently not ideal for primary suturing. Dead frayed portions should be excised. Save blood supply running alongside nerves. Tag and cover for later suturing or grafting. A microscopic excision is usually helpful in determining normal tissue in nerve. Secondary procedures are usually done in 7 to 10 days.

Bone

Attempts are made to remove as little bone as possible. Only small, loose, denuded fragments should be washed out or removed. Larger fragments are left in situ especially if they contribute to stability of the fracture. All other fragments attached to muscle or periosteum should be left. Bone should be covered by periosteum, muscle or skin whenever possible. Otherwise it dries up and parts of it slough away. In general, open fixation is not done in open fractures. In exceptional cases soft tissues tend to heal quicker if there is stability - even if minimal.

Muscles

Dead muscle is good soil for bacteria. The one type of sepsis unwanted is clostridial infection which can be fatal in a fairly short space of time. All devitalized muscle should be excised using the principle of the Cs:

- Circulation - living muscle bleeds when cut
- Contractility - living muscle contracts when pinched
- Colour - living muscle glisters and is redish brown in colour.

Traumatized muscle which is left and where sepsis does not occur (if at all) will form a scar. This leads to loss of bulk, adhesions from loss of gliding mechanism inherent in skeletal muscles, reduces contractility and subsequent diminished function. On the other hand, infected muscle can easily lead to uncontrollable sepsis and death. The formed appears a better alternative. Therefore muscle that is devitalized should be ruthlessly excised and whenever in doubt about muscle viability, cut it out.

Blood Vessels

In closed injuries diagnosis of vascular compromise can be very difficult to make. All existing diagnostic modalities should be employed as soon as this is suspected. Clinical examination, checking puls proximal and distal to the suspected area, should be thorough. Doppler, angiography and later exploration have all proved useful. The presence of a pulse or its absence does not exclude or confirm injury to vessels.

Vascular injury types can be:

- spasm
- complete rupture
- intimal rupture
- thrombosis
- puncture holes
- compression by external pressure

The exact problem should be identified. Enough visualization should be the rule - this may call for further dissection distally and proximally. Vascular clamps are used. Avoid clamping with mosquito forceps, etc. Dead tissue is carefully trimmed until normal intima and wall are visualized. Local heparin and/or systemic heparin are used. Embolectomy may be

necessary. The ends are then sutured, remembering to avoid tension. If undue tension is present, a graft should be considered. Graft if gap is greater than 2 cm. Techniques of vascular surgery should be employed at all times. Repaired vessels should always be covered with soft tissue like muscles or skin.

Where vascular injury is associated with a fracture or a disrupted joint, it is generally accepted to stabilize the disruption first. Vascular surgery is delicate and often takes long. What a waste to spend many hours repairing a vessel, only to have the suture line dismantled by an unstable fracture!

Joints

Penetrating injuries to joints may appear innocuous at first. Left alone, the result is often disastrous. For this reason and the fact that open synovial membranes lead to sepsis and adhesions, injured joints should be debrided. The joint is exposed widely and excision of all devitalized tissue is done.

All loose and foreign bodies are removed. Osteochondral fractures are repaired and fixed appropriately. These procedures are preceded by meticulous washing and irrigation as described above.

Before skin closure tha capsule and synovial membrane are meticulously closed to prevent leakage of synovial fluid. This will result in fistula formation. If the capsule is deficient and difficult to close, a fascial graft/patch can be done. Prophylactic antibiotic is an adjunct treatment.

Besides trauma, joints have been debrided for osteoarthritis, degeneration and degeneration following repeated haemorrhage (haemophilia).

Routine arthrotomy is done. All debris is removed. This includes degenerated synovium, cartilage, bone and in joints like the knee, menisci may be removed too. Washing is done, haemostasis effected. The large raw area may make conventional haemostatic control impossible. In these cases fibrin-seal technique may be employed.

Bowel

Bowel "excision", usually called resection, is done for blunt, open and sometimes vascular insult. Leaking bowel or dead gangrenous bowel usually ends in death if left unattended. At laparotomy, the abdomen is washed of blood and soiling. The site is identified. Arcades are examined carefully before segments are removed.

The remaining segment should receive a reasonable sized vessel which will not be compromised after dividing the mesentery. The anastomotic end should not be cleared of mesentery for more than 0.5 cm.

Mesentery should always be divided between clamps/ligatures. The remaining bowel is then either sutured in the routine way or exteriorized, as the case may be. If in doubt of bowel viability the abdomen may be closed and re-exposed in 24 hours time.

Brain: Debridement

The regeneration capability of the brain is very limited, therefore aggressive debridement may end up with the patient being a cabbage. A burr-hole is made after a careful documentation of clinical finding as well as X-ray and scan findings.

Foreign bodies are gently removed if easily accessible. The tract or remaining cavity is gently irrigated and sucked; bleeding is controlled.

This procedure is carried out in cases of brain abscess as well. Aspiration and irrigation are done till the aspirate is clear. Antibiotics are used, and the dura repaired.

Debridement Following Sepsis

The notorious invasion of muscle by clostridial infection frequently needs radical debridement. Muscles, fascia, fat and subcutaneous tissues may all be affected. This condition is a local as well as a systemic disease. On many occasions it ends fatally.

Local management comprises wide excision of the area. Work along limb or muscle plane axis. Fascia is opened wide or excised if diseased. Bulging muscles are removed if dead. Continue excision till a normally bleeding operative site is reached. The wound should be left open, packed with wet packs, and larger bleeding vessels are controlled by ligation. This wound heals well by secondary intention. It may need secondary suturing or grafting later.

The systemic disease is managed by general supportive measures and antibiotics. Renal failure is prevented, ventilatory support given as necessary and an aggressive nutritional support, orally or intravenously, should be given.

Early diagnosis is the key to successful treatment. X-rays may help in the diagnosis by detecting the presence of gas in tissues.

In necrotising fascitis all necrotic fascia, dead skin and muscle are removed. Meticulous dissection to save nerves, larger vessels and especially perforating nutritive vessels to normal skin and subcutaneous tissues should be carried out. The areas most affected are the limbs, perineum and abdomen. Limbs are usually spared. Secondary debridement should always be done under anaesthesia.

Debridement by Using Maggots

Maggots were introduced into wounds to eat away necrotic tissue. This was a successful method, for an average load of maggots would copnsume 10-15 g of necrotic tissue per day (loads varied from 200 to 600 maggots). Several theories were advanced to explain the mechanism of action of maggot debridement:

- increased alkalinity in wounds
- destruction of bacteria in guts of maggots
- enzymatic liquefaction of necrotic tissue
- mechanical cleansing of bacteria by serous exudate caused by irritating effect of maggots in wounds
- secretion of agents like allantoin, ammonia and $CaCO_3$
- allantoin has long been known to have a healing effect on wounds.

The disadvantage of this method was mainly the irritating and tickling or itching sensation caused by worms crawling in the wound, skin or clothing of the patient.

Comment

Debridement

R Grabe

As regards debridement following trauma, I would suggest that it is mandatory to use a theatre hose connected to a tap. This will ensure a larger flow of water and contribute more effectively to the mechanical cleansing of the wound.

To remove the grease within the wound by ether or acetone will cause more damage to the tissue by these chemicals then the tar or grease in the wound itself. Nothing more than water and antiseptic soap should enter the wound.

The surgeon should scrub as for a major operation and put on surgical gloves before doing a debridement. The wound should be covered with large sterile swabs while the surrounding skin is scrubbed and cleansed. After this has been done, the surgeon should carry on with irrigation of the wound itself. This is followed by the formal debridement operation and the surgeon should scrub again and put on gloves and gown as for a major procedure.

Even large fragments of bone totally devitalized should be excised and not left in the wound. Leaving large fragments contribute to secondary infection, and these fragments do not really serve any purpose. In these specific cases, the healing of soft tissue is paramount and is encouraged by the use of an external fixator.

Chapter 6.2: Closure of Wounds and Materials for Suture

A Lamont

Introduction

Many wounds never need to be sutured, they heal spontaneously because they are small and very superficial.

Definition

Wound suture is the temporary mechanical stabilization of tissues in apposition so that disruption to the wound healing process is prevented.

Indications for suturing are those wounds where the tissues need to be immobilized in contact because of the tendency to separate due to surrounding elasticity or mechanical factors such as epithelial surfaces near to joints. This process will only be successful o rfree of complications in clean, healthy tissues shortly after wounding or disruption.

The contra-indications to wound suture are important and are stated briefly as they have been discussed in a preceding chapter. These include:

- acute wounds where necrosis or infection is predicted

- subacute or chronic wounds which are infected and/or contaminated by foreign material or necrotic tissue

- defects in tissue in which tissue closure will cause excessive tension

Technique

After thorough debridement, where necessary, and cleansing, adequate haemostasis is established. The various structures must now be brought into apposition. The closure of the skin and subcutaneous tissues will be discussed in detail as these are the commonest tissues to be sutured. The suture of specialized tissues and structures will also be considered.

The deep fascia, if divided, may or may not be sutured. If it gives support to the overlying skin it will help take the strain during healing if it is sutured. It is wise to close the structure if leaving it divided will lead to a large cavity or "dead space" where haematoma or seroma may form. Small holes in the deep fascia over muscle compartments must be enlarged or closed to prevent herniation of muscle. The deep fascia over muscle compartments must not be closed if it will lead to raised pressure within the muscle compartment i.e., causing the so-called "compartment syndrome".

Fat sutures are controversial. In general, fatty tissue has a poor blood supply and sutures easily cause necrosis so that they are perhaps best avoided. Scarpa's fascia in the abdominal wall is of some use as it lies within the fatty and can take sutures well. A minimal

number of sutures should be employed to bring the tissues together. More harm may be done by leaving large volumes of suture material in tissue with a poor blood supply.

The most useful way to use subcutaneous sutures is to include the deeper layer of the dermis. This will ensure good support to the overlying skin where this is necessary. It is best to place the sutures so that the knot is buried away from the dermis and epidermis, i.e., start the suture away from the skin surface and end deep.

Suturing the Skin

If a semi-fluid substance is bound by a flexible band, the area within the band will tend to take the form of a circle and the higher the pressure which this band exerts on the entrapped area, the stronger the circle tendency becomes, as a circle is the shortest perimeter for a given area.Most tissues are semi-fluid substances, to a greater or lesser extent, and therefore all suture materials will tend to form circles when used to bind these tissues. Added to this is an important time factor, in that once the suture is tied, it is relatively inelastic around a volume of tissue which will usually undergo some degree of swelling postsuture. The pressure within the suture will therefore rise, thus increasing forces which form circles.

A further matter of great importance is that if the pressure within this bound area exceeds the tissue perfusion pressure, either initially or later because of swelling, necrosis will result in the tissues being thus bound. This area of necrotic tissue is a focus for infection and it can be said that the cause of so-called stitch abscesses is more iatrogenic than due to organisms or the nature of suture materials.

Avoiding this complications requires a great deal of self-discipline on the part of the surgeon, especially as it is human to attempt to achieve better closure, improved healing and sounder apposition with greater physical input i.e., tightes sutures.

If circles are to be the inevitable and most natural outcome of sutures in tissues, then it is obvious that we should place our sutures in such a way as to form circles at the outset, thus causing minimal deformity within these tissues. With curved needles and a little manipulation this is a relatively easy matter.

The point of entry into the skin is near to the wound edge and the needle is directed so as to take a semi-circle of tissue i.e., epidermis, dermis and some subcutaneous tissue. It will be found that it is best to confine the suture largely to the dermis taking relatively little of the subcutaneous fat. It will be found that it is best to confine the sutures largely to the dermis taking relatively little of the subcutaneous fat. It is usually unwise to attempt to pass the needle through the second half of the wound in one movement, but rather to regrasp the needle and approach the other edge of the wound as a second movement. Here again, the needle is directed so as to form a semi-circle in the tissues coming out on the skin surface close to the wound edge. When the know is tied, two semi-circles come together in a relatively tension-free circle with perfect skin apposition.

Skin Overlap

Why, when simple suture is attempted, is skin overlap such a common occurrence? The common mistake is that the needle enters the skin too far from the wound edge and is directed through the tissues in a direction which is more horizontal than vertical in relation to the skin surface.

When the suture is tied and the circle forms, the skin surface is too long to fit into the circle without folding and overlapping. To avoid this, the mattress suture is employed to hold up the edges of the wound. When the suture is tied, it will again attempt to form a circle and not a square as erroneously shown in so many diagrams. The pressure on the skin surface is highest at the point of entry and exit and if tight enough will cause skin necrosis. The common scars seen in abdominal wounds are the central surgical scar with suture marks at right angles to it, widest at the outer limit away from the central scar. This is because the skin cannot fit into the circle. Mattress sutures are not the solution to the problem. Well-placed simple interrupted sutures will give perfect apposition of skin edge with minimal pressure on the skin itself and even if marks are formed, the skin below the suture is of minimal length.

The thickness and texture of the skin varies a great deal on the body surface. Generally the thicker the skin, the bigger the semi-circle i.e., on the back skin 3/0 or 4/0 suture material is suitable. Eyelids consisting of very thin skin require 6/0 or 7/0 suture material with very small semi-circles. Other forms of interrupted suture such as horizontal mattress sutures, etc have similar disadvantages to those of the vertical mattress suture and further discussion of these is not justified as they need seldom be used.

Continuous Sutures

Here again a simple continuous suture placed in the tissues like a coiled spring in circles with relatively little suture material on the skin surface will give good apposition with an acceptable scar. Continuous mattress sutures and the like should not be used for the reasons stated above.

Subcuticular Sutures

This term indicates a continuous suture within the dermis just below the epidermis and is popular because, if an absorbable material is used, the patient is spared the rigours of suture removal. This is a highly successful and atractive form of wound closure and the best results are obtained as follows:

- Subcutaneous support should be good so that there is minimal tension on the subcuticular suture in the skin edges to be apposed.

- Regular semi-circles of tissue should be taken in a horizontal plane alternating from side to side of the wound.

- The material must pass across the wound at right angles to the wound direction so that when the suture is pulled up, a since curve is formed within the dermis. If the suture

jumps too far ahead or goes too far back, the skin edges are distorted in relation to each other.

Knots

Knots may be tied by hand or with instruments and both methods should be mastered in such a way as to be able to tie consistent square reef knots followed by a locking half-knot. The main aim is to avoid the so-called tumble "granny" knot which slips. A double throw on the first half-knot will help to prevent its slipping before it is secured by the second half-knot. If the wound still separates under a double half-knot, this should serve as a warning of excessive tension.

Suturing of Special Structures

Intestines

A watertight closure must be achieved to avoid chemical and bacterial contamination of surrounding areas and spaces. Within this closure healing of mucosal and muscle layers must take place to restore the integrity of the tubular structure. The inner layers of mucosa and muscle are accurately opposed to encourage rapid primary healing. It is unwise to attempt water tight closure here as this may jeopardize blood supply and lead to necrosis, breakdown and fistula. These layers generally heal so rapidly that a good closure is rapidly attained. The watertight protective layer is usually placed outside this first layer of sutures in the serosa or surrounding tissues outside the muscle layer where blood supply is a less critical factor. More than two layers is not recommended as this can cause infolding of too much tissue bulk with resultant obstruction or stricture within the lumen.

Nerves

The frustrating analogy facing the surgeon confronted by a severed nerve is that of a cut telephone cable containing thousands of wires and which can be repaired only by joining the outer plastic coating. There is no hope that any but a very small number of telephones will work. Fortunately nature is forgiving in that if the outer coat is repaired failry accurately, the telephone wires will repair themselves. There is evidence to suggest that a degree of chemotactical interaction can make it possible for axons growing from the proximal cut sheath to select between sensory and motor sheaths in the distal end. Added to this is the CNS's ability to compensate for nerves which reach the wrong end-plate or end-organ. Accurate suture is obviously of prime importance in assisting these processes. This is achieved, to some extent, by the recognition of vesicular patterns within the nerve and the presence of blood vessels on the nerve surface. Such work can be done properly only by using magnification and an operating microscope is mandatory for good results.

The question of epineural versus vesicular repair i.e., repair of the separate bundles within the larger nerves, is dependent in the end on the degree of disruption that growing axons experience in finding the appropriate tubes in the distal nerve. If a great deal of mechanical distortion takes place during vesicular repair, more harm will be done than by a good epineural repair, i.e., repair of the outer fibrous lining alone. The repair must be done

with very fine suture material with no tension at the suture site and no overbunching of the contents of the fibrous linings by overzealous suture.

Tendons

Tendons are highly mobile parts of the anatomy, particularly within the tendon sheaths of the palms and fingers where they are not only mobile within a relatively immobile sheath, but isolated from tissue whence a rich blood supply could come. Tendons fortunately do not need a rich blood supply because of their relatively low metabolic activity. The blood supply usually enters the tendon vie mesenteric connections with the surrounding area across the synovial space. The synovial sheath is essential for allowing a gliding action of the tendon. In the case of the tendons in the hand these attachments are very tenous in the form of strings called vinculae. Within the tendon the blood vessels are centrally located running along the length of the tendon.

This whole delicate balance of function and blood supply is upset by trauma and the need for mechanisms of healing. The blood supply is easily disrupted and any damage to the tendon surface or synovial lining, or both, will tend ot cause adhesions between the tendon and the sheath. This results in characteristic loss of function. The suture of damaged or cut tendons must comply with the following requirements:

- it must be as atraumatic as possible
- all "raw surfaces" should be avoided
- the tendon blood supply must be preserved
- the tendon sheath must also be repaired with atraumatic methods.

Problems encountered are:

- the tendon ends are often difficult to find
- the damage to the tendon and sheaths may be excessive and not easy to repair
- closure of the sheath after opening to find and repair tendons can be difficult
- the technique of atraumatic tendon suture is demanding.

Best results are obtained with a laced suture of 4/0 nonabsorbable material in the tendon to pull it into position, followed by a 6/0 continuous suture around the cut edges to neaten it. The sheath must be repaired in its entire length to avoid contact between the tendon and tissues outside the synovial lining. Magnification in the form of an operating microscope will again improve results.

Blood Vessels

From Poiseuille's law for fluid flow in a hollow tube

$$BF = (P \times D^4 \times c) / V \times L$$

it can be seen that the diameter of the vessel is by far the most critical factor. It is now possible to suture vessels from 0.5 mm in diameter as well as the larger vessels. The

diameter of a vessel becomes a significant factor when the vessel is less than 3 mm in diameter.

Muscle

Muscle heals so well that suture is seldom necessary. Even when a muscle is cut across completely, it will heal by fibrosis with return of a surprising degree of function. Any attempt at suturing which will cause further damage to muscle with necrosis, must be avoided as muscle once lost is not replaced. Furthermore, tight sutures within the muscle are painful and are responsible for postoperative morbidity. When muscle layers must be opposed, this should be done with loose sutures within the muscle or preferably suture of the fascialo investing layers only.

Suture Materials and Instruments

Threads

Absorbable Synthetic Threads

The advent of polyglycolic acid (Dexon) and later polyglastin 910 (Vicryl) was a significant advance for surgery. The advantages were their increased strength, minimal tissue reaction and longer absorption time. These are usually presented as braided sutures. Early disadvantages with knot stability and visibility were solved by coating the suture and by introducing colours into the material. Initial claims that sutures on the skin did not need removal proved ill founded as these materials stay in situ too long in the upper dermis and epidermis and must be removed just as non-absorbable sutures. The degree of tissue reaction is very low and any reaction is more likely to be caused by surgical technique and infection.

A more recent material, polydioxanon (PDS), has an even longer absorption time. It is presented as a clear or coloured mono-filament thread and may be successful in preventing scar spread. It is strong, but tends to "pigtail" if treated roughly. Avoiding this disadvantage may have advantages for the tissues which will be handled with less trauma.

Absorbable Natural Threads

Surgical practice was for a long time dependent on catgut prepared from intestinal submucosa of sheep. The plain form had a very short absorption time while the treatment of the material with chromic salts have a prolonged tensile strength of 4-7 days. The major disadvantage of these materials is the gross tissue reaction to a foreign protein substance. These materials are still favoured for their excellent handling characteristics. However, the short absorption time, the limited shelf-life, their weakness and particularly the tissue reaction make their continued use difficult to justify.

Non-Absorbable Synthetic Threads

Nylon was the first of many plastic materials prepared as monofilament as well as braided threads. The advantage of braided thread is its greater flexibility. It is softer and

usually knots more easily and securely. The "memory" in the monofilament material is a disadvantage.

Because of their inert nature there is seldom any adverse reaction and the monofilament designs show less tendency for infection.

Non-Absorbable Natural Threads

The disadvantage fairly consistently quoted is the wick action due to its braided form which allows the spread of infection from epithelial surfaces into deeper tissues. It is probably unwise, with the availability of alternative absorbable materials, to leave silk sutures in deep tissues as they do act as potential foci of infection which can cause complications even long after healing has stabilized.

Clips, Staples and Other Mechanical Devices

They are very convenient and cost-effective.

Adhesive Tapes

This is a useful atraumatic way of establishing primary healing where no tension is present.

Tissue Adhesives

Unfortunately, most substances are toxic or cause foreign-body reactions. The ideal is a bio-degradable adhesive with a good tensile strength. The nearest preparation available is a fibrinogen preparation with its catalyst which is applied to surfaces as for skin grafts or adhesion of large flaps, or for the lower tension aposition of nerves in cable grafts. When used for the correct indication, this method has good haemostatic and adhesive properties with good clinical results. Due to its low tensile strength and expense it is proving slow to gain widespread application.

Instruments

The tissue should be so handled as to cause minimum iatrogenic trauma. The most common offenders are forceps and tissue-holding forceps. Fine-toothed forceps are recommended if used delicately.

Comment

Closure of Wounds and Materials for Suture

K Bofard

"It doesn't matter what you do, so long as you do it properly."
Prof Lee McGregor

All wounds will heal, given the right circumstances. All that is necessary is the accurate apposition of tissues. When wounds fail to heal, it is usually due to:

- Ishaemia. This occurs either as a result of too much tension, or as a result of local ischaemia, i.e. due to diabetes.

- Infection. The presence of infection is not necessarily a contraindication to suture (i.e. as in bowel) but combined with ischaemia will result in failure to heal.

- Neoplasia. Malignant neoplasia fails to heal.

Where the circumstances obtain, suturing shoul not be attempted. The wound should instead be dressed with saline, and allowed to begin healing. Once the condition of the wound has improved, it may then be possible to close - delayed primary closure, ideally carried out between the third and fifth day. This situations pertains especially in contaminated wounds, i.e. dog bites, and would apply where the initial viability of the tissues is in some doubt such as in bullet wounds. Infected wounds should be allowed, if necessary, to granulate closed rather than be sutured (i.e. diabetic lesions).

When suturing, it must be remembered that the correct tension is that which will be present at the time of maximal tissue oedema, usually around 48 hours post-injury, rather than at the time of suture.

The problem often associated with monofilament synthetic sutures as polypropylene, is knot slippage. Correctly placed, square knots should not slip. Care should also be taken not to err on the side of too many knots which could form a sizable braided foreign body. In general, five "throws" in a 2-1-2 combination will lock any suture.

Vascular and intestinal repairs can both be done using interrupted sutures and this is particularly important in situations where the lumen is narrow. Interrupted sutures allow the lumen to "stretch", whereas a continuous suture dictates the maximum circumference that a lumen can achieve.

Finally, skin closures, i.e. steristrips, should be used only if there is adequate support from the underlying tissue. Failure to do so will leave an unsupported cavity. Additionally, a gap roughly equal to the width of the skin closure should be left between each strip. This allows serum to drain. Failure to do this results in a collection of fluid between the skin edge and the strip, and a poorer scar.

Chapter 6.3: Bites

R G Golele

Introduction

Among the emergencies that a doctor may have to treat are those resulting from bites. The seriousness of these bites varies from minor scratches through prolonged morbidity to ghastly injuries which may result in the death of the victim. The occurrence of bites will vary from place to place throughout the world and is determined by such factors as urbar or rural environment, victim's age, whether it is near the coast or near a game reserve, social activities of victims concerned, etc.

This chapter intends discussing bites under the following headings:

- Human bites
- Animal bites
    - Domestic animal bites
    - Wild animal bites
    - Marine animal bites
- Insect bites.

Before detailed discussion, let us first define what we mean and understand by "bites".

Definition

According to Dorland, a bite can be defined as seizure with teeth, a wound or puncture made by the teeth or other mouth parts of a living organism.

There is an entity that is sometimes confused with bites viz. stings. This is an injury caused by the venom of a plant or animal introduced into the individual by contact or otherwise, together with the mechanical trauma caused by the introducing organ.

Human Bites

This is a very old problem through various sections of societies and age groups. However, it it commonly seen in the age group 15-30 years, i.e. adolescent and young adults. The victim is often a male.

Human bites are typically seen in two common sites, namely the face and the hand. Other areas like the neck, trunk and lower limbs have been bitten. Even more unusual sites like penile amputations have been seen. The man I saw eventually committed suicide. The bite was from a rape victim.

The bite is usually from an assailant, often upon the assailant's fist, and, not uncommonly, self-inflicted on tongue and lower lips upon falling or talking while eating. Circumstances surrounding human bites are usually fights, assault and drunkenness.

Facial Human Bites

In the head region commonly bitten sites are the:

- ear - partial or complete
- nose
- lips - lower lip commonly
- cheeks
- eye lid.

Wound type:

- puncture wounds
- linear tears
- tissue flaps still attached
- complete avulsion of an area, i.e. ear, nose and lips
- skin defects of differing sizes
- abrasions and contusions
- upon healing these lesions are always a tell-tale to an experienced eye.

A fight is often the usual history. Injured sites are finger tips, mid finger region and the knuckles (metacarpo-phalangeal joints), the mechanism being that of a blow to an opponent's teeth or an actual bite by the assailant.

Injury Types

They include:

- lacerations
- amputations - complete or partial
- penetrating teeth wounds with/without fractures
- joint penetrating wounds.

The victim usually brings the amputated part along, for it is often unpleasant to swallow!

Clinical Presentation of Human Bite

Some differences exist in the presentation of facial and hand bites by a human. They are both anatomical and cosmetic. In face bite wounds, where the blood supply is abundant, patients tend to bleed a lot and therefore delays are not frequent. Secondly, the anxiety of later cosmetic appearances drives the patient to hospital earlier, particularly if an ear lobe, part of the nose or lip is lying in the patient's hand.

On the other hand bites on the hand may sometimes look innocuous - thus fooling the patient temporarily - yet they often lead to serious infections and subsequent complications.

Hand bites tend to present in different ways:

- fresh wounds
- cellulitis
- sepsis like
    - abscesses (finger or hand)
    - tendon sheath infection
    - arthritis
    - gangrenous finger, skin or sometimes whole hand
    - toxicity and severe pain.

In my experience more than 90% of hand bites present with either cellulitis or sepsis.

Management

Some patients may not reveal the nature of the cause because of the embarrassing nature of being bitten. A good history is essential for establishing, among other things, the time interval between bite and presentation as this may alter the management. Attention should be paid to the general care of an injured patient for airway, breathing or circulation check.

Furthermore, a detailed general examination should be done to confirm or exclude:

- diabetes
- alcoholism
- steroid therapy and
- depressed immunity, as these will adversely affect the result of treatment.

Local examination should then be done to determine the extent of the damage, and the type of wound.

Intact skin is relatively easy to handle. The abrasions and contusions are simply cleaned with an antiseptic solution. The pain is relieved by analgesics.

An aggressive approach should be followed for open wounds to prevent sepsis and to provide good functional and cosmetic result.

In both face and hand injuries the initial phase of wound management is thorough cleansing, including the surrounding area.

Wound irrigation, which should be copious, remains the mainstay in the early management of bite wounds. The solution to be used is still a point of contention. Ideally one needs a solution with antiseptic and detergent properties without harming the tissue. There are many on the market, the effect of which still need scientifically controlled studies. It is generally said that you must not put in a wound what you cannot put in the eye. Normal saline, though it lacks these properties, is still widely used. Plasmalyte may be a better physiological solution.

After irrigation, removal of devitalized tissue should be done, taking care not to remove excessive amounts of tissue. Only devitalized tissue should be removed plus about 1-2 mm of tissue surrounding the wound edges. The hand and face do not have excess skin to spare, hence this precaution.

The third phase is the decision to suture or not. It is generally agreed that bites on the hand should be left unsutured. These should heal by secondary intention. On the other hand, facial wounds are usually sutured because there is a low risk of infection plus good blood supply. In addition cosmesis on the face is of significant importance. Despite the above advice, there will be grey zones where one is never sure at the outset whether to suture or not. If in doubt, it is probably wise to delay wound suturing for several days and suture later when the chances of sepsis will be reduced.

Skin grafts and flaps may be used. More than one operation may be needed.

Cartilaginous areas like the nose and ear may be a problem to handle. Small partially avulsed tissues should be reattached. Larger totally avulsed pieces, especially of the ear, can be denuded of skin and buried behind the ear for later staged reconstruction. For the nose, flaps in the neighbourhood are often used.

Prosthetic implants may also be used, i.e. for nose, ear, etc. The early management of hand bites requires irrigation, debridement, open wound treatment, elevation and immobilization. Patient co-operation is essential for good results.

Prophylactic antibiotic and tetanus toxoid are usually given when the patient is first seen. Literature is deficient somehow on prophylactic use of antibiotics done prospectively. But because sepsis rate is significant in human bite, antibiotic should be used. This should be based on bacterial isolates.

The common bacteriae cultured in human bites include *Staphylococcus aureus* and streptococcal species. However, gram negative and anaerobic types have been cultured with varying frequency. The antibiotic used should therefore include a penicillin G, plus a penicillinase resistant type like cloxacillin or a cephalosporin as an initial therapy pending culture results. To cover the anaerobes metronidazole has been used as well. For penicillin-sensitive patients erythromycin is often a good substitute.

A further adjunct therapy for treating human bites is to give anti-tetanus toxoid where there is unknown or an incomplete history of primary immunization. Syphilis and hepatitis B have been known to be transmitted by "biters" who are carriers. In such cases hepatitis B immune serum globulin should be used as a prophylactic measure and syphilis should be treated with penicillin.

Infected Wounds

If the infection is obvious, the approach would follow the routine of:

- admission
- incision and drainage
- debridement - removing all non-viable tissues
- elevation (for hands)
- antibiotics - usually those used for prophylaxis. Additional antibiotics may be added according to the results of gram stain, culture and sensitivity.

In addition factors which may compromise the patient's response should be sought and treated accordingly.

Complications of Human Bites

Hand

- osteitis
- arthritis
- stiffness - permanent functional disability
- loss of digit/hand mainly associated with diabetes mellitus
- tenosynovitis.

Face

- disfigurement from complete or partial loss of parts, i.e. ear
- multiple stage operations
- very rarely death from severe sepsis.

General

Hepatitis B, especially from human biting carriers. Prophylaxis: avoid or run from imminent assailants.

Animal Bites

Domestic Animals

It is difficult to define domestic animals today as virtually all kind of animals have been domesticated and made pets, or are cared for in captivity.

However, the dog and cat remain the commonest animals kept in the home or the neighbour's home. Horses, camels, donkeys, etc are others. The dog is the common offender, while the victim is often a child. Other dog victims include: breeders, veterinary surgeons, zoo keepers, postmen, laboratory workers and intruders.

Sites Bitten by Dogs

These include:

- head
- neck
- trunk
- extremities.

As mentioned above, most victims are under 15 years. These are ideal victims to be bitten on the face and head because they come face to face with the dog as they are of the same height as the large breeds.

Injuries Caused

Puncture wounds - usually multiple.

- Lacerations of varying sizes and depths
- Tissue loss - lips, nose, ears, fingers
- Abrasions
- Haematomas
- Fractures - skull, fingers, therefore X-rays may be necessary
- Mixed injuries.

Management

Broadly it should follow the same principle as for human bite, viz. thorough cleansing and debridement. All face wounds are usually closed primarily. Wounds on the hand and puncture wounds should be left open after debridement for secondary suture or to heal by secondary intention.

Special attention should be paid when patients are smaller children, because penetrating wounds in the skull have been described. These are easy to miss because of the hair and tendency of a puncture mark to "seal". All penetrating wounds to the skull should be enlarged, dura inspected and dural patch graft done. X-rays may not show anything.

It is also important to suspect rabies which usually presents 3-8 weeks after the bite by a rabid dog or other animals like cats, bats, etc. History is essential here. Clinical presentation is that of abnormal sensation on the affected area, or that of sympathetic overstimulation. Paralysis, especially of muscles of swallowing, follows later.

Prevention is most important for avoiding the serious consequences of this deadly disease.

Antibiotics and Bacteriology

An average dog harbours more than 60 different species of bacteria, including known human pathogens such as *Staph. aureus*, streptococcus and gram negative anaerobes. The

Pasteurella multocida is also commonly found though it is more important in cats and larger animals of the cat family.

Fungi like human blastomycosis have been reported. Antibiotics used should therefore cover these species pathogenic to man, i.e. they are basically the same as those used for human bites. Other unusual bacteria are the *mycobacterium fortuitum* which responds to rifampicin.

Complications/Prognosis

Early

- septicemia
- DIC
- rabies
- brain abscess
- haemorrhage.

Late

- special reconstructive problems, especially in face bites
- repeated surgical procedures
- scar and graft problems.

The earlier the extent of the injury is noted and the necessary initial treatment is carried out, the better the prognosis.

Wild-Animal Bites

Bites from:

- lion
- tiger/leopard
- rats
- hippopotamus (on land and in water)
- monkeys, and many others have been described.

The victims are the same as for dogs with the exception of age and those who hunt animals. The larger animals usually cause more serious injuries and these can be on any part of the body.

Injury Type

- large wounds (lacerations)
- haemorrhage
- visceral injuries
    - lungs
    - bowels
    - brain
- fractures from
    - firm bites
    - shaking manoeuvers
    - trampling.

Diagnosis is usually not a problem, but particular attention should be paid to hidden injuries (visceral), therefore a thorough examination is imperative. X-rays may be needed to establish presence of fractures.

Treatment

Treatment should follow the same principle as outlined for human bite injuries. However, the resuscitative measures will have to be intensified because of the large wounds and haemorrhage caused.

Bacteriology and Antibiotics

Here again a multitude of organisms has been isolated, including:

- *Pasteurella multocida*
- *E. coli*
- *Diphtheroids*
- *Streptococcus*
- *Staph. aureus.*

Past. multocida infections are serious human pathogens. They have caused serious arthritis, meningitis, etc. and therefore need aggressive antibacterial therapy. They are sensitive to penicillin G.

Complications/Prognosis

If the patient survives the initial trauma, the prognosis is good. Complications are:

- haemorrhage
- large tissue defects
- deformities - bone and soft tissues
- death is the most serious complication encountered.

Rat Bites

The "rat" family is large and includes rodents, mice, etc. These have been known to bite humans. Victims are usually bitten at night while asleep. Laboratory workers are the other group of victims.

Sites

Rats attack exposed areas. These include face and extremities. Sometimes many different areas may be bitten.

Wound Type

Multiple Punctures

- puncture wounds - single or many
- bruising
- haematomas
- mixed wounds
- occasionally fractures of small bones like phalanges.

Diagnosis

History and specimen (dead rat) are important.

Treatment

Good surgical cleansing of wounds is necessary. The sepsis rate in rat bites is very low. Clean wounds can therefore be closed primarily especially on the face. Doubtful wounds should be left open for later suturing or healing by secondary intention.

Bacteriology

Staph. epidermidis is commonly isolated. Other species include diphtheroids, *B. subtilis, Strep. haemolyticus.*

Prophylactic antibiotics should be given to cover the above bacteria. If wounds are already infected a cephalosporin plus a penicillinase-resistant penicillin should be given. Tetanus prophylaxis is mandatory.

Complications

Complications are very rare if appropriate prophylactic measures are taken. Diseases such as rat rabies and many plagues have been known to be transmitted by rats. These should be prevented.

Snake Bites

By definition snake bites conform more to "stings" than bite. However, some important surgical points will be highlighted. It is a very wide subject meriting its own chapter. The variants of this subject include type of snake (venomous and non-venomous), marine or those on land, geographical distribution, fanged snakes, spitting snakes, etc.

There are many types of snakes in southern Africa. Broadly, one can look at these under two major groups:

Venomous and Non-Venomous Snakes

Non-venomous snakes have no fangs, only small sharp teeth. These usually cause small lacerated wounds and teeth may remain in these wounds. Venomous snakes, on the other hand, have fangs. These fangs have grooves on them while others have canals through which the poison flows or is injected into the victim. The venom is stored in venom sacs and is released as and when required.

Venom Composition and Mechanism of Action

According to White and Goodwin, these have been classified as follows:

A. Enzymatic Proteins

- Phospholypids

Lyse cell membranes allowing easy penetration of toxins into muscles, nerves and blood vessels.

- Phosphatidases

Destroy nucleoproteins and nucleotide, i.e. phosphate compounds.

- Proteases

Contribute to fibrinolytic, antithromboplastic and anticoagulation activity of snake venoms.

- Amino-acid esterases

Initiate coagulation leading to microclots which are later lysed.

- Acetylcholinesterases

Contribute to the myasthenic effect of many snake venoms.

B. Non-Enzymatic

- Cytotoxins (haemorrhagins)

The target cells of these are the endothelia of small vessels and lymphatics. Within minutes following a bite a histamine-like reaction is observed and spreads rapidly. There is oedema which is very painful. Histamine, serotonin, kinins and prostaglandins are released. There is also local interference with coagulation. If the bite is by a larger adder, this loss of blood into compartments can continue for more than 48 hours, and may be followed by slow onset of severe blood volume deficit which may culminate in organ failure.

Cytotoxins are also absorbed immediately through the lymphatics leading to a painful regional lymphadenitis.

- Cardiotoxins

These affect nerve and muscle tissues of the heart and may cause local necrosis of tissues, haemolysis and damage to nerve cells of the central nervous system leading to cardiorespiratory failure. The above effects are commonly observed in Indian cobra bites.

- Neurotoxins

These cause a post-synaptic, curare-like effects on the neural endplates especially of the ocular, oral, pharyngeal and respiratory muscles. The period of onset of paralysis may be as short as 10-15 minutes after a mamba bite, but may be as long as 3-4 hours after a cobra bite. The effect of paralysis may last from between 12 hours from cobra bites, to 72 hours following green mambra bites and up to seven days following a black mamba bite.

Clinical Presentation

The common victim is a boy rather than a girl. Boys being more adventurous, working as herd boys and enjoy walking barefoot. Other victims include barefoot adults walking at night, and snake handlers. The bite follows after a snake is frightened, handled or trodden upon.

Sites

Usually extremities, hand and feet. Eyes, from spitting snakes. Occasionally, head and neck or trunk is bitten.

After non-poisonous snake bites onfe finds small wounds, scratches, minimal local reaction, and small teeth may be found in the wounds.

Following poisonous snake bites:

- there is usually local pain
- swelling
- discoloration
- local bleeding
- blistering
- oedema
- necrosis of skin, fascia and muscles
- conjunctivitis from spitting snakes.

Systemic Findings

- shock
- multiple organ failure
- muscle paralysis
- neurological disturbances.

Diagnosis depends on good history. If the snake is brought along, diagnosis is much easier, the only remaining problem being to identify the snake as poisonous or not, which can be difficult.

Examination should be thorough. Blood for hypoxia, anaemia, and DIC should be taken.

Treatment

The aim should be to limit venom, which reaches circulation via the lymphatic system; absorption and spreading. Massive doses of antivenom (usually the polyvalent type) 20-60 mL IVI diluted in 5% dextrose should be given intravenously. Up to 200 mL have been given. An initial test dose should always be given. This is not a guarantee that there will be no untoward reaction. Adrenalin (1:10000), hydrocortisone (100-200) should always be at hand. The venom is fixed to tissues as soon as it is injected, therefore late administration of the antivenom is not always useful.

Tourniquets applied inappropriately have caused more damage than the venom left alone would have caused. Therefore only a firm bandage should be applied. The limb should be immobilized. Immediate care should include:

- washing of excess venom with water
- remove broken teeth
- avoid incision, cautery and herbal application on the bitten area
- sedate patient (select sedatives which do not cause respiratory depression)
- avoid oral fluids for they can cause aspiration if muscle paralysis is present.

Prognosis and Complications

- septicaemia
- tetanus
- anaphylaxis
- compartment syndrome
- inappropriate fasciotomies plus tourniquet application
- failure to control aerobic and anaerobic sepsis
- death from organ failure.

Common cause of death is hypoxia and aspiration.

Prognosis may be improved if:

- the patient is bitten on extremities
- he is near a hospital
- he is an adult
- bite is incomplete
- bitten through clothing
- snake is immature, etc.

Lastly, prognosis can improve if the general public can be educated in the care immediately following a snake bite. A warning is that even a dead snake's mouth and fangs can still be dangerous if handled carelessly.

Marine Animal Bites

Water, being both a source of life and a friend to man, has always attracted people and animals. Not infrequently fatakl or serious injuries have been inflicted on man by animals living in water. Among others, the most notorious water animals, in both the sea and rivers, include the shark, alligator, crocodile and the hippopotamus. These are large animals and consequently the injuries caused are often severe. Sharks have been studied quite extensively and will be used here as a prototype.

It has been noted that sharks are inclined to bite humans at certain times of the year, viz. in the warmer seasons, and these attacks have coincided with sharks' natural food shortage. Occasionally, they have bitten in terror or when cornered. The victim is usually a swimmer or angler. Laboratory workers and aquarium workers have also been bitten.

Injury Type

As mentioned above, these are usually major injuries. The grey shark produces "clean"-cut lacerations while the ragged-tooth shrak produces ragged, dirty bites.

Fractures, vascular laceration, eviscerations and even amputations are other serious injuries caused by shark bites. Because these victims are usually bitten in water, they have to swim ashore. This results in severe exhaustion when added to the injuries and the struggle

with the shark. In addition the victim may drown, or swallow so much water that resuscitation is complicated, whereas it would have been easier to treat only the injuries.

Clinical Presentation

The injuries are easily visible. Shock and signs of drowning should be looked for. This demands a critical clinical assessment. There is usually no need for special investigations as the emergency on the beach or river bank is treated first. Special investigations will come later.

Treatment

- Prevention comes first, i.e. shark nets are often used.
- Life savers are usually taught the first-aid measures for accidents of this nature.
- Many a death has been caused by hasty transporting of a patient to hospital before proper "first-aid" measures were taken. Whenever possible, a doctor should be summoned.

Once in hospital, a more detailed assessment of the patient should be made. Intensive anti-shock treatment must be given. Wound cleansing (copious irrigation) should be done. Debridement, laparotomy and fracture treatment can then be done when the patient is in a more stable condition.

Proceed as follows:

- Remove devitalized or dead tissue.
- Remove lacerated bowels widely and do an exteriorization.
- Skin grafting can be done to cover exposed bone, nerves, tendons and joints.
- If in doubt about a wound, don't close, rather do secondary suturing.
- Several wound inspections in theatre may have to be undertaken.

Antibiotics

Prophylactic antibiotics should be used. Cultures from shark wound bites and mouths of sharks, especially the great white shark, have grown a vairety of vibrio species, viz. *V. alginolytica, V. fluvialis* and other genera. These species are sensitive to the tetracyclines and chloramphenicol antibiotics. The cephalosporin and aminoglycosides have also been used successfully. In addition, prophylaxis for tetanus should be given.

Prognosis

Swimmers, anglers and even herdboys bitten by crocodiles are usully fit, healthy people. Once they reach the hospital, the chances of recovery are usually very good depending on the extent of the bite.

Complications

The most serious is death. Others include shock, haemorrhage, drowning, infections and disfigurement.

Insect Bites

There are many smaller insects and arthropodes which bite humans. To name a few common ones:

Spiders

- Paralysis may result from a spider bite.
- Local tissue necrosis and haemolysis have been caused by spider bites.
- Abdominal crisis and respiratory failure may result.
- Spider anti-venom is available for treatment.

Scorpion Bites

In hot climates these are commonly seen at night. Pain, spasms, paralysis and even cardiac and respiratory arrests have occurred. Treatment is by a firm bandage and application of ice, sometimes for up to two hours.

Ticks

The most important effect of tick bite is paralysis - ascending type. Treatment consists of removing the thick including the teeth.

Mosquitoes

Malaria is well known in this country. Awareness of the disease is the keystone of diagnosis, prevention and treatment.

Comment

Bites

G S Fehrsen

- When there is a minor injury to a finger like a skin laceration which is becoming painful and showing signs of early infection, a first-aid treatment of dipping the finger for 10 minutes at atime in the hottest water one can tolerate with intervals of about three times, will often cure that infection.

- I think that it is impractical to give prophylactic antibiotics that are based on bacterial isolates. In the first place one is unlikely to isolate bacteria, and secondly very few people will wait for bacterial culture before giving prophylactic antibiotics. In general, prophylactic antibiotics should be based on probabilities and risk.

- I just wondered what the cost would be of giving Hepatitis B immune serum globulin tyo all the people presenting with human bites. In addition one would have to mention the possibilities of developing AIDS in the same breath today.

- I think the fact that with minor bite wounds, the damage almost always exceeds the exterior appearance of a few puncture wounds, should be emphasized. The tissue tearing or rip effect from i.e. a dog bite leaves quite a lot of damage below the skin that is not immediately visible on inspection when the patient presents soon after the bite.

- I think some mention of rabies and when to investigate its possibility is necessary in the chapter on bites. Any animal that is behaving inappropriately i.e. a wild animal behaving like a tame one or vice versa, would need to be investigated for possible rabies.

- With regard to snake bite, I think it should be emphasized that the venom progresses into the circulation and the rest of the body via the lymphatic system. Much emphasis should be given to firm compression bandages, using any available cloth at the site of injury before transport is undertaken, as a replacement of tourniquets. This will stop the flow of lymph, giving extra time.

- With regard to insect bites, I think it should be emphasized that many insect bites are erroneously treated as boils. Although there may be fluid collection and a lot of tissue reaction, incision and drainage may not necessary improve the matters. The surgeon should also be alerted to the extensive skin necrosis that can result after certain insect bites. These bites should not be treated lightly because of the debilitating effect of such extensive skin necrosis. Early immobilization of the limb might be a factor in preventing further damage.

Chapter 6.4: Prophylaxis of Gas Gangrene and Tetanus

C van der Merwe

Gas Gangrene

Introduction

Gas gangrene is an infection caused by the gas-forming organisms of the *Clostridia* species. It is mainly limited to muscle tissue, but can spread to surrounding tissues. It still remains an exceptionally serious condition. Exotoxins of the causative organism are responsible for myonecrosis which not only spreads rapidly locally, but is also responsible for the life-threatening systemic complications. *Clostridia* thrive in an environment with low oxygen tension and particularly in damaged tissue. In damaged tissues the vascular supply is compromised and tissue oxygen tensions are lowered. If this tissue is contaminated with soil or other foreign matter containing *Clostridia,* an ideal environment is created, for the organisms can multiply and liberate dangerous exotoxins, with serious local and systemic complications.

The incidence of gas gangrene has not fallen significantly in recent years. The mortality and morbidity rates are influenced by the standard of patient care. However, it has been estimated that the annual occurence in the USA is stil in excess of 3000 cases per annum. Reliable worldwide statistics are unavailable.

Aetiology

The causative organism responsible for gas gangrene can be any of the 150 species of *Clostridia*. *Clostridia* are gram-positive, spore-forming, encapsulated, obligate anaerobe bacilli which can be motile or non-motile, depending on the species. Most are soil or foreign material contaminants but the organism has also been isolated from the gallbladder, stomach, small intestine, colon, vagina and occasionally, the skin of normal healthy individuals.

Although *Clostridium* species thrive under anaerobic conditions, some are reasonably aerotolerant. Some strains of *Clostridium perfringens* can survive exposure to oxygen for as long as 72 hours. This is important when therapy is planned.

Clostridia are gram-positive organisms, but many species appear to be gram-negative, especially in clinical material or in late cultures. For this reason the staining characteristics should not be an absolute guideline in diagnosis.

Clostridium perfringens has been implicated, either alone or in combination with other organisms, in the majority of gas gangrene infections. The clostridial species reported as causative agents of gas gangrene are listed in table 6.4.1.

Table 6.4.1. Clostridial species causing or associated with gas gangrene

Group I Toxogenic and Proteolytic

C. perfringens
C. septicum
C. novyi

Group II Proteolytic

C. histolyticum
C. bifermentans
C. sporogenes
C. fallax

Group III Contaminant

C. tertium
C. butyricum
C. paraputricum
C. sartagoforum
C. sordelli
C. capitovales

- Group I includes the species that cause the classical syndrome. They have toxogenic and proteolytic capabilities.
- Group II augment an infection by their proteolytic capabilities, but are not toxogenic.
- Group III are wound contaminants only.

Gas gangrene has often erroneously been associated (almost exclusively) with trauma. A surprisingly large number of gas gangrene cases in which trauma was not a factor have also been reported.

Aetiological factors can be divided into post-traumatic, post-operative (non-traumatic) and spontaneous (neither injury nor surgery). A review of 284 reported cases of gas gangrene revealed the following distribution of aetiological factors:

- traumatic 49%
- surgical 35%
- spontaneous 16%.

Posttraumatic aetiological factors can include compound fractures, gastro-intestinal trauma, burns, criminal abortions and even bee stings, injections and venepunctures.

Clostridial sepsis of the abdominal wall can occur post-operatively (no trauma) and it can follow, in descending order of importance, surgical procedures involving the appendix, biliary tract, small intestine or upper gastrointestinal tract. Endogenous sources of *Clostridia* such as the skin or gastrointestinal tract are implicated in elective surgical cases. Lastly, it must be noted that spontaneous clostridial sepsis (no injury or surgery) can occur. The underlying conditions implicated in the development of this spontaneous sepsis include colonic malignancies, diabetes mellitus, burns, peri-rectal abscesses, ileus, acute cholecystitis and arteriosclerotic peripheral vascular disease.

Pathophysiology

If tissue (mainly muscle tissue) is contaminated with *Clostridia* and anaerobic conditions exist, or are created for any reason whatsoever, the organisms then have an environment in which they can multiply and liberate the exotoxins which initiate the fulminant phase of the process: an intense oedema develops around the area of necrosis with little or no inflammatory response. This swelling in itself comprises the blood supply, reducing the availability of leucocytes and lowering the oxygen tension of the tissues, leading to rapid spread of the necrotizing process, with a fulminating toxaemia as a result of the exotoxins.

Clostridium perfringens is known to produce 12 toxins that are active in the tissues, as well as an enterotoxin. In infections by this organism, five major and a number of minor exotoxins responsible for local and systemic changes have been identified. The alpha-toxin, a C-lecithinase (a phospholipase), is the major lethal toxin that splits lecithin. It causes haemolysis, platelet destruction, capillary damage and necrosis. It is also oxygen stable, i.e. it remains active when exposed to hyperbaric oxygen at 2 or 3 atmospheres absolute (ATA).

The haemolysis leads to anaemia, haemoglobinuria, jaundice, oliguria and renal failure. The remainder of the exotoxins are responsible for destroying, liquifying, and dissecting into adjacent unaffected tissue, thus promoting rapid fulminating spread of the process. Other enzymes involved include fibrinolysins, hyaluronidase, collagenase, haemolysin and cytolysin. The oedema and development of gas in the fascial compartments cause expansion with raised intracompartmental pressure. At first the muscles are haemorrhagic and friable. Later the

muscle tissues change to a dark colour and lose their contractility with secretion of a brick-red, foul-smelling fluid and the development of gas-containing bullae.

Systemic effects such as cardiotoxicity, brain dysfunction and renal failure may be the result of proteolytic and saccharolytic enzymes which are responsible for the production of hydrogen sulphide. The various species of *Clostridia* liberate their own specific endotoxins, i.e. the alpha-toxin of *Clostridium novyi* predominantly increases vascular permeability.

Clinical Presentation

The incubation period in the trauma and post-surgical groups ranges from eight hours to 20 days, with an average of four days after the initiating event.

It must be noted that patients with gas gangrene remain remarkably alert with extreme sensitivity to their surroundings, despite profound shock, impairment of renal function and advancing palpable crepitus. They realise their impending doom and a sense of terror becomes evident in their facial expression. Just before death they mercifully lapse into a toxic delirium and coma.

Successful management depends on early diagnosis of this condition - thus a high index of suspicion should be maintained to ensure recognition of the early signs of the infection, which include subtle changes such as:

- pain in a wound or a surgical incision which increases progressively in intensity and is disproportionately severe
- a tachycardia disproportionate to the fever
- shiny oedematous skin around a wound, and
- apathy.

Suspicion should also be high in any situation where the patient is compromised by shock, vascular impairment, oedema, tight-tension skin sutures, abdominal distension or a tight plaster cast. Progressive changes can take place towards the classic presentation of the disease.

These include:

- oedema
- a discoloured wound with brown to brick-red watery discharge with a "foul-sweet smell"
- haemorrhagic bullae and rapid extention of the surrounding erythema.

Palpable crepitations are only found later. Subsequently, shock and renal failure ensue.

Diagnosis and Special Investigations

- The diagnosis is made by clinical assessment.

- Gram staining of wound drainage is a rapid means of confirming a suspected diagnosis of this condition. If gram-positive bacilli are found in a patient with early signs or a classic presentation as described above, it should be considered to be gas gangrene until proven otherwise and treatment must be initiated even before culture results are available.

- Blood cultures are required for a final diagnosis. It must be borne in mind that the organism can take 48-72 hours to grow, hence treatment is initiated on clinical grounds.

- Other valuable laboratory investigations include serial leucocyte and platelet counts and bilirubin measurements.

- Renal function measurement is not an early diagnostic aid, but it is helpful as a prognostic assessment and as a monitor of progress.

- Radiographs will not necessarily consistently demonstrate the presence of gas in the tissue and the evidence of gas on X-ray is not pathognomonic of clostridial gas gangrene.

The diagnosis can thus not be confirmed by gas in the tissue alone, as a variety of other organisms can produce gas, i.e. *E. coli,* anaerobic streptococci, bacteroids infections and even aerobic aerogenic infections like haemolytic staphylococcal fasciitis and haemolytic streptococcal gangrene. Other organisms implicated include *Klebsiella, Enterobacter* and *Pseudomonas.* Gas in soft tissue seen on X-ray can also be the result of the mechanical effect of trauma, hydrogen peroxide irrigation, barotrauma and surgery. In myonecrosis, the radiograph usually demonstrates a feathery pattern, indicating dissection along muscle fascicles as opposed to large gas bubbles, which are usually associated with the soft-tissue gas of open wounds.

Differential Diagnosis

Gas in tissues and a necrotizing myositis are not pathognomonic of clostridial infections. Other organisms, as mentioned, can also be responsible for the formation of soft-tissue gas. The history and clinical presentation assist in the differential diagnosis (table 6.4.2). Gram stains may be helpful.

Table 6.4.2. Differential Diagnosis of Soft Tissue Gas

Bacterial

I Aerobic aerogenic infections

- Haemolytic streptococcal fasciitis
- Haemolytic streptococcal gangrene
- Coliform

II Anaerobic Streptococcal infections

III Bacteroides Infections

IV Clostridia

V Mixed aerobic and anaerobic infections

Nonbacterial

- Mechanical effect of trauma
- Air hose injury injection
- Hydrogen peroxide irrigation
- Injection of benzene
- Barotrauma, dysbarism
- Postoperative
- Aberrant sexual activity.

Prophylaxis and Treatment

In the injured patient prophylaxis is aimed at proper wound care, including thorough debridement where indicated and recognition of high-risk patients. This includes careful inspection and cleaning of wounds prior to a decision on surgical closure and careful observation of patients with a risk of developing gas gangrene. The effectiveness of antitoxin has not been proved and its possible allergic reactions and side-effects do not justify its use.

Careful attention to resuscitation is essential, since some of the toxins released by the histiotoxic organisms cause increased vascular permeability with intravascular volume loss to the interstitial compartment. Haemolysis and/or surgical losses can result in sizeable volumes of blood loss and several units of blood must be made available. A subclavian and arterial line are required to adequately monitor and medically manage the patient. The four cornerstones of prophylaxis and treatment remain:

- suspicion
- early recognition
- effective early surgical debridement
- administration of antibiotics.

Hyperbaric oxygen has proved to be a beneficial adjunct. It is important to realize that antibiotics alone, in the absence of an adequately debrided wound, will not prevent gas gangrene. More frequent use of delayed primary and secondary closure in suspected cases could play a major role in preventing gas gangrene.

Surgical Debridement

Timely surgery is the primary and essential component of a combined management regime. The aim is complete surgical excision of infected and necrotic tissue. This may involve repeated exploration and incisions and the performance of fasciotomies to ensure decompression and drainage of muscle compartments. Even amputations may be necessary. The appearance of involved muscle is characteristic. It must, however, be viewed by direct surgical exposure, since many of the changes are not apparent when superficially inspected through the edges of a wound. On surgical exploration the muscle is initially pale and oedematous (looking like a piece of steak seared over a fire) and does not contract when incised; on further dissection a red, non-viable muscle is found.

Surgery also allows the essential establishment of haemostasis and the evacuation of haematomas. This prevents the release of catalase by disintegrating erythrocytes, which in turn would have been used by the anaerobes in the wound to metabolize would-be-self destructing peroxides formed within the bacteria. In this way the bacteria may prevent self-destruction.

Antibiotic prophylaxis and therapy

Individual circumstances should be taken into account when prophylactic antibiotic administration is considered. Aerobic and anaerobic bacteria are invariably components of any suppurative infection that involves *Clostridia* and should be treated with penicillin and broad-spectrum antibiotics designed to suppress both. Similarly, prophylactic antibiotics are indicated in cases of contamination from bowel perforation where surgery was delayed. For aerobes and anaerobes in this setting, an aminoglycoside (i.e. gentamycin, tobramycin or amikacin) and metronidazole respectively are indicated.

When gas gangrene is evident, judicious surgical debridement is required and antibiotic therapy is indicated only when the process extends into adjacent tissues or when systemic signs of fever and sepsis are present. Penicillin G acts against nearly all strains of *Clostridium perfingens* and it is the drug of choice for prophylaxis and treatment of gas gangrene.

The sodium salt of penicillin is preferred to the potassium salt, because the latter will exacerbate the hyperkalaemia that may already be present due to haemolysis, tissue necrosis and renal failure. The recommended dosages are 10 to 24 million units per day. In the presence of proven penicillin allergy, tetracycline is recommended instead (dosage 2-4 g IV per day). Chloromycetin (in high doses), erythromycin and clindamycin have also been recommended, although clindamycin has been reported to be somewhat less effective.

Hyperbaric Oxygen (HBO)

This is the third component of the therapy of gas gangrene. Both bactericidal effects (in vitro) and bacteriostatic effects of *Clostridia* have been reported with use of HBO. HBO therapy interferes with the process of exotoxin liberation, in that the tissue oxygen tension is elevated. Tissue oxygen tension of 20 kPa (150 mm Hg0 inhibits production of the exotoxin C-lecithinase, but does not neutralize existing toxins. HBO therapy supresses the growth of *Clostridia,* in that a less favourable environment is created for the growth of anaerobes. With

HBO therapy patients are exposed to 100% oxygen at a pressure of 3 atmospheres for one to two hours. The procedure is repeated 5-12 hourly for six treatments.

The frequency of complications related to HBO is low. Oxygen toxicity, barotrauma, decompression sickness and damage to the lungs have been mentioned in the literature. One study reported over 20000 hyperbaric compressions without a chamber-related death. An important role of HBO is to counteract the hypoxic environment in which *Clostridia* thrive, in that peroxides are formed within the organism, killing it. The presence of catalase in erythrocytes and muscle cells inactivates the peroxides. Surgery thus complements the effects of HBO by removing the necrotic tissue and red blood cells which may release catalase. *Clostridium perfringens* stops producing toxins in ambient oxygen tension of 10.7 kPa (80 mm Hg) or greater. With HBO, tissue oxygen tension of 33.3 kPa (250 mm Hg) at 2 ATA oxygen and 60 kPa (450 mm Hg) at 3 ATA are achieved. Tissue oxygen tension while breathing air is in the range of 4-5.3 kPa (30 to 40 mm Hg). HBO treatment at 2.5 bar (36.7 psi), in order to achieve an oxygen tension of 33.3 kPa (250 mm Hg) in the infected area and yet not exceed the cerebral oxygen toxicity threshold, is suggested. The management of clostridial myonecrosis can be summarized as follows:

1. Diagnosis

- high index of suspicion
- history
- disproportionate wound pain
- toxicity
- examination of the wound:
    - erythema
    - brick-coloured watery discharge with a bad mousey odour
    - haemorrhagic bullae - possibly crepitations
- laboratory
    - gram-positive organisms on staining
    - fascicular gas pattern on X-ray

2. Initial Management

- resuscitation
    - IV and arterial lines
    - Swan-Ganz catheter placement
- antibiotic cover
    - sodium penicillin drug of choice or
    - tetracycline or
    - chloramphenicol or
    - clindamycin

3. Transfer to Major Hospital

4. Combined Management

- fluid and blood administration
- surgical debridement
- antibiotic therapy continued
- hyperbaric oxygen therapy.

5. Reconstruction/Rehabilitation

Prognosis

In spite of remarkable advances in medical, surgical and hyperbaric oxygen therapy, gas gangrene still often results in disfigurement and death. Mortality in gas gangrene is 40-60%. Since the organisms causing clostridial myonecrosis are ubiquitous, every attempt must be made to prevent its occurrence. Aggressive treatment, as was described, improves morbidity and mortality but still does not provide the ultimate solution in management. Prophylaxis rests on the careful management of wounds, including proper cleaning and irrigation with peroxide solution and primary suturing only when it is safe to do so. Early recognition of changes compatible with possible gas gangrene is of paramount importance because the described sequence of events can lead to a rapid spread of the necrotizing process causing fulminating toxaemia and death within 12 hours. Signs indicating a poor prognosis are:

- leucopaenia
- a low platelet count
- evidence of intravascular haemolysis
- severe liver or renal impairment.

Tetanus

Tetanus is caused by tetanospasmin, a potent neurotoxin elaborated by the organism *Clostridium tetani.*

Tetanospasmin is responsible for the clinical manifestations of muscle rigidity and reflex spasm. The disease is fatal in a high percentage of cases. Tetanus is prevented by immunization and is therefore predominantly seen in underdeveloped countries. However, it has not even been totally abolished in developed countries such as the USA.

Aetiology

The causative organism, *Clostridium tetani,* is amotile, gram-positive, anaerobic, non-encapsulated, spore-formin rod. At least ten species exist, but they all produce the same neurotoxin, tetanospasmin. The vegetative forms of *Clostridium tetani* can be inactivated by disinfectants, heat and penicillin. A number of other antibiotics are also effective against the organism. The spores are, however, very resistant to physical heat and chemical disinfection. They can survive autoclaving at 121 °C for 10-15 minutes and they can withstand exposure to phenol, mercurochrome and other generally used effective disinfectants.

The natural habitat and the most common source of infection is soil. *Clostridia tetani* are found in high concentrations in faeces of man and animals, particularly large animals such as horses and cows.

The incidence depends on the possibility of exposure to the infection and the above explains the reason for the increased incidence of the disease among farm workers, gardeners, barefooted individuals and those with primitive lifestyle. Men show a higher incidence than women and no age group is immune. Neonatal tetanus has the highest mortality. All age

groups, races, and both men and women are susceptible if effective prophylaxis is not ensured. In a person with inadequate immunity, essentially any wound or closed infected area may serve as a portal of entry. The organism can be isolated from wounds without the development of tetanus in the individual.

A wound of the lower limb is the most common portal of entry of the organism, but any wound is susceptible (often very minor wounds!) and often no apparent portal of entry is detectable. The history of an already treated wound is therefore of great importance. Minor trauma, requiring no surgical repair, is the commonest type of wound acting as a portal of entry. Spores of the tetanus organism are usually introduced into the body by injury to the skin produced by puncture wounds, cuts or burns. Other routes of entry include chronic leg ulcers, skin infections (including skin lesions caused by vaccination), middle-ear infections and paronychiae. In fact, the presence of other organisms and necrotic tissue enhances the reversion of spores to the vegetative form. Other aetiological factors are tooth extractions, injections used by narcotic addicts, post-abortion, post-confinement, infestation and neonatal tetanus (occurring in the first 28 days of life) which is due to unhygienic obstetrics. Postoperative tetanus following elective surgery occurs most commonly after bowel surgery. Operating room floors, catgut, or bandages may be the source of the organisms. It is well known that tetanus often occurs without a demonstrable wound. This is in contrast to gas gangrene, in which a wound with necrosis is evident. It must therefore be borne in mind that a small insignificant wound incurred three to 21 days previously and now healed, could have been a portal of entry.

Pathophysiology

After the spores of the tetanus organism have gained access into the body by injury to skin, they revert to the toxin-producing vegetative form if a reduction in local oxidation is produced by trauma and local suppuration. The tetanus toxin is disseminated through the bloodstream or through lymphatics, mainly those closely associated with nerves.

Once converted, the vegetative form produces two toxins: tetanolysin and tetanospasmin. Tetanolysin produces haemolysis in vitro and plays no significant role part.

Tetanospasmin reaches the nervous system by either intra-axonal or bloodborn routes. The tetanus toxin usually enters the peripheral nerve endings and ascends into cells of the central nervous system by retrograde axonal transport. This toxin acts on several different levels of the central nervous system: it interferes with neuromuscular transmission by inhibiting the release of acetylcholine from nerve terminals in muscle; the toxin interferes with the functioning of synaptic reflexes in the spinal cord, leading to inhibition of antagonists causing the characteristic spasms of the disease; fixation of toxin by cerebral gangliosides may result in the typical seizures; a disturbance of the autonomic nervous system manifested by excessive sweating, fluctuating hypotension, episodic tachycardia and other cardiac dysrhythmias and increased secretion of catecholamines in the urine is often found.

Clinical Presentation

The incubation period is variable, ranging from three to 21 days between the incident during which the patient was inoculated with spores and the appearance of initialsigns and

symptoms of the disease. The incubation periods are generally related to the site of injury: portals of entry further away from the central nervous system result in a longer incubation period, while those near it, i.e. cephalic wounds, have a short incubation period.

Tetanus can manifest itself clinically in three different forms:

- **Generalized tetanus** which is the most common form and accounts for 80% of cases.

- **Local tetanus** in which case the patient has persistent localized muscular contractions in the same anatomical area as that of the injury responsible for the disease. These contractions may persist for a long period. Local tetanus may precede general tetanus, but it is generally a milder form and less frequently fatal, having a mortality of one per cent.

- **Cephalic tetanus** is rare and is usually associated with chronic otitis media or head injuries. This form may present with dysfunction of any of the motor cranial nerves, but the seventh cranial nerve is most frequently involved.

Symptoms and Signs

Prodromal symptoms of malaise, restlessness, diphoresis, rigors, sore throat and headache may manifest in the initial phase. The characteristic initial sign is trismus, which may be accompanied by dysphagia, neck stiffness, rigidity of the abdominal muscles and a raised temperature of 2-4 degrees above normal.

Generalized tetanus mainly affects three muscle groups: the masseters, the abdominal muscles and the extensors of the back. In severe cases, trismus may progress during the following 24 hours or longer until it becomes persistent, with the manifestation of the characteristic facial expression, *risus sardonicus.*

Opisthotonos, due to spasm of the back muscles, may develop. Tetanus is not the only disease causing trismus and a variety of conditions must be considered in the event of a patient presneting with the sign:

- dental abscess
- mandibular fracture
- tonsillitis
- diphtheria
- mumps
- retropharyngeal abscess and even
- serum-sickness after tetanus toxin prophylaxis with horse serum producing temporomandibular arthritis.

Strychnine poisoning, rabies and even perforated peptic ulcer may cause opisthotonos. Various other conditions such as meningitis, hypocalcaemic tetany, retroperitoneal haemorrhage, epilepsy or narcotic withdrawal can mimic tetany.

The management can be complicated by the aforementioned autonomic hyperactivity which is, if untreated, associated with a higher mortality.

The disease may manifest with intermittent spasms of muscle groups and the duration of the periods between attacks depends on the severity of the infection. In patients with a mild form of tetanus, spasms may be caused by stimuli such as noise. Even lightly touching the patient may cause reflex spasms.

Diagnosis and Special Investigations

The usual sequence of events is: it starts with involuntary jerking, followed by opisthotonos, trismus, flexion and adduction of the arms (fists held clenched across the chest) and extension of the lower extremities. Dysphagia is often present. The general condition of patients usually remain reasonably satisfactory and they are mentally normal.

This is a clinical diagnosis, rather than a laboratory diagnosis. Bacteriological studies may confirm the clinical suspicion by demonstrating spores or vegetative forms of *Clostridium tetani* on gram-staining of material from wounds. The organism may be identified in anaerobic cultures. However, the organism is frequently not detected by either gram stain or culture, even in the presence of definite tetanus.

Prophylaxis

- Active immunization

The association of tetanus with various kinds of trauma and other causes of penetration of spores has been emphasized. As these problems will continue to exist and since the spores of *Clostridium tetani* are ubiquitous, the only real answer is to improve the immunity of everybody. The disease can theoretically be eradicated by immunotherapy and the recommendations for active immunization are given in table 6.4.3.

Table 6.4.3. Schedule of active immunization against tetanus

Dose	Age/Interval	Vaccine
	Age less than 7 years	
1st dose	Age 6 weeks or older	DPT
2nd dose	4-8 weeks after first dose	DPT
3rd dose	4-8 weeks after second dose	DPT
4th dose	About 1 year after third dose	DPT
Booster	4-6 years of age	DPT
Additonal boosters	Every 10 years after last dose	Td
	Age 7 and older	
1st dose	First visit	Td
2nd dose	4-6 weeks after first dose	Td
3rd dose	6 months-1 year after second dose	Td
Boosters	Every 10 years after last dose	Td

DPT - Diphtheria and tetanus toxoids and pertussis vaccine absorbed.
Td - Tetanus and diphtheria toxoids absorbed (for adult use).

Prophylaxis After Trauma

- Prompt and thorough debridement of wounds is of paramount importance in preventing the proliferation of *Clostridium tetani.*

- Active immunization with booster toxoid is recommended for the previously immunized patient, provided that the period lapsed since the test booster dose is more than 12 months. Well-immunized patients will develop antibodies rapidly following the administration of a booster. Absorbed tetanus is superior to fluid toxoid as it induces higher antitoxin titers, thus ensuring durable protection. The combined diphtheria-tetanus-pertussis vaccine (DPT) is recommended for persons of under 7 years of age. For those 7 years and over, the preferred preparation is combined tetanus-diphtheria (Td) vaccine to avoid possible reactions to the pertussis component.

Patients with minor clean wounds need no prophylaxis, but it is administered as the initial dose if the patient has not been immunized or where prior full vaccination is in doubt, or to provide a booster if the most recent toxoid injection was administered more than ten years ago. For patients with all other wounds, a booster of absorbed toxoid is recommended if there has been primary vaccination but no booster for five years or longer. Administration of a booster is indicated in all cases where vaccination is in doubt.

- Passive immunization: Passive protection with human tetanus immune globulin (TIG), in doses of 250 to 500 units, is recommended for patients with suspicious wounds (other than clean wounds), if the vaccination history is uncertain. Equine and bovine antitoxin are not recommended because of the risk of anaphylactic reaction and serum sickness.

Excessive levels of antibodies have no benefit and may be harmful. Guidelines for the indications for the administration of tetanus toxoid (Td) and human immune globuline (TIG) are shown in table 6.4.4.

The incidence of reactions to TIG is minimal and it is safer than the risk of developing tetanus.

Table 6.4.4. Guidelines for the use of tetanus toxoid (Td) and human tetanus immune globulin (TIG)

A. Unimmunized patients, or those with uncertain incomplete (1 or 2 doses of toxoid) immunization

1. Low risk wound: One dose of Td followed by completion of immunization; thereafter, booster every 10 years.
2. Tetanus-prone wounds or wounds neglected < 24 hours: One dose of Td plus 250 to 500 U TIG, followed by completion of immunization.

B. Full primary immunization with booster within 10 years of wound

1. Low-risk wound: No toxoid necessary.
2. Tetanus-prone wound: If more than 5 years since last dose, give one dose of Td. No toxoid necessary if less than 5 years.
3. Wound neglected for < 24 hours: One dose of Td plus 250-500 U TIG.

C. Full primary immunization, but no booster doses or last booster dose < 10 years

1. Low-risk wounds: One dose Td.
2. Tetanus-prone wounds: One dose Td.
3. Wounds neglected > 24 hours: One dose Td, plus 250-500 U TIG.

NB. Use separate syringe and sites for TIG and toxoid.

- Antibiotics: Penicillin G or tetracycline are effective against the toxin-producing vegetative form of *Clostridium tetani*. The effects of antibiotic therapy alone in prevention of tetanus are minimal but, as was mentioned earlier, the presence of other organisms enhance the reversion of spores to the vegetation form. Inflammatory oedema due to infection causes local tissue hypoxia. Antibiotic therapy will not eradicate the spores of *Clostridium tetani* and they may continue to revert to the vegetative form with elaboration of additional toxin. In serious wounds which continue to be infected, it may therefore be advisable to repeat passive immunization with TIG after three to four weeks.

Principles of Treatment

Once tetanus develops, it calls for aggressive medical treatment and meticulous nursing care. Recovery is almost always assured if the patient can be kept alive for three weeks with effective supportive therapy, as tetanus is a self-limiting disease.

The management is aimed at:

- eradication of the organism from the wound to prevent toxin production
- neutralization of existing toxin before it reaches the nervous system
- reduction of the sensitivity of the nervous system to the toxin.

The latter is most important in the event of covulsions and frequent tetanic spasms. General consideration in management include:

- Prevention of external stimuli. Any stimulus such as noise or movement should be limited to a minimum, as it may precipitate convulsions. A bladder catheter and nasogastric tube are placed early and routine nursing procedures such as heart-rate counts should be limited. The treatment area should be semi-dark (twilight).

- Wound debridement is important but should be delayed until several hours after the patient has received antitoxin, as free tetanospasmin is mobilized into the bloodstream during surgical intervention.

- Assisted ventilation and administration of muscle relaxants such as pancuronium bromide (Pavulon) or other curare-like drugs.

- Intravenous electrolyte replacement therapy based on laboratory findings.

- Sedation is important - administration of a short-acting barbiturate such as pentobarbitone is useful. Alternatively, diazepam given intravenously every two to eight hours may control or prevent seizures and is probably the drug of choice.

- Cultures are obtained at the time of debridement. Antibiotics are administered to destroy the remaining organisms. Penicillin is administered in a dose of 1.2 million U of procaine penicillin daily in 1 million U of penicillin G IV six-hourly. Tetracycline and erythromycin are also effective.

- Tracheostomy may be necessary to maintain adequate ventilation and to ensure satisfactory bronchial toilet especially in patients with opisthotonos and involvement of the thoracic muscles.

- Active and passive immunization is indicated, as described. Intrathecal administration of TIG may be considered in severe cases.

- Severe sympathetic hyperactivity can be controlled with a beta blocker such as propranolol.

It must be noted that the patient should not receive any form of oral administration, due to the almost inevitable danger of aspiration. Even nasogastric tube insertion is replaced, by some with early gastrostomy. Parenteral hyperalimentation may provide an alternative means of maintaining nutrition during prolonged episodes.

Careful nursing care is the essential feature.

Complications and Prognosis

Possible complications include:

- respiratory involvement such as bronchopneumonia
- aspiration and arrest
- dehydration
- pulmonary embolism
- serious effects of sympathetic overactivity, including severe hypertension
- drug overdose during treatment, i.e. with barbiturates
- compression fractures of the vertebrae due to spasm of the back muscles
- fracture of the long bones
- anaphylaxis to antitoxin (bovine type)
- exhaustion.

The risk of pulmonary embolism is greater in the presence of dehydration and insensible fluid loss plays an important role in the aetiology thereof. On the other hand,

pneumonia, the major cause of death, is more often a complication of overhydration. Weighing the patient, measuring fluid input and output and determining the SG of the urine are probably the best parameters to control fluid balance.

Prognosis depends on many factors. The disease is more severe when it occurs at either extreme of life. The mortality of patients over 70 years is above 60% and approaches that of neonatal tetanus. The prognosis is worse if the clinical manifestation follows the original injury within seven days or less.

The frequent appearance of severe generalized tetanic seizures indicates a poor prognosis. Autonomic manifestations, when present, complicate the management and are associated with increased mortality. Elderly people and drug addicts are more prone to these complications.

Comment

Prophylaxis of Gas Gangrene and Tetanus

G S Fehrsen

The detail in this chapter is extensive and not only gives different actions to be taken in the prophylaxis of gas gangrene and tetanus, but also the background information which makes sense of different policies to be followed.

I feel the classification into traumatic and surgical causes of gas gangrene is artificial as both can be classified as trauma when looking at it from the point of view of the tissue and the person being affected. In general, I think, it has a positive effect on us when we operate to realize that we are busy traumatizing tissues.

In terms of early diagnosis I think a little bit more emphasis on what exactly a high index of suspicion means, would be helpful. Many times in the chapter some incongruities are mentioned between the level of alertness of the patient, for instance, and the seriousness of the disease. I think that a high incidence of suspicion is assisted by realizing that the early warning signs such as undue pain in a wound that might look innocent at the time may not be a reflection of the patient's poor ability to tolerate pain or on the abilities of the surgeon concerned and thus be ignored; but rather something that needs to be taken notic of seriously and watched skillfully for a few hours to see if any further deterioration will follow. It remains better to be hyper-alert and to do some high-intensity observation unnecessarily than to sign a death certificate of someone who's warning signs were neglected.

In the section on tetanus, I would hold that one should entertain the possibility of an incubation period being much longer than 21 days, even up to three months have already been seen.

Obviously, again the emphasis is on primary prophylaxis, but I think a useful tip in the early diagnosis of neonatal or infant tetanus is the inability of a child to suck, or the child that is easily frightened.

Chapter 6.5: Burns and Their Management

J F Scholtz

Introduction

Heat energy is one of the essential requirements for human survival but, when abused, it can lead to a life spent as a cripple or cause a fatal injury. Due to work situations, customs, economics and accidents there is a high incidence of burns amongst the South African population.

The majority of burns are minor and moderate but the psychological, economic and emotional situation gives rise to great concern. Burn treatment is a team approach and in severe burns this requires a lot of skill in the management of victims.

Definition

Heat of whatever cause damages the protective surface of the body namely the skin in variable depth and may even include deeper structures. This influences the protection against infection, temperature changes and the maintenance of body fluids by endangering the human body in its adjustment to the outside environment.

Aetiology

The cause of burns varies from one part of the world to another and it also differs between the different population groups in the same area. Hot water, open fire, petrol, paraffin and explosives are some of the most important causes in southern Africa. But other causes are candles, cigarettes and cooking and heating accidents which occur as well as burns with motorbike exhaust, suicide, chemical and electrical burns.

There is a variation amongst age groups, races, sexes and occupations.

Radiation burns form a separate group because of the associated effects caused by radiation.

Classification

There are various classifications in use. The most common one is that of first, second and third degree burns (Boyer 1814). In Africa it is a problem that we do not see sunburn in our black population, therefore I would suggest that we use the following classification:

- superficial or partial thickness burn
- full-thickness burns
- mixed burns.

It means that the superficial burn will heal by itself usually within two weeks if no infection or other complication intervenes. One often sees a mixed burn where patchy areas

are deep with superficial areas around. With full-thickness burns the whole dermis and sometimes the deeper tissues are damaged.

Pathophysiology

A chain of reactions follow the destruction of the skin by thermal injury. In the local wound physiological occur as well as systemic changes affecting nearly every organ system in the body.

The exposure to heat causes the destruction but it depends on the time of exposure as well as the extent of what damage will follow.

Cell death as well as vascular changes and chemical responses in the local wound have a systemic effect on the rest of the body as well as a local effect. Prostaglanding causes vasodilatation with leakage of protein and fluids causing oedema which can lead to further cell damage.

Local Effects Following a Burn

- loss of water regulation by the skin (direct or by water evaporation)
- loss of protein
- loss of electrolytes
- wound infection
- vascular thrombosis (deep burns)
- development of necrotic tissue
- blisters
- oedema.

Systemic Effects Following a Burn

- shock - hypovolaemia
- increased blood viscosity
- pulmonary effects
    - toxic gases (direct)
    - oedema (indirect)
    - airway obstruction
    - hyperventilation
- increased hormones
    - catecholamina
    - cortisone
    - glucagon
- gastric effects
    - acute gastroduodenal mucosal lesions
    - prolonged gastroduodenal mucosal lesions
    - duodenal ulcer induced by surgery
    - stomach dilatation.

The coagulation and a fall in blood pressure can contribute to cell damage and increase of cell death. It is therefore difficult to estimate the depth of the burn shortly after injury because further deterioration of the local tissues can worsen the situation. Hormonal and inflammatory mediators may follow endotoxin release and with an afferent neural communication serve as major stimuli that "trigger" catabolic responses.

Clinical Symptoms and Signs

It is usually obvious that the patient has burned him/herself by any of the causes mentioned but sometimes we need to differentiate this from other blister-forming diseases.

How do we know we have a superficial burn? By:

- blister formation
- erythema
- pain elicited byy pin prick
- not possible to pull out hair in burned area.

In the deep burn the following may help to diagnose it:

- white to brown colour (if severe, pigskin appearance)
- thrombosed superficial vessels
- charcoal area (if very deep)
- easy to pull out hair
- no pain with pin prick.

Superficial burns will heal by themselves within two weeks but the deeper burn will form slough that requires sloughectomy or removal with enzymatic debridement and skin graft. It is sometimes difficult to judge the depth of the burn in the early stages and therefore re-evaluation every 48 to 72 hours is imperative.

The following requires monitoring:

- Urine output

If there is no renal impairment a satisfactory urine volume is about one-half to two-thirds (or more) of the normal output (i.e. 30-50 mL per hour in an adult and 20-30 mL per hour for children, 0,5 mL per kg per hour).

- Body mass

Daily measurements of mass is essential.

- Vital signs

Pulse rate, blood pressure, respiratory rate monitoring are mandatory while an ECG is desirable.

Diagnosis and Special Investigations

It is important for the diagnosis to get a good history and to examine the patient like any other patient to see if there are any systemic abnormalities caused by the burn (i.e. shock, anaemia, respiratory problems, renal output, etc). Existing illness or associated injuries should also be sought for. Locally the depth, extent and areas of burn must be examined.

Special Investigation

- Full blood count

The Hb and hematocrit are important to see if anaemia exists or what the degree of dehydration is.

- Urea and electrolytes

The electrolyte balance helps in monitoring the fluid therapy and indicates the type and amount needed.

Watch out for hypercalaemia following the cell damage in the acute stage as well as a low sodium and chloride level that could develop

- Plasma proteins

Especially the serum albumin that leaks out needs attention.

- Osmolarity

The serum and urine osmolarity is helpful in the detection of early renal failure. A high osmolarity with a low volume of urine may indicate too little transfusion whereas a persistent low osmolarity approaching that of serum (300 mmol) suggests renal failure.

- Blood gases

These must remain within normal limits unless respiratory injury or pre-existing cardio-pulmonary diseases are present.

- X-rays of the lungs

Must be performed on admission to give an idea of the lung condition and to compare with follow-up X-rays.

- Bacterial cultures

Either a wound swab, full thickness biopsy or blood culture may be required.

Treatment of Burns

As with any severely injured patient the following must be evaluated first:

- Airway

It is important in all head and neck burns to maintain or establish a patent airway. If any doubt exists an oro-pharyngeal airway or an endotracheal tube may be used. A tracheotomy is the last resort and should be avoided.

- Breathing

The patient may have suffered a head injury or multiple fractured ribs and may not be able to breathe.

- Circulation

In addition to the vital signs the urinary output is the best indicator of adequate circulating volume. A Foley's catheter must be inserted in all burns of > 10% in children and 15.2% in adults and the urine output measured hourly.

Fluid Replacement

The formula used in adults and children is 4 mL/kg% TBSA, for the first 24 hours. Fluid used is Plasmalyte L (Ringer's lactate) or 0.9% saline. It is given as follows:

- 50% of the total fluid is given in the first 8 hours
- 50% over the following 16 hours

The first 24-hour period is counted from the time of injury; that means the calculation must be adjusted accordingly.

Approximately half of the first day's fluid is given in the next 24 hours as maintenance but the intake and output must be monitored carefully. The urinary output must be maintained at minimum of 1 mL/kg/hour.

Fluid is administered to all patients with burns of 15% or more in adults and 10% or more in children.

The head and neck area in an infant is 21% of body size and in an adult only 9%.

History

It is important to determine the cause of the burn and whether the patient was trapped inside a burning house or perhaps inhaled poisonous fumes from burning substances.

Blood/Plasma/Colloids

These are usually not essential and therefore not administered in the first 24-48 hours, but can be given after this period in addition to the routine intravenously administered fluids.

Prophylactic Antibiotics

These are not routinely used. Only when blood cultures or wound swabs indicate infection, should their use be considered.

Local Wound Care

- The wounds may be washed with diluted Savlon solution.

- Blisters may be left intact for 24 hours and then opened, the skin over the blister must not be removed as this serves as a biological cover; it will gradually become dry and can then be removed.

- Topical wound treatment: The ointments most commonly used are Betadine and Flamazine. Dressings should be done at least daily. The wounds are normally covered with gauze dressings and bandages.

- Early tangential excision and skingrafting of deep wounds are indicated to cover the wounds as soon as possible.

- Skingrafting: It must be the aim of the surgical team to cover the burned area as soon as possible. Split-thickness skin graft is the first choice but biological dressings like porcine or cadaver skin can save a patient's life if operation must be delayed due to systemic illness. Artifical material like Bioborane may also be used.

For the future it seems that skin cultures will play an important role in extensive burns.

- Tangential excision of already formed granulation tissue can be covered by a split skin graft in large segments or it can be meshed (1:1 to 1:3). The *Beta haemolytic streptococcus* is the most dangerous organism and it needs treatment before a skin graft is done - a shiny granulating wound is the clinical manifestation of this type of infection.

Determination of Depth of the Burn Wound

In the early stages it is often not possible to assess the depth of a burn. Normally superficial and partial-thickness wounds maintain some sensation while the full-thickness burn is insensitive and has a thick leathery appearance with coagulated blood vessels visible. Partial-thickness burns will heal spontaneously while full-thickness burns will need skingrafting at some stage once the eschar has been removed, either surgically or with medication.

A partial-thickness burn will progress to full thickness if infection supervenes or gross oedema develops.

Burns of Specific Areas

Prevention of complications is the key.

Upper Limb

Axilla

A splint is absolutely necessary to keep the shoulder in abduction (90 degrees);

Cubital fossa

Splint the elbow in 180 degrees;

Hand

Prevent the following:

Oedema

By elevation above the level of the heart it can be prevented or improved.

Immobilization

Continuous mobilization of all the joints is mandatory but it must be combined with physiotherapy and occupational therapy.

Deformity

Deformity could be prevented by the correct use of splints and active/passive mobilization. The splint must keep the wrist in 15 degrees extension, the MP joints at 90 degrees and IP joints at 180 degrees.

The first web space must be kept wide open.

Check the peripheral circulation

A thick eschar on the fingers/arms may constrict the extremity and diminish the blood supply leading to compartment syndrome and gangrene.

Should such eschar constriction be present an escharotomy must be done in the ward as an emergency procedure. Biobrane or even plastic bags assists in achieving early mobilization.

Early excision and skingrafting of hand burns are recommended within 48 hours after injury if possible. Split-thickness skin grafts or 1:1 mesh grafts can be used. If open joints or bone are present, covering by skin flap must be considered.

Lower extremity

- Elevation reduces oedema and prevents prolongation of healing or deepening of a superficial burn.

- Splints keep the knee in extension and the foot in dorsiflexion.

- Regular mobilizing exercises are mandatory.

All splints may only be removed for dressing purposes and during exercise programmes. It is essential that they be worn for months to prevent contractures from developing.

Head and neck area

One should suspect injury to the respiratory tract if the following is present:

- burns of head/neck area
- burns of nasal vibrissae
- red/swollen mucosal surfaces
- respiratory distress
- carbonaceous material coughed up.

A negative chest X-ray in the first 24 hours does not indicate that no damage has been done. Repeat the chest X-ray after 48 hours.

Always keep carbon monoxide poisoning in mind, which may manifest as disorientation or coma. Blood-gas analysis in this situation is ofno value and estimation of the carboxy Hb levels in the blood must be done. Oxygen in high concentrations is indicated for these patients.

Thoracic Wall

Thick eschar in this area will restrict chest wall excursion and an escharotomy is indicated.

Perineum

Insert a Foley's catheter. Make a splint to keep the thighs apart to facilitate dressing and healing.

Gastric management

Prevent gastric ulceration (Curling ulcer) with antacids or H_2 receptor antagonists (Cimetidine).

If there is gastric dilation, insert a gastric tube. Early feeding prevents this because it suppresses the formation of toxic substances in the bowel from enteric bacteria.

Nutrition

General consensus is that during the treatment of severe burns, patient require a large caloric intake up to 5000 cal per 24 hours. The requirements include proteins, lipid, carbohydrates, minerals and vitamins. Mixtures that combine the abovementioned nutrients can be used orally. In special units intravenous feedings may be necessary.

The daily loss of water and nitrogen may reach 3000-5000 mL and 309-509 mmol/L respectively, and the reduction of body mass may be 1 kg per day in severe cases. Generally speaking, proteins supply 15% of the total calorie intake, fat 35% and carbohydrates 50%. Early oral feedings with fluid of patients with extensive burns are usually acceptable during the first 48 hours post-burn. Nutrients can be added to the drink whereby a hyperalimentation regime can be reached within a few days. Accurate monitoring is essential and up to 5700 cal can be administered per day.

In Summary

- maintain an airway
- fluid resuscitation essential
- local wound care
- monitor intake and output, urea and electrolytes, full blood count, plasma proteins, chest X-ray
- prevent complications (i.e. contractures)
- nutrition must be adequate

Diagnosis and Treatment of Burn Wound Sepsis

Suspect infection if the following are present:

- high temperature
- progression of a superficial wound to a deep wound
- gram-negative shock
- black discolouration of the wound
- eschar that separates rapidly
- metastatic abscesses
- red wound margins
- vesicular lesions in healing wounds.

Diagnosis

Wound swabs are often not accurate or diagnostic of wound sepsis. However, this is the accepted method most commonly used. A more accurate method is to do a wound biopsy and culture the specimen as well as determine the presence of bacteria in unburned tissue microscopically (Diagnosis of invasive sepsis).

Blood cultures must also be done if invasive sepsis is anticipated. High dosis of culture specific antibiotics are indicated if sepsis is proved.

If a patient with a burn wound becomes confused, the following must be kept in mind:

- sepsis
- electrolyte disturbance
- acid-base disturbance
- CO poisoning
- unsuspected diabetes, epilepsy, etc (always keep other conditions in mind)
- **Prevention of tetanus:** Tetanus toxoid if previously inoculated. If not, tetanus antitoxin (3000 units in adults and 3000 units in children) becomes necessary.

Prevention and Treatment of Hypertrophic Scars and Keloids

Once the burn wound or skin graft has healed, constant pressure on the wound with tubigrip or a Jobst elastic garment may prevent a keloid or a hypertrophic scar from forming. These garments have to be worn continuously for a long time (12 months to two years) in order to have an effect on the scars. Once the keloid has become established, steroid injections in conjunction with the garments may soften the hypertrophic scars/keloids. In extreme cases the keloid has to be excised and steroid injections together with pressure garments used to prevent a recurrence. As a last resort radiotherapy can be given as adjunct therapy.

Prognosis and Complications

Various factors can influence the prognosis. Among others are the degree of burn, surface area burned, age, existing illnesses, complications like sepsis, pneumonia, inhalation oedema, shock, etc, therefore each patient must be evaluated separately, examined properly and monitored thoroughly, especially if any risk factor or factors are thought to be present.

Infection is the most frequent cause of death and always needs to be taken into account.

Local wound infection can develop from the organisms present on the skin or it can be introduced from the surrounding wound infection. It may change a superficial wound to a deep wound.

Septicaemia can follow an invasive systemic infection and cause the death of the patient.

Rehabilitation

Long after the initial injury the patient may still need follow-ups and rehabilitation to continue with a normal life. Splinting, pressure garments, physiotherapy, occupational therapy and release of contractures may be necessary to rehabilitate the patient. This entails continuous management starting from the time of injury until a satisfactory result is achieved.

Post-burn contractures are the most common problem that needs reoperation after healing, and it can influence the rehabilitation programme. Prevention is still the best if this could be achieved.

Chemical Burns

Contact with chemicals, whether accidental or through abuse, has become a significant cause of injury in out industrialized society.

The morbidity incurred with thermal injury is well known and prevention of such injuries is the ultimate goal of all who have to deal with the disabling sequelae. Until this goal is reached, we must seek better methods to manage chemical burns that will result in the lowest possible morbidity and the greatest degree of rehabilitation.

Pathophysiology

As a rule chemical burns differ from other thermal injuries, principally in the action of the causative agent, which may continue to destroy tissue for a prolonged period if not removed or diluted by copious irrigation.

Systemic effects may be caused by a few chemicals:

- Hepatic necrosis
    - phosphorus
    - tannic acid
- nephrotoxicity
    - phosphorus
    - oxalic acid
    - hydrofluoric acid.

Importance of Local and Systemic Management

Acids produce coagulation necrosis. If the injury is diffuse and/or a partial thickness burn results, debridement and later skin grafting may be necessary. For limited areas, primary excision and skin grafting are recommended.

Alkali burns produce colliquative or liquefaction necrosis. A running shower for extensive burns of hand and arm with irrigation of the wounds for several hours is required. Tanks for hydrotherapy may be necessary in some cases. Do not use Gentamycin ointment which can cause an exothermic reaction.

Early excision of deep localized injury with primary closure or skin grafting is ideal for smaller injuries.

Acid Burns

Characteristically the wound is extremely painful, often for long periods. The appearance of the wound varies according to the depth of burn, from erythema in the superficial injury to grey, yellow, brown or black in the deeper lesions. Although deep burns have a leathery texture in most instances, superficial wounds are usually soft to the touch.

As a group, acid burns should be treated with immediate and copious water irrigation. Phenol burns should be rinsed with ethylalcohol, if immediately available, or with water. Neutralization may be done secondarily after copious water irrigations. The most common neutralizing agent for acid burns is a diluted solution of sodium bicarbonate. The notable exception to bicarbonate neutralization is hydrofluoric acid injury, the treatment for which will be detailed later.

Early debridement of blisters and non-viable tissue is imperative, and strong consideration should be given to early excision in the smaller localized injuries, especially of the dorsum of the hands.

If the injury is diffuse and/or partial thickness, non-excisional treatment with topical chemotherapeutic agents, debridement, and then skin grafting (if necessary) are recommended.

Hydrofluoric Acid

It is a highly corrosive liquid at room temperature. Cell damage is produced by coagulation necrosis and liguifaction with marked vasoconstriction, resulting in ischaemic necrosis.

The recommmended treatment is as follows:

- Copious dilution with water.

- Moist dressings with iced Zephiran or Hyamine chloride.

- If the hydrofluoric acid concentration was less than 20%, continue soaks for 1-2 hours and repeat the first three steps.

- If the concentration was greater than 20% or if the burns appear deep or are exceptionally painful, 10% calcium gluconate is injected cautiously using a 30 gauge needle and regional anaesthesia. The amount to be injected should be $\pm$ 0.5 mL/cm^2 of burn tissue.

- Debride if necessary, including the nails if there is exquisite pain in the ungual areas where acid may be trapped beneath the nails.

- Excise small burns or, for more extensive burns, treat with topical chemotherapy, debride and graft.

Alkali Burns

Injuries produced by exposure to alkalis are similar to acid injuries. The general principles of prompt exposure and water irrigation, optional neutralization with 0.5-5% acetic acid or 5% amonium chloride, and immediate debridement of non-viable tissue are applicable. Early excision of deep localized injury with delayed primary closure or grafting is ideal for the smaller burn injury.

The production of liquefactive (colliquative) necrosis by strong alkali may be due to the fact that alkaline proteinates are soluble and the OH- ion may pass through tissue without becoming inactivated. The mechanism of injury is that of exothermic reaction, cellular dehydration with saponification of fat and protein precipitation. As with hydrofluoric acid, these mechanisms of necrosis are not static, and the extent of the injury obviously depends on the length of time the agent has to act without being diluted or neutralized. If untreated, the lesion resulting from lye exposure will usually encompass the full thickness of the skin and only by chance will it space ectodermal remnants, hair follicles, or sweat glands.

The mainstay of treatment for alkali injury should be water irrigation which can be easily accomplished by placing the patient on a chair in the shower stall and irrigating the wounds continuously for several hours. In hand injuries, hydrotherapy can be instituted easily with hand and arm tanks and continued for several hours without inconvenience.

If the burns involve more than 20% of the total body surface, the physician's concern for good wound care must not make him neglect appropriate resuscitation therapy. In those wounds to be treated by means other than excision the application of topical antibacterial agents may assume special significance in alkali burns. Edgerton recommends Mafylon because of its dual effect of being bacteriostatic as well as combining with active lye in the wounds to form sodium acetate and sulphamylon radicals. He has recommended the avoidance of gentamycins since sulphate radicals are produced with an exothermic reaction and can lead to further wound injury.

Phosphorus Burns

The physical and chemical properties of phosphorus dictate an approach towards anb initial therapy régime which differs from that employed for acid or alkali injuries.

Phosphorus ignites spontaneously when exposed to air, and is rapidly oxidizing to phosphorus pentoxide. Inorganic acids of phosphorus are formed but apparently are not as locally destructive as the heat of reaction, which is the initial and most severe destructive force. Since the ignition point of phosphorus is 34 °C, the removed particles should be kept under cool water; they may ignite when dry and exposed to air, creating a surgical hazard.

It is crucial to emphasize that patients with phosphorus burns should receive **immediate** operative removal of the phosphorus granules from all wounds. This may be difficult, because the phosphorus particles can burrow deep into the wound. Smoke is frequently present, and may facilitate localization of the granules.

The use of 1% copper sulphate solution has been advocated for a brief washing of the wounds prior to operative exploration. This will facilitate further identification of small phosphorus particles. The copper reacts with the phosphorus to produce cupricphosphide which appears black.

Summary

The prompt recognition and management of chemical burns may prevent injury to the deep structures. It may make all the difference between satisfactory rehabilitation and crippling deformities. Immediate irrigation with water is the single most important treatment that can be carried out and should be continued for at least an hour and often for several hours, depending on the severity of the injury. Precious time should not be wasted by hunting for the specified neutralizing agent. Hydrofluoric acid injuries and phosphorus injuries are two exceptions to this principle.

After copious irrigation and debridement, small superficial burns may be treated without dressings or topical therapy. Large partial-thickness burns are best treated with Sulfamylon burn cream and then with biological dressings until healing is achieved. Full-thickness injuries of limited extent should be excised and grafted to regain maximum function; more extensive burns are treated with a non-excisional regime.

Electrical Burns

Electrical burns are more severe than thermal burns because they alswo affect blood vessels, muscles, nerves and other tissue away from the area of contact. Observation in a hospital followed by debridement and re-evaluation is necessary. Patients with severe electrical burns may need more fluid than those with thermal burns.

The factors that affect the passage of an electrical current through a body are the following:

- voltage (or tension)
- type of circuit
- resistance offered by the body
- pathway
- duration of contact.

Types of Electrical Burns

- Enter and exit wounds
- arc burns
- flame burns.

Complications that may Follow an Electrical Burn

- Infection
- neurological injuries
- cardiac injuries
- respiratory injuries
- vascular injuries
- gastro-intestinal injuries
- electrolyte imbalance
- orthopaedic injuries
- ophthalmic injuries.

A thorough examination and monitoring of the patient and all these systems is therefore most important.

Comment

Burns and Their Management

E J Theron

Injury caused by excessive heat occurs commonly in South Africa and has become a severe burden on medical, nursing and paramedical resources in the country.

Classification of burns may be more appropriate when divided into superficial, partial-thickness and full-thickness burns. These are descriptive terms and probably more applicable than the previously used Boyer classification by degrees.

Pathophysiology

In addition to the changes described in the text, the following could be mentioned:

- haematological changes, i.e. haemolysis, initial thrombocytopenia followed by a high platelet count, white cell response and the often quite significant bone marrow suppression.

- Immune system impairment resulting in overwhelming wound and systemic infections.

- Liver-function derangement in severe burns with resultant blood coagulation disturbance.

Systemic Effects of Burns

Apart from the effects mentioned in the text, some others are of considerable importance i.e.:

- Massively increased metabolic rate with fat mobilization, glycogenolysis and elevated glucose flow, resulting in a progressive catabolic state, with rapid mass loss due to mobilization and eventual exhaustion of protein and fat reserves.

- Hypercoagulability initially due to plasma loss, but followed within two to three days by an impaired clotting mechanism, which may last 10 to 14 days.

- Anaemia is an early and progressive problem, and is due to haemolysis and a suppressed bone marrow. In large burns in adults about 500 mL of blood is lost every three to four days.

- Evaporative water loss at a rate of up to 200 mL per m^2 burn area per hour also leads to severe heat loss and often fatal hypothermia.

The lesions in the gastric mucosa are due mostly to a prolonged shock state with hypoxia of mucosal cells, and breaking down of the mucosal barrier. This leads to acute "stress" erosions, and also ulceration. Any further events where stress predominates (i.e. debridement, skin grafts, etc), could give rise to further damage to the mucosa.

Fluid Replacement

A clear difference should be made between maintenance fluid therapy and resuscitation fluids. Maintenance fluid can be administered per os in many cases, but must be measured to be adequate (40 mL per day); if for some reason the intravenous route becomes necessary, maintenance fluid should be used (same formula) every day. Resuscitation fluid we agree should be Ringer's lactate or Plasmalyte B solution, in a formula of 2 mL per kg per % TBSA which could be supplemented by bolus Ringer's infusions of 200-500 mL in response to changes in parameters. The 4 mL/kg/% TBSA formula should be used only in an environment where regular and efficient monitoring of fluid balance is possible. Blood, plasma and other colloids are utilized from the second day onwards.

Antibiotic Therapy

This should be used only when indicated, i.e. prophylactically in extensive burns (> 40% TBSA); therapeutic use of antibiotics should be restricted to specific indications, i.e. obvious septicaemia, positive blood cultures and specific systemic infections, i.e. pneumonia. A major problem in early eschar excision and immediate skingrafting is the difficulty of determining the actual depth of the burn in the early stages. Errors of judrgement in this area could lead to unnecessary blood loss and morbidity.

Tetanus prophylaxis should be managed by the administration of human immune globulin 250-500 mg in the non-immunized individual for immediate coverage. Tetanus toxoid should be given simultaneously as the first of three doses intended as active immunization.

Special types of burns and their management and also burn wound sepsis are both discussed adequately. However, it would not be possible to give full credit to these aspects within the limited scope of this chapter.

Covering all aspects of burns in a concise yet comprehensive manner is a formidable task. The author has managed to achieve this to a large extent.

Chapter 6.6: The Principles of Wound Cover, Free Grafts and Flaps

A Lamont

Many young surgeons and practitioners are filled with anguish when a wound cannot be closed primarily. Fortunately most wounds can be closed primarily, but if this is not possible, a range of alternatives can be considered starting from simple skin grafts and progressing to complex flaps. In traumatic tissue loss or after resection the simplest and most convenient form of cover must be selected as indicated in each case. The following is a resumé of the alternatives, and the indications and contraindications of each technique will be discussed.

Non-Primary Wound Cover

- Delayed primary or secondary suture (discussed previously)
- Skin grafts
    - split skin grafts
    - full-thickness skin grafts
    - composite tissue grafts
- Flaps (local tissue or distant staged flaps)
    - cutaneous - random pattern
      - axial or arterial flaps
    - myocutaneous
    - fasciocutaneous
    - free flaps (with microvascular anastomis)
- Tissue expansion.

Surgical Anatomy

There are many names for grafts and flaps, some of which will emerge in the discussion, but the fundamental classification of flaps is based on blood supply and tissue survival and is reflected in the summary above. The major skin layers are the epidermis and the dermis which in turn is divided into the papillary dermis superficially and the deeper reticular dermis. The subcutaneous tissue comprises mostly fat held in a fibrous network of varying strength. Below is the deep fascia. Over most of the body the skin and deep fascia lie on muscle with only a small area directly over bone or modified deep fascial structures.

Within these layers are distinct patterns of blood vessels or plexuses:

- the superficial or subpapillary plexus comprising capillary loops just under the epidermis

- the deep dermal plexus lies below and in relation to the base of the hair follicles and skin glands

- the fascial plexuses both deep to and superficial to the deep fascia. The signficance of this fascial pattern of vessels has only very recently been appreciated in its application to clinical practice.

These plexuses form a so-called random pattern in a plane parallel to the skin and fascial layers. They are in free communication with each other and are supplied by larger axial pattern vessels and by vertical musculo-cutaneous perforators. Thsi background will introduce some logic into the following definitions and discussion of the various forms of wound cover.

Skin Grafts

Split Skin Grafts (Thiersch grafts)

The thickness of these partial-thickness grafts varies with the actual level within the dermis at which the skin is split. In thin partial-thickness grafts, the level is within the area of the dermal papillae taking some germinal layer but leaving some behind. This gives a relatively poor quality cover to the grafted area, but a rapidly healing donor site with minimal resultant scar. Thick partial-thickness grafts cut within the dermis will give a better quality cover, but will leave a donor site which is dependent largely on the epithelium from hair follicles and glands to achieve healing. The healing time is prolonged with a poor quality scar which is more prone to hypertrophy or keloid formation.

It must be emphasized that this form of grafting gives only an epithelial covering and is not normal skin as the full thickness of the dermal layer with its skin organelles is not transplanted. The major indication for this form of graft are:

- fresh wounds of viable tissue with a good blood supply after adequate haemostasis
- for chronic wounds once the granulation tissue has been excised
- the entire wound area must be covered
- the skin must be taken from the patient. Homo- and heterografts can be regarded as a temporary benefit only.

These four criteria reflect the work of Thiersch who worked before the rejection phenomena was understood and in an area when pinch grafts were still employed as islands of skin within granulating surfaces. These grafts survive initially on the humeral content of the plasma exudate in the wound and rapidly establish a circulation by a process of revascularization and neurovascularization, discussed in a preceding chapter.

More important than the indications is a clear understanding of the contra-indications. Split skin grafts will not "take" in the following circumstances:

- areas of poor blood supply such as some fatty tissue, bare cartilage, bone or tendon, and on chronic granulating or necrotic surfaces
- active haemorrhage will give rise to haematoma which will separate the graft from its healthy bed
- certain infections in more chronic wounds such as *Beta haemolytic streptococcus* or heavy infections of *Klebsiella* or *Pseudomonas.*

Skin grafting is a most useful surgical procedure and has a high success rate with a low morbidity which can be performed with relatively simple apparatus. Small grafts can be taken with an open straight cutting edge. For larger grafts a band knife or automated dermatome should be used. Both have a disposable blade which must be kept free of contact with all objects to protect its very fine cutting edge. The commonest reason for failure to cut a good graft is a blunt or damaged blade. These devices are adjustable so that the skin can be taken at different levels. In general it is set fine for infants and elderly skins and thicker for donor areas such as the thigh in adults.

The donor area can be dressed by two methods. First a homeostatic environment can be established using one of the semi-permeable adhesive plastic layers. This works well and is relatively pain-free giving gradual dehydration of the sero-sanguineous exudate and it can be left in situ for two weeks. The disadvantages are that it is prone to leaks and may become infected, with resultant pain and recourse to more conventional dressings. The more conventional dressings are in the form of paraffin gauze layers which establish an artifical scab within a few days postoperatively. The healing and epithelialization then occurs as under any normal scab. The disadvantage is the discomfort caused by such a large hard surface each time the skin is moved.

The graft should be placed onto the recipient site as soon as possible after rinsing in a tissue fluid to remove lubricating fluids such as liquid paraffin. The graft should not be placed into a saline solution as is so widely practised. This will cause electrolyte disruption of the superficial viable cells. Valuable substances such as liberated kinins will be lost so that the graft loses its haemostatic and adhesive qualities seen in its fresh state.

Meshing or perforating the graft is essential to prevent formation of haematoma or seroma beneath the graft which will lift it off its potential blood supply.

Many forms of dressings are employed to keep the graft in place until it "takes". These should prevent dehydration and avoid sticking when removed. The use of sutures or staples is advised in areas where the graft could be shifted and grafts near joints require the immobilization of the joint for approximately five days.

The graft should beleft undisturbed for five days. However, if infection is predicted, it may be opened on the third day.

Grafts may be carried out using the so-called open method. This is when the graft is taken and stored to be put onto the defect in the ward within the next 24 hours. This is successful in specialized units, but is subject to loss due to poor storage of the graft, dehydration or removal by staff who do not understand the method.

The commonest complication is loss of the graft due to infection or a low haemoglobin level. The haemoglobin level of less than 10 gms% will cause poor take or evel loss of the graft after initial good take.

The disadvantage of split skin grafts is the poor epithelial quality and appearance and the tendency to form contracture. Donor areas once healed, may be used again. Useful donor areas for cosmetic considerations are the buttock and the scalp.

Full Thickness Skin Grafts

These grafts, sometimes called Wolfe grafts, are more difficult to carry out, but have several advantages over split thickness grafts. The full thickness gives better quality cover and in the face the colout match is better if a donor area in the head and neck area is used.

Only thin full-thickness skin can be used because thick skin will undergo necrosis before satisfactorily circulation is established in the graft. Typical donor areas are the retro-auricular fold, supra-clavicular skin, eyelid skin (if present in excess), inner arm, groin or prepuce. Though Wolfe described small areas of thick skin as transplant, large areas can be covered if the graft is thin. A critical factor is the volume of tissue per unit area of exposure to plasma circulation and rapid revascularization.

These grafts are usually small enough to allow primary closure of the donor defect. However, split thickness grafts can be used if a very large area is required. The retro-auricular area can yield a surprisingly large area of skin if the total area between the helical rim and hairline is used. Suturing the ear to the scalp gives a very minor cosmetic deformity.

The skin should be taken on the interface between the dermis and the subcutaneous fat. This can be done quite easily using a skin hook and a scalpel. Too much poorly vascularized fatty tissue will act as a barrier to the re-establishment of circulation and can make the graft volume too excessive. The graft should be sutured carefully into place to give a neat edge. Vascularization will take place from the edge as well as from the under surface. On convex surfaces the graft can be covered by a relatively simple dressing. Concave surfaces will need to be packed or tied over to prevent the graft tenting over the space with resultant non-take. The graft will take longer to revascularize than a split-thickness graft, and is usually left undisturbed for ten days. Many grafts which look as if they have been lost have a good take and the superficial loose layer is caused by epithelial desquamation within the graft.

Composite Grafts

Small blocks of tissue, i.e. less than 1 mL in volume, can be employed for defects in areas with an excellent blood supply. An example would be the outer edge of the ear comprising two skin layers as well as cartilage, as a wedge excision to replace defects in the ala rim of the nose. This concept is closer to the concept of the original Wolfe grafts in that the tissues survive because of a small volume in contact with good healthy tissue.

Flaps

Definition

The term flap can be defined as being a volume of tissue mobilized from its normal attachments to fill an adjacent or distant defect, but retaining an attachment through a base of pedicle through which an adequate blood supply to its substance can be assured.

The simplest flaps of whatever design or however nourished are those taken from local tissue adjacent to the defect. Distant flaps must be used when there is no or inadequate local

tissue and must usually be staged in order to allow the flap to establish a blood supply before the original attachment or pedicle is divided.

Flaps are most often mobilized for the purposes of wound cover in which simpler methods such as primary closure or split skin grafts would not be possible, i.e. to cover exposed joints, bone, etc. Flaps therefore consist largely of skin and subcutaneous tissue. However, for various reasons these flaps can also contain deep fascial muscle and bone according to pre-requisites for the blood supply to a flap and the requirements of the defect. The aim is to cover the denuded structures with tissue of good blood supply. This promotes healing and prevents loss of vital structures through exposure, for instance tendons and cartilage. Skin flaps could, under certain circumstances, be less preferable to pure muscle flaps for cover with a split skin graft to give epithelialization over the muscle flap.

When a flap is mobilized, a secondary defect is created. In areas where there is enough skin, the flap can be designed in such a way as to allow primary closure of the secondary defect. These defects are usually in healthy tissue so that a split skin graft can be used to cover a large secondary defect. Thus the origin of the so-called Bater system where the advantages of flap mobilization are traded for the ability of the simpler closure of the secondary defect - borrowing from Peter to pay Paul without rendering Peter destitute.

In summary, therefore, flaps have the advantage of providing full-thickness cover and padding, they can import good blood supply and can be lifted again if reoperation to underlying structures is required. The disadvantages are that they are often bulky in appearance. There is a significant secondary defect. More than one procedure may be required and they sometimes lead to a degree of loss of function. The design of flaps can be classified as follows:

Local Flaps

- Transposition of a required shape.
- Rotation of a large area.
- Advancement with or without removing two skin triangles.
- Z-plasty.
- Z-plasties are actually small transposition flaps designed to increase length in a given direction.

Distant Flaps

These require a base to be tubed or grafted, i.e. the use of tubed inguinal flaps for hands or cross-leg flaps. This base can often be returned to its original area thus leaving a smaller secondary defect.

As indicated in the resumé, flaps are classified according to their means of blood supply.

Random Flaps

These are skin flaps mobilized anywhere on the surface of the body which depend on the dermal plexuses for their blood supply. The integrity of this system varies over the body surface so that the plexuses of the lower leg skin makes the design of random flaps unwise. By contrast, in the face where the dermal blood supply is good, random pattern flaps are highly successful. The haemodynamics of this microcirculation is such that the flap should not be designed with a base or pedicle less than one half the length of the mobilized tissue. This situation can sometimes be improved by using the so-called "delay phenomenon" in which a degree of axiality of the blood supply can be encouraged by staged mobilization of flaps over a three-week period.

Axial or Arterial Flaps

It was found, surprisingly recently, that the length and mobility of flaps could be greatly increased by introducing an arterial venous system along the axis of the flap. The classical example was the inguinal or groin flap using the superficial circumflex iliac artery. The axial pattern extends from the femoral artery medially in a supra-lateral direction to roughly the anter or mid-axillary line over the iliac crest. The length can be increased by including an area of the skin with a random pattern blood supply beyond the area of the axial supply.

Other areas using the same principle are the dorsum of the foot, the pectoral area, the temporal area and the mid-forehead area. These are areas where the skin is supplied by constant relatively large vessel systems.

A refinement of this principle is the so-called island flap. Here the skin is dissected loose as an island and remains attached by the vascular pedicle only. This greatly enahnces the mobility of these flaps and the pedicle can be buried under skin or tunnelled into adjacent areas without the need for tubing the skin or the base of the flap or for grafting of its raw surface.

Myocutaneous Flaps

Only a small area of skin is supplied by constant vessels as above. Most of the skin is supplied by perforators which reach the skin via underlying muscle or via fascial septa. The recent discovery of the musculo-cutaneous perforating system lead to the concept that muscles could be used to carry skin flaps.

Myocutaneous flaps are therefore composite flaps of muscle with its overlying fascia, subcutaneous tissue and skin. The use of a muscular hylus to carry blood supply also increases the length and manoeuvrability of flaps particularly if the muscle origin as well as its insertion can be mobilized. The major disadvantage of these flaps is the large secondary defect and the large bulk of the flap. There is almost no loss of function in the normal individual after mobilization of a single muscle. Some muscles are taken as muscle flaps only to be covered by a skin graft. This cuts down muscle bulk and leaves an insignificant secondary defect. If the muscle is denervated, it will loose bulk, but retain a layer of excellent blood supply. This has been well utilized in the cover for chronic infections in bone.

Examples of myocutaneous flaps are:

- Latissimus dorsi.
- Gluteus maximus.
- Gastrocnemius.
- Pectoralis major.
- Sternomastoid.
- Rectus abdominis and others.

Fasciocutaneous Flaps

The most recent clinical benefit from applied anatomical study has been the defining of the blood supply that reaches the skin via various fascial planes. Many surgeons noticed that flap survival could be encouraged by including the deep fascia. It is not known that the deep fascia as well as the intramuscular septa are valuable carriers of blood supply to the skin as well as having arterial and venous plexuses within their structures which communicate with those of the skin. The full extent to which this knowledge can be used clinically is still being explored but these flaps have largely displaced the bulky myocutaneous flaps.

Examples are: the medial and lateral longitudinally orientated fasciocutaneous flaps of the lower leg. The ventral aspect of the forearm is a further area with a fasciocutaneous element and indeed the older axial skin flaps will probably be included in the classification of various flaps of fasciocutaneous blood supply. If the vascular axis is parallel to sensory nerve supply to the skin, these nerves can be transposed intact or resutured in order to provide sensation to the flap. This is a particularly useful technique when protective sensation is required, for instance, for covering of selected pressure sources.

Free Flaps

A massive step forward for the reconstructive surgeon was taken when the technique and technology for microvascular anastomosis was made available. It is a logical progression to be able to isolate these axial vessels described above and use them to transplant blocks of tissue to distant areas of the body without the need for staged procedures for the pedicle, i.e. the blood vessels are reanastomosed. Indeed the disadvantage of many staged pedicle flaps is that the axial pattern is eventually divided so that the flap is entirely dependent for its survival on the local blood supply and its ability to form microconnections with the transported tissue. In chronically scarred areas this can be a major disadvantage with atrophy or loss of the flap due to necrosis. If the pedicle vessels could be reanastomosed, this blood supply would be maintained.

The term free flap was used to indicate that for a short while a selected volume of tissue with its arterio-venous system is cut free fo the body to be reattached to the vessels in the recipient area. Similar methods of closure of the secondary defect are used as for other flaps. However, these flaps are designed in areas where tissue is relatively expendable and it is usual that primary closure can be achieved leaving a relatively minor linear scar.

The principles of these procedures therefore is to define the defect and exclude other easy methods of repair. The indications and contra-indications for these operations depend

very largely on the experience of the surgeons involved. A suitable donor area is selected and explained to the patient. Two surgical teams are usually employed. One to prepare the defect and the blood vessels in the area. The second team removes the flaps and hands it over to the first team after all preparations for the anastomosis are completed. These operations are demanding in that they take a great deal of preparation, man power and theatre time. However, the advantage are significant and cost-effective when patient selection is correct, for instance the reconstruction of a lower leg which would otherwise have been lost in a young person, has great socio-economic implications.

These flaps can be briefly classified as follows according to those in common use at present:

- skin flaps
    - scapular and parascapular
    - forearm
- muscle flaps
    - latissimus dorsi
    - gracilis
- Myocutaneous flaps
    - latissimus dorsi
    - tensor fascia lata
    - rectus abdomonis
- bone flaps
    - iliac crest
    - fibula
    - radius
    - scapula.

These are usually composite tissue blocks giving one-stage reconstructions of complex defects such as in the lower leg with bone and skin loss after cancer resections, i.e. mandibular reconstruction with resurfacing and oral lining. Replant surgery uses similar microsurgical techniques but is not within the scope of this chapter.

Tissue Expansion

We are aware that tissue will respond to expansion, i.e. during pregnancy or in response to rapidly growing tumours. However, in reconstructive surgery this characteristic has only recently been utilized. Whether the tissues undergo true growth and hypertrophy or just stretching is not yet fully understood. Tissue can be made available for elective cover of large defects by expanding the tissues, mainly skin and subcutaneous tissue, in adjacent areas. This is done by placing an expandable prosthesis under the skin with a filling port over a convenient injection site nearby. Over the ensuing weeks after the wound has healed and at variable rates, the prosthesis is expanded by injection of saline into the filling port. Various formulae can be used to predict how much expansion is required to yield adequate skin cover in a given defect. At a second stage the prosthesis is removed and the expanded tissue used to obtain skin cover on the defect. Examples of advantages are expansion of hair-bearing skin for scalp defects or during breast reconstruction.

In conclusion it is hoped that the information contained in this chapter will encourage the surgeon not to attempt to force wounds toclose primarily when such closure is not possible.