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

Chapter 2: Intraoperative Care

J A L van Wyk: 2.1 Aseptic and Antiseptic Techniques

An accidental break in skin or mucosal surface as well as surgical incisions or puncture sites may cause infection.

An increase in the duration of the operation as well as the length of pre- and postoperative hospital stay increases the rate of infection. Hospital acquired infections give a higher rate of wound infection than community acquired infections. Some of the most important nosocomial infections are caused by *pseudomonas* and *serratia,* while fungi and anaerobes have also become very important. The number and virulence are of great importance, and the presence of foreign material in a wound adds an additional risk.

Vascular disease with diminished blood supply to tissue, diabetes mellitus, liver disease, certain blood diseases, renal disease and certain immunosuppressed states lead to an increase in infection rates. Nutritional disorders and advance tumour have been associated with reduced resistance to infections.

Techniques to Reduce Wound Infection

Preparation of the Patient

- The nutritional state of the patient should be optimized for elective surgery.

- Any existing bacterial infective site should be treated and healed prior to operation and the pre-operative stay in hospital should be as short as possible.

- The patient should be bathed with an antiseptic soap prior to operation.

- The site of incision should be prepared in the operating room and includes removing of hair and scrubbing of the skin with a detergent such as a chlorhexidine compound. Thereafter an antiseptic solution such as chlorhexidine or iodine should be applied to the skin and left on skin for at least two minutes.

- Sterile drapes should cover those areas which are not required for the operation and the anaesthetic.

- If adhesive covering of the skin is used, great care should be taken in wiping off the cleansing solution to render the skin completely dry.

The Surgical Team

- Proper theatre attire which consists of a set of clothes not worn outside the theatre should be worn by every individual entering the operating room area. A mask which covers the mouth and nose; a hood that covers hair and beard; and shoe covers should be worn.

- The operating team should scrub their hands and arms up to the elbows with an antiseptic solution before the operation for at least two, but preferably five minutes. Chlorhexidine and iodophores are recommended for this purpose.

- Sterile gowns and gloves are worn for the duration of the operation.

- Punctured gloves should be replaced promptly.

Operative Technique

Careful and meticulous surgical technique is one of the cornerstones involved in reducing the incidence of wound infection. It should therefore include the principles listed below.

- Blood supply to the tissues is of the utmost importance and should not be jeopardized by excessive clamping of blood vessels and similar operative trauma.

- Tension caused by suturing should be reduced to a minimum.

- Excessive bleeding and hematoma formation should be avoided at all costs. The latter interferes with the blood supply due to the tension which it creates, and form an ideal culture medium for bacterial growth.

- Devitalized tissue plays a similarly negative role in wound healing.

- Foreign material including sutures should be kept to an absolute minimum to avoid foreign body reaction.

Care of the Wound

Clean or uninfected surgical wounds should be closed with meticulous care to ensure precise approximation of the epithelial edges.

- If drainage is necessary, the drain should be brought out through a separate stab wound.

- Contaminated wounds should be cleaned by thorough but gentle mechanical irrigation with a non-irritant solution.

- The postoperative care of the wound should be of the same aseptic quality which was employed at the time when the incision was made. This means that personnel should scrub their hands or wear sterile gloves when handling wounds.

- The care of the infected wound should be similar to the abovementioned methods in order to avoid superimposed infection.

- Surveillance of both the operating room environment and the incidence of infections should be carried out regularly. This will enable the surgical team to pinpoint defects in such

environmental factors as ventilation and theatre cleaning, as well as patient preparation, wound care, operative technique and preparation of the surgical team.

Comment

Aseptic and Antiseptic Techniques

C J Grobelaar

A wrong impression may be created when only pseudomonas and serratia, fungi and anaerobic organisms are quoted as the **most important** nosocomial infections. In practice we know that although gram-negative organisms such as pseudomonas and anaerobes can be difficult to treat, requiring parenteral antibiotics, we have to acknowledge the most *common* organisms, i.e. gram-positive organisms where staphylococci at areas distant from the perineum still remain a very common headache.

The pre-operative stay - although it is true that the length of pre-operative stay can be associated with a higher incidence of wound sepsis - has to be seen in the right perspective. We have to weigh the pre-operative period against the reasons for pre-operative admission. For instance, it is important to evaluate the patient pre-operatively as far as the following is concerned, and for that at least two days are required. A good idea would be to do these tests at an outpatient clinic if at all practical:

- ESR, FBC & CRP.
- Three urine specimens for MKS on a daily basis.
- Lung examination, X-rays, ECG, etc.

Sometimes admission is imperative to correct these findings pre-operatively and can take two to three days or more.

Scrubbing of hands and arms should be limited to nails and fingers and must only be done until visibly clean. Only washing is recommended to elbow level for 5 minutes before the first operation of the day and thereafter 3 minutes for the remaining operations.

We may add that we should not dissect, but rather operate. Do not expose more than is essential to do what we have to do. Time will thus be saved and haemorrhage limited so that a lower infection rate can be expected.

Suction drainage is used to obliterate dead spaces by creating negative pressure within the closed wound.

Asepsis in total joint replacement (TJR) is of vital importance. The introduction of foreign materials such as metal, plastics and bone cement increases the sepsis risk. There are also dead spaces around the implant which will invite haematoma formation and sepsis if asepsis is not of a very high standard.

Sepsis can originate basically from three possible sources: - the environment; the surgeon and the patient, and it is imperative that we should look at all three of these factors.

The Environment

Clean air introduced into the theatre in a laminar fashion is essential in total joint replacement. Organisms are carried on dust particles of approximately 1 mm and we have to filter only down to 1 mm pre-size. Although today we use filters of 0.3 m pore size (submicron) this is optional and more expensive.

The surgical staff use so-called "space suits" to prevent air contamination from this source. Not only are these gowns impenetrable for organisms but the immediate body environment like shedded skin and expiration is removed from the operative area. Temperature should be regulated to + 18 °C to prevent sweating and shedding of organisms from the skin of the patient and staff. After a septic procedure in the laminar air theatre (which should be prevented whenever possible), it is feasible to leave the theatre unoccupied and fully ventilated for 12-18 hours.

The Surgeon

The surgeon is the second important link in the prevention of contamination. Shaving of the patient is done immediately pre-operatively in the operating theatre block, preferably in the anaesthesia room, using an electrical shaver. Iodine 3% has been proven to be the most effective cleaning solution in our bacteriological department and is applied twice from the incision area, radially outwards including the entire limb. Draping is done twice using a different colour of drapes for each stage and avoiding contact with the first stage of drapes, when the final draping is done.

Research indicates that up to 25% of gloves can be porous, so that two pairs of gloves are recommended in TJR. The first pair of gloves are donned before gowning, so that the distal ends of the cuffs when donned never touch the forearm skin **which is never sterile.** Gown cuffs are thus left on top of the deep gloves, finally covered with the second pair of gloves.

Instrument tray changing is done in six stages during TJR to ensure minimum possibility of contamination. Surgery must be well rehearsed. The surgeon should never waste time about the open wound; at the same time he should keep the exposure limited only to the essential extent. He should thus operate and not dissect the patient.

Wound closure must be meticulous, preventing involution of skin edges at all cost. Vacuum suction drainage is essential and should be limited to two or three days.

Lastly the surgeon should invite all members of the staff to watch out for weaknesses in the asepsis technique and not to hesitate to volunteer any possible problem areas.

The Patient

Preventive measures must start eight days pre-operatively. Thorough examination of the patient for septic foci on the skin, throat and lungs must be done. Three urine specimens for MCS, FBC, ESR and CRP must be taken and normalized if necessary.

Blood serum, proteins and liver function and especially serum iron must not be overlooked as these can limit host resistance severely.

Prophylactic antibiotics are recommended for all cases where foreign materials are incorporated. Anticoagulants are a serious threat to asepsis and should only be used in selected cases.

In Conclusion

All these stringent asepsis measures may seem an overkill, especially in cases of lower risk surgery. That these measures are effective, however, is not in question at all - the 1% incidence of deep infection in TJR that we experience today certainly is a far cry from the original sepsis incidence of 10% reported by Sir John Charnley in 1963.

It seems reasonable therefore to take a serious look at these measures when any surgery is undertaken - we simply owe that much to our profession and our patients. And although these thoughts still remain largely unproven, it nevertheless commands the agreement of most arthroplasty surgeons and it certainly represents our hopes.

Chapter 2.2

Electricity and Radiation in Operating Theatres

T P Lombard, T C Downes

Part I

Electrical Safety and Hazards

In the modern operating theatre, patients are exposed to a myriad of highly sophisticated equipment such as monitors, diathermy, lasers, and electroconvulsive devices. The obvious advantages that these devices offer should not blind us to the fact that electricity and electrical apparatus, if incorrectly or injudiciously used, pose hazards to both patients and medical staff.

This section covers the standards which should be maintained with respect to electrical supply to and wiring of operating theatres, maintenance of electromedical equipment and electrical faults which may cause macroshock or microshock. The use of diathermy and defibrillation has become routine, the latter especially in the cardiac theatre. It is important to understand how this equipment functions in order to use it safely and responsibly.

Factors responsible for causing burns to surgical patients will be discussed and brief mention will be made of explosion hazards.

An increasing percentage of the population has received permanent pacemakers and sooner or later these patients are likely to present for surgery. The patient with a pacemaker presents a number of potential problems to the surgical team in the operating theatre. The

preparation of these patients for surgery as well as their intra-operative management will be discussed in some detail.

Part II will deal with the hazards posed by diagnostic radiation in the operating theatre.

Definitions

These definitions may not be exactly what you will find in standard physics texts, but will hopefully serve as a basis for the understanding of electrical safety and the hazards associated with the use of electrical equipment in the operating room.

- **Electrical current:** When there is a flow of electrons in a conductor (or a flow of ions in a fluid medium such as the body), an electric current is said to flow.

- **Direct current (dc):** This is a unidirectional flow of electrons in a conductor. The flow of electrons is due to a potential difference (PD) or voltage between two ends of a conductor. The potential difference is usually supplied by a battery (fig. 2.2.1). The amount of current, measured in amperes, that will flow as a result of the voltage is described by Ohm's law (I = V/R) which states that the current (I) is directly proportional to the voltage (V) and inversely proportional to the resistance (R) in the circuit.

- **Alternating current (ac):** Mains electricity provides an alternating voltage, i.e. the voltage on the live wire alternates between positive and negative with respect to the neutral wire (which remains at about zero volts). The alternating voltage causes an alternating current to flow, and the electrons exhibit a "to and fro" movement in the conductors. The variation of the amplitude of the voltage (and hence the current) is represented by a sine wave (fig. 2.2.2). In South Africa the voltage rises to a maximum of 325 V, but because it is at a peak for only a short while, the effective or RMS (root mean square) value of 230 V is used. The average of a sine wave is zero, but the power output of electrical equipment is definitely not zero - think of an orthopaedic drill turning at 1000 revolutions per minute. It makes a hole in the bone easily, i.e. it has power. The RMS principle is a mathematical manipulation of the ac wave to compute this power output. **When referring to mains voltage, the RMS value is always used.** In South Africa the current alternates at a frequency of 50 cycles per second or 50 Hertz (Hz), hence one cycle takes 20 milli-seconds to complete (fig. 2.2.2). In the USA mains electricity has a voltage of 120 V RMS and a frequency of 60 Hz.

The relationship between current, voltage and resistance as given by Ohm's law is equally valid for ac and dc.

- **Current density:** This is the amount of current per unit cross-sectional area of the medium through which current flows. The current density is thus high at the tip of a diathermy probe but low at the neutral plate where it is spread over a large area.

- **Potential difference (voltage) (V):** For the purposes of this chapter potential difference and voltage have the same meaning. The potential difference is the driving force which causes electrons to flow. For any given resistance, the higher the voltage the greater the current. If you were to touch the live wire of an electrical instrument with one hand and an earthed conductor with the other, the potential difference of 230 V (existing between the

live wire and earth) would cause a current to flow through you, which you would experience as an electric shock. The severity of the shock and the damage caused would be proportional to the magnitude of the current, which would be determined by the voltage and the resistance of your body, according to Ohm's law.

- **Resistance:** This is the opposition to flow of current. It is measured in ohms.

- **Inductance:** An inductor is formed by a coil of wire and it exhibits a frequency-dependent opposition (called reactance) to alternating current. The higher the frequency, the higher the reactance. Because its reactance depends on frequency, an inductor is often used in a filter to remove unwanted signals, such as high frequencies at the input of a monitor.

- **Capacitance:** A capacitor is formed by two metal plates separated by an insulator. When connected to a dc source, the plates of the capacitor will be charged to the voltage of the source. The capacitor will store the charge, even when disconnected from the source voltage, because no current can flow across the insulator separating the capacitor plates. Should the capacitor be connected to a load (resistance), it will discharge, causing current to flow from the positive plate, through the load, to the negative plate. The current will decrease to zero as the charge on the plates diminish. The dc defibrillator uses a capacitor (vide infra).

- **Impedance:** The overall opposition to the flow of alternating current at a particular frequency, i.e. the sum of the resistive, capacitive and inductive elements in an electrical circuit, is called impedance.

- **Earth/Ground:** This is the zero potential to which all other potentials are referred. An electrical ground or earth is an object capable of receiving large amounts of electrical charge. Every item of equipment used in theatre should have its casing earthed and the earth wire connecting the earth sockets of all mains supply outlets, should be physically connected to earth by means of a low-resistance wire or conduit with one end planted in the ground.

- **Leakage current.** This is a non-functional unwanted and potentially harmful current which arises due to a fault in equipment or imperfect insulation between conductors.

- **Earth leakage current:** This is leakage current that flows from the live part of electrical equipment to earth via the protective earth conductor (earth wire). An earth leakage unit, installed in all modern buildings, will "trip" to stop further current flow when the earth leakage current exceeds a threshold value, usually 20 mA.

- **Patient leakage current:** A leakage current from any source passing through the patient to earth.

Effects of Electric Current on Humans

The effect that an electric current has on the body depends on the skin resistance, the voltage and the frequency of the ac source. Assuming that the voltage and frequency are constant in a given situation, then the only factor which can change the amount of current that will flow, is a change in the skin resistance. The resistance of skin, depending on its dryness and the thickness of the horny layer, can vary from a few kilo-ohms to a few hundred kilo-

ohms. Skin resistance is reduced by moisture (i.e. perspiration) and especially when an electrolyte solution or conducting jelly is applied.

When an ac voltage at a frequency of 50 Hz (mains frequency) is applied to the body, it causes violent muscle contractions. If a current should flow through the heart, then the cardiac muscle might contract in an un-coordinated manner leading to ventricular fibrillation. At high frequencies muscle contractions do not occur and the current causes the tissues to heat up. At a frequency of 100 kHz there is no longer any sensation of electric shock, but the thermal effect can be felt. This is the principle on which diathermy works and it will be discussed later.

The threshold of sensation for electric current at 50-60 Hz is about 1 mA. As this current increases to about 10-15 mA, pain is felt. At about 25-30 mA, the stronger flexor muscle contract so the person cannot let go. At about 50 mA the respiratory muscles go into spasm, a situation that cannot be endured for long. Ventricular fibrillation can occur at currents above 80 mA. These data are shown in table 2.2.1.

Mains Electricity Supply to Theatres

Electricity supply to major hospitals is usually 3-phase, 11000 V. Once the high voltage has been transformed down to 3-phase, 230 V, the neutral wires from each of the secondary windings of the step-down transformer are bounded together and connected to earth to provide a common neutral wire. It will be noted that the neutral wire is earthed and thus should be at the same potential as earth, with the potential in the live wire of each phase varying from +235 to -325 V in relation to the neutral wire (see fig. 2.2.2).

The electrical supply to operating theatres, heart catheterization and intensive care sections differs from that to the rest of the hospital. The supply to these areas is isolated, i.e. the neutral and earth wires are **not** connected and the secondary winding of the isolating transformer is not physically connected to the primary winding. This is achieved by means of an isolation transformer (fig. 2.2.3). Each theatre has its own isolation system. Isolation systems were introduced to reduce the risk of microshock in areas where intravenous catheters are used and to reduce the risk of macroshock in theatres with conductive flooring. The reasons for installing conductive flooring are discussed later in the section on burns and explosions.

To understand how an isolation transformer diminishes the chance of macroshock one must understand how shock occurs with an unisolated supply. If a person should accidentally touch the live wire, current will flow through him to ground and then via ground back to the voltage source (fig. 2.2.4, top). The current has a return path to the voltage source because somewhere in the system the neutral and earth are connected. If the person is wearing thick rubber-soled shoes and the floor is non-conducting, he will probably not be severely shocked. However, if the floor is conductive, a much larger current can flow, increasing the severity of the shock.

Should a person accidentally touch a live wire (point A, fig. 2.2.4, bottom) of an isolated system, the current cannot flow through him to earth because there is no return path

for current back to the voltage source (point B, fig. 2.2.4, bottom), therefore he will not be shocked, even if the floor is conducting).

The scenario for microshock is described later, in the section under microshock.

Between the isolation transformer and the mains wall sockets in the theatre, a distribution board is installed. Every line going to a wall socket has a circuit breaker (trip switch) which disconnects the wall socket from the supply when the socket is overloaded or if a short circuit occurs. The trip switch can be reset once the fault has been rectified.

The three wires in the cable to an electrical device have different colours. The brown wire must be connected to the live (L) pin of the plug, the blue to the neutral (N) and the yellow-green to the earth (E).

To ensure that the isolating system is intact, a safety device is installed in the distribution board. This device, called the line isolation monitor (LIM), monitors the resistance between every live wire and earth, in other words it monitors the degree of isolation between live and earth. An alarm is triggered if the resistance between live and earth falls below a set values might occur if someone who is earthed touches a live wire or if moisture reduces the isolation between live and earth.

When power to a hospital is interrupted, continuity of electrical supply to critical areas within the hospital (i.e. ICU and the operating theatres), is vital if unconscious or paralysed patients are to survive. There must be a diesel-driven emergency generator which switches on automatically in the event of a power failure. This generator should supply power to all lights and mains wall sockets in the operating theatre, recovery rooms and intensive care units. The generator should also supply power for low-level lighting of wards and corridors to avoid darkness which may create panic. This generator must be regularly checked and tested by trained technicians to avoid potential catastrophe should the power fail and the generator not function properly.

Safety of Electromedical Equipment

All modern electromedical devices that come into direct contact with the patient must meet certain minimum safety requirements:

- All circuits connected to the patient should be floating: i.e. they should not be connected to earth in any way and should be separated from the mains supply to the unit by a transformer which steps down the mains voltage and isolates the primary and secondary (patient) circuits.

- The casing of the unit should be earthed. By casing we mean any part of the apparatus which can be touched by a patient or medical personnel.

- Earth or patient leakage currents under normal and single fault conditions must be below certain limits, especially for equipment which could be connected via leads or catheters directly to the heart. These limits vary from country to country and are constantly under review. Leakage current should not exceed 0.01 mA under normal conditions and must be less

than 0.05 mA when a single fault occurs. When ordering electrical equipment, ensure that specifications regarding earth and patient leakage currents are adhered to.

- Impedance (resistance to ac current) of patient leads must be as high as is compatible with good signal pick-up to avoid or reduce the possibility of leakage currents, which flow from faulty equipment via the patient leads, through the patient to earth.

- The resistance between the live mains conductor and any part of the patient circuit should be very high - at least 10 mega-ohms.

Relevant Data from SABS Recommendation for Prevention of Electrical Hazards in the Operating Theatre

- The electrical system should have an isolated power supply, because this provides considerable protection against electric shock. A power supply is isolated when neither of the wires from the output of the isolating transformer is connected to earth. This prevents shock because no current can flow to earth; however, it will not prevent shock due to current flowing between two or more connected devices, nor will it prevent the hazard of microshock from capacitive or inductive coupling. Each isolated supply system should have a line isolation monitor to monitor the resistance between the isolated mains wires and earth. While this value is above 5000 ohms the green light outside the operating theatre should stay on. When this falls below 5000 ohms an alarm should sound and the red light outside the operating theatre should come on. The current should not automatically be cut off as this may endanger the life of patients relying on the function of electrical apparatus for life support.

Each operating theatre should receive electric power from a separate branch circuit of an isolated supply system.

- In each operating theatre a patient earth point terminal bus-bar should be as close as possible to the head of the operating table. The earth terminal of each plug point should be connected to this bus-bar. All conducting objects with which the patient may come into contact should be connected to this so as to provide a low-resistance, common earth point to drain away any static electricity build-up, or earth leakage currents, which could otherwise flow through the patient. This bus-bar is intended to ensure that all conductors that may come into contact with the patient are at the same electrical potential.

This is important in the prevention of microshock (vide infra).

- The operating theatre floor should be constructed so as to be antistatic or conductive, however, there should be a certain minimum resistance. In Britain, it is suggested that this should be between 20 kilo-ohms and 5 mega-ohms. A higher resistance increases the possibility of static build-up which might pose the danger for microshock in patients with intra-cardiac catheters. On the other hand, if the floor had a very low resistance, current from faulty equipment or earth leakage current may flow more easily causing microshock. The higher the current, the more dangerous it is for the patient, therefore the floor should have a certain minimum resistance to reduce this danger.

- All electrical sockets and plugs in anaesthetizing locations should be fitted with a device having an interlocking switch to prevent withdrawal of the plug while the switch is in the ON position.

- The wires of all electrical cables connected to plugs should be according to specifications and SABS recommendations: **brown = live, blue = neutral, and yellow/green = earth.** This should be checked by the operating theatre technician or engineer before the equipment is used.

- The wiring, earth indicator circuits, earth-to-earth resistance across the room, and the functioning of electrical equipment should be regularly checked by a qualified technician or an electrical engineer, and a record should be kept of these maintenance checks.

Role of Clinical Engineering in Operating Theatres

Any hospital of over 800 beds should employ a full-time clinical engineer and a staff of clinical engineering technicians to meet the needs of the hospital. Before any new electromedical equipment is purchased, the specifications should be checked by the engineer or senior technician to ensure that the equipment is suitable. If the engineer or technician is suitably qualified, he may advise on the purchasing of equipment. The engineer or technician should also be present when new equipment is demonstrated and installed and certain safety and acceptance tests may be performed before payment for the equipment is approved.

Any hospital which runs more than three operating theatres should have a minimum of one clinical engineering technician whose duties, other than checking of new equipment, include routine maintenance checks of mains and emergency supply, preventive maintenance of equipment, checking and repair of faulty equipment and assisting with the setting up of sophisticated electronic equipment for use by medical personnel in the operating theatre and intensive-care complexes.

Diathermy - How It Works and How to Use It Safely

Diathermy uses high-frequency current to coagulate or cut through tissue. This current is discharged into the tissue from the tip of a fine electrode producing a high current density that burns, desiccates or coagulates. An arc of 1000 °C is discharged from the tip of the electrode to the tissue causing destruction of the tissue in a small area. The frequency used for coagulation is between 250 and 750 KHz while that for cutting is between 500 and 2000 kHz (0.5-2 MHz). The effect of this high frequency current on the body is totally different from that of a 50 Hz current. A 50 Hz current causes violent muscular contractions which are inconvenient to the surgeon and the current may even cause ventricular fibrillation. For frequencies above 50 kHz, muscular and cardiac effects are greatly decreased, but the conscious patient may still experience sensations of electric shock. Frequencies above 250 kHz do not cause the sensation of electric shock, and if unanaesthetized, the patient would feel only the thermal effects of the current. Current at this frequency does not cause ventricular fibrillation even if it passes through the heart.

Coagulation currents in old diathermy machines are generated by a spark-gap generator which produces damped sinusoidal wave-forms of approximately 400 Hz. Current for cutting

is produced by a valve oscillator that generates a pure sine wave at a suitable amplitude and frequency. Most modern diathermy machines are transistorized. Coagulation is achieved by bursts of high-frequency sinusoidal current with short intervals at about 15 kHz. To cut, the device produces a continuous sine wave. Transistorized sets with a blend facility (coagulate and cut) produce a continuous sine wave current upon which bursts of higher intensity are superimposed.

Diathermy current will flow when there is a conducting path between the live and the neutral or indifferent wire. This circuit is always (hopefully) completed from the diathermy-active electrode tip, via the patient to the plate and back to the machine. The high current density at the active electrode makes the tissue heat to very temperatures, resulting in either cutting or coagulation. It is essential that the indifferent plate should make good contact, over a large area, with the patient (fig. 2.2.5). The current is spread over a large area (low current density) and thus no tissue heating occurs. If the indifferent plate makes contact with the patient over only a small area, a high-current density might result which could cause burns.

In earlier years there was confusion and disagreement about what the terms monopolar and bipolar meant. Today it is agreed that in monopolar diathermy there is a single live lead from the diathermy, held in the hand. Current from here coagulates or cuts and then returns to the diathermy machine via the plate (indifferent electrode). If cautery is used on a part of a pedunculated organ, the current will flow through the narrow pedicle or mesentery to the plate. This might result in a high current density in the mesentery which could coagulate the blood vessels in the peduncle and cause necrosis of the entire organ. Resting the peduncle on a wet conductive material such as a saline-soaked swab will reduce the current density and prevent this problem. Another way of circumventing this problem is to use bipolar diathermy. This consists of a pair of forceps, the two arms of which are insulated from each other. The ends of each arm are conducting and the current flows through the patient from one arm of the forceps to the other. Current therefore only flows through a highly localized area and it cannot cause damage elsewhere. Bipolar cautery is the only cautery that should be used in patients with permanent pacemakers.

The diathermy plate (indifferent electrode) in the old machines is connected to earth. The current generator is therefore not isolated. If the plate has not been connected to the patient or if there has been a break in the earth wire, current will flow from the active electrode, through the patient and possibly through ECG electrodes (or some other conductor he was touching) to earth, causing burns at the site of contact. Burning occurs because the current exits the patient's body at sites of small area (high current density) instead of the large area of the indifferent electrode. In modern machines the diathermy plate is not earthed (in other words it is isolated or "floating"). However, it is still important to ensure good contact between the plate and the patient over as wide an area as possible so that the current density where the diathermy current leaves the body is as low as possible and therefore has minimal heating effect. The patient may get burnt if there is poor contact between the plate and the patient. Should there be no contact at all between the diathermy plate and the patient, a number of possibilities could arise:

- Some machines will not function and an alarm will sound.
- Some machines will not function but no alarm will be given.

- Some machines will function, usually poorly, as the diathermy current finds another path through the patient back to the machine. In this case it is likely that the patient will be burnt.

Safety Precautions

- Ensure good contact between the diathermy plate and the patient.

- Check for damage to the lead from the plate.

- Check for damage from the cables to the footswitch.

- Position the plate in such a way that the current does not flow from the active lead to the plate through the heart.

- Ensure that the patient does not touch the metal of the operating table. Wet towels or drapes which can provide a conductive path from the patient to the operating table should be replaced.

- When a new machine is delivered, ensure that the mains cable and plug are correctly connected. The plug should be checked from time to time to ensure that the earth wire is intact. (The machine will not work if either of the other wires is disconnected, but may still function if the earth is disconnected.)

- When coagulating tissue in infants and small children, a lower power should be used.

- Should a machine not cut or coagulate properly at the level that is normally used, the natural inclination to increase the power should be suppressed. A fault condition is usually indicated and increasing the power increases the likelihood of burning the patient. Firstly, the diathermy plate and its lead should be examined, as well as the lead for the active electrode. Check alarm conditions and the mains cable and plug. It should be noted that the power of a spark-gap generator does decrease with age but this occurs over a long period. When the cutting and coagulation of these machines is no longer adequate they should be condemned.

- The surgeon should place the footplate in a position where it cannot be accidentally activated.

- Special precautions should be observed when using diathermy in patients who have permanent pacemakers (vide infra).

- **It is the responsibility of the surgeon** (the person who is using the diathermy) **to ensure that the correct procedures are adhered to.** It is the surgeon who is most likely to be held responsible for any mishaps due to incorrect or unsafe operation of the diathermy equipment.

When should the surgeon stop using the diathermy, switch off and get a new machine?

Firstly, and most obviously, when it does not cauterize - the plate should be checked. All wires and switches should then be checked and if no fault is found, the machine should be changed. Should the second machine not work, the plate, together with its lead, and the active electrode with its lead should be replaced.

Secondly, when higher power values than normal need to be used to cut or coagulate. The machine should be changed and checked by the theatre technician or clinical engineer.

Thirdly, if the machine alarms for any reason, even though it may still work, heed the alarm, investigate and correct the fault or, if no fault is obvious, replace the machine.

A mild shock or warm feeling reported by someone touching a patient indicates a fault condition. This could cause a burn if the fault is not rectified and the patient is touched by only a finger (high current density). Bear in mind that the anaesthetized patient cannot object or even indicate that something is wrong and thus at the first sign of malfunction the equipment should be disconnected from the patient.

Defibrillators

These devices work on the theory that if a heart in ventricular fibrillation can be completely stopped by an overwhelming electric current, a normal rhythm will resume after the short period of asystole. Obviously, for this to be successful, the cause of the ventricular fibrillation must be removed.

There are two types of defibrillators, ac and dc. The ac defibrillators are seldom used nowadays because of the possibility of damaging the heart. With ac defibrillation a very high current is passed through the patient and therefore there is higher incidence of burns than with dc defibrillation. The dc defibrillation comprises a charged capacitor which is discharged through the patient by pressing a button.

The current waveform described by a capacitor discharging has an exponential shape (fig. 2.2.6). This exponential decay has been slightly modified by adding resistance and inductance in the circuit to give a waveform that is most effective in defibrillating the heart, but at the same time supplying the least quantity of electricity to the patient and reducing the possibility of tissue damage.

The problems associated with defibrillators are mainly related to their high power output. Burns and tissue damage have already been mentioned. About 400 Joules (J) are dissipated in 0.005 s. This gives a power output of 80 kW. Another problem is that attendants may get burnt or shocked if they touch the patient or the bed during defibrillation. This should not occur if the defibrillator is working properly, but should a fault occurs, someone touching the patient could connect the high current to earth through his body and could be shocked or burnt. The operator should also wear rubber shoes with a high resistance to reduce the possibility of significant current flowing through him to earth.

If the patient is defibrillated while he is connected to an ECG monitor, the large current often polarizes the ECG electrodes, blocking the ECG signal. This polarizing action might last for quite a few seconds and the enthusiastic doctor might be tempered to

defibrillate a second time even though the heart has already returned to sinus rhythm. If there is any uncertainty, check the pulse and wait at least 7 seconds before attempting to defibrillate a second time.

What should be done if the defibrillator has discharged but the patient remains in ventricular fibrillation?

This could occur if the electrode gel has been applied too liberally and there is a highly conductive pathway from one paddle, through the gel, to the other paddle. The defibrillation current flows through the gel instead of the patient's heart. This is termed creepage. Make sure that the gel is not continuous between the paddles and that the paddles are firmly applied to the chest. Unsuccessful defibrillation can also occur if the patient's heart is not able to respond to the defibrillation. The most common causes of unresponsiveness are the duration of the fibrillation (the longer the fibrillation, the less the likelihood of a successful defibrillation) and the condition of the myocardium. Hypoxia and acidosis have been reported to reduce the success rate of defibrillation, but various authors have found these to be less important than the duration of the fibrillation and the state of the myocardium. Success is also higher and myocardial damage less if larger paddles (12 cm diameter) are used.

What Energy Level Should be Used for External Defibrillation?

Between 200 J and 300 J should be used the first time, the higher value being employed if the patient is obese or has a very thick or hairy chest wall. As the thoracic wall impedance is reduced by the first shock, more current during the second defibrillation will penetrate through the heart so the output of the defibrillation should not be increased for the second attempt. Remember that defibrillation will not be successful if the patient is in asystole. If the second attempt is still unsuccessful, continue with external cardiac massage and ventilation, correct any biochemical abnormalities and then try again at a 10-20% higher energy output.

If the defibrillator does not charge or if it charges but does not appear to discharge, change the paddles and try again. If still unsuccessful, the defibrillator should be replaced.

Macroshock

Macroshock is produced by the flow of current in the milli-ampere (mA) range through the body. Although the total current might be 10 mA or more, the current is usually distributed through a large area and the current density is low, therefore only a small percentage of this current might pass through the heart. It is possible that ventricular fibrillation will not occur. When the total current applied to a person passes through the thorax (i.e. hand-to-hand shock) than a current of about 50 mA could cause ventricular fibrillation. Macroshock can arise when a fault occurs in an electrical device. (Usually two or three faults must occur simultaneously before one gets shocked.) This could come about if a device has been incorrectly wired, i.e. a diathermy machine where the earth (yellow/green) wire has been connected to the live pin of the mains plug. This would render the cabinet of the diathermy live and a person touching the casing would conduct current to

earth and receive an electric shock. (In an operating theatre, with its isolated supply, there should ideally be no current to earth, hence no shock.)

A typical double fault situation occurs when the earth wire to a device has come loose, usually at the plug (fault 1), and a short occurs with the live wire touching the casing of the device (fault 2). The potential for shock from a live casing as described above has thus arisen.

Patient circuits of modern electromedical apparatus are transistorized and run on low voltages (5-20 V). As long as these devices are earth-free the chance of macroshock occurring as a result of a fault in the device is very small as the voltages are too small to allow significant current to flow through the patient.

Microshock

The internal body resistance is approximately 500 ohms, which is about 100 times smaller than the skin resistance. Invasive techniques bypass the protective skin resistance and any shock under these circumstances will be correspondingly severe. Even a very small voltage source applied to the heart can generate a current large enough to cause fibrillation. This is known as microshock. A current as low as 100 microA (0.1 milliA) flowing through the heart could cause fibrillation.

The measures advocated for the prevention of macroshock are not necessarily effective for the prevention of microshock. To avoid microshock two precautionary measures must be adhered to:

- All electrical devices that make contact with the patient must have isolated (floating) patient circuits.

- All equipment that is grounded should be grounded to a common earth point with as short a lead as possible.

The operating theatre with its isolated mains supply should ideally not allow current to flow from live to ground, even under fault conditions. However, as a result of capacitive coupling between live and ground, leakage currents do flow and can cause microshock during a fault situation. Normal leakage currents in properly designed equipment are not large enough to cause microshock (fig. 2.2.7 A). However, when a live to casing short occurs (fault 1), in a device that has a direct connection to the patient's heart, and the patient is earthed (fault 2), the circumstances are created for microshock (fig. 2.2.7 B). In this situation, if someone touches the live casing and the patient, or worse, the conductor to the patient's heart, then dangerous currents are bound to flow, notwithstanding isolation of both the mains supply and the patient circuit. The short circuit (fig. 2.2.7 B) has removed the resistance (capacitance) between live and the casing, the person connecting the casing and the heart catheter has effectively shorted the live directly to the patient's heart. The patient is earthed and current flows through the heart to the casing of the ECG, through the capacitive coupling and back to neutral. These circumstances show how patient isolation has been reduced, which increases the patient leakage current by a factor of three. Fortunately, due to isolation of the power to the theatre, some degree of isolation is still maintained. This might not be sufficient,

however, to prevent microshock. If the power had not been isolated the neutral and earth at the ECG would have been joined and a very large current would have resulted.

Most countries have standards for the maximum permissible leakage current under normal and single-fault conditions. Leakage current should not exceed 10 microA (0.01 mA) under normal conditions and must be less than 50 microA (0.05 mA) when a single fault occurs.

Burns and Explosions

Burns to patients under anaesthesia have become a widely publicized and emotional issue. They can be prevented if a little time and care is taken to check equipment which might burn a patient, before it is used. The most common sources of burns in the operating theatre are malfunctioning diathermy apparatus, warming blankets or a burn as a burn as a result of a fire or explosion. Fortunately the latter is rarely seen today as explosive anaesthetic mixtures are seldom used.

It has already been explained how diathermy can burn a patient. Remember, a poorly connected plate can cause burns if only a small area is in contact with the patient. Secondly, if the neutral or indifferent electrode is not connected to either the patient or the diathermy machine, the current must find another route back to the machine which could be via a low impedance monitoring lead of an outdated (earthed) ECG machine. These burns are deep, take a long time to heal, and cause permanent scars on the patient's chest.

A warming blanket may overheat if its thermostat (which controls the temperature of the circulating water) should malfunction. The heater will continue to heat the water until the blanket is switched off. Burns due to this fault are usually extensive but are superficial unless very high temperature are reached. They usually occur on the "pressure points" - heels, calves, buttocks and shoulders.

An explosion sometimes occurs when a cleaning solution ignites. This can occur when a solution containing alcohol has been used for wound site preparation in cavities, i.e. mouth, nose, ears, and then cautery is used soon after. A case has recently been reported from a local hospital where such a solution was used to clean the mouth. When the cautery was used there was still a pool of alcohol in the oropharynx, which ignited and led to nasty intra-oral burn.

Another instance when an explosion might occur is when cautery is used in the presence of a recently opened large bowel. The colon produce hydrogen and methane. However, their concentrations are usually very low and the chances of an explosion occurring are small, but explosions might occur if gas such as N_2O, which is flammable and supports combustion, is used to expand the peritoneal cavity for laparoscopy. If a hot light source is used for laparoscopy and by mistake the colon is perforated, all the ingredients for an explosion, i.e. a gas which supports combustion, a flammable agent and a source of ignition are present.

The flammability of the older inhalation anaesthetic agents (ether, trichloroethylene) might still pose an explosion hazard in rural areas where these cheap agents are still used. The ideal situation is to avoid diathermy when these agents are used. A zone of risk has been

defined within which the risk of explosion is significant, especially if waste anaesthetic gases are not scavenged. In the UK this zone is defined as 25 cm around the breathing circuit. However, the SABS still recommends that in any room in which flammable anaesthetics may be administered, equipment within 1.5 m of the floor should be flame proof, explosion proof, intrinsically safe and gas tight or non-sparking. All mains switches should be at least 1.5 above the floor. When filling the vapourizer, care should also be taken not to spill flammable agents. If the skin is cleaned with ether to remove residue left by adhesive strapping, then this area should be cleaned with water and dried before a source of ignition is used.

As we all know, static electricity can cause sparks. Therefore patients and theatre staff should all wear cotton clothing to avoid the build-up of static charge. For the same reason non-conducting anaesthetic circuits and other equipment should be made of antistatic rubber, the theatre floor should be antistatic (conducting) and all new floors should have resistance between 20 kilo-ohms and 5 mega-ohms.

Pacemakers - Hazards and Special Precautions

As the quality of medical care improves, more people with coronary artery disease and other cardiac diseases will live longer, and more patients will have permanent pacemakers inserted for various cardiac conduction defects. The incidence of patients with cardiac pacemakers presenting for surgery is increasing. The presence of a permanent pacemaker in a patient presents extra electrical hazards to the patient when undergoing surgery. If sudden pacemaker power failure or sensing failure is to be avoided, certain pacemaker functions should also be checked before the patient comes to the operating theatre.

Pacemakers fall into one of the following groups:

- Fixed-rate pacemakers
- Pacemakers which sense the patient's cardiac-electrical activity:
    - ventricular triggered
    - ventricular inhibited
    - atrial synchronous
- Special-purpose pacemakers.

Owing to the multiplicity of pacemaker types, all patients are issued with a card which states the type of pacemaker, the serial number, when it was inserted, the type of leads, the preset rate or magnet rate and the name of the patient's physician.

The patient should be examined pre-operatively with respect to his underlying disease and the function of the pacemaker should be assessed. The most common problem is exhaustion of the power supply. Firstly, the patient's pre-implantation symptoms should be assessed. If these have returned, it is probable that the power supply is either failing or has failed. A one-minute ECG strip must be obtained and the peripheral pulse counted. A variation of greater than 10% of the preset rate when the pacemaker is pacing consistently also implies failing power function. It is not quite so easy to check the sensing and pacing functions on the ventricular triggered and inhibited pacemakers. However, this can be done by cardiac technologies and it should be done before the patient comes to surgery.

What special precautions should be taken by the surgical team when operating on a patient with a pacemaker?

The hazards of microshock and the circumstances through which it may occur have already been discussed. Suffice it to say that very low currents (50-150 microA) flowing to the pacemaker wires may cause ventricular fibrillation. Thus, the surgical team and clinical engineering technician must ensure that all electrical apparatus in contact with the patient have floating (isolated) patient circuits and that the earthing of all casings is in good order. Ensure that the patient is at no time in contact with any earthed object and that the cautery plate (if monopolar diathermy is used) makes good contact with the patient.

Be aware that any applied electrical signals (i.e. neuromuscular transmission monitors, evoked potentials assessments) or biological potentials (i.e. suxamethonium fasciculations) might be sensed by the pacemaker. If it is a ventricular inhibited (demand) pacemaker than it will not function during the above stimuli. Cautery and equipment driven by electrical motors (i.e. orthopaedic drills) may have the same effect.

The reference (neutral) lead of all diathermy apparatus should be connected to the patient, as far away as possible from the pacemaker, on the side of the active lead away from the pacemaker (fig. 2.2.8). Bipolar cautery forceps will greatly reduce interference from diathermy units. If interference or inhibition is still a problem, then the use of diathermy should be abandoned or limited to 2-3 second bursts interrupted by long pauses to allow the pacemaker to regain normal function.

All leads to and from electrical equipment should be as short as possible to reduce electromagnetic interference.

A strong magnet should always be available if the pacemaker is not of the fixed rate type. The magnet, when applied over the pacemaker, reverts it to a fixed rate mode which is much less sensitive to extraneous applied electrical stimuli. Most pacemakers inserted today are not fixed rate because of the possibility that they themselves may cause ventricular fibrillation. This might occur when one of the pacemaker stimuli falls on the "sensitive" part of the T wave of a preceding normal beat. The strong magnet should accompany the patient to theatre and the implanting site of the pacemaker must be known.

Pacemakers can be damaged by the high output of a dc defibrillator. If a patient with a pacemaker in situ requires defibrillation, the paddles should be placed at least 12 cm away from the pacemaker pulse generator. Pacemaker function should be assessed as soon as possible after defibrillation.

Adequate monitoring of the patient with a permanent pacemaker is extremely important so that any interference with function can be immediately observed and acted on. ECG monitoring and palpation of the peripheral pulse are mandatory. A peripheral pulse detector may be used instead of palpation. This is important as it is vital to know whether electrical activity on the ECG is followed by mechanical activity.

Part II

Radiation Hazards in the Operating Theatre

There is a wide spectrum of radiation that has both electrical and magnetic properties (fig. 2.2.9). Visible light and X-rays cover only small portions of the electromagnetic spectrum. This section deals with radiation that is used in the operating theatre for either diagnosis or therapy, namely X-rays and gamma rays. Exposure to these rays can be harmful and their use is therefore governed by statutory proclamation and by SABS recommendations.

X-rays are absorbed to a certain extent by tissue, and the dose, or amount of X-rays absorbed, is measured in gray (Gy) (1 Gy = 1 Joule/kg = 100 rad). The concept of dose equivalent is used to indicate the effect of radiation on biological tissue. Dose equivalent = absorbed dose x quality factor, where the quality factor depends on the type of radiation, i.e. X-rays, gamma rays, etc. Radioactive materials, producing gamma rays which are similar to X-rays, are sometimes also used in theatre for therapeutic purposes.

The sievert (SV) is used to quantify radiation for the purposes of radiation protection for people working with X-rays.

Effects of Radiation on Tissue

Certain tissues in the body are more sensitive to radiation than others. Probably the most sensitive is bone marrow, where rapid cell-division occurs. Other tissues that are particularly sensitive include the thymus, lymph tissue, ovaries and testes.

There are several factors that determine the extent to which a worker's body is exposed to radiation:

- the frequency and time of exposure
- the proximity to the X-ray source
- the position of the worker in relation to the primary beam (fig. 2.2.10)
- the energy of the radiation beam (proportional to the kV)
- the amount of protection, in the form of shielding and/or lead garments.

Radiation can have both somatic and genetic effects on living tissue. The somatic effects can be immediate or they might be delayed. Immediate effects include impotence, sterility, epilation and skin ulcers which may occur soon after excessive irradiation. The delayed, long-term effects include leukaemia, bone tumors, cataracts and various forms of cancer. Genetic effects, which are usually recessive and deleterious, are especially important for pregnant women, because the fetus is more susceptible than adults to the effects of radiation, and excessive radiation may cause fetal abnormalities.

Practical Radiation Protection

No X-ray equipment should ever be used by persons not qualified to do so. All persons working with X-ray equipment, and those who are regularly exposed to scattered radiation (vide infra), must be registered as radiation workers and should therefore wear a

film- or TLD badge to monitor their exposure to radiation. Internationally it is accepted that radiation workers should not be exposed to a dose of more than 50 mSV per year.

The main source of radiation for persons who are present when X-rays are taken, is scattered radiation. Whenever an X-ray beam strikes an object, some of the X-rays are absorbed, some pass through (to form the image on the film), and some are reflected or scattered (fig. 2.2.10). Scattered X-rays have a lower energy than the primary X-ray beam but exhibit the same properties and therefore present the same hazards. Note that scattered radiation is least at right angles to the primary beam. It is obvious that one should never unnecessarily expose oneself to primary radiation; equally so, one should take care to minimize exposure to scattered radiation as well. If one has to hold a cassette, lead-impregnated rubber gloves and a lead apron should be worn. One should stand as far away as possible from the primary beam or should use tongs to hold the cassette.

The further away one is from the source of radiation (remember that objects in the primary beam become sources of scattered radiation), the lower the exposure. The intensity of radiation decreases with the square of the distance from the X-ray source. Thus the exposure to a person 2 metres from the X-ray source is four times less than that to a person 1 metre away, and a person 3 metres away receives one ninth of the exposure.

When fluoroscopy is performed, the beam should be restricted to as narrow an area as possible by the use of collimators and cones. The time of exposure should be as short as possible and lead aprons of at least 0.25 mm thickness, increasing by 0.01 mm for each kV above 100 kV, should be worn. Intermittent exposures should be used in preference to continuous exposures. With modern image intensifiers the quality of the fluoroscopic image has improved a great deal. In addition the newer machines can hold the last image, even though the machine is not producing X-rays, which reduces radiation to both patient and operator.

When using mobile equipment, the operator and assistant must be at least 2 metres away from the tube and as much at right angles to the beam as possible. Obviously lead aprons must be worn.

Patient protection is also important and gonads and other body areas sensitive to irradiation - thyroid, skull and sternum (in children) and eyes, should where practical be covered with protective lead sheeting. The beam should be limited to the smallest possible area.

Comment

Electricity and Radiation in Operating Theatres

K D Boffard

It is always easier to avoid trouble than to extricate oneself from it. A surgeon should strive to understand when problems might arise, and to anticipate them. A thorough understanding of the equipment in an operating theatre environment, and how it works, is basic to this.

Prior to the widespread use of antistatic flooring, static electricity was a problem, and there are several cases on record of patients being "defibrillated" via a central venous pressure (CVP) line. Where possible, lines used for CVP measurement should be used with dextrose 5% rather than an electrolyte solution.

A major and recurrent problem cropping up in the insurance society statistics is that of burns following over-generous use of skin preparation solution, if alcohol-based. Whenever possible, care should be taken to avoid pooling of skin preparation solutions (i.e. chlorhexidine in alcohol) in skin creases, or between buttocks. A particular area of concern is under the pad of the diathermy.

Every effort should be made to avoid spillage and if this occurs, to mop up the residue and to dry the skin before surgery is commenced.

The diathermy pad should not be placed in a drainage area before skin prep, but should either be attached in an area remote from the dependent area or the patient should be dried prior to the application of the diathermy plate. Patients in the lithotomy position are at particular risk.

Poor contact will result in sparks which ignite the alcohol, and the resulting conflagration is disturbing, to say the least.

Chapter 2.3

Wound Healing and Care of Wounds

A Lamont

Introduction

Epithelial cells and allied tissues retain the power of true regeneration while most meso-dermal tissues are restored by the formation of new tissue which is of less specialized nature and leads to fibrosis with impaired function, varying degrees of morbidity and even mortality. The organ of repair, so-called "granulation tissue", is based on the fibroblast and its products and is concomitant with new blood-vessel formation.

Definition

Wound healing is a combined phenomenon of **removal** of denatured and foreign material, **regeneration** of remaining tissues and **repair** of other tissues by new tissue formation in several overlapping processes which, in varying degrees of severity, result in a scar with varying degrees of loss of function. The varying degrees of response depend on the extent of the injury or the degree of wounding or damage and are explained by the concepts of primary and secondary wound healing.

Primary Union

This takes place in the "ideal" surgical aseptic wound after haemostasis. The normal tissues are in apposition and stuck together by a thin layer of coagulum. This layer is absorbed by a limited number of leucocytes and a thin layer of collagen fibres is produced by a small number of fibroblasts. New vascular ingrowth is minimal. Epithelial continuity is restored on the surface by migration and mitosis. After five or six days the process is complete, leaving a thin layer of young connective tissue which, after maturation, leaves a thin, pale, soft and cosmetically acceptable scar.

Secondary Union and Healing by Granulation

Factors which may prevent or disrupt the ideal of primary union are:

- tissue damage or necrosis
- large disrupted wounds with serious discharge
- infection and resultant excessive inflammatory process
- mechanical disruption during healing and
- altered healing such as after irradiation or metabolic inhibiting factors

The wound is then said to heal "from the bottom up" by a process of **granulation, contraction** and **advancing epithelization.** The essential components of this characteristic granular tissue are fibroblasts migrating into an exudate in conjunction with buds of new vessel formation at right angles to the underlying capillaries. There is usually a marked inflammatory response with a large number of polymorphonuclear leucocytes. The leucocytes respond to the presence of denatured matter and micro-organisms. With heavy infection these micro-organisms may inhibit the growth of the granulation tissue. Once a robust layer of granulation tissue has formed, the penetration of toxins and bacteria into the tissue is checked. The wound becomes sealed off and the systematic effect decreases. The process of fibrosis then commences with a characteristic contraction of the edges of the wound due to the active contraction of specialized fibroblasts with smooth muscle properties, the so-called myofibroblasts. The collagen fibres produced with their matrix then maintain the position and after epithelialization this collagen undergoes a process of maturation and softening. In very large wounds a chronic ulcer can result with a static layer of granulation, with excessive underlying fibrosis and poor epithelialization. The migration of the epithelial edge has its limitations and the thickness of the granulation tissue is limited by its ability to form new blood vessels.

Once epithelialization is complete the inflammatory response is altered to the activity of cells such as mononuclear leucocytes, plasma cells and lymphocytes. The epithelium thickens though it never forms normal skin with hair follicles and sweat glands. The underlying collagen mass is devascularized and remodelled in a process of maturation. The cicatrization can take a considerable time and can be delayed by factors as yet not fully understood. Other factors can further lead to hypertrophy of scar tissue and delayed maturation with so-called keloid formation.

<center>**Physiology of Healing (E-P-P-Ne-Ma-C-T)**</center>

Several processes can be observed which overlap or occur simultaneously:

- Epithelialization
- Productive or preparatory healing phase
- Proliferative or collagen phase
- Neovascularization and revascularization
- Maturation phase
- Contraction
- Tensile strength increase

<center>**Epithelialization**</center>

Though clinical epithelialization is obvious on the third day only, within hours, loosening of intercellular connections at the wound edges occur in preparation for two processes, i.e.,:

- **Epithelial cell migration.** Cells migrate actively from the wound edge down the side of the defect, then through or across the fibrous clot towards the wound centre.

- **Active mitosis.** This takes place in the basal layer at a distance from the edge of the defect.

This process of migration and proliferation produces a sheet of cells which covers the open areas of the wound and then stops when cover is complete, by a mysterious process of "contact inhibition" involving substances called *calones*. Once the wound is covered, the epithelium assumes stratified squamous characteristics, but without papilliform dermal processes or skin organelles if these have been destroyed with the deep dermis.

The quality of the epithelial cover decreases the further it migrates and the centre of large wounds can consist only of a fragile single layer of cells easily stripped from the underlying fibrous tissue. **It must be stressed that in full thickness skin loss, the normal epithelium and dermal configuration is never regained leaving only fibrotic tissue covered by an epithelial layer.** In the partial thickness skin loss the quality of the resultant cover is dependent on the depth of the remaining dermal element. In partial loss of the epidermal and germinal layer such as in superficial abrasions, healing will leave a very thin scar which is almost not detectable. In deep dermal damage epithelialization can only occur from the lining of remaining skin organelles such as sweat glands and hair follicles. Resultant scar is more obvious and more prone to hypertrophy and keloid or simple distortion due to wound contracture. Most wounds, particularly burns with a large surface area, will demonstrate a spectrum of the above differences.

<center>**Productive or Preparatory Healing Phase (old term lag phase)**</center>

First three to five days. During this phase, also called the substrate phase, damaged tissue undergoes necrosis, vessels thrombose and plasma and lymph exudes into the wound. Inflammatory cells move out of the capillaries into this fluid substrate and commence the

removal of dead tissue and foreign material. There is also the early interaction with colonizing organisms. The exudate becomes rich in mucopolysaccharides.

Proliferative or Collagen Phase (4-21 day)

By the third day fibroblasts begin to appear in large numbers which introduces a so-called fibroblastic phase of three to five days. From the fifth day and into the third week a rapid synthesis of collagen takes place. The "healing ridge" appears in this phase and the wound is characteristically warm and erythematous.

The **fibroblast** is the dominant cell in healing tissue and in the collagen phase it is plump and endowed with an abundant protein synthesizing endoplasmic reticulum and ribosomes. These fibroblasts have two possible origins which attest to their multi-potential nature. They may arise from activated local resting fibroblasts or from cells transported via the bloodstream. Collagen synthesis takes place on the ribosome network of the fibroblast. Hydroxylation of prolines and lysines into tripeptide sequences is an essential step and requires the presence of iron, ascorbic acid, molecular oxygen and alpha-keto-glutarate. Three coiled polypeptide $delta_1$ and $alpha_1$ and $alpha_2$ chains join in a left-handed configuration to form tropocollagen of 3000 angstrom x 15 angstrom units in size. This substrate is soluble in diluted salts and acids. These molecules then join with others in a right-handed supra-helix with 25% overlap to form pro-collagen. The can be split off again by means of pro-collagen peptidase. Increasing crosslinks between molecules make the collagen more and more insoluble so that mature collagen forms as a relatively stable substance. A muco-polysaccharide matrix, also synthesized by the fibroblasts, is attached to and around the collagen network.

It must be emphasized that along with the process of synthesis, there is an active process of lysis of collagen. This allows the initial random and disorganized network to mature in the wound after the relatively short collagen phase.

Neo-Vascularization and/or Revascularization

Healing and the development of granulation is accompanied by vascularization. Buds from existing capillaries grow out at right angles into the empty spaces. These are at first blind ended and circulation is limited. In primary healing or in the small space between skin grafts and bed, a "plasma circulation" is formed with occasional red blood cells passing into and across the space. Eventually these vessels meet across the space by endothelial link-ups. The bloodflow in this link-up or revascularized graft increases to a peak between the second and third week, and then decreases. In secondary healing the formation of new blood vessels is essential. These take the form of capillary loops growing further and further outwards from the capillary bed.

By whatever process blood supply is restored to the dead space or grafted tissue without circulation, there seems to be a limit to how far this micro-circulation can reach out beyond the original vascular bed. This is supported by the clinical observation that granulation does not continue to develop to an indefinite thickness, but reaches a static phase in which it outgrows its ability to form new blood supply. Similarly grafts of tissue which are too large and too thick will undergo necrosis before revascularization can take place. It would appear

to be possible to overcome this problem by repeated grafting provided that a healthy vascular bed is established after each graft.

Maturation Phase or Phase of Differentiation

This phase is a continuation of the collagen phase and lasts with ever decreasing activity for months or up to two years until a stable balance is established between collagen synthesis and collagen lysis. During this phase the collagen is afforded the opportunity to remodel with ever increasing insolubility and decreasing cellular content. The vascular element recedes and there is an increase in tensile strength. There is a significant bio-mechanical influence on the wound so that the initial random collagen network is remodelled into a more parallel arrangement along the mechanical stress lines. It is in this phase that the tissues such as facial layers and tendons attain the tensile strength required for their function. Maturation is affected by vit C, pressure (mechanical), blood supply and cortisone and some drugs.

Contraction

The remarkable phenomenon of wound size decrease was only fully explained when it was found that fibroblasts could adopt the characteristic of smooth muscle cells and that the change from one cell form to the other was possible. Fibroblasts could by the synthesis of contractile proteins establish an active process of mechanical shortening, similar to the process seen in smooth muscle cells. These so-called myofibroblasts contain a contractile protein of the actomyosin type and occur beyond the edge of advancing epithelial cells in that part of the granulation tissue which is undergoing changes seen after epithelialization has covered it, i.e., flattening and collagen remodelling. The activity of these cells gave rise to the "picture frame" concept of wound contracture. By active shortening, the tissues are apposed and this position is maintained by the collagen which is laid down. **Contraction is an intracellular process and is not a function of the collagen fibres.** This shortening is registered by the surrounding nerve endings sensitive to stretching and particularly in flexor areas this whole process is enhanced by the reflex, muscle, pain-sparing activity, thus leading to the characteristic deformities seen in large, deep burn injuries.

Tensile Strength

Collagen is the main element contributing to wound strength and permanent soundness of repair. Scar tissue is weaker and more brittle than normal tissue. After five months it has only half the strength of normal tissue to resist rupture. This explains the stretched scars seen in young individuals with a pronounced elastic element to the normal surrounding tissues. In studies of tensile strength in wounds it is only at approximately 100 days that the curve starts to flatten out at a significant level. This increases to approximately 70% of normal tensile strength after one year. **At the time of conventional suture removal, from five to 15 days, only a fraction of the eventual strength has been attained.**

Etiology

Lacerations and Sharp Penetrations

These wounds are usually clean separations of tissues with little tissue damage or contamination. In practice most of these are superficial and heal by primary intention.

Abrasions

Here the surface of the skin is usually involved to a varying extent both in depth and in area. So-called "road burns" can be included, where mechanical abrasion is combined with the generation of heat by friction with resultant surface necrosis as in a burn wound. Healing is normally by epithelialization from surrounding skin elements and can be seriously hampered by the presence of incorporated materials as contaminants and by infection.

Crushing, Avulsion and Degloving

These injuries are usually more serious though the true picture can be difficult to assess early after trauma. There are often underlying fractures which serve as an index of the severity of the trauma. Degloving injuries can be particularly deceptive with large apparently undamaged areas of skin separated from the underlying anatomy and blood supply with a large space into which vast volumes of blood and fluid can be lost. Crush injuries can also be deceptive in that deep delicate structures may be damaged or severed with little skin damage. Avulsions can cause widespread injuries of different tissues at different levels in a vertical plane.

High-Energy and High-Velocity Wounds

These injuries are associated with high-speed motor vehicle accidents and gunshot wounds respectively. Both are serious injuries in that large areas of tissue damage occur which can often be accurately defined only after more than one attempt at debridement.

These wounds, along with the wounds caused by crushing, avulsion and degloving usually need surgical intervention and corrective measures to achieve healing. In many cases the healing process is followed by lengthy programmes of rehabilitation.

Burns

These injuries have many causes such as:

- dry heat or fire burns normally causing varying areas of deep skin loss
- hot fluids or scalds with burns of a widespread more superficial nature
- flash burns of short duration but intense heat
- electrical burns
  - chemical burns and a large spectrum of radiation burns, from common sunburn to high-energy radiation injuries.

Human and Animal Bites

These are wounds demanding special attention because of the high risk of infection.

Pathology

Most wounds will have some tissue loss, however minimal, with release of factors such as prostaglandins, histamines and vasoconstrictors which will usually give rise to an early triple response of vasoconstriction followed by vasodilation and swelling.

The inflammatory response is a consideration in all wounds. Subsequent pathological changes will depend on the amount of damage and necrotic tissue and by the presence of infection. Most wounds, particularly burn wounds, demonstrate three zones. The zone of coagulation or necrosis is where the injury has been most severely felt causing death of cells. The zone of stasis is immediately adjacent to this area of necrosis and is usually lost along with the coagulation zone. This is why some wounds appear to become "deeper" after initial injury. The zone of inflammation is where vital tissue reacts to the injury and whence the body mobilizes its forces of healing.

Infection

Infection is established once the organism population exceeds 100.000 per mm^3 of tissue. The ability of organisms to multiply unchecked is enhanced by factors such as foreign material, necrotic tissue and poor blood supply. It is thus imperative that the wound is cleaned and debrided not just to remove the organisms already present, but to remove the substrate in which they will flourish. Any surgical manipulation which will damage tissues will do more to encourage infection than the presence of a low count of organisms in healthy tissue.

The common organisms are:

- the streptococcus and of particular importance, the Beta haemolytic strains (Lancefield group A) which are lethal to skin grafts
    - staphylococcus with its numerous resistant strains, particularly in chronic wounds
    - pseudomonas which tend to heavy infections in the presence of necrotic tissue
    - *klebsiella* and *E coli* contaminants of hollow organs
    - clostridia strains with all their dangers
    - the dangers of opportunistic infection and chronic infection caused by fungus strains.

Infection is the most important cause of defective healing.

The antibiotic era has not removed infection in wounds, but has only changed its character. In clean wounds antibiotics are not necessary, as risk of infection is minimal. If contamination of such a wound is predicted, the antibiotic should reach the wound at the time that the organism does, and should be administered in effective dosages for a short period only, not longer than two days. This is true and effective prophylactic use, with little risk of encouraging resistant strains. The use of antibiotics is of limited value in contaminated wounds and will not save the patient from infection. This is a surgical problem and is best treated by early effective surgical wound care and antisepsis.

The sepsis in a wound is best treated on two fronts, i.e., direct wound care with antiseptic agents or even antibiotic substances and systemically in effective dosages via the bloodstream. Systemic administration loses its effect as the wound becomes sealed off by fibrosis as in chronic ulcers. Antibiotics should thus only be used to counter systemic effects of infection. **It cannot be overstressed that the bland use of broad-spectrum antibiotics, locally or systemically, in the hope of containing unidentified organisms is incorrect and leads to the development of resistant organisms.**

Factors Inhibiting Healing

Age

Healing may be significantly slower in advanced age while the scar tissue reaction is more energetic in the young with hypertrophy after healing being more common. Because of reduced tissue elasticity, scars are usually more cosmetically acceptable in the aged than in the young.

Congenital Factors

Wound healing is impaired in some connective tissue disorders such as osteogenesis imperfecta and cutis laxa or in specific enzyme deficiency such as absent lysil hydroxylase.

Starvation and Protein Deficiency

It is a clinical observation that after loss of 20% of normal body weight, healing may be impaired though specific reasons such as hypoproteinaemia are seldom demonstrated biochemically. In patients with inverted globulin/albumin ratio, healing is usually normal.

Vitamin C

It is known that after trauma, above-normal ascorbic acid intakes of up to 1 gram/day are fully utilized, yet healing is not grossly impaired by mild ascorbic acid deficiency and vit C free diets have not been useful in inhibiting the tendency to hypertrophic scar formation.

Oxygen

Clinically we are aware of better healing and tissue survival in areas with good quality blood supply such as the face. While fibroblasts, macrophages and neutrophils can survive low oxygen tensions, they can only perform their specialized functions at a PO_2 level of around 100 mm Hg. With normal wound perfusion, satisfactory PO_2 levels can be found up to 70 microm from the nearest capillary. Perfusion is itself dependent on factors such as cardiovascular status, viscosity and respiratory exchange. These can be affected by severe generalized trauma.

Mechanical Factors

Repeated trauma directly as a result of poor immobilization will delay the rapid healing of an area and usually leads to excessive fibrosis.

Metabolic Diseases

Diabetes mellitus, jaundice and uraemia are associated with impaired healing of unpredictable degree. Particularly in DM the utilization of carbohydrate is impaired and poor micro-circulation plus susceptibility to infection make matters worse.

Corticosteroids

Cortisone inhibits collagen synthesis and stimulates collagen lysis. By depressing the inflammatory response the start of healing in the preparatory phase is delayed, but once healing is started, the effect of cortisone is reduced. Cortisone also inhibits wound contracture. These effects are reversed by vit A.

Radiation and Cytotoxic Drugs

Any suppression of cellular activity will inhibit healing. Ionizing radiation has two effects. Firstly, directly on the healing process by depressing cells engaged in the process and secondly, in post-irradiated tissue which has a scarred appearance, the blood supply is impaired. Cytotoxic drugs can only impair healing in high doses when applied locally in such a way as to damage normal tissues.

Other Drugs

Drugs which would impair healing in a controlled predictable way would be a useful therapeutic tool in hypertrophic scar. However, all chemicals such as beta amino proprionitride (BAPN), mono-amine oxidase inhibitors or colchicine and penicillamine are too toxic. They are unpredictable in their ability to cause clinically beneficial scar-tissue control or lathyrism - the lysis or reduction of connective tissue.

Zinc and Other Trace Elements

Many elements and radicals such as zinc, magnesium and phosphate have been proved to be "essential" for healing, however, administration of these elements seldom give rise to increased healing, probably because they are never truly absent in clinical situation.

Classification of Wounds

- clean surgical wounds
- contaminated wounds
- infected wounds
- chronic wounds

Clean surgical wounds can be expected to heal easily by primary intention as described above.

Contaminated wounds are fresh wounds in which foreign material is present and in which tissues have been rendered non-vital. The danger of infection and suppuration is very high unless sound, early surgical debridement is carried out.

Infected wounds are those wounds which have been contaminated primarily or secondarily and where inadequate debridement and antiseptic measures have allowed organisms to flourish causing typical suppuration.

Chronic wounds are slow-healing injuries demonstrating established fibrosis. This fibrosis has sealed off the wound so that the systemic effect is minimal with reduced surrounding inflammatory response. There is a layer of granulation tissue of varying thickness which is subject to colonization by a wide spectrum of organisms. Epithelialization is slow or can even recede with enlargement of the extent of the wound as in lower leg ulcers.

The time scale for acute, surgical and contaminated wounds is usually up to five days. Infection is characteristic of a subacute phase lasting from six days posttrauma until approximately three months. Chronic wounds are usually older than three months and may be present for years.

Clinical Factors

A useful question is "what stopped the blow or penetrating agent?" This will enable us to predict the extent of the injury in underlying areas with surprising accuracy. These predictions are then confirmed or dispelled by a careful systematic examination of the wound locally and the patient as a whole to assess systemic effect and exclude other injuries.

Diagnosis

Signs such as profuse bleeding, rapid swelling, poor blood supply (locally or to distal parts), nerve function disorders (both sensory and motor), bruising and discoloration and, in more subacute wounds, inflammation and suppuration, must all be carefully followed up and the reasons sought. Systematic evaluation for problems such as shock, respiratory or excretory function, pyrexia and loss of consciousness must be considered.

The rapid assessment of a seriously injured patient by a traumatologist is of immeasurable value so that all systems affected can be dealt with during or soon after resuscitation by the various specialists who will be involved as a team for any particular patient as indicated. Again, radiological special investigations as well as laboratory backup for electrolyte, respiratory and renal functions is essential.

Treatment

Prevention

Avoiding unnecessary accidents is the plea of most medical, nursing and paramedical personnel. This applies particularly to children and the infirm. Child safety units have achieved great success in public education, particularly with respect to burn injuries. Again alcohol and drug misuse must be mentioned and more should be done to avoid the ravages of alcohol as a major etiological factor, particularly in motor vehicle accidents.

First Aid

In serious injury rapid care of respiratory emergencies and blood loss with rapid transport to specialized trauma units by experienced teams has preserved life and minimized morbidity.

Most important is the maintenance of the airway. This is a simple act in most cases and is the most urgent emergency if the airway is compromised by trauma positioning or by blood and fluids.

In small wounds, non-traumatic pressure haemostasis by bandaging, with elevation, is of prime importance. In small burns the application of cold water in clean dressings has two advantages: firstly, it has a powerful analgesic effect and secondly, the energy effect of a heating agent is reduced, particularly if the cold water is applied immediately after heat exposure. This reduces tissue damage and it is claimed with justification that the suppression of the vigorous inflammatory response has a significant effect on the early healing process.

Administration of oral fluids is unwise as this can delay the administration of a safe general anaesthetic which is particularly useful in rapid early treatment of children. Personnel trained in the administration of intravenous fluid should not hesitate to do so, particularly in larger burns and these should be in the form of crystalline solutions. More harm is done by withholding or delay in fluid administration particularly if the injured person has to be transported to a distant centre.

Heavy analgesia is necessary only if the transport of the patient with serious injuries and fractures to the treatment centre will be complex and traumatic.

In case of the amputations, after attention to haemostasis the amputated part should be placed in a dry plastic bag and rendered water tight. No attempts at cleansing and antisepsis should be made as this may damage tissues and delicate anatomy. The plastic bag should then be placed in a mixture of ice and water. The mixture will ensure that the ice is not below freezing and will significantly prolong the viable ischaemic time especially in tissues proximal to the MP joints, i.e., muscle. Rapid transport to a centre capable of assessing these injuries and repairing them when indicated, should follow with a phone call to warn the centre of the arrival so as to expedite mobilization of the receiving staff and the therapeutic team.

Haemostasis

Haemostasis by blind use of instruments such as artery clamps is dangerous as serious unnecessary damage can be done to structures such as mixed nerves, especially in the upper limb. Pressure by bandage with elevation is adequate, or digital pressure to larger arteries is indicated until accurate anatomical haemostasis can be achieved surgically. The use of a tourniquet is unwise as it can cause ischaemia if left in situ for too long, pressure injury to nerves if applied too tightly or serious blood loss if applied so as to occlude the venous drainage and the arterial inflow.

Surgical haemostasis is usually required for larger vessels only and the capillary and small-vessel haemorrhage will have stopped by the time surgery is carried out. These methods should be atraumatic, i.e., ligature or diathermy of the vessel only and not the vessel plus a mass of surrounding tissue.

Injury to large vessels such as in the neck and groin requires local digital pressure and the urgent attention of a vascular surgeons, as they are life threatening.

Cleansing Antisepsis and Debridement

A useful clinical approach in extensive wounds is the so-called "48 hour look-see" after initial radical debridement. If the first surgical debridement has been carried out effectively, most wounds will soon be ready for delayed primary suture or other cover by graft or flap, as indicated, within 48 hours. However, many high-energy and high-velocity wounds need to be re-inspected after 48 hours to be sure that only vital tissue has been left. This regime should be actively planned and the wounds should not be opened in the ward. They should be well covered with sterile dressings which are left in situ until the next inspection under anaesthesia in the operating theatre. This will prevent infection for up to six days - leaving adequate time for planning elective reconstructive procedures which may be contra-indicated in infected wounds.

Cleansing solutions and antiseptic agents should be used in large volumes, be effectively antibacterial and be totally non-toxic to tissues. The best solution is a balanced electrolyte with a low concentration of soluble iodine. Even saline causes tissue damage in that it will disrupt electrolyte balance in the superficial cells. Any agent which causes a layer of dead cells, no matter how powerfully antibacterial, will enhance infection because once the antibacterial effect has passed, the non-vital tissue is a fertile medium for organisms which cannot be controlled by this tissue. It is human to use thick, dark coloured agents to kill organisms, but if this also kills tissues, more harm will eventually be done.

Closure and Suture

These techniques are dealt with in detail in a subsequent chapter.

Secondary Suture and Delayed Primary Suture

The obvious ideal is the immediate safe closure of a wound to promote primary healing, but this should not become an obsession that clouds good surgical judgement. The decision to leave a wound open is a difficult and responsible one. The primary and most basic reason is in a situation where tissue shortage or severe swelling does not allow closure. If a wound will not close with reasonable measures, then it must not be forced to close as this will cause iatrogenic complications. These complications include ischaemic skin flaps and sutures which will cut into and damage healthy tissue. Strong suture material is not a rational solution and so-called "tension sutures" should be employed with great circumspection. Other means of cover must be sought and will be considered in another chapter.

The second problem is the disadvantages of closure over tissue which is not certain to be free of contamination and which may require further inspection and debridement. This

delay in closure as described above is quite reasonable, followed by delayed primary closure. This takes place in a phase in which the wound would in any event only have completed the preparatory phase of healing or just entered the collagen phase and is as yet free of infection.

The reclosure of wounds which are non-infected several days after primary suture and which have been opened to drain haematoma or have suffered dehiscence, demonstrate similar healing capability and indeed may "catch up" or appear to heal faster than a primary closure. This is because the collagen phase has already been initiated and the preparatory phase is over.

Secondary suture is quite a different concept. This attempt at closure takes place when the collagen phase is well established or even into the maturation phase in subacute or chronic wounds. The wound edges demonstrate a marked degree of immobility compared to earlier wound phases in that fibrosis, contracture and oedema have caused shrinkage and fixation of tissues around the defect. This can take place without any tissue loss and obviously is worse when tissue is missing. This tissue must be remobilized and will "give" to some extent, once tension is applied. However, the procedure is not without its dangers for the following reasons:

- Firstly, the tissue is swollen and generally holds suture material poorly.

- Secondly, complete mobilization is difficult and if too much tension is applied in an attempt at complete closure, ischaemic changes can be produced in skin flaps or within sutures which will cut through the tissue and often leave a defect as big as or bigger than the defect in its unsutured state.

- The last danger is that of enclosing infected granulation tissue or cavities under skin flaps, thus producing an abscess situation despite surgical drains, with or without irrigation. Secondary suture is a useful maneuver if carried out with the above dangers in mind, but its success cannot be taken for granted and cover for such wounds is often best achieved by other methods.

Special Type of Wounds

Human Bites

The dangers of infection in bites has been mentioned particularly in penetrating wounds which may appear relatively trivial on the surface. These wounds must be explored thoroughly, cleaned and debrided and sutured only if primary healing can be predicted. Generally it is wiser to leave them open before delayed primary suture is done. Secondary healing with elective repair as a delayed procedure is often best. This delayed surgical procedure or reconstruction takes place once the maturation phase is well established or past.

Phosphorus Burns

Contamination of wounds with this element will lead to extensive damage if the metal is allowed to continue its reaction with the air. The first-aid treatment of these wounds is thus some form of air-tight occlusive dressing such as thick dressings, wet or dry, or paraffin gels.

Specific chemicals such as a copper sulphate in 1% aqueous solution may be of value. Early radical surgical debridement is the measure of choice with particular care to avoid further injury to the patient and injury to the therapists concerned.

Chemical Burns

These injuries can be effectively treated if a specific antidote to the agent is known, provided that this antidote is not in itself harmful. For instance, neutralization of strong acids or alkali burns is best carried out with diluted solutions of a weaker alkali or acid respectively. In general, large volumes of bland substances or water will help to arrest the damage.

Radio-Active Contamination

Specific steps are followed to achieve decontamination without further spread of radio-activity and then to store and ultimately dispose of the radio-active products. This can all be done prior to or during any surgical treatment which may be indicated in the acute phase.

Very High-Velocity Bone Wounds

This is included to emphasize the well-known need for adequate exploration and debridement of tissues devitalized by the shock waves after soft-tissue penetration and particularly in combination with the injury to bone.

Fat Necrosis

Injury to fatty tissues can follow relatively minor blunt injury with minimal damage to overlying skin. The devitalization of this tissue with its relatively poor blood supply can lead to collection of sterile lipid, which may further undergo calcification or which may become infected. Loss of tissue then leads to indentations of the skin as a late sequel. Such injuries are common in the breasts and thighs.

Complications

The immediate complications of haemorrhage, sepsis and necrosis have been dealt with in preceding paragraphs.

Wound Breakdown and Dehiscence

Superficial wound breakdown carries little danger and can safely be left to heal spontaneously. More serious are breakdown in surgical structures such as intestines or large vessels. Abdominal wound breakdown is a useful model. It is seldom complete and when resutured, heals quickly because, as stated previously, the healing process is well under way. It is thus not a disturbance to the healing process, but rather a mechanical disruption. The most common cause of wound disruption is surgical technique. There is a wide spectrum of problems ranging from minor expelling of suture materials, as in any foreign body reaction, to major disruption of anatomical layers. The reasons, apart from infection and factors before, are early absorption of material such as catgut, ischaemia due to overstressed sutures or the

suturing of poor quality traumatized tissue. The basic principle is atraumatic suture technique with tension-free, mono-filament, non-absorbable, interrupted sutures placed in healthy tissue, using thin material in not too generous quantities.

Fulminating and Complex Infections

Septicaemia and Bacteraemia

Bacteraemia may be a transient symptom-free presence of organisms in the bloodstream.

Septicaemia is persistent with pyrexia, tachycardia, etc, indicating the self-propagation organisms in the bloodstream. Toxaemia gives similar signs and symptoms of illness as a result of toxins produced by organisms in a wound or in the bloodstream.

Tetanus

The clostridium of tetanus is a non-invasive saprophyte which remains close to or within the wound and its effects are due entirely to the effects of its exotoxin. It enters the wound as a contaminant usually from the soil exposed to animal excreta. The main action of the toxin is to combine with and increase the conductivity of synapses in the peripheral and central nervous system resulting in characteristic tonic and clonic spasms of voluntary muscles. This starts early in the muscles of mastication and later spreads to the whole body.

This disease is entirely preventable by prophylactic immunization, thorough cleansing of wounds and the use of booster doses of toxoid. In established tetanus the use of human globulin, sedatives and muscle relaxants are useful in tiding the patient over the effects of the toxin in the nervous system.

Gas Gangrene

This is a fulminating infection caused by anaerobic spore-bearing organisms of the clostridium group. They include *clostridium welchii* alone or in combination with *clostridium septicum, oedematiens* and *sporogenes.* The classic picture is that of a toxic patient with a contaminated penetrating wound of a limb with little apparent inflammatory reaction and the presence of gas bubbles in the wound and along muscle planes along with a copious, watery, foul-smelling discharge. The proteolytic enzymes produced cause rapid spread, especially in devitalized muscle, with resultant colour changes to a green-black hue, softening and putrefaction. Treatment includes antibiotics specific to the organisms, antitoxins, general supportive treatment for shock and early radical surgical removal of all affected tissue.

Necrotizing Fasciitis (Fournier's Gangrene, Cancrum Oris)

Fournier first described the occurrence of rapidly advancing unexplained gangrene of the scrotum. Later Meleney recognized the role of synergistic infection as a major aetiological factor in a set of syndromes characterized by progressive cellulitis and obliterative endarteritis caused by the synergistic activity of a wide range of organisms. These organisms are often enteric in origin, but principally anaerobic, streptococci and *Bacteroides* strains.

Affected tissue requires effective and often repeated debridement. The condition often occurs with a systemic disease such as acute severe infections or DM, but can be a fulminating illness in healthy young individuals with a significant mortality depending on the effectiveness of treatment.

Stretched Scars

Scar tissue is weaker than normal tissue because of stress imposed by surrounding elastic tissue, especially in younger persons. These scars often undergoes stretching. This results in spreading and depression of the scar with an unacceptable cosmetic result. The appearances are similar to hypertrophic scars, but they have a different aetiology. The prevention of this stretching can be achieved only by the use of non-absorbable material to support the dermis during healing. These sutures should be left in place until the wound has attained the required tensile strength. This period usually is in excess of three months so that for practical purposes the sutures are left permanently.

Hypertrophic Scar and Keloids

At the end of the collagen phase the production of collagen slows and comes into balance with the lytic processes, thus introducing the maturation phase. The reasons why some scars continue to lay down inordinately excessive amounts of collagen with delay of the maturation phase also remain unexplained.

Age

Collagen production appears to be more aggressive in younger individuals and only appears to alter in the third decade.

Body Area

There is a greater tendency for keloid formation over areas such as the sternum, deltoid, neck and ear lobes and lower abdomen. Keloids do not occur on mucus membranes and are rare on areas such as the lower leg.

Race

Negroid and darker coloured European races are more prone to scar hypertrophy, but it is not uncommon in the fairer northern European races.

Chronic Infections

Repeated stress in a wound appears to delay the maturation phase especially over scars on flexor areas. Differences between hypertrophic scar and keloid can be demonstrated biochemically and histologically, but in clinical practice appear to form a spectrum of severity which ranges from hypertrophy with slight delay in maturation to frank keloid formation in a continuous process with no sign of maturation or softening. Hypertrophy thus occurs within the bounds of the original wound while keloid is more aggressive collagen production which

gives rise to a lesion which is larger than beyond the original wound. The worst form is similar to a progressive benign local tumour. The clinical appearances are well known, i.e.:

- pain and discomfort with or without itch
- raised firm tumour covered by thin epithelium with telangiectasis
- a hyperaemic appearance which becomes synergistic in the cold and pole on pressure.

There are four main treatment modalities and the best results seem to be obtained by combinations of these:

- surgery
- pressure garments
- cortisone injections
- radiation.

Surgery

Surgery can consist of complete excision into normal surrounding tissue or take the form of intralesional excisions thus reducing the bulk of the lesion without disturbing normal tissue.

Pressure Garments

This form of treatment is gaining more and more acceptance because of its non-invasive nature, and increasing degree of success seemingly, if used correctly and for an adequate length of time. Lesions most suitable are those overlying bone and in the early state of the phase in which hypertrophy becomes apparent. Once the scar has softened and matured and is no longer hyperaemic in appearance, pressure is no longer indicated.

It has been shown that the O_2 tension in keloid is reduced despite the hyperaemic appearance. With pressure this PO_2 rises. It is postulated that pressure on dilated blood channels within the tumour increases the speed of blood flow and thus a rise in PO_2. Once the PO_2 is above 80% the pressure is said to be ideal and if, after removal of the garment, the PO_2 is maintained at 80% or above, then the pressure therapy can be stopped.

Cortisone

Cortisone is often used in the form of Triamcinolone 40 mg/cc or less (maximum dose 120 mg in adults) diluted with a local anaesthetic agent such as 1% Lignocaine. The solution is injected into the scar with a fine needle under pressure being careful to avoid infiltration of surrounding normal dermis and subcutaneous fat. When a blanching effect is seen, the injection is deemed adequate. The solution can also be introduced using a springloaded jet apparatus percutaneously.

Radiation

The use of superficial X-ray treatment for keloids was popular in the past, but fell into disrepute because of the development of malignant changes in later years. This modality is used post-excision.

Malignant Changes in Scars

The first description of malignant ulceration in burn-scar tissue is attributed to Marjolin early in the nineteenth century. This malignant change is attributed to repeated breakdown with repeated stimulation of mitotic activity in epithelial layers. It occurs many years after the initial injury and is most commonly seen in burn scars. However, this change can follow any chronic ulceration of the skin, for instance after chronic osteomyelitis infection. Various series have shown that this form of squamous cell carcinoma is particularly virulent with a high incidence of rapid mortality once the lesion has metastasized. The development of metastasis is said to be delayed by the surrounding scar tissue with relatively poor lymph drainage.

Healing of Special Tissues

Peripheral Nerves

It is well known that when the axons of a peripheral nerve are interrupted, the distal part of the axon undergoes degeneration called wallerian. The proximal axon then regenerates along the part of the distal neural sheath until the end organ is reached. The speed of this regeneration is said to be approximately 1 mm per day. When the neural sheaths are divided, the axons grow out of the end of the proximal sheath and seek a neural sheath within which to continue growth. In mixed nerves this leads to wastage of axons in that sensory axons can grow down motor sheaths and vice versa. More and more evidence is emerging to indicate that there is some form of chemotaxis which will allow axons to select the correct sheath, either sensory or motor. Because of axonal wastage and the problems of exact mechanical repair, results are never 100% normal. The best time for nerve repair is immediately, in order to give the best chance for anatomical realignement. The sooner the axon can reach its end organ, the less the chance of irreversible regeneration of such end organs, particularly with reference to motor endplates.

Tendon

Because tendon has a relatively poor blood supply its healing is to some extent dependent on ingrowth of granulation tissue from the paratenon. This granulation tissue unfortunately remains after the healing process as adhesions within the tendon sheath thus seriously disrupting the gliding action and function of the tendon. Tendons will heal as a result of blood supply along the axis of the tendon though this is generally poor. Attempts are therefore made to suture the tendon i such a way as not to strangulate this blood supply and to keep the tendon as mobile as possible after suturing in order to minimize the development of adhesions around the repair site.

Intestine and Mucous Membranes

The healing of these epithelial surfaces is similar to that described for skin and improved healing can be achieved by surgical technique which will promote primary healing mechanisms.

Bone

The healing of bone is a specialized subject because of the variables which affect the calcification for structural repair.

Comment

C Bloch

A primary sutured wound will heal by the deposition of collagen, a partial-thickness wound heals by epithelialization and an open full wound by contraction and collagen deposition.

Certainly, there is no known way at present that the normal process of healing can be **accelerated.** However, providing the correct environment for optimal healing will allow maximal efficiency of healing in normal subjects, the following will play a part in this:

Hydration

Moist wound allow more rapid healing by increasing re-epithelialization and the regeneration of the underlying connective tissue. The use of modern semi-occlusive dressings help to retain the moisture, thereby expediting epithelial closure in 24 hours, as opposed to 72 hours in wounds exposed to the air. Scabs do not form, the inflammatory cell infiltration is reduced, there is minimal desiccation and a lesser mitotic response.

Thus, blisters should be left intact, i.e., in burn wounds, as the epithelial cover will occur twice as fast under an intact blister.

Temperature

In humid climates wounds heal better, probably due to less wound desiccation.

Infection

Few wounds become infected mainly due to the accumulation of leucocytes just under the wound surface. It is only later (24 hours) that bacteria gain entry - hence the danger of razor shaving the night before an operation. By the same token, topical antibacterial agents will reduce or prevent infection if used shortly after wounding.

Nutrition

A low protein, poor calorie intake will reduce normal healing, as will low levels of vitamin C, copper and zinc.

Wound Contraction

All wounds undergo some degree of contraction which helps in reducing the size of the defect. The amount of contraction is highly variable. It is effected mainly by the myofibroblast which has both fibroblastic and smooth muscle in its constitution and has strong contractile properties.

Contraction works best in uninfected well-oxygenated and vascularized wounds which have not been subjected to radiation nor to systemic steroids or chemotherapeutic agents. Good nutrition is also helpful.

Radiation

When the skin is severely injured by radiotherapy, as evinced by hard woody fibrosis, telangiectasis and skin atrophy, the wound will heal poorly. Less severely irradiated skin (dosage less than 1600 rads) will show little evidence of radiotherapy but still heals poorly.

Smoking

This is a most potent cause of poor wound healing. Apart from its long-term effects on lungs, etc. flaps have a great propensity to necrosis - certainly nicotine is anathema to a microvascular flap mainly because of its vasospastic effects.

General Diseases

Diabetics are much more prone to wound infections.

Prostaglandins (the arachidonic acid cycle)

There are also many substances which can influence this cycle, including steroids and non-steroidal anti-inflammatory drugs (NSAIDs).

Immunimodulation

The part played by the T and B lymphocytes, complement, interleukin, oxygen radicals, fibronectin and EPA in stimulating or depressing the immune system will become one of the most important aspects of wound healing in the future, especially when associated with multiple and severe injuries which may culminate in multiple organ failure (MOF).

Chapter 2.4

The Perioperative Management of Patients with Diabetes Mellitus

C J C Nel and S P Grobler

Introduction

Diabetes mellitus is a syndrome caused by a heterogenous group of disorders characterized by *hyperglycaemia, an absolute or relative insulin deficiency* and the tendency to develop certain *long-term complications.*

The diabetic is uniquely susceptible to the adverse metabolic consequences of stress.

Pathophysiology

Metabolic Homeostasis

Normal individuals, whether fed or fasting, maintain their blood glucose concentration between 3.3 and 6.7 mmol/L. This remarkable precision depends on a complex interaction of metabolites and hormones balancing anabolism and catabolism. In the postprandial state, insulin exerts an anabolic, storage-producing effect, preventing hyperglycaemia. In the fasting state the catabolic hormones reverse the situation to provide a continuous supply of glucose and other fuels such as fatty acids and ketone bodies. It should be noted that basal insulin secretion continues in the fasted state and is of key importance in restraining catabolism, i.e., damping down lipolysis, proteolysis, glycogenolysis and gluconeogenesis. Total insulin deficiency results in loss of these restraints with uncontrolled hypercatabolism leading to diabetic ketoacidosis.

Metabolic Stress

The catabolic hormones are secreted in response to any stress. These hormones include adrenaline, noradrenaline, cortisol, growth hormone and glucagon. Their effects may be further amplified by the concurrent administration of exogenous pressors and glucocorticoids.

Normally these changes in metabolism are balanced by a subsequent increase in insulin release that leads to increased peripheral glucose utilization and inhibition of alternative fuel mobilization.

Hyperglycaemia, hyperosmolarity, ketoacidosis and shock may develop in a previously well-controlled patient with insulin-dependent diabetes mellitus. The net protein catabolism that occurs in response to surgery is compounded by the effects of insulin deficiency. It is important to note that surgical patients are commonly starved so that blood glucose rises will not necessarily be massive, even in the diabetic person, but this may mask large increases in lipolysis, ketogenesis and proteolysis, as well as excessive fluid and electrolyte losses.

Classification of the Diabetic Syndromes

Diabetes is not one disease but it is rather a family of metabolic derangements that have hyperglycaemia in common.

Table 2.4.1 Classification of Diabetic Syndromes

1. Diabetes mellitus
   - a. Insulin-dependent diabetes mellitus (IDDM or Type I)
   - b. Noninsulin-dependent diabetes mellitus (NIDDM or Type II)
   - c. Secondary and other types of diabetes, including diabetes associated with
     - pancreatic disease
     - hormonal abnormalities
     - drug or chemical agent intake
     - certain genetic syndromes
     - insulin receptor abnormalities
     - other
2. Impaired glucose tolerance
3. Gestational diabetes
4. Statistical risk classes
   - a. Previous abnormality of glucose tolerance
   - b. Potential abnormality of glucose tolerance

Insulin-Dependent Diabetes Mellitus

In IDDM the insulin-producing beta cells in the pancreatic islets are destroyed and an absolute deficiency of insulin ensues. IDDM develops abruptly in individuals of normal weight, typically in childhood and adolescence. A patient with untreated IDDM can neither store nor utilize glucose and hyperglycaemia, hyperlipidaemia and ketosis develop. The situation is further complicated by secondary fluid and electrolyte shifts and typical diabetic ketoacidotic coma ensues.

Noninsulin-Dependent Diabetes Mellitus

Patients with NIDDM comprise the majority (80%) of the diabetic population. NIDDM is characterized by a relative lack of insulin. The beta cells are often hypertrophic and plasma insulin levels are normal or even elevated. The hyperglycaemia in NIDDM is caused primarily by insulin resistance. Even when untreated, there is usually sufficient insulin to minimize lipid mobilization and to prevent ketosis.

Intercurrent stress, as typically occurs in the surgical or critically ill patient, can lead to extreme hyperglycaemia, hyperosmolarity, coma and lactic acidosis. Obesity is present in 80% of patients with NIDDM. The disease develops insidiously, usually in individuals who are more than 40 years old. It may go undetected for many years, only to be discovered incidentally or during the stress of surgery or another illness. They may or may not require insulin to control hyperglycaemia and its attendant complications, but they are not prone to ketoacidosis. Although many patients with NIDDM are well controlled with an appropriate

diet and oral hypoglycaemic agents, and most obese diabetics respond to weight reduction alone, the small group of lean patients tends to require insulin therapy.

Impaired Glucose Tolerance (IGT)

This designates patients with mildly elevated blood-glucose concentrations when fasting or a glucose tolerance test that is borderline abnormal. This group of patients progresses to overt diabetics at the rate of 1-5% per year. The rest may remain glucose intolerant or even revert to normal. Individuals with IGT will often become frankly diabetic in times of stress. They are at higher risk of atherosclerotic (macrovascular) cardiovascular disease but tend to be free of microangiopathic retinal and renal disease.

Diagnosis

The diabetes may be suspected on the basis of the history, signs and symptoms of the disorder or of its complications, or the result of urine examination. The diagnosis is confirmed by the presence of hyperglycaemia and, if necessary, a glucose tolerance test.

Recommended criteria for the diagnosis of DM and IGM are not identical and remain somewhat arbitrary. The values presented below are for venous plasma glucose in non-pregnant adults and are based on the recommendation of the National Diabetes Data Group (NDDG) and the WHO Expert Committee on Diabetes Mellitus.

Diabetes is diagnosed if:

- a subject has classical symptoms of diabetes (polyuria, polydypsia, ketonuria, rapid weight loss) and unequivocal hyperglycaemia i.e.,

fasting > 7.8 mmol/L (8 mmol/L)
random > 11.1 mmol/L (11 mmol/L)

- a subject without classical symptoms

fasting > 7.8 mmol/L on more than one occasion

The WHO recommendation suggest that a fasting plasma glucose (FPG) of < 6 mmol/L or a random value of < 8 mmol/L excludes the diagnosis of diabetes.

For subjects with equivocal values a simplified two-hour blood-sugar screen (2HBSS) or formal glucose tolerance test (OGTT) are required:

2HBSS   < 6 mmol/L normal
      > 12 mmol/L diabetic

OGTT

normal:	fasting	< 6.4 mmol/L
	2-hour	< 7.8 mmol/L
	0.5 1 1.5	< 11.1 mmol/L

diabetic: two-hour sample and one intermediate sample < 11.1 mmol/L

IGT: 3 criteria must be satisfied

FPG	< 7.8 mmol/L
intermediate	< 11.1 mmol/L
2-hour sample	7.8-11.1 mmol/L

Management

Goals

Although optimal control with near-normal blood glucose levels is highly desirable, levels of 8-14 mmol/L are probably safer for the surgical and critically ill diabetic patient. Values lower than 8 mmol/L pose the threat of hypoglycaemia and provide little margin for safety. Glucose concentrations in excess of 14 mmol/L have certain short-term consequences viz:

- disturbance of host defence mechanisms with increased susceptibility to infection
- fluid and electrolyte imbalances with intracellular dehydration
- impaired wound healing

Smooth control with avoidance of ketoacidosis, hyperosmolarity, electrolyte disturbances and undue protein catabolism are required.

Preoperative Evaluation

Diabetic patients appear to be older physiologically than chronologically. It has been suggested that one should add the duration of the diabetes to the chronological age of the patient to get an approximation of the patient's physiological age (Steinke).

It is always preferable to admit the diabetic patient three days or more preoperatively for complete evaluation and optimal stabilization. Besides the usual careful history and clinical examination, preoperative evaluation of the diabetic should be directed at certain specific problems:

- Determine the precise nature of the therapy being used to control the diabetes i.e., diet, oral hypoglycemic agents or insulin. Other medications such as Beta-blockers, diuretics, antihypertensives and digitalis may also influence the surgical management and outcome.

- Evaluate the degree of control of the diabetes, particularly the few days before admission:

- symptoms of poor control (polyuria, polydypsia, nausea, vomiting, fatigue)

- hyperglycaemia, glycosuria or ketonuria on home glucose monitoring programme

- hemoglobin A_1C (glycosylated hemoglobin) levels may provide an index of the average blood glucose concentration prevailing over the preceding 6-8 weeks

A history of poorly controlled diabetes, particularly IDDM, carries implication of poor nutrition, impaired resistance to infection, poor wound healing and vitamin deficiency (i.e., thiamine).

- Whether the patient is ketosis-prone or a "brittle diabetic" i.e., subject to frequently hypoglycaemic reactions to insulin and hyperglycaemia. Patients who have long-standing IDDM often develop extreme sensitivity to regular insulin, which may contribute to increased brittleness.

- Assess the patient for any of the chronic complications of diabetes viz

- cardiovascular system - ischemic heart disease, cardiac autonomic neuropathy, peripheral vascular disease and hypercoagulability

- nephropathy

- neuropathy, both peripheral and autonomic

- infections, particularly occult infections i.e., plantar ulceration, gingivitis, sinusitis, urinary tract infection

- electrolyte imbalance particularly potassium depletion, magnesium and phosphorus deficiency

- malnutrition

- Evaluate the expected degree of stress, whether the procedure is major or minor, elective or emergency and the status of gastro-intestinal tract function in the postoperative period.

Essential laboratory evaluation includes an ECG, determination of blood glucose, urea, creatinine, electrolytes, calcium, phosphorus, lipids, liver function tests, urinalysis, chest X-ray and haemogram. Routine measurement of urinary glucose levels coupled with blood glucose levels may provide an indication of the renal threshold for glucose. Unless the urinary measurements is from a catheterized or double-voided specimen, it may reflect the blood glucose level hours before. Urine specimens may be difficult to obtain at exact times. The acute changes that occur in the perioperative period make this method misleading. Blood or plasma glucose measurements are thus preferable.

Perioperative Management

Anaesthesia

The operation should preferably be scheduled for early in the morning. Regional anaesthetic techniques are not contraindicated by the presence of diabetic neuropathy. Careful monitoring of blood pressure levels is essential: oxygenation and pulmonary ventilation should be optimal and hypoglycaemia must be prevented. Diabetic patients should receive low-dose subcutaneous heparin in an attempt to prevent thromboembolism.

Elective Surgery: Insulin-Dependent Patients

Patients should be admitted to hospital three days or longer before the intended operation. Long-acting insulins should be stopped and patients stabilized on twice-daily mixtures of short- and intermediate-acting insulins or three or four times a day short-acting insulin. Insulin requirements in the hospitalized patient generally change because the patient is less active, complies more closely to the prescribed diet and better glycaemic control is desired than is generally achieved on an ambulatory basis.

Blood glucose should be monitored frequently - at least four times per day (fasting, 11 - 15 - 21 hrs).

Several protocols are available for the administration of insulin.

Split Normal Dose Method

The most commonly used method is to give subcutaneous insulin on the morning of the operation. The dose is generally a fraction of the usual morning dose (half to three-quarters) of intermediate- and short-acting insulin. Breakfast is replaced by an infusion of 5% dextrose in water at a rate of 200 ml per hour. The remainder of the usual dose of insulin should be given immediately postoperatively. Postoperative control is further met with short-acting insulin subcutaneously every 4-6 hours according to blood-glucose levels. An example of a blood-glucose sliding scale system is presented below:

< 8	mmol/L	O U
8 - 10	mmol/L	5 U
10 - 12	mmol/L	10 U
12 - 15	mmol/L	15 U
> 15	mmol/L	20 U
ketones		// 5 U

Intermediate acting insulin is resumed once the patient is stable and eating.

Constant Intravenous Infusion of Insulin and Glucose

These methods involve the continuous infusion of short-acting insulin coupled with dextrose, water and sometimes potassium. Dextrose in electrolyte solution (i.e., Maintelyte, Electrolyte No 2) are usually administered according to the basic fluid requirement of the

patient. There is some argument as to whether 5% or 10% dextrose should be used, but the latter is generally favoured because more calories and insulin are given for the same infusion rate and this will favour anabolism. Insulin requirements may vary and 0.3-0.4 U/g glucose have been suggested to maintain the blood glucose at 10 mmol/L. In certain predictable situations insulin requirements may be increased, i.e., liver disease, obesity, severe infection, steroid therapy and cardiopulmonary bypass.

An intravenous solution of 10% dextrose in water is started at 18.00 hrs on the evening before the operation. The aim is to maintain the blood-glucose concentration at 10 mmol/L. Before starting the insulin infusion the blood-glucose level must be determined. To prepare the infusion 20 U of short-acting insulin is added to a 200 ml container of normal saline. Insulin is administered via a microdip set (60 drops/ml) according to the blood-glucose concentration. If the blood-glucose level is > 25 mmol/L 3 U/h (30 drops/min); if 15-25 mmol/L 2 U/h (20 drops/min); if 10-15 mmol/L 1 U/h (10 drops/min); and if < 8 mmol/L no insulin is given. The preparation of insulin and saline must be renewed 12-hourly.

It is necessary to repeat the blood-glucose estimation at 22.00 hrs. The insulin infusion is adjusted to maintain the blood glucose level at 10 mmol/L. A repeat blood-glucose determination is required on the morning before the operation and insulin should be given according to the guidelines given above.

It is important not to stop the infusion of insulin and glucose during the operation; the insulin infusion rate is adjusted according to the blood-glucose level.

Using the intravenous insulin/glucose regimen outlined above, postoperative care is straightforward and flexible. Blood glucose is monitored 2-6 hourly and the insulin infusion regulated to maintain the blood-glucose level at 10 mmol/L. Once the patient is stable and the insulin requirements has been established, determination of blood glucose can be done 12-hourly. The infusion is continued until the patient starts eating normally. The appropriate dose of insulin, assessed according to the preoperative regimen or as determined by the insulin requirements while on the intravenous regimen, is given subcutaneously one hour before the infusion is stopped. If the postoperative course is complicated and oral feeding is delayed, amino acids can be added after 48 hours and full TPN begun at the fourth or fifth day. In this situation insulin requirements may rise to as much as 20-40 U/h, particularly as 40 or 50% dextrose solutions may be used to provide calories.

Elective Surgery: Noninsulin-Dependent Diabetic Patients

Most of them are above the age of 40 years and are particularly liable to cardiovascular complications postoperatively.

Preoperative Management

Minor Operations

Well-controlled diet-treated diabetic persons do not require any special therapy before and during minor surgery. If fasting blood glucose is less than 8 mmol/L they can be treated

as a nondiabetic patient. If the glucose is higher, and particularly if it is higher than 10-12 mmol/L, one should treat as for an insulin-dependent diabetic.

In patients on oral hypoglycaemic agents, the agent should be discontinued on the evening before the operation. Even for minor operations, it is advisable to change all long-acting sulfonylureas (i.e., chlorpropamide) to short-acting agents (i.e., tolbutamide, glibenclamide) because of the very prolonged hypoglycaemic action of long-acting agents. ALl biguanides must be stopped 2-3 days preoperatively. If the fasting blood glucose is less than 8 mmol/L, therapy is omitted on the day of the operation and the patient is treated as a non-diabetic. If the glucose is greater than this value, an insulin infusion is used as in insulin-dependent diabetes. Postoperatively the usual therapy is reinstated at the time of the first meal. Blood glucose should be monitored regularly and small doses of subcutaneous insulin added if necessary (i.e., values above 10 mmol/L).

Major Operations

The modern tendency is to convert from sulfonylureas to regular insulin preoperatively. Patients are admitted at least three days preoperatively and all sulfonylureas and biguanides are stopped. The biguanides carry a real risk of precipitating lactic acidosis, particularly if there is impaired hepatic or renal function. The patient is stabilized on 3-4 times daily short-acting insulin. Perioperatively the patient is managed according to the intravenous insulin/glucose regimen. The infusion is continued until the first meal, whereafter it is probably wise to treat with 3-4 times daily . . before changing back to oral hypoglycaemic agents.

Emergency Surgery

Acute surgical emergencies, i.e. sepsis, acute cholecystitis, perforated peptic ulcer and trauma in the diabetic patient are likely to cause metabolic decompensation with the rapid development of hyperglycaemia, hypokalaemia, dehydration, ketonemia, ketoacidosis and death. IDDM not uncommonly is first detected at the time of an acute illness and delay in the recognition of the diabetic state may lead to severe acidosis and death.

Appropriate blood and urine specimens are taken and medical therapy for hyperglycaemia and ketoacidosis instituted immediately. Fluid and electrolyte imbalances and acidosis must be corrected and insulin given by conventional subcutaneous techniques or the preferred continuous low-dose intravenous infusion. Four hours of therapy should suppress ketogenesis and partially correct hyperglycaemia. The blood glucose level should decline by 4-6 mmol/L/hour. In the presence of sepsis the insulin dose required to produce such a fall averages 10-30 U/hour. Operation should be undertaken after a period of three to five hours for fluid replacement and insulin therapy. One cannot afford to wait longer to fully correct the ketoacidosis, as the underlying surgical problem that caused it will continue to progress and vicious circle will develop. During operation insulin and fluids are continued, the amounts being determined by period checks of the blood glucose, electrolytes and arterial blood gases.

Lactic acidosis and aketotic hyperosmolar states may also occur and are treated accordingly.

The Pregnant Patient

The need for insulin is raised in pregnancy. It is estimated that diabetes occurs in 1-2% of all pregnancies. Most of these patients have gestational diabetes - only 10% of pregnant patients are diabetic before pregnancy. All patients should be controlled by insulin administration. Blood-glucose levels should be kept at 3-7 mmol/L until the birth of the child. The intravenous insulin/glucose regimen can be ... during the labour and surgery. Insulin requirements ... the baby is born and care must be taken regarding the dose and duration of the action of the insulin that was controlling the disease, as there is a great danger of hypoglycaemia occurring after delivery. The neonate being exposed to too high a concentration of insulin in utero should also be watched for the development of hypoglycaemia and may require intravenous glucose.

Conclusion

The key to successful care of the diabetic surgical patient is careful monitoring. These patients all have a defect in normal metabolic regulation, and in the face of the additional metabolic stress of surgery or critical illness, only the physician can compensate for the diabetes. Constant monitoring and the application of the guidelines presented should enable the physician to minimize the increased risk that is faced by all diabetics who experience the stress of severe illness or surgery.

Comment

G Decker

- In the pre-operative evaluation of diabetic patients the clinician must recognize that myocardial ischaemia and infarction may be silent because of functional denervation of sympathetic afferent fibres associated with autonomic neuropathy. Up to 60% of adult diabetic patients without symptoms of coronary artery disease have been shown to have both abnormal exercise electrocardiography and myocardial perfusion scintigraphy. Orthostatic hypotension is a reliable indicator of cardiac autonomic neuropathy.

- Poor gastric emptying is another manifestation of autonomic neuropathy. This necessitates a longer period of pre-operative fasting or insertion of a nasogastric tube prior to induction of anaesthesia.

- The acute abdomen may be disguised by the lack of pain, probably due to diabetic neuropathy. On the other hand an important differential point between an acute surgical abdomen and diabetic ketoacidosis is that in the latter anorexia, nausea and vomiting always precede the abdominal pain.

- Beta-adrenergic antagonists are used commonly in the management of hypertensive patients. Adrenalin secretion in hypoglycaemia may be deficient in 40% of patients with IDDM, as a result of the adrenergic neuropathy. Beta-adrenergic antagonists can further impair glucose recovery from hypoglycaemia by blocking the catecholamine stimulation of beta 2 receptors of the pancreas, thereby inhibiting glucagon release. This effect has also been seen with the cardioselective beta blocker, Metoprolol. Beta-adrenergic antagonists are

contraindicated in diabetes with peripheral vascular disease because of the blockade of the beta 2 mediated vasodilatory response.

Perioperative Control of Blood Sugar

Certain principles must be recognized. "Tight" control of the blood sugar in the perioperative period may cause episodes of dangerous hypoglycaemia. Tight control of the diabetes pre- and postoperatively does not reduce morbidity in diabetic patients. This must not be confused with the advantages of "tight" control of the chronic diabetic state. Inhibition of lipolysis and gluconeogenesis is very sensitive to insulin.

When insulin is given subcutaneously, the levels build up very slowly in the circulation and flexibility is lost because of the long duration of action. Insulin intramuscularly has a half-life of 2 hours. The initial level rises slowly but small hourly injections thereafter can maintain steady insulin levels in the circulation without building up significant intramuscular depots. Intravenous insulin has a very short half-life of 4 minutes and has therefore to be given by constant intravenous infusion.

Continuous low-dose intravenous insulin regimens to control the diabetic patient perioperatively has risen from the realization that even in diabetic ketoacidosis the large doses of insulin given previously were unnecessary. True insulin resistance is rare. Intravenous insulin has the advantage over intramuscular regimens that insulin action begins at once and a steady concentration can be maintained or terminated almost immediately because of its short-life.

If insulin is added to the vacolitre of standard intravenous solution it is absorbed in part by the plastic tubing. The insulin binding sites on the plastic can be saturated by running through about 60 mL of fluid before starting the infusion. A concentrated solution of insulin may also be administered with an infusion pump and a plastic syringe. There is a 90% recovery of insulin with this system.

Walts et al. have demonstrated that if the split normal dose method is used, there was no better control of the perioperative blood sugar than patients receiving no insulin or substrate.

It must be emphasized that if a constant intravenous infusion of insulin and glucose is given, hourly intra- and postoperative glucose monitoring is required.

I would be reluctant to use electrolyte no 2 solution for volume replacement in the perioperative period because it contains 25 mmol lactate. The lactate is a gluconeogenic substrate which will aggravate the stress-induced hyperglycaemia. Saline rather than lactated Ringer's solution should be used for third-space replacement, for the same reason.

Maintenance dextrose and potassium is given at the rate of 0.1 g dextrose/kg/hour. Maintelyte is a 10% solution of dextrose containing 35 mmol potassium, 35 mmol sodium and 65 mmol chloride. A 10% solution of dextrose in water to which 40 mmol potassium chloride has been added, can also be used. The dextrose solution controls metabolism and maintains blood sugar in the 5.5 to 11 mmol range.

Comment

P Bauling

AODM and JODM

Terminology often used are the terms adult onset diabetes mellitus (AODM) and juvenile onset diabetes mellitus (JODM). JODM is an auto-immune disease, T-cell mediated against the beta cells of the pancreatic islets, ultimately leading to pancreatic islet destruction. However, this process is not an immediate and sudden event. The insulitis induced by the auto-immune activity (most frequently starting during the adolescent period) precedes the clinical symptomatology of the disease and only when enough islets have been destroyed does the patient become metabolically unstable (diabetic). However, at the time when the patient presents with the classical symptoms of diabetes, the islets are not all destroyed. In fact, several large studies have shown that, should immunosuppressive therapy be instituted at this time, the onset of diabetes can be delayed or aborted.

Blood-Glucose Control by Intravenous Method

Inherent dangers exist in administering a predetermined dose of insulin (say three units per hour) even with a dextrose infusion running. A wide range of dosages may be necessary to control a patient's blood glucose. The only way to decide on the appropriate hourly infusion rate of insulin is to do blood-glucose determinations as often as two-hourly or even hourly. Using urine-glucose determinations for the control of diabetes should be condemned.

Pre-Operative Assessment

Regarding the autonomic neuropathy, important complication is gastroparesis, either preoperatively or postoperatively, even in diabetics who are well controlled. This complication may lead to aspiration during induction of anaesthesia due to a large volume of fluid in the paralysed stomach despite a 12-hour fast. It may lead to prolonged postoperative gastric ileus.

Emergency Surgery

Only a three to five-hour period of intense resuscitation and insulin infusion may be necessary in the majority of patients before surgery is to be done, particularly for sepsis. The presence of the septic state is responsible for metabolic derangement.

Vascular Disease

There is a continued and ongoing debate with regard to the tight control of diabetes and the long-term risk of progression of vascular disease. There is tremendous support for this theory. However, and excellent article by Raskin reviews the controversies involved comprehensively.

Chapter 2.5: The Perioperative Management of the Patient on Steroid Therapy

C J C Nel, S P Grobler

Introduction

The ability of a patient to withstand stress depends on the physiological response of the adrenals by increasing glucocorticoid production. The magnitude and duration of the response is related to the severity of the stress, but it can be up to ten times basal secretion. This physiologic response is deficient in patients who have been on long-term steroid therapy, patients who undergo surgery for adrenal hypercorticolism, in primary adrenal insufficiency associated with auto-immune diseases, and following bilateral adrenalectomy.

Glucocorticoids are commonly used systemically for the treatment of a variety of diseases including asthma, ulcerative colitis, rheumatoid arthritis, collagen vascular disorders, nephrotic syndrome, chronic active hepatitis, thrombocytopenia and sarcoidosis. The relative potencies of commonly used steroids is listed in table 2.5.1.

Pathophysiology

Glucocorticoids and Stress

An intact hypothalamic-pituitary-adrenal (HPA) axis is essential for the normal physiological response (fig. 2.5.1). Normal adults secrete about 20 mg (12.5 mg/m^2) of cortisol and 0.1 mg of aldosterone per day. Glucocorticoid secretion is directly controlled by pituitary adrenocorticotropic hormone (ACTH) activity, which in turn is regulated by a corticotropic releasing factor (CRF) of hypothalamic origin. ACTH and CRF release are governed by three factors: glucocorticoid levels (negative feedback), the sleep-wake cycle and stress. Acute stimulation of the hypothalamic-pituitary-adrenal (HPA) axis occurs with trauma, pyrogens, hypoglycaemia, hyperthermia, burns, cold exposure, intense exercise, hypotension, hypovolaemia and acute anxiety.

The metabolic consequences of the adrenocortical response to surgical stress include hyperglycaemia, increased protein breakdown, sodium and water retention, and numerous permissive actions in intermediate metabolism. Corticosteroids are essential as well for maintenance of normal cardiocirculatory function through their effects on contractility of muscles.

Impaired adrenal function is seldom significant if steroids are stopped within about 10 days, unless very high doses are employed. After prolonged glucocorticoid administration there is profound suppression of the HPA axis and atrophy of the adrenals. After withdrawal of therapy there is a slow recovery of the axis: the hypothalamus and pituitary recover their ability to respond to stress (such as hypoglycaemia) first, and the adrenal last. Basal plasma cortisol levels may return to normal relatively rapidly, but the cortisol response to hypoglycaemia may take up to 12 months to return to normal; earlier recovery of the stress response - within two months - seems, however, to be the rule. Patients with contralateral adrenal suppression by an adrenocortical tumour may require periods of up to two years to recover fully from adrenal insufficiency. For practical purposes adrenocortical insufficiency

should be considered in all patients who are receiving corticosteroids or who have received them for more than one to two weeks within six to twelve months prior to the operation.

Clinical Presentations

Hypercortisol State

Chronic administration of glucocorticoids share many of the complications found in Cushing's syndrome such as obesity, psychiatric disorders, hyperglycaemia, oedema, poor wound healing and immunosuppression. Hypercorticolism secondary to exogenous steroids has several unique side effects including aseptic necrosis of bone, benign intracranial hypertension, glaucoma, posterior subcapsular cataracts and pancreatitis. In contrast, striae, purpura, plethora, hypertension, acnae, hirsutism, menstrual irregularities and impotence are more commonly associated with endogenous hypercorticolism. When present, these features may aid in distinguishing the aetiology of the hypercortisol state.

Withdrawal of Steroid Therapy

Withdrawal of glucocorticoids can be associated with exacerbation of the underlying disease and with symptoms of adrenocortical insufficiency, including anorexia, fever, nausea, lethargy, myalgias, arthralgias, weakness and weight loss. These symptoms can occur despite a gradual decrease in dosage.

Adrenal suppression may also occur in patients who manifest no obvious signs of hypercorticolism and lack symptoms of steroid excess or steroid withdrawal.

Biochemical Evaluation

The integrity of the HPA axis can be evaluated by creation of a standardized stress situation. The need for perioperative corticosteroid supplementation can be evaluated especially in patients where steroid use may be detrimental due to its immunosuppressive and other side-effects. The insulin hypoglycaemia test (table 2.5.2) produces a standardized stress situation and the cortisol response reflects the ability of the adrenal to respond. A normal response to insulin hypoglycaemia indicates that the reintroduction of perioperative steroid therapy is not required. If the patient has been placed on steroid cover prior to the tests, dexamethasone should generally be substituted as it does not interfere with the laboratory method for plasma cortisol estimation.

Adrenal responsiveness to ACTH can also be evaluated by the Synacthen test. If this test indicates that the adrenals can respond acutely to exogenous ACTH, it is assumed that the rest of the HPA axis must also be intact as the adrenals are the last part of the axis to recover after suppression by steroids. In this test synthetic ACTH - a dose of 0.25 mg - fails to increase low blood cortisol levels to above 18 mg/dL if the adrenals are unresponsive.

Table 2.5.2. The insulin hypoglycaemia test

- Overnight fast
- Indwelling intravenous cannula
- Basal sample for glucose and cortisol
- IV injection of soluble insulin 0.1 unit/kg
- Further samples for glucose and cortisol at 20, 40, 60, 90 and 120 minutes
- Adequate hypoglycaemia should be 40 mg/dL or less (2.2 mmol/L). The patient should have mild symptoms, i.e. sweating
- Increase in plasma cortisol of more than 5 microg/100 mL to greater than 20 microg/100 mL is the "normal" response
- Synthetic steroids, i.e. prednisolone, are usually not detected in plasma cortisol estimations

NB. A doctor should be in constant attendance to administer 50% glucose if necessary.

Management

Minimizing Steroid Suppression

Ideally, steroids should be given in a manner that eliminates side effects but maintains anti-inflammatory and immunosuppressive activity. The most feasible and effective schedule is alternate-day therapy using intermediate-acting steroids such as prednisone and prednisolone given at 7 to 8 am on alternate days. It is based on observations that the anti-inflammatory effects of glucocorticoids persist longer than the unfavourable metabolic side effects. Longer-acting glucocorticoids such as dexamethasone given on alternate days tend to produce HPA axis suppression.

Relative to the disease being treated, initially a high dose daily or more frequent steroid schedule may be necessary to accomplish satisfactory disease control. Once control is gained, an attempt should be made to titrate the dosage gradually over several weeks to months to an alternate-day schedule.

Titration schedules must be individualized with close observation of the patient's clinical status, avoiding disease recurrence or adrenal insufficiency and permitting optimal recovery of the HPA axis. Periodic evaluation of the HPA axis's responsiveness should be undertaken to determine the need for continued steroid replacement.

Anticipating Steroid Suppression

Patients on chronic glucocorticoid therapy at either pharmacological or replacement doses or who have received daily steroid replacement for a period exceeding one week during the past year are at risk of pituitary-adrenal suppression. Steroids administered by other than the oral route may also exert systemic effects and result in adrenal suppression, i.e. inhaled beclomethasone, topical dermatological steroids or prednisolone retention enemas. Subjects with proven primary or secondary adrenal insufficiency and patients who are to undergo adrenal surgery also need to be covered with supraphysiological doses of steroids. It may be beneficial to assess the adrenal status in other high-risk surgical patients in whom occult

adrenal insufficiency may be a problem, i.e. patients with severe or prolonged infections or those with organ-specific auto-immune diseases, such as pernicious anaemia, thyroiditis, or primary hypopituitarism.

All patients at risk of adrenocortical insufficiency should receive additional glucocorticoid cover during periods of stress. The steroid supplementation regimens attempt to imitate the normal cortisol response to surgery and stress. Under severe stress the maximum cortisol production rate in normal individuals is 300 to 400 mg per day. Most schedules include a planned excess of glucocorticoids given my more than one route to ensure continuous availability during the period of maximal stress (table 2.5.3). These short-term excesses of glucocorticoids are relatively harmless.

Table 2.5.3. A typical steroid therapy schedule for patients undergoing major surgery uses appropriate volume replacement with

- Two hours preoperatively: hydrocortisone 100 mg IM

- At induction of anaesthetic: hydrocortisone 100 mg IM plus 100 mg IV

- Day 1 to 2 of surgery: hydrocortisone 100 mg 6-8 hourly IV

- Gradual tapering postoperatively by 50%/day until maintenance doses of daily steroids are achieved.

- When the patient can tolerate and retain oral feeds without difficulty, oral corticosteroid medication can be given.

- Amounts of cortisone or hydrocortisone greater than 100 mg daily exert adequate mineralocorticoid effects. When the daily dose is reduced below 100 mg or if methylprednisolone or dexamethasone is the only drug available for parenteral therapy, fludrocortisone or deoxycorticosterone acetate should be added to the regimen.

- This schedule is a general guide, to be modified to each patient's needs, as judged by clinical status (particularly the presence or absence of fever, pain or other stress), blood pressure and serum electrolytes.

For patients undergoing minor surgery, less initial steroid replacement could be given, but the guidelines in the above schedule allow the physician confidently to assume that adequate steroid levels are present during stress. Short procedures like endoscopy, lumbar puncture and dental work can be covered with hydrocortisone 100 mg IM or IV prior to the procedure plus a doubling of the evening oral maintenance dose.

Patients with chronic adrenal hypofunction who are subjected to protracted minor stresses should receive three to four times their usual daily oral dose of steroid during the stress. They should liberalize their salt intake and adjust their mineralocorticoid dosage upward if necessary, particularly if primary adrenal insufficiency is present.

The steroid requirements of normal women roughly double during the second half of pregnancy, so patients with adrenal insufficiency should receive two or three times their usual daily dose. As gestational steroids possess mineralocorticoid activity, the dose of fludrocortisone may need to be lowered. During labour or caesarean section, steroid therapy should be instituted in doses appropriate for major surgery to be safe. After an uncomplicated delivery, however, cortisol requirements drop dramatically and rapid tapering of maintenance doses can be achieved.

Conclusion

Steroid therapy necessitates careful and prudent clinical evaluation and use of ancillary laboratory testing to ensure adequate disease control, to avoid iatrogenic hyper- and hypocortisol states and their attendant complications and to ensure safe cover during periods of stress and increased adrenocortical requirement.

Comment

Pre-Operative Management of a Patient On Steroid Therapy

P Bauling

- The first case of suspected steroid-induced hypoadrenalism leading to death was described by Frazer et al in 1952. This was followed by the first confirmed report of adrenocortical depression due to steroid therapy in 1962. This patient had a serious fall in blood pressure during surgery. Later the pituitary-adrenal axis was shown to be suppressed in the patient.

- In 1969 an excellent overview of this problem by Plumpton et al was based on a study of 40 patients on steroid therapy and 20 patients previously treated with steroids. This report has formed the basis of our approach to supplemental steroid therapy in patients undergoing surgery since that time. What is quite remarkable in this very old study is the fact that despite reasonably prolonged steroid therapy, the majority of patients showed a near normal to normal response to surgical stress when cortisol levels were determined during surgery. This relatively old paper is extremely well prepared and deserves attention. Furthermore, in this early paper on preoperative steroid supplementation there was already a hint that supraphysiological doses were

- unnecessary
- possibly harmful

leading to immunosuppression, infection and delayed wound healing.

- In 1981 Symreng et al approached this problem on a more scientific basis by using corticotropin response measurements to address the following problems:
- Can responders be separated from non-responders?
- What is the cortisol response pattern of responder patients to major surgery?
- Is a very low dose of steroid administration sufficient in non-responders?

The answers to these questions were provided experimentally in this paper:

- responders could be identified by using corticotropin stimulation
- the majority of patients on long-term steroid supplementation retain their pituitary adrenal axis
- responders respond with a cortisol response curve almost identical to normal controls
- very low dose steroid supplementation (total of 75 mg hydrocortisone per 24 hour period) produced peak values even higher than normal individuals undergoing major surgery. This is supported in studies by Kehlet et al 1981 showing that surgery-induced stress in man produced maximum cortisol levels ranging between 75 and 160 mg in the first 24 hours. Both these authors hint that the majority of patients on steroid therapy may in fact not have required steroid supplementation during surgery, provided that the clinician involved remained alert to the symptoms and signs of cortisol insufficiency. Recent data in animals, however, suggest that undersupply of cortisol during stress may in fact be just as disadvantageous. This has not been confirmed in man. In summary, therefore, the administration of small (physiological) doses of corticosteroids in the perioperative management seems to be the most prudent approach.
- With the progressive expansion of organ transplantation, the organ recipient may soon be the most common patient requiring this management. Exactly the same principles apply in these patients with regard to steroid supplementation. In addition, however, strong emphasis should be placed and attention paid to asepsis in the organ recipient undergoing any form of surgical intervention - as should be the case in all patients on steroid therapy.

Comment

Pre-Operative Management of a Patient On Steroid Therapy

G Decker

Anaesthetists now seem much less fussed about patients who have received steroids in the past. Kehlet collected from the world literature 44 possible but only three confirmed cases of adrenal failure in these patients. Nevertheless, in a patient with systemic disease who has received treatment with glucocorticoids, it may take 9 months for recovery of the hypothalamic-pituitary-adrenal cortical function. Until complete recovery has occurred it is advisable to assume that the patient will need basal steroid therapy as well as supplementary therapy when he has a serious illness or an operation.

Inducing hypoglycaemia with insulin to test pituitary adrenal competence is impractical and probably a more dangerous practice than simply administering glucocorticoids.

The potential risk of peri-operative cortisol are said to be aggravation of hypertension, fluid retention, inducement of stress ulcers, abnormal wound healing, increased prevalence of infection and precipitating a psychiatric disturbance.

The risks of these complications developing are probably minimal in patients who receive steroid cover during the peri-operative period only, particularly if modest doses of steroids are given. Symreng et al recommended hydrocortisone 25 mg intravenously with induction of anaesthesia and a continuous infusion of a 100 mg hydrocortisone for the

following 24 hours. They found that this low dose of hydrocortisone in patients with proven adrenal insufficiency resulted in plasma cortisol concentrations considerably greater in the first four hours after induction of anaesthesia than that of healthy controls subjected to the same degree of operative stress.