Orthopedic surgery is the branch of surgery concerned with preserving and/or restoring the skeletal system—its functions, articulations and structures. For example, reconstructive procedures are performed on pediatric patients for congenital problems and on the aged for correction of degenerative processes. Factors that need to be considered include the preoperative condition of the patient, intraoperative hemorrhage, fluid and electrolyte management, controlled hypotension, fat emboli, acrylic cement, and the proper use of tourniquets. Although all the above factors may not be pertinent in every case, each procedure has unique anesthetic complications.

Preoperative assessment

Prior to the induction of anesthesia, a thorough preoperative assessment is essential. For example, if the patient is elderly, he may have compromised respiratory and cardiovascular systems. If the patient has chronic obstructive pulmonary disease (COPD), pulmonary function tests will be warranted. Some patients may tolerate a general anesthetic poorly, so consideration should be given to an epidural or spinal anesthetic when applicable. Some patients may be on a broad spectrum of pharmacological agents, thus, consideration should be given to the problems associated with these drugs. Patients on steroids for a prolonged period of time may require a steroid cover. Finally, a patient on long-term salicylate therapy may have pre- and post-operative coagulation complications.

Hemorrhage is a complication often associated with orthopedic surgery. In cases where tourniquets cannot be used, an adequate amount of cross-matched blood should be readily available. Allowance should be made for postoperative bleeding, which can be substantial and which may even exceed the intraoperative blood loss. When a large quantity of blood is transfused, the blood should be warmed and delivered through a microfilter.

Three measures can be taken to offset the hazard of hemorrhage. The first is to ensure an adequate fluid volume preoperatively; the second is to reduce blood loss intraoperatively by employing a hypotensive technique; and the third is to reduce blood loss intraoperatively by the use of pneumatic tourniquets. It must be noted that the last two methods have a restricted application.

In order to reduce the hypotension associated with hemorrhage, the patient should arrive at the opening room in a well-hydrated state. However, this is rarely the case for several reasons. In most cases, the patient has had nothing to drink since the evening prior to surgery. If the patient is elderly and has a compromised cardiovascular sys-
tern, he may be taking diuretics which will increase obligatory water losses. Patients who experience pain with movement may intentionally limit their fluid intake in order to prevent additional trips to void at night.

**Fluid management**

Normal fluid requirements for a 70 kg adult generally fall between 2.4% of body weight per day;¹ 2500-3000 ml is considered a normal daily fluid intake. If a patient has been given nothing orally (NPO) for eight hours, he may have a 1000-2000 cc negative H₂O balance prior to incision. Since H₂O losses from the respiratory mucosa, sweat, stool and urine are mainly obligatory, the replacement solutions should be hypotonic with respect to sodium concentration. Jenkins and Giesecke suggest that an adult scheduled for elective surgery, who has no fluid or electrolyte imbalance, should initially receive 500 ml of D₂W.²

The rationale for administering this solution is that when a patient has been NPO, a negative H₂O balance results, which in turn causes a slight increase in serum osmolality. This increase triggers the osmoreceptors located in the hypothalamus. These receptors send impulses to the posterior pituitary gland, causing an increased release of antidiuretic hormone (ADH). The ADH causes the distal convoluted tubules and the collecting ducts in the kidney to become more permeable to H₂O. Water moves into the hypertonic interstitium and back into the circulation, while the urine becomes more concentrated. The infusion of 500 cc of D₂W should depress further ADH secretion and avert oliguria or anuria unless there are other subsequent changes to the patient's fluid balance mechanisms.

It should be stressed that fluid replacement is dependent on the current status of the patient and the length or type of procedure. Jenkins and Giesecke suggest that an adult patient without pre-existing fluid or electrolyte imbalance undergoing a total hip arthroplasty should receive an initial 500 cc D₂W, then a solution of D₅LR at the rate of 12-15 ml/kg/hr body weight during the first hour.² If the operative procedure continues into the second hour and if there is no undue loss of blood or unusual extent of tissue manipulation or trauma, the fluid administration is slowed to 6-10 ml/kg/hr. If the operation continues past three hours, lactated Ringer's solution without dextrose is substituted. Hourly urinary output should be monitored in these longer operations and fluids should be administered at a rate to assure an output of about 50-100 ml/hr or 1 mg/kg.

Urinary output is perhaps the most valuable guide to continued fluid therapy. Central venous pressure (CVP) gives one information about the manner in which the heart manages the fluid load which is being infused. It is not a direct measure of the adequacy of volume replacement. A patient in congestive heart failure can have an elevated CVP even though the extracellular fluid volume is deficient.² In a healthy patient, serious overloads of isotonic salt solutions can be given without significant elevation of the CVP. The overload is more likely to be manifested by interstitial edema and diuresis.

Volume replacement, not the use of vasopressors, is mandatory on the successful management of hypovolemic shock. Balanced salt solutions should not be used as a substitute for whole blood.⁴

Blood loss in excess of 20% of the estimated blood volume must be replaced with whole blood. Estimated blood volume ranges from 60 ml/kg for adult males, 55 ml/kg adult females, and 80 ml/kg for infants.³ Balanced salt solutions are intended to replace functional losses of extracellular fluid which occur following trauma and shock.⁵ As blood is lost, fluid shifts from the interstitial space into the blood vessels. This shift results in a deficit of functional extracellular fluid, which should be equivalent to the amount of blood loss.

In shock states, fluid also shifts from the interstitial space into the cells, resulting in a further diminution of the functional extracellular fluid volume.⁶ The shift into the blood vessels and cells eventually diminishes the interstitial pool sufficiently so that it can no longer effectively stabilize the vascular system and the patient becomes hypertensive. Proper therapy involves administration of simultaneous whole blood and balanced salt solutions. To reiterate, administration of blood alone or colloids alone fails to correct the deficit of functional extracellular fluid.

**Blood transfusions**

Although blood replacement is essential for hypovolemic shock, it carries innate dangers. Blood transfusions carry a hazard to life estimated at one death per 1,000-3,000 transfusions; non-fatal reactions occur in 3-6% of the population.⁷ Some of the potential dangers include transfusion reactions, post-transfusion hepatitis, bacterial contamination and coagulopathies secondary to reduced platelets and clotting factors. Such complications should not be acceptable in an elective surgical procedure.

There are four ways to circumvent the above mentioned complications. The first is mechanical
Intraoperative salvage, such as a cell saver. Blood loss is collected, filtered, and later reinfused into the patient. The apparatus needed for this is expensive; blood cannot be used if it comes from an infected site and it is not reinfused if the patient has cancer. The other methods are acute preoperative hemodilution, the use of deliberate hypotension and autologous transfusions.

Frozen autologous blood has been shown to be effective replacement therapy even when stored for as long as seven years. Refrigerated blood can be stored up to a maximum of 21 days. A study of Echandt, Gossett, and Amstute showed the advantages of this technique in 49 patients scheduled for total hip arthroplasties. One major advantage is that post-transfusion hepatitis can virtually be eliminated.

Patients who require revisional procedures may have received previous transfusions and are now difficult to cross-match because of the presence of circulating antibodies. Preoperative autologous banking assures available blood at low risk of transfusion complications. Blood can be drawn several weeks prior to the surgery and patients can still have acceptable hematocrits the day of surgery. Although a 5-15% red cell mass loss occurs during the freezing, thawing and washing process, it has been shown that the *in vitro* survival for frozen washed erythrocytes is equivalent to that of fresh erythrocytes. Finally, the cost of autologous blood transfusion has not proven to be excessive. Such transfusions can essentially eliminate the need for homologous transfusions and the costly complications they induce.

**Hypotensive anesthesia.** Another method used to reduce hemorrhage and the complications of blood transfusions is the use of deliberate hypotension. Davis and Mallory have shown a 50% decrease in blood loss in patients undergoing primary total hip replacement using hypotensive anesthesia. Besides reducing the need for blood replacement, this technique provides several other advantages. A decrease in bleeding will allow the surgeon to see better and thus perform a more definitive operation. It allows better wound healing and less wound infection, since there are less blood ligatures and cauterized tissue in the wound.

In Mallory's study, operative time was reduced by approximately 25% with hypotension due to improved hemostasis. There were no postoperative problems with wound hemorrhage when the patients were returned to normotensive values. Especially relevant is that subsequent blood replacement following surgery was again reduced by approximately 50%. Additionally, there were no problems postoperatively with coronary insufficiency, myocardial infarction, cerebral edema, or renal dysfunction.

An acceptable method of producing hypotension is the combination of halothane and sodium nitroprusside (Nipride®). While halothane used alone will decrease whole body O2 consumption, the cardiac index is depressed more than the oxygen consumption, resulting in an increased capillary oxygen extraction. In contrast, sodium nitroprusside has been reported to increase the cardiac index by about 20% during anesthesia. Another advantage is that there does not appear to be any significant change in whole body oxygen consumption. Finally, Bland and Lowenstein showed that halothane decreases the severity of experimentally induced myocardial ischemia in the non-failing canine heart. This suggests that nitroprusside-induced hypotension, supplemented by a myocardial depressant, may be a desirable approach.

Besides anesthetic drugs and adjuvants, body position and the surgical site will affect blood pressure and thus blood loss. For each 2.5 cm vertical height, blood pressure falls 2 mmHg. If a patient is in the lateral position, the surgical site is uppermost, blood pressure in the wound is hydrostatically lower, and blood is pooled in capacitance vessels in the dependent portions of the body. If surgery is performed on an extremity and the extremity is elevated, blood loss will be reduced, resulting in less stress and fewer complications to the patient.

**Pulmonary and fat embolism**

The development of pulmonary emboli is another complication that might be seen after orthopedic surgery. Deep vein thrombosis with subsequent pulmonary embolism is relatively common in orthopedic cases because of the high average age of patients, frequent obesity, prolonged immobilization and circulatory occlusion during surgery. Venous thromboembolism is the most common complication of hip surgery in adults and fatal pulmonary embolism occurs in approximately 2% of patients undergoing elective procedures of this type. Preventive measures include care in positioning for surgery, compression stockings, early ambulation, very early postoperative leg exercises, and maintenance of hydration.

The origin and frequency of fat embolism syndrome (FES), on the other hand, is controversial. Ross revealed that clinical cases of FES are relatively uncommon and usually occur unexpect-
edly. Sevitt, however, stated that 80-100% of patients dying after a fracture were found to have fat embolism syndrome.

There are two theories regarding the origin of fat emboli. The first is that fat is released from the marrow of the fractured bone into the venous and lymphatic channels and, thus, into the systemic venous circulation. The mechanism by which emboli are found in the arterial circulation and move to the brain is difficult to explain, since the lungs act as a natural filter between venous and arterial blood.

The second theory has been proposed by Lehman and Moore. They state that trauma alters the natural emulsion of fats in the blood stream leading to the formation of droplets about 5-10 microns in size that can act as emboli. They are coated with sticky adhering platelets and thus block smaller arterioles and capillaries. To support this theory, Hillman and LeQuire hypothesized that trauma causes large amounts of catecholamines to be released. The catecholamines mobilize free fatty acids (FFA) which are taken up to the liver, synthesized and released as low density lipoproteins. This results in fat emboli.

Regardless of the origin of the fat globules, the major insult occurs in the lungs. Peltier demonstrated the toxic effect of FFA on parenchymal cells in lung tissue with resultant edema and hemorrhage. Lung surfactant is altered and electron microscopy studies have shown a disruption of the structure of the alveolar capillary membrane. Heandon states that the mechanical block in the capillaries of the lung leads to a diffusion block and increased AV shunting. To complicate matters, the platelets release their amines, such as serotonin, and thus enhance the vasoconstriction, bronchoconstriction and passive congestion of the pulmonary vascular bed.

The monitoring of serum lipase or coagulation parameters does not appear to be a reliable early indicator of the occurrence of fat embolization. The most reliable parameters appear to be temperature, pulse and respiratory rate. As Allandys reports, “Respiratory rate exceeding 30 per minute will almost certainly be associated with severe pulmonary fat embolism and hypoxemia.”

Treatment includes mechanical ventilation and corticosteroids in the dosages used in the treatment of septic shock. Although the use of heparin is controversial, heparin 10-15 mg IV every 4-8 hours is also advocated. This may decrease platelet adhesiveness and prevent release of amines, which appear to play an etiological role in the development of fat emboli. Low molecular weight dextran (40,000 in doses of 1000 ml per 24 hours) decreases viscosity, thus improving blood flow.

It is the delay in the clinical symptomatology that makes treatment of fat emboli difficult. By the time symptoms of hypoxemia become evident, the pathologic changes of endothelial damage and interstitial edema have been progressing for 24-72 hours. Treatment is usually started after the damage is done. Prophylactic therapy has been advocated so that if the pathologic process should begin, so too has the treatment.

Stoltenberg and associates observed that femoral shaft fractures are associated with FES twice as often as tibial shaft fractures. Other fractures, unless combined with either femoral or tibial shaft fractures, rarely are associated with fat embolism syndrome.

In their study, patients with tibial and femoral shaft fractures were randomly placed in three groups. One group served as control; another received 50 ml of 50% dextrose IV every four hours for four days; and the third group received one gram methylprednisolone every eight hours for a total of three doses. None of the patients treated prophylactically with methylprednisolone developed FES. Of 64 patients, five developed FES and each of these had femoral shaft fractures. Thus, methylprednisolone given prophylactically may reduce the incidence of FES and deserves consideration when administering anesthesia for acute repair of femoral shaft fractures.

**Acrylic cement**

One of the factors involved in the success of joint replacement is the use of acrylic bone cement, to secure the prosthesis in place. The material is methylmethacrylate. It is supplied as an ampule of the monomer and an envelope of the powdered polymer. The setting process generates considerable heat about 6-7 minutes after mixing.

There are essentially three untoward reactions attributed to the use of acrylic bone cement. The first is fat embolism. The fatty marrow of older individuals makes them more susceptible to the development of fat embolism. In bone, the capillaries are relatively wide—25 microns as opposed to 6-10 microns in the systemic circulation. These capillaries empty into wide sinusoids that are supported by a delicate fibrous network attached to the intramedullary trabeculae. These sinusoids remain distended rather than collapsing as do the veins in the surrounding soft tissues. Early in life the marrow is filled with a considerable amount of hematopoietic tissue. In the older adult, this is gradually replaced with large fat cells.
composed primarily of olein, which tends to liquify at body temperature.\textsuperscript{37,38}

The pressure within the marrow cavity lies midway between the arterial and venous pressure.\textsuperscript{38} An increase is likely in pressure within the marrow cavity, which might be caused by packing the cement on the bone marrow to secure the prosthetic device. This increased pressure may rupture the delicate sinusoidal structures, allowing fat to enter the open venous collecting system and, thus, be transported to the lungs.

The expulsion of fat from the marrow cavity as a result of increased pressure has been directly and indirectly observed. Breed was able to artificially raise the intramedullary pressure using saline.\textsuperscript{39} This resulted in transfer of the fat and marrow elements into the venous system with lodging in the pulmonary vasculature.

The cement is not a glue because it has no adhesive qualities. It does not bond to the polished surfaces of the components, but it does bond to some degree to the slightly rough surface of non-polished stems. It bonds securely to cancellous bone if while in the creamy or doughy state it is forced into the interstices of the bone. A secure bond is extremely important because it prevents motion at the bone cement interface. Any motion here will cause absorption of the bone and eventual loosening of the cement. Recent laboratory evidence suggests that methylmethacrylate attaches to bone better in a dry surgical field.\textsuperscript{39} A dry surgical field is more likely to be seen when using a hypotensive technique.

According to Wilde and Greenwald, the strength of the cement is about half that of compact bone.\textsuperscript{40} It is three times stronger under compression than under tension.\textsuperscript{42} If the cement is not tightly packed between the bone and the components and gaps or spaces are left between the surfaces, the cement will break because it is subjected to shear and tension rather than compression.

A second untoward reaction attributed to the use of acrylic bone cement is hypotension. According to McMaster and associates, the hypotension that sometimes follows the introduction of polymethylmethacrylate into the femoral canal, but not into the acetabulum, may be the result of peripheral vasodilatation without myocardial depression.\textsuperscript{41} The hypotension seems to correlate not with the level of monomer or the circulation, but rather with the \textit{deficit} in blood volume at the given moment.\textsuperscript{42} This unexpected hypotension should be alleviated if the fluid management previously outlined is adhered to and urine output closely monitored.

If a hypotensive technique has been employed, circulating volume must be carefully maintained since these patients cannot tolerate large volume loss. Charnley stated that under a hypotensive technique with the blood pressure levels of 60 torr, no further fall occurred due to the cement.\textsuperscript{43} He reported that the greatest falls came when the pressure was relatively high (110-120 torr) and that the \textit{least} effect occurred when the starting pressure was low (60-80 torr).

Various mechanisms have been suggested to explain the cardiovascular effects of the cement. These would include a reaction to the heat generated when the cement sets, a toxic or vasodilator effect of free monomer absorbed into the circulation, cement anaphylaxis, autonomic effects secondary to the pressure rise within the femoral shaft, embolism from the medullary cavity of air, polymer particles, fat embolisms, platelet aggregation, and finally, thromboplastins.

To reiterate, it is believed that impaction of the stem of a prosthesis into the cement-filled cavity of a long bone has a piston effect which forces various embolic materials into the circulation. In order to reduce this pressure effect, it is usual to vent the medullary cavity by drilling the bone cortex or more commonly, by passing a catheter down the shaft prior to inserting the cement prosthesis. It has become common to defer insertion of the cement for at least two minutes after mixing so that it has a stiffer consistency, less heat production, and less free monomer present. After the mixing is completed, it should be handled as little as possible so that monomer from the interior of the bolus is not brought to the surface since it is the surface monomer that is absorbed. As stated previously, mixing should not go on right up to the moment of insertion; rather, time should be allowed for the monomer to evaporate from the surface of the bolus.

A third untoward reaction attributed to the use of acrylic bone cement is a transient fall in arterial oxygen tension.\textsuperscript{44,45} Park and associates found a sudden sharp transient decrease in \(
\textit{PaO}_2\) following application of acrylic bone cement during total hip replacement.\textsuperscript{46} The greatest decrease occurred without any change in blood pressure or pulse rate, making pulmonary emboli an unlikely cause. They postulated that the decrease in \(
\textit{PaO}_2\) seemed to be directly related to the amount of acrylic polymer absorbed into the systemic circulation through the bone. In patients who had the operation for the second time, changes in \(
\textit{PaO}_2\)
were greater with application of the bone cement into the acetabulum than changes observed with insertion of the cement into the femur. It was felt that the acetabulum was more vascular, since it was easier to remove more old bone cement from it than from the femur. There is no real evidence for the conclusion that the more monomer absorbed, the lower the \( \text{PaO}_2 \). Finally, Park suggests that until further investigations determine the exact cause of the \( \text{PaO}_2 \) changes, patients undergoing total hip arthroplasty should be given enough oxygen to maintain \( \text{PaO}_2 \) at twice the normal values for a few minutes before and after the application of acrylic bone cement to the bone.

**Tourniquets**

Operations on the extremities are made easier with the use of a tourniquet. It provides the surgeon with a clear surgical field and intraoperative blood loss is significantly reduced. There are numerous complications that may occur if the instrument is used without proper knowledge and care. The upper arm or the thigh is wrapped with several thicknesses of cotton case padding or a 6-8 inch bias cut stockinette applied smoothly. Then the deflated pneumatic tourniquet is applied to the extremity smoothly and evenly. If this is not done, wrinkles will appear during inflation which will pinch the skin and cause blisters.

Every effort should be made to save tourniquet time. The extremity may be prepped and draped before the tourniquet is inflated. The extremity should be elevated for at least two minutes prior to application of the rubber or Esmarch\textsuperscript{TM} bandage. The extremity may be prepped and draped while the extremity is elevated. To apply the rubber bandage, begin at the finger tips or toes and tightly wrap the extremity proximally to within 2-5 cm of the tourniquet. The edges of the bandage should overlap layer upon layer in a spiral fashion.

The tourniquet should be inflated quickly to prevent filling of the superficial veins before the arterial blood flow has been occluded. The correct pressure will depend on the age of the patient, blood pressure and size of the extremity. Prior to inflation, the valves, gauges, couplings and rubber tubes should be checked for leaks before each individual application.

In the adult, the general range for an upper limb is 275-300 torr and 500-550 torr for the thigh. In applying solutions to the skin, one must be careful to prevent any of the solution from running beneath the tourniquet. This may result in a chemical burn.

A tourniquet may cause vascular injury and/or paralysis if excessive pressure is applied above the previous guidelines. Insufficient pressure results in passive congestion in the wound with hemorrhagic infiltration of the nerve, causing injury. Also, keeping the tourniquet on too long or the application of a tourniquet without considering the local anatomy will cause injury.

There is no absolute rule as to how long the tourniquet may be safely inflated. The time will vary with the age of the patient and vascular supply of the extremity. The average healthy adult should tolerate a tourniquet time of 1-2 hours for an upper extremity and 1.5-2 hours for a lower extremity.\textsuperscript{21} It is generally agreed that on an upper extremity in good condition, the time should not exceed 2 hours.\textsuperscript{21} If the operation requires more time, the tourniquet is deflated for ten minutes. During this time hemostasis should be secured. Safe limits after reinflation are unknown.

For IV regional anesthesia such as a Bier Block, lidocaine HCl 3 mg/kg may be used. In a study by Tucker and Boas,\textsuperscript{49} peak plasma levels of lidocaine after cuff release during IV regional anesthesia were 20-80\% lower than those found when the same dose was given directly into a vein over three minutes. Peak levels after cuff release were inversely proportional to tourniquet application time. Peak plasma levels also tended to be lower by about 40\% when the same dose was given in 0.5\% solutions instead of 1.0\% solution.

During the first few minutes after cuff release, distribution of lidocaine within the pulmonary system buffers the vital organs against high blood levels of the drug. Calculations indicated that after a 10 minute cuff time of a 1.0\% solution, release of the drug from the arm followed a biphasic pattern. There was a fast release of about 30\% of the dose, followed by a much slower release of the remainder. The pattern after a 45-minute cuff time was significantly different from that observed after a 10-minute cuff time. Initial release of the former was much slower and about 55\% of the dose was estimated to remain in the arm after 30 minutes.

If anesthesia is to be reestablished following cuff release, this may be possible 10-30 minutes after initial release by the injection of about half of the original dose following reinflation of the cuff.

Complete arrest of circulation of an extremity is obviously contrary to physiology. During the ischemic phase, there is a fall in the temperature of the skin and muscle. In two hours of tourniquet ischemia, the venous p\textsubscript{H} falls from 7.4 to 6.9 in a linear fashion to reach a value which is close to that of the intercellular p\textsubscript{H}.\textsuperscript{40} At the same time,
the venous \( \text{pO}_2 \) falls from 45 mmHg to 4 mmHg. The venous \( \text{pCO}_2 \) rises from 35 to 104 mmHg and around this critical period, irreversible muscle fatigability develops.

Striated muscles rendered ischemic for two hours show evidence of cell damage and there is atrophy of muscle tissue. There is also a decrease in the control of myokin, water-soluble protein and non-protein nitrogen. After release of the tourniquet, there is a transient increase in blood flow of the post-ischemic limb. There is an increased capillary permeability of the post-ischemic limb to both fluid and protein. This may be the cause of postoperative edema. The plasmin activity on the blood is increased, probably because of release of a kinin factor produced during the hypoxic phase. There is a prolonged rise on the fibrinolytic activity on the systemic circulation which lasts for about 15 minutes. There are no changes in Factors V or VII, fibrinogen and platelet count.

Certain dosages of lidocaine have been implicated as causing grand mal seizures. Crampton reported a grand mal seizure when lidocaine was infused at a rate of 6.7 mg/min (86 \( \mu \)g/kg/min). Diazepam is one of a series of benzodiazepine compounds that suppress seizures or alter discharges arising from subcortical loci, notably the amygdala and hippocampus. The benzodiazepines appear to be especially effective in arresting drug-induced seizures. Considerable clinical experience with acute as well as chronic administration attests to diazepam's safety. Based on its efficiency as an anticonvulsant and its wide margin of safety, DeJong and Heavener recommended judicious clinical trial of diazepam as a local anesthetic premedicant.

### Other considerations

**Choice of an anesthetic.** Although both inhalational or neurolept anesthesia is acceptable for orthopedic surgery, the use of \( \text{N}_2\text{O} \) narcotic anesthesia with neuromuscular blockade and intermittent positive pressure ventilation is preferred for the more major orthopedic procedure. This ensures good oxygenation, relaxation and carbon dioxide elimination. Recovery is more rapid and bleeding is less than with spontaneous respiration techniques based on volatile anesthetic agents.

Epidural or spinal analgesia has two advantages in that hemorrhage is reduced and postoperative pain can be well controlled. For orthopedic surgery on an upper limb, intravenous regional analgesia under a tourniquet provides both a clean surgical field and reduces intraoperative blood loss. Thus, there are multiple techniques and agents available to the anesthetist for the management of orthopedic anesthesia.

**Position.** Several procedures, such as spinal fusion, spinal decompression and intervertebral disc removal, require the patient to be in a prone position. The technique of anesthesia should incorporate intermittent positive ventilation via an endotracheal tube. A secure intravenous infusion should be established beforehand. If the operative site is to be infiltrated with adrenaline, a compatible anesthesia technique should be selected.

Intraabdominal pressure should be kept low in order to promote venous drainage from the operative site. If abdominal pressure is not kept low, then venous bleeding arising from epidural vessels and cut bone surfaces will hinder the surgeon's view. Abdominal pressure may be reduced by correct positioning. Pillows should be placed under the patient's thorax and pelvis. Additionally, abdominal pressure can be reduced by the use of muscle relaxants which maintain full abdominal relaxation.

### Summary

In summary, patients scheduled for major orthopedic cases should be hemodiluted. This requires 500 cc \( \text{D}_5\text{W} \) plus 12-15 ml/kg of \( \text{D}_5\text{LR} \) on the first hour, then 6-10 ml/kg for the second hour, and if the operation continues past three hours, fluids such as lactated Ringer's solution should be administered at a rate to assure urine output of 50 cc per hour.

Blood loss in excess of 20% of the estimated blood volume must be replaced with whole blood. When possible, the use of preoperative autologous blood banking assures available blood at a low risk of transfusion complications.

Hypotensive anesthesia for total hip arthroplasty offers several advantages. Intraoperative and postoperative blood loss can be reduced by 50%. A cleaner surgical field is also attainable, which can decrease the operative time. Methylmethacrylate attaches to bone better in a dry surgical field.

Pulmonary and fat emboli are complications that can occur after orthopedic surgery. Adequate hydration, early ambulation and elastic compression stockings are prophylactic measures that may prevent pulmonary embolism. The prophylactic use of methylprednisolone deserves consideration on cases involving fractures of the femoral shaft.

Acrylic cement is frequently used in joint replacement of the lower extremities. Fat embolism, hypotension and a transient fall in arterial oxygen tension are untoward reactions attributed.
to acrylic cement. Venting the femoral shaft, ade-
quate hydration and maintenance of PaO₂ at
twice the normal values for a few minutes before
and after application of the cement are measures
that can offset these untoward reactions.

Finally, IV regional anesthesia under tourni-
quet provides a clean surgical field and reduces
intraoperative blood loss. In addition, these pa-
tients can be safely sedated with diazepam.

REFERENCES

(1) Finlayson PC. 1972. Fluid and electrolyte requirements dur-
ing anesthesia and surgery, Anesthesia and Analgesia, 51:7-6.4.
(2) Jenkins MT, Giesecke AH. 1974. Balanced salt solutions in clin-
ical anesthesia, Anesthesia and Analgesia, Phil-
(3) Smith RM. 1980. Anesthesia for Infants and Children, 4th edi-
tion, the C.V. Mosby Company, St. Louis, pp. 553.
(6) Cunningham JN, Baker CRF, Reeder JF, et. al. 1972. Alter-
ations in cellular membrane function during hemorrhagic shock
(7) Guyon VL, Reynolds JT. 1958. The use and abuse of blood
(8) Valeri CR, Szymanski IO, Runck AM. 1970. Therapeutic
effectiveness of homologous erythrocyte transfusions following
frozen storage at -80° C for up to seven years, Transfusion
10:162.
(9) Valeri CR, Zaroulis CG. 1972. Rejuvenation and freezing of
(10) Eckardt J.J, Gossett TC, Amstutz HC. 1978. Autologous
transfusion and total hip arthroplasty, Clinical Orthopaedics
(11) Bryant I.R Jr. 1976. Use of frozen-thawed erythrocytes in
a community hospital, In Griepe, JA (ed.) Clinical uses of frozen
(12) Sumida C. 1968. Autologous blood for surgical auto trans-
fusion, J. Bone Joint Surg 50 A:834.
(13) Davis NJ, Jennings TJ, Harris WH. 1974. Induced hypo-
tension anesthesia for total hip replacement, Clin. Orthop.
101:93-98.
(14) Mallory TH. 1973. Hypotensive anesthesia in total hip repla-
cement, JAMA 224:248-249.
(15) Eckenhoff JF. 1978. Editorial views deliberate hypoten-
sion, Anesthesiology 48:87-88.
(16) Stone JG, Sullivan SF. 1972. Halothane anesthesia and pul-
(17) Styles M, Coleman WJ, Leary WP. 1973. Some hemody-
(18) Adams AP, Clarke TNS, Edmonds-Seed J. et. al. 1974. The
effects of sodium nitroprusside on myocardial contractility and
(19) Hamilton WK. 1976. Do let the blood pressure drop and do
use myocardial depressants, Anesthesiology 45:273-274.
(20) Bland JH, Lowenstein E. 1976. Halothane induced de-
crease in experimented myocardial ischemia in the non-failing
canine heart, Anesthesiology 45:297-293.
(21) Gray CF, Nunn JF, Utting JE. 1980. General Anesthesia,
Comparison of warfarin, low molecular weight dextran iasprrin
and subcutaneous heparin in prevention of venous thrombo-
embolism following total hip replacement, J. Bone Joint Surg.
56 A: 1552-1562.
(23) Ross APJ. 1977. The fat embolism syndrome—with special
reference to the importance of hypoxia in the syndrome, Ann. R.
(25) Lehman EP, Moore RM. 1927. Fat embolism including
(26) Hillman JW, LeQuire VS. 1968. Lipid metabolism and fat
embolism after trauma—the contribution of serum lipo-
(27) Hamilton RW, Hustead RF, Pelzler CF. 1964. Fat emboli-
— the effect of particulate embolus on lung surfactant surgery.
56:55-56.
(28) Pelzler CF. 1969. Fat embolism—a current concept, Clinical
(29) Heandton JH, Rischebof EF, Rischer JE, 1971. Fat em-
bolism—a review of current concepts, Journal of Trauma 11:673-
689.
Pulmonary capillary blood volume on the dog—effects of 5
Increasing our knowledge of the pathogenesis of fat emboli-
s— a prospecitve study of 43 patients with fractured femoral shafts,
J. I. Trauma 14:955-962.
(32) Gardner AMN, Harrison M.M. 1957. Report on the treat-
ment of experimental fat embolism with heparin, J. Bone Joint
Surg. 39B:538-541.
JAMA 194:899-901.
(34) Stoltenberg JJ, Gustilo RB. 1979. The use of methylpred-
nisolone and hypertoxic glucose on the prophylaxis of fat em-
bolism syndrome, Clinical Orthopaedics and Related Research
143:211-251.
(35) Bloomenthal ED, Olson WH, Nechols H. 1952. Studies on
damage to bone due to blunt trauma, Intraosseous Phlebography
(37) Scuder CS. 1942, Fat embolism—a clinical and experimental
(38) Vance BM. 1931. The significance of fat embolism, Arch.
Orthop. 102:227.
(40) Wilde AH, Greenwald AS. 1975. Shear strength of self-
sure lowering effect of total hip replacement, J. Bone Joint
Surg. 56A: 834.
oxygen tension during total hip replacement, Anesthesiology
39:642-644.
(43) Zauder HJ. 1980. Anesthesia for Orthopaedic Surgery,
F.A. Davis Company, Philadelphia.
(46) Mulliken S. 1978. The tourniquet in operations upon the exter-
(52) Crampton RS, Oriscello RG. 1968. Petit and grand mal convulsions during lidocaine hydrochloride treatment and ventricular tachycardia, JAMA 204:201-204.

AUTHOR
David W. Hoerneman, CRNA, BSN, received his BSN from Marquette University in Milwaukee, Wisconsin and graduated from the Wausau Hospital Center School of Anesthesia in 1981. Mr. Hoerneman is currently a staff nurse anesthetist at the Marshfield Clinic in Marshfield, Wisconsin. This article was prepared while Mr. Hoerneman was a senior nurse anesthesia student.