Miller Anaesthesia for Orthopedics

Orthopedics

SPECIFIC PROBLEMS OF THE ORTHOPEDIC PATIENT

Rheumatoid Arthritis

Rheumatoid arthritis is a disease of unknown origin characterized by immune-mediated synovitis. <u>1</u> The patients who present the most significant challenge to the anesthesiologist are those with advanced disease having deformity, instability, and destruction of many joints throughout the body. The cervical spine, hips, shoulders, knees, elbows, ankles, wrists, and metacarpophalangeal joints may all be affected. <u>1</u> Although cardiac valvular lesions, pericarditis, and pulmonary interstitial fibrosis do occur, these secondary features of the disease are usually not clinically significant. On the other hand, there is an increased incidence of ischemic heart disease (presumably secondary to corticosteroid treatment), cancer (secondary to chemotherapeutic agents), and infections, all of which contribute to only a 50 percent 5-year survival in advanced cases. <u>2</u> These patients also have an impaired immune system, wasted musculature, and underlying hypermetabolism. All these factors contribute to an increased rate of postoperative infections and other complications. <u>3</u>

The anesthesiologist's immediate concerns, however, tend to be technical. Arterial lines may be difficult to place because of small calcific radial arteries that may be inaccessible, owing to flexion deformities of the wrist joint. These patients have a high incidence of carpal tunnel syndrome, which may predispose them to recurrent symptoms postoperatively if radial artery lines are inserted. <u>4</u> Central venous lines may be difficult to insert because of fusion and flexion of the neck. The lumbar spine, however, is not often affected in rheumatoid arthritis, so spinal anesthesia and epidural anesthesia are usually straightforward. <u>5</u>

Other technical problems of concern are airway management and cervical spine instability. <u>6</u>, <u>7</u> The trachea may be difficult to intubate for a number of reasons (<u>Table 60–1</u>) that are most prominent in those with juvenile rheumatoid arthritis. Atlantoaxial instability (<u>Table 60–2</u>) develops in many patients with adult onset of rheumatoid arthritis. <u>8</u>, <u>9</u>, <u>10</u>, <u>11</u> Symptoms include neck pain, headache, or neurologic symptoms in the arms or legs with neck motion. <u>10</u> Atlantoaxial subluxation develops from erosion of ligaments by rheumatoid involvement of the bursae around the odontoid process of C2 (<u>Fig. 60–1</u>). Acute subluxation may result in cord compression and/or compression of the vertebral arteries with quadriparesis or sudden death. Subluxation occurs with flexion of the neck (<u>Fig. 60–2</u>). Anesthetic management must prevent flexion of the neck and maintain stability of the cervical spine. This may be accomplished by tracheal intubation using a flexible bronchoscope under topical anesthesia and positioning the patient while the patient is still awake. Regional anesthesia with the patient minimally sedated and the neck stabilized is a reasonable perioperative alternative.

TABLE 60-1. Orthopedic Patients in Whom Intubation of the Trachea May Be Difficult

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DIAGNOSIS CAUSE(S) OF DIFFICULTY Ankylosing spondylitis Fusion of cervical spine Juvenile rheumatoid arthritis Ankylosis of cervical spine Hypoplasia of mandible Adult rheumatoid arthritis Multiple deformity Ankylosis and instability of the cervical spine Prior spine fusion Ankylosis and limited extension of the cervical spine Congenital deformities of the cervical spine

Epiphyseal dysplasia

Dwarfism (achondroplasia)245 Fractured cervical spine246 Limited motion Risk of quadriplegia Copyright © 2000, 1995, 1990, 1985, 1979 by Churchill Livingstone

TABLE 60-2. Causes of Atlantoaxial (C1-C2) Instability

TABLE 60-2. Causes of Atlantoaxial (C1-C2) Instability

Rheumatoid arthritis Down syndrome Ankylosing spondylitis Mucopolysaccharidoses (e.g., Morquio disease) *Note:* Patients are stable in extension, but flexion may compress the spinal cord or medulla oblongata.

FIGURE 60–1 Magnetic resonance image of a patient with advanced rheumatoid arthritis demonstrating invagination of the odontoid process of C2 (arrow) through the foramen magnum compressing the brain stem. Note the degeneration of C4 and C5, a common problem in rheumatoid arthritis. FIGURE 60–2 Computed tomographic scan of the neck demonstrates moderate subluxation of C1 and C2. The odontoid (single arrow) tends to compress the spinal cord (double arrow) against the posterior arch of C1, especially during neck flexion.

Patients with severe rheumatoid arthritis may develop airway obstruction postoperatively from narcotics or sedatives. <u>12</u> Therefore, judicious use of narcotics or epidural analgesia for pain relief should be considered postoperatively, together with the administration of nasal oxygen and pulse oximetry if feasible. Cardiopulmonary resuscitation is difficult in rheumatoid patients (<u>Ch. 25</u>), and emergency tracheotomy is almost impossible in severe cases (<u>Ch. 39</u>). Jet ventilation by means of a percutaneous catheter through the cricothyroid membrane may be required. <u>13</u>

Ankylosing Spondylitis

Ankylosing spondylitis, which is more common in men than women, involves ossification of ligaments at their attachment to bone. Progressive ossification involves the joint cartilage and disk space of the axial skeleton, with eventual ankylosis. Arthritis and ankylosis may also develop in the hips, shoulders, and costovertebral joints. <u>14</u>

Lung function is somewhat impaired from the development of rigidity of the rib cage. Vital capacity is minimally reduced if diaphragmatic activity is preserved. <u>15</u> Aortic regurgitation and bundle branch block may develop, necessitating aortic valve replacement or pacemaker insertion. <u>16</u> There is an ever-present risk of spine fracture and cervical spine instability in these patients, so careful positioning in the operating room is important <u>17</u> (Tables <u>60–3</u> and <u>60–4</u>).

Anesthetic considerations include (1) use of fiberoptic techniques for tracheal intubation, <u>13</u>, <u>18</u>, <u>19</u> (2) positioning the patient while the patient is awake, and (3) the choice of axillary rather than interscalene blocks when using regional techniques in the upper extremity. Caudal anesthesia can be readily obtained. The vertebral column is usually fused, making lumbar epidural or spinal anesthesia difficult or impossible. In patients who can still move their neck, fusion of the lumbar spine may be incomplete, <u>20</u> enabling epidural or spinal anesthesia to be performed successfully.

TABLE 60-3. Anesthetic Problems of the Prone Position

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Airway

Endotracheal tube kinking or dislodgement

Edema of upper airway in prolonged cases may cause postoperative respiratory obstruction.

Blood vessels

Arterial or venous occlusion of the upper extremity (check with pulse oximeter on the finger)

Kinking of the femoral vein with marked flexion of the hips; this may predispose to postoperative deep vein thrombosis

During lumbar laminectomy, abdominal pressure increases may cause elevation of epidural venous pressure, contributing to intraoperative bleeding.

Nerves

Brachial plexus stretch or compression

Ulnar nerve compression due to pressure medial to the olecranon

Peroneal nerve compression due to lateral pressure over the head of the fibula

Lateral femoral cutaneous nerve trauma due to pressure over the iliac crest

Head and neck

Gross hyperflexion or hyperextension of the neck247,248

External pressure over the eyes may result in retinal injury due to compression.26,249

Lack of lubrication or coverage of eyes may result in corneal abrasion.

Headrest may cause pressure injury of supraorbital nerve.

Excessive rotation of the neck may contribute to brachial plexus problems or kinking of the vertebral artery.250

Lumbar

Excessive lordosis may lead to neurologic injury.251,252

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TABLE 60-4. Sites of Peripheral Nerve Injury in Orthopedics

NERVE INJURY SITE/ CAUSE/ COMMENT

Upper extremity

Brachial plexus/ Abduction, external rotation, or extension of shoulder /Usually resolves within several months Traction of shoulder/

Ulnar nerve/ Pressure at the elbow/ Not uncommon Traction of C8–T1 dermatomes over the 1st rib/ Postoperative palsy results in numbness of ring and fifth fingers

Radial nerve/ Pressure behind the arm /Results in wristdrop

Anterior interosseous nerve/ Pressure at the distal elbow laterally /Bandages or external pressure253

Head

Supraorbital nerve/ Pressure on the supraorbital ridge when lying prone /Results in numbness of forehead

Lower extremity

Lateral femoral cutaneous nerve of the thigh/ Pressure over anterior iliac crest in lateral or prone position or over lateral thigh/ Results in numbness of the lateral aspect of the thigh and knee

Femoral nerve/ Pressure to the groin of the dependent limb in the lateral decubitus position /Results in numbness of the anterior thigh and medial aspect of lower leg

Common peroneal nerve /Pressure below the head of the fibula /May be due to compartment syndrome/ Results in footdrop

Ankle Pressure from Esmarch bandage/ Pressures beneath Esmarch bandage can be much higher than believed

Checklist:

- 1. Check for preoperative nerve dysfunction.
- 2. Check tourniquet problems-duration and pressure.
- 3. Check postoperative position, splints, tight bandages; rule out compartment syndrome.
- 4. Check intraoperative surgical factors.
- 5. Risk of neuropraxia is more common in prolonged surgery.

POSITIONING FOR ORTHOPEDIC SURGERY

Patients are placed in a variety of positions for orthopedic procedures (<u>Ch. 26</u>). Improper positioning may result in intraoperative or postoperative problems such as those described below.

Air embolism (Ch. 52) can occur when the operative field is above the level of the heart. This is a potential problem in surgery of the cervical spine or the shoulder in the sitting position, in total hip replacement in the lateral decubitus position, or in lumbar spine surgery in the prone position. Air embolism should be considered if untoward circulatory compromise occurs in any of these settings, although it is rare. 21, 22, 23, 24

Stretch or malposition of joints may occur during anesthesia and might account for a variety of nonspecific postoperative discomforts in the back or the extremities. Patients with rheumatoid arthritis, osteoporosis, osteogenesis imperfecta, or contractures must be carefully positioned to avoid ligamentous or bony injury.

Direct pressure, especially over bony prominences, may cause tissue ischemia and/or necrosis, particularly after prolonged surgery when hypotensive anesthesia is used. Direct pressure on the soft tissues of the orbit when lying prone may lead to retinal artery occlusion, <u>25, 26, 27, 28</u> and direct pressure over other peripheral nerves may result in postoperative neurapraxia. <u>29, 30, 31, 32</u>

Compression of the veins and/or arteries supplying either the upper or lower extremity may occur. Prolonged venous obstruction at the axillary vein is best alleviated with an axillary roll positioned beneath the upper thorax. Similarly, with patients in the lateral decubitus position, stabilizing posts must be positioned carefully over the groin so as not to interfere with venous return at the level of the femoral vein. Arterial obstruction of a limb may be checked by the use of a pulse oximeter <u>33</u> or by palpating the pulse of a distal artery. Venous obstruction may lead to a compartment syndrome with edema, neurapraxia, elevation of creatine phosphokinase level, and myoglobinuria postoperatively. <u>34</u>

Positioning of the rheumatoid patient is very important; care must be taken not to flex the neck excessively. Regional anesthesia is particularly suitable for these patients, because neck stability can be maintained by the patients themselves, particularly if only light to moderate sedation is given. In addition, other joints that should not be moved beyond the normal range of motion will be protected by the conscious patient. Excessive motion may occur when these patients are moved in the anesthetized paralyzed state, resulting in neurapraxia, joint dislocation or stretch, or muscle trauma.

MAJOR ORTHOPEDIC PROCEDURES

Major operations require special preparation on the part of the anesthesiologist, increased attention to the details of intraoperative monitoring and fluid management, and active participation whenever possible in postoperative pain management.

Management of Blood Loss

Major orthopedic procedures are associated with significant blood loss 38 (Chs. 46 and 47). Public awareness of the dangers of transfusion has increased dramatically as a result of the acquired immunodeficiency syndrome epidemic as well as a recognition of other risks of transfusion, such as hepatitis, malaria, and, more recently, bovine spongiform encephalitis in Great Britain. 39, 40, 41 A plan to minimize homologous transfusion for all patients undergoing joint replacement, tumor resections, or major spine surgery should be established. This may begin with predonation of autologous blood, 42 preoperative erythropoietin 43 (e.g., in Jehovah's Witnesses 44 or patients with preoperative anemia), intraoperative hemodilution, <u>45, 46</u> induced hypotension, <u>47</u> conduction anesthesia, use of a cell saver, 48, 49 preservation of normothermia, 50 or by tolerating lower hematocrit values postoperatively (lowering the socalled transfusion trigger 51) and the use of antifibrinolytic agents. 52, 53 In practice, combination of several of these modalities is most effective in reducing homologous transfusion. At The Hospital for Special Surgery, most patients predonate autologous blood and receive hypotensive anesthesia, and the transfusion trigger has been lowered into the mid 20s. Cell savers, which are expensive and have certain risks, 54 are used selectively in the operating room for major spine surgery for scoliosis. Antifibrinolytic drugs and postoperative cell salvage are probably best used when expected blood loss exceeds 2 L but is unwarranted in more minor procedures due to the risks of aprotinin. 52, 55, 56, 57

Total Hip Replacement

Background

Anesthetic management of total hip replacement varies according to the complexity of the surgery, complications that may arise during the surgery, and the medical status of the patient. Complex procedures such as those involving acetabular bone grafting, insertion of a long-stem femoral prosthesis, <u>58</u> removal of a prosthesis, revision surgery, or surgery in patients with acetabular protrusion (which entails a risk of entering the pelvic cavity and/or the iliac vessels) complicate the management of the anesthetic.

Anesthetic Management

Monitoring

Because most candidates for total hip replacement have only a limited ability to exercise, their cardiopulmonary function can be difficult to assess. This often elderly population frequently has underlying systemic diseases. Fluid administration must be carefully managed during this type of extensive surgery. Furthermore, there is an increased likelihood to develop hypoxemia and/or pulmonary edema due to pulmonary endothelial injury from fat or bone marrow emboli <u>59</u> and from ventilation/perfusion mismatching (see <u>Positioning</u> and <u>Tables 60–3</u> and <u>60–4</u>). It is, therefore, reasonable to use invasive hemodynamic monitoring perioperatively in the elderly or medically compromised patient undergoing total hip replacement, especially when this involves complex or revision surgery.

Extensive studies during and after total hip replacement show that use of either hypotensive or regional (epidural or spinal) anesthesia reduces the blood loss by 30 to 50 percent. <u>60</u>, <u>61</u> Lowering intraoperative mean arterial pressure to 50 mm Hg reduces blood loss more effectively than a mean arterial pressure of 60 mm Hg. <u>62</u>

Blood loss during total hip replacement is significantly greater during revision surgery. In these patients, several modalities to reduce the risk of homologous transfusion should be used, including preoperative donation of autologous blood, induced hypotension, or hemodilution. Cell savers can be used if blood loss is expected to be greater than 1 L. With the use of hypotensive epidural anesthesia, intraoperative blood losses of more than 300 mL are unusual, limiting the need for the cell saver or antifibrinolytic agents.

Positioning

The majority of total hip replacements are performed with the patient in the lateral decubitus position. This creates a potential ventilation/perfusion mismatch with resultant hypoxemia, a problem that appears most often in patients with underlying lung disease. The lateral decubitus position can create neurovascular problems as well because the dependent shoulder presses on the axillary artery and brachial plexus, <u>63</u> and the anterior stabilizing post compresses the femoral triangle. <u>33</u> These problems can be minimized by placing an axillary roll beneath the upper thorax and by careful positioning of the anterior stabilizing post at the dependent groin. Patients who are given hypotensive anesthesia may be at greater risk of neurovascular injury, because less extrinsic pressure is required to compress a less tense vessel. <u>26</u>

Cement Fixation

The quality of the cement–bone interface is improved if there is no blood covering the cancellous bone as the cement is applied. Hypotensive anesthesia has been shown radiographically to improve the quality of cement bone fixation, 64 because it reduces bleeding from bone. 62

Intraoperative Hypotension

Profound hypotension immediately after insertion of cemented femoral prostheses has resulted in cardiac arrest and death 58, 65, 66, 67 (Ch. 41). These events are not seen with noncemented prostheses. Nowadays, this is distinctly uncommon during elective primary total hip replacement but not uncommon in certain high-risk groups (Table 60-6). Therefore, it seems likely that hypotension is related in some way to the use of cement (Fig. 60-3). Two possible explanations are that (1) it may be caused by direct vasodilation and/or cardiac depression from methyl methacrylate or (2) it may be due to the forced entry of air, fat, or bone marrow into the venous system with resultant pulmonary emboli. 65 Large echogenic emboli have been described after insertion of femoral prostheses; this supports the concept that the circulatory collapse is embolic 68 rather than from a toxic effect of the methyl methacrylate. Attempts to minimize this complication have included (1) the use of a plug in the femoral shaft to limit the distal spread of cement in the femur, 69 (2) venting of entrapped air, and (3) waiting for cement to become more viscous before its insertion. These maneuvers minimize the disruption of bone marrow in the distal femur, thereby preventing catastrophic fat/bone marrow pulmonary embolism. Embolization can also be reduced by carefully lavaging the femoral canal. 70 By contrast, in the high-risk group, extensive reaming of the femur occurs, disrupting more bone marrow; and with pressurization with cement, significant marrow is forced into the circulation. This can be seen with transesophageal echocardiography 65, 68, 71, 72 (Fig. 60-4).

TABLE 60-6. Patients at Risk of Acute Hypotension/Cardiac Arrest During Total Joint Replacement

Long-stem cemented primary total hip replacement

Metastatic cancer71,254

Removal of hardware after intertrochanteric hip fracture58

Spiral fracture of femur58

Revision of long-stem cemented femoral component

Cemented prosthesis to distal femur

Fracture below femoral component

Cemented long femoral stem total knee replacement86,87

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FIGURE 60–3 An 85-year-old woman undergoing primary total hip replacement, status post Richard screw with a long side plate. She had a history of chronic obstructive lung disease and osteoporosis. Pulmonary

and radial artery pressures were stable until a 200-mm cemented femoral component was inserted (point A). One minute after impaction of the femoral component, pulmonary artery pressure increased acutely (point C) and arterial pressure decreased to 30 mm Hg. Epinephrine, 25 ?g, was injected through the distal port of the pulmonary artery catheter (point D). This resulted in a rapid restoration of arterial pressure, a transient tachycardia, and stabilization of the pulmonary artery pressure. The hip was relocated at point B, which resulted in no change in pulmonary artery pressure. (From Sharrock et al<u>256</u>) FIGURE 60–4 Right atrium during echocardiography. (*A*) Multiple small emboli in the right atrium. (*B*) A large embolus 7 cm in length, which is probably a cast of the femoral vein. (From Christie et al73)

Echogenic material is noted with reaming the femur, after insertion of the cemented femoral component, <u>65</u>, <u>68</u>, <u>71</u>, <u>72</u> and with relocation of the hip joint. <u>73</u> Large emboli may be observed in the right side of the heart, obstructing the right ventricular outflow tract leading to right-sided heart failure, hypotension, and cardiac arrest. Small emboli traverse the right side of the heart and embolize to the lung. These may increase pulmonary artery pressure but are less likely to cause intraoperative cardiac arrest. In patients with a patent foramen ovale, emboli may pass into the systemic circulation, causing myocardial infarction or stroke. <u>71</u> It is curious that although patent foramen ovale is common, severe systemic manifestations of fat embolization during surgery. Fat may also pass through the pulmonary circulation, contributing to systemic manifestation of fat embolism after surgery. <u>74</u> Whether this contributes to acute delirium or persistent decline in cognitive function is unclear. <u>75</u>, <u>76</u>, <u>77</u>, <u>78</u>

Hypotension may occur with impaction of the femoral component or after relocation of the hip joint (see Fig. <u>60–3</u>). With impaction of the femoral component, the embolic material can be forced directly into the venous system. On the other hand, obstruction of the femoral vein occurs during insertion of the femoral component so that fat or bone marrow is usually retained in the femoral vein until the hip joint is relocated. As soon as the obstruction of the vein is relieved, the emboli pass toward the right side of the heart. This process can be monitored with transesophageal echocardiography or by using oximetric pulmonary artery catheters <u>79</u> (Fig. <u>60–5</u>). An acute decline in mixed venous oxygen saturation occurs in conjunction with a rise in pulmonary artery pressure with release of an obstructed femoral vein containing embolic material. FIGURE 60–5 Pulmonary artery oxygen saturation during total hip replacement is recorded from an oximetric pulmonary artery catheter (American Edwards, Baxter Healthcare Corporation, Irvine, Calif). Note the acute fall in pulmonary artery oxygen saturation after relocation of the hip at minute 62 after trial reduction (T) with 8 minutes of potential venous occlusion and minute 74 after final reduction (F) with 12 minutes of potential venous occlusion. (From Sharrock et al<u>79</u>)

An effective treatment for the acute hypotension is intravenous epinephrine (4-50 ?g) as soon as the hypotension is noted. <u>58</u> The dose depends on the degree of hypotension. In the high-risk group, epinephrine (10-20 ?g) is injected through the distal port of a pulmonary artery catheter the moment any reduction in arterial pressure is noted after impaction of the long-stem cemented femoral component. If cardiac arrest occurs, much larger doses of epinephrine will be needed to resuscitate the patient.

Hypoxia has been described immediately after insertion of a cemented femoral prosthesis and for up to 5 days into the postoperative period. In the event of hypoxemia, one must first ascertain whether it has a specific cause, such as atelectasis of the dependent lung, hypoventilation, or fluid overload. Even with no specific cause, hypoxemia may persist for some days after hip surgery. <u>80</u> It may be a result of the embolic effects of femoral shaft cement or fat embolism. <u>81</u> Pulmonary emboli with cement or bone marrow increase pulmonary artery pressures in dogs <u>82</u> and humans, <u>79</u> but it is unclear whether this is directly responsible for the hypoxemia observed. Postoperative management should include nasal oxygen, pulse oximetry (if necessary for several days), <u>59</u> judicious use of narcotics to provide analgesia and yet avoid hypoventilation or airway obstruction, appropriate fluid management, and diuresis. Hypoxia and fluid overload may further increase pulmonary pressures and thus increase the likelihood of pulmonary edema or right-sided heart failure. Postoperative hypoxia is more common in patients who snore.

Total Knee Replacement

Background

Patients who need total knee replacement frequently have severe rheumatoid arthritis, degenerative osteoarthritis, or other significant comorbidities that compound the difficulties of the operation. The average duration of the procedure is 1 to 3 hours (see <u>Table 60–5</u>).

TABLE 60-5. Suggested Management of Major Orthopedic Procedures

PROCEDURE/ AVERAGE DURATION (H)/ RANGE OF BLOOD LOSS (UNITS)/ SUGGESTED MONITORING OR SPECIAL TECHNIQUES Total hip/ 1–4/ 1-6/ Autologous blood donation/ Arterial cannula Central venous pressure Foley catheter (optional) Epidural catheter (optional for postoperative pain treatment) Induced hypotension Cell saver (revision) Total knee/ 1.5–3 /0-2 (with tourniquet)/ Autologous blood donation Arterial cannula Folev catheter (optional) Epidural catheter (for postoperative analgesia) Maior spinal surgery/ 3–8/ 1-10/ Autologous blood donation Arterial cannula Central venous pressure Foley catheter Induced hypotension Evoked potential monitoring Wake-up test Major allograft surgery/2-8/2-10/ Autologous blood donation Arterial cannula Central venous pressure Foley catheter Induced hypotension and/or hemodilution Epidural catheter (for postoperative analgesia) Major pelvic resections/2-8/2-10/ Autologous blood donation Arterial cannula Central venous pressure Folev catheter Induced hypotension and/or hemodilution Epidural catheter (for postoperative analgesia) Evoked potential monitoring

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Anesthetic Management

Unilateral Versus Bilateral Total Knee Replacement

There is considerable debate in the orthopedic literature about the wisdom of performing one-stage bilateral total knee replacement. <u>84, 85</u> Knee arthritis is frequently bilateral, and patients appreciate the opportunity to have both replacements performed at the same time so they have only one period of rehabilitation and can "get it over with." On the other hand, patients undergoing bilateral total knee replacement have a higher rate of postoperative complications and require much more perioperative interventions so some selection process is required. However, if patients have appropriate hemodynamic monitoring, postoperative epidural

analgesia, and a 24- to 48-hour period of surveillance in a high-dependency unit, the outcome is usually excellent.

Cement

When acrylic cement is applied to the cavities of the tibia, femur, and patella, acute hemodynamic responses seldom follow. Such responses do occur, however, when *long-stem* femoral prostheses are inserted after extensive femoral reaming. <u>86</u>, <u>87</u> Lesser degrees of femoral reaming may reduce the incidence of embolic events, but the significance of these events is unclear. Pressures in the femoral canal of 300 mm Hg or more have been recorded during impaction of the femoral component, <u>88</u> although this does not appear to adversely affect arterial oxygen or pulmonary artery pressures. <u>89</u>

On release of the tourniquet after insertion of a cemented total knee replacement, showers of fine emboli are detectable in the right side of the heart. This has been associated with an increase in pulmonary vascular resistance during general anesthesia. <u>90</u> Both the severity of metabolic injury <u>89</u> and the echogenic material <u>90</u> were similar with intramedullary and extramedullary fixation, suggesting that the emboli are thrombi rather than bone marrow. <u>90</u> Echogenic material has also been observed during total shoulder replacement. <u>91</u>

Blood Loss

The intraoperative use of tourniquets makes blood loss negligible, but postoperative drainage averages 500 to 1,000 mL per knee. <u>92</u> Therefore, postoperative monitoring, possibly in the postanesthetic care unit, for 24 hours or more may be necessary in high-risk patients until wound drainage slows. Patients undergoing bilateral procedures are at additional risk of becoming hypovolemic during the first few hours after the operation. Preoperative autologous blood donation can minimize homologous transfusions in this setting.

Fibrinolytic activity increases when the tourniquet is inflated. <u>93</u> This observation provided the rationale for the use of antifibrinolytic agents to minimize blood loss after total knee replacement. Although aprotinin has not been clinically proven to be useful in this setting, <u>94</u> tranexamic acid is cost effective in unilateral total knee replacement. <u>53</u>, <u>95</u>, <u>96</u>

Postoperative Pain Management

Total knee replacement is associated with significantly more pain than total hip replacement, and the use of continuous passive motion devices or early mobilization of the knee increases the pain (<u>Ch. 69</u>). Epidural analgesia for 24 to 72 hours effectively controls the pain. <u>97</u>, <u>98</u> Femoral/sciatic blocks or intrathecal morphine can be used to provide analgesia for 8 to 24 hours.

Femoral nerve catheter technique can be used for several days. The particular technique depends on the availability of postoperative pain services.

Thoracolumbar Spine Surgery

Major thoracolumbar spine surgery is usually used to correct deformity (e.g., scoliosis), to stabilize fractures, <u>99</u> or for resection of tumors. <u>100</u>

The basic aim of surgery for scoliosis is to prevent progression of the curvature of the spine, maintain posture, and prevent progression of pulmonary dysfunction. Scoliosis can be congenital or can develop during adolescence or later in life. Comorbidities in adolescent scoliosis include restrictive lung disease, which may lead to pulmonary hypertension, and an increased incidence of malignant hyperpyrexia. Patients with congenital scoliosis may have congenital heart disease, airway abnormalities, and preexisting neurologic deficits. Patients with neuromuscular disease such as Duchenne muscular dystrophy, poliomyelitis, dysautonomia, spinal-cord injury, and neurofibromatosis may also develop scoliosis, which can lead to extremely complex management problems. <u>101</u>

Perioperative considerations include intraoperative positioning, spinal-cord monitoring, minimizing of blood loss, and postoperative respiratory care. Many of these patients undergo both anterior and posterior

procedures, which may be staged or performed under one anesthetic. The anterior procedure is often performed by thoracotomy. Postoperative ventilatory support and pain management are more complex in patients who have had double procedures and in those with preoperative neuromuscular disorders or dysautonomia.

Problems of surgery and anesthesia in the prone position are listed in <u>Table 60–3</u>. Particular attention should be focused on positioning of the neck, arms, and eyes to protect pressure points adequately, particularly if hypotensive anesthesia is to be used in longer surgical procedures. Patients may be moved slightly as a result of surgical manipulation after a wake-up test or after alterations in the position of the table. Therefore, reassessment of patient positioning is advisable at regular and pertinent intervals intraoperatively.

TABLE 60-3. Anesthetic Problems of the Prone Position

Monitoring

Somatosensory Evoked Potentials

Intraoperative spinal-cord function monitoring is important if correction of spinal curvature is to be undertaken. Distraction of the spine may lead to ischemia of the spinal cord because anterior spinal artery flow may be compromised.

There are two approaches to spinal-cord function monitoring: (1) somatosensory evoked potential (SSEP) monitoring <u>102</u> (<u>Ch. 35</u>) and (2) the so-called wake-up test. Disruption of spinal cord function results in changes in both amplitude and latency of SSEPs. <u>103</u> The SSEPs, however, can also be altered by the use of inhalation agents. On the other hand, they are less disturbed by the nitrous oxide–narcotic-relaxant technique and minimally disturbed by intravenous anesthesia. It is less clear whether deliberate hypotension or moderate hypothermia influences the interpretation of SSEPs, but profound hypotension and shock do cause significant inhibition of the responses. SSEP monitoring may be also used when placing pedicle screws <u>104</u> or using other spinal instrumentation or during cervical spine surgery. <u>105</u>

Wake-Up Test

SSEPs assess posterior spinal cord function. A reduction in anterior spinal artery blood flow, however, produces ischemia of the anterior regions of the cord, which may result in motor weakness of the lower extremities. In some cases this may occur without observed alterations in SSEPs. For this reason, a wake-up test has been extensively used in many centers in addition to SSEPs during scoliosis surgery. <u>106</u>

Patients receive nitrous oxide-narcotic-relaxant anesthesia throughout the procedure. Volatile anesthetics are not administered. The wake-up test can be performed by discontinuing nitrous oxide and, using peripheral nerve stimulation, ensuring that neuromuscular blockade is relatively shallow (two or three twitches on train-of-four stimulation). Within 3 to 5 minutes from discontinuation of nitrous oxide, patients will usually respond to verbal commands to move their hands and their feet. The presence of motion in the feet suggests that there is not complete ischemia of the spinal cord.

Use of potent anesthetic vapors may delay wake-up for as much as 30 minutes. Intraoperative antagonism of narcosis or of neuromuscular blockade should not be done, because this may cause overly sudden alertness and dangerously excessive movement on the operating table. In the partially paralyzed narcotized state, however, this technique is easy to perform and has worthwhile predictive value regarding the safety of spinal-cord distraction. It is not psychologically traumatic to the patient because amnesia is nearly complete and there is no recollection of pain or discomfort. Anesthesia is reinduced as soon as movement is demonstrated. Motor-evoked potential monitoring has recently been introduced because it should be a better monitor of ischemia in the anterior spinal cord, avoiding the need for a wake-up test. <u>107</u>, <u>108</u>

Conservation of Blood Resources

Because major blood loss is to be expected, autologous blood donation (3 to 4 units if possible), <u>109</u>, <u>110</u> intraoperative hemodilution, use of the cell saver, and induced hypotension should all be considered <u>102</u>,

110, 112 (Chs. 41 and 47). Antifibrinolytic agents such as aprotinin reduce blood loss and homologous transfusion in selected cases.

Hemodynamics

Because blood loss during spinal procedures is considerable (2 to 10 units, depending on how many segments are to be fused), moderate levels of hypotension (to a mean of 55 to 60 mm Hg) will effectively reduce blood loss, limiting the likelihood of homologous blood transfusion. Invasive monitoring should be carried out with arterial lines, central venous or pulmonary artery catheters, and Foley catheters for fluid management and replacement therapy.

Postoperative Care

Postoperatively, patients with significant comorbidity should be carefully monitored. 113 Patients with neuromuscular diseases, significant restrictive pulmonary disease, congenital heart disease, or evidence of right-sided heart failure may require ventilation for 24 hours or longer. Admission to an intensive care unit should be planned in advance to ensure precise hemodynamic and fluid monitoring and to allow maximum therapy for pain relief during this time. Intrathecal narcotics 114, 115 or epidural analgesia may also be used for postoperative analoesia. Postoperative hyponatremia sometimes occurs and has been attributed to inappropriate antidiuretic hormone secretion. 116

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Cervical Spine Surgery

Surgery on the cervical spine for trauma, tumor, arthritis, spinal stenosis, or instability has a number of complications. 117, 118 Posterior cervical decompression may be performed in the sitting position, increasing the risk of air embolism, or in the prone position, increasing the risk from external pressure on the eyes. 26 Intubation may be difficult, owing to instability of the spine (as in neck fracture or C1-C2 instability) or to a complex airway due to neck deformity as in rheumatoid arthritis. 8, 11 If any question exists, these patients are best intubated with the aid of a fiberoptic bronchoscope and positioned while they are "awake" (in fact, sedated and amnesic) to be certain that the anesthetic maneuvers have not compromised the spinal cord. An armored endotracheal tube is often preferable to minimize kinking due to retractors or movement during surgery.

Cervical spine surgery may be complicated by injury to the spinal cord during surgery, leading to postoperative guadriplegia with respiratory impairment. 8 For this reason, SSEPs are often used during these procedures to monitor surgical manipulations. Patients with rheumatoid arthritis undergoing cervical spine surgery in the prone position should receive minimal intraoperative fluid, be intubated with the aid of a fiberoptic bronchoscope, and have their neck maintained in a neutral position; and after surgery, they should be kept head up for 3 to 5 hours to minimize the risk of upper airway obstruction after extubation. 119, 120 The potential respiratory difficulty is presumably from upper airway edema secondary to trauma during intubation, excessive fluid, and prolonged dependency. 120 Cervical spine surgery can be performed using local anesthesia to avoid some of these problems. 19, 121 Copyright © 2000, 1995, 1990, 1985, 1979 by Churchill Livingstone

Lumbar Spine Surgery

Modern lumbar spine surgery ranges from disk excision through tiny incisions to extensive anterior/posterior fusions with allografts, 122, 123 bone grafts, pedicle screws, 124 and so on, which may be associated with excessive blood loss, prolonged dependency, positioning problems, and neurologic monitoring with SSEPs and/or electromyograms. The anesthetic techniques must be adapted to the particular surgery. Although traditionally, these cases have been performed with general anesthesia, spinal or epidural anesthesia is being more commonly performed for simple disk excision. 125 Lumbar spine fusion can be performed with low thoracic epidural anesthesia (usually in combination with general anesthesia). The virtues of epidural anesthesia are less bleeding and improved postoperative epidural analgesia. 126 Epidural catheters can also be placed by either the anesthesiologist or surgeon during scoliosis surgery to facilitate postoperative analgesia.

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Major Allograft or Autograft Transplantation Surgery

Background

Major segmental skeletal defects, particularly in the long bones of the extremities, may occur as a result of tumor resection, <u>127</u> trauma, or osteonecrosis. Repair of these lesions to eventually permit weight bearing in the legs or restoration of mechanical function to the arms requires bone grafting. Either banked allografts obtained from living or dead donors or vascularized autografts, usually of the fibula, are used to bridge resultant bony gaps <u>128</u>, <u>129</u> (Fig. 60–6). The duration of these procedures is long (2–10 hours), and many candidates may be debilitated as a result of radiation therapy, chemotherapy, or chronic infection. FIGURE 60–6 Three months after en bloc resection and reconstruction with a vascularized fibular graft augmented with autogenous cancellous graft using external fixation for immobilization. (From Hsu et al<u>129</u>)

Frequently, tumor resection is followed by immediate replacement grafting. The surgical procedure consists of two phases for the anesthesiologist: first, the surgical resection is often bloody, requiring attention to details of fluid management and blood conservation and replacement; second, subsequent fitting of the graft and fixation to adjacent structures followed by wound closure may require several hours of reconstructive surgery. Major anesthetic considerations are monitoring requirements, fluid and transfusion therapy, and postoperative pain relief (see <u>Table 60–5</u>). In longer procedures, meticulous attention to prevention of pressure necrosis, neurapraxia, joint stiffness, and arthralgia is necessary.

TABLE 60-5. Suggested Management of Major Orthopedic Procedures

Anesthetic Management

Whatever the choice of anesthetic technique, intraoperative hemodilution combined with deliberate (induced) hypotension should be strongly considered because it is desirable to limit blood loss and to provide as dry a surgical field as possible during the procedure (Chs. <u>41</u> and <u>47</u>). Preservation of the vascularized graft is vitally important. <u>130</u>, <u>131</u> Patient temperature, circulatory blood volume, and cardiac output must be maintained and, if possible, graft flow augmented by a sympathetic blockade. Other measures include intravenous mannitol and anticoagulation with heparin. Postoperatively, these patients should be kept in special care units so that the wounds can be monitored for graft patency by visual inspection, Doppler flow probe, and pulse oximetry monitoring.

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Pelvic and Sacral Resections/Fractures

Background

Major bony resections of the sacrum or pelvis are performed as primary treatment of usually cancerous bone tumors. Major pelvic surgery is also done for repair of complicated pelvic or acetabular fractures. As in major spinal or hip surgery, care in positioning for a long procedure is important, because many of these operations are done in the lateral or prone positions (see <u>Table 60–3</u>).

TABLE 60-3. Anesthetic Problems of the Prone Position

Anesthetic Management

Measures for conservation of blood resources and body heat should be carefully followed (Chs. $\underline{37}$, $\underline{41}$, and $\underline{47}$). Invasive monitoring may be essential, and improved postoperative analgesia by means of epidural catheters should be strongly considered $\underline{132}$ (see Table 60–5).

TABLE 60-5. Suggested Management of Major Orthopedic Procedures

If the dissection is to involve major pelvic vasculature or nerve roots, the following additional measures might be taken: pulse oximetry in the lower extremity (toe) will aid in judging adequacy of circulation, and SSEP monitoring of L4–L5 to S2 nerve roots may help to lessen the possibility of nerve damage during en bloc

dissections of the sacrum (see <u>Table 60–5</u>) or during repair of pelvic fractures. If SSEP monitoring is used, epidural anesthesia and inhalation anesthetics may be contraindicated. Additional large-bore intravenous cannulas may be needed in anticipation of rapid fluid and blood infusion during major resections. Copyright © 2000, 1995, 1990, 1985, 1979 by Churchill Livingstone

POSTOPERATIVE ANALGESIA IN ORTHOPEDICS

Pain after orthopedic surgery depends on the site and extent of surgery and the preoperative use of analgesics by the patient (<u>Ch. 69</u>). The techniques used are further defined by the facilities available in the hospital. Without a postoperative pain service, systemic narcotics may be the mainstay of therapy. On the other hand, if a postoperative pain service is available, a variety of continuous infusions or patient-controlled analgesia modalities can be used to optimize pain therapy and augment recovery.

Orthopedic surgery lends itself to regional anesthesia. Peripheral blocks with bupivacaine or ropivacaine can provide 12 to 24 hours of significant analgesia, which is often sufficient to eliminate the need for intramuscular or intravenous narcotics. Alternatively, infusions of local anesthetic by means of catheters inserted into the femoral, <u>152</u> popliteal, <u>153</u> or brachial plexus <u>154</u> may provide significant postoperative analgesia. Intra-articular injections of local anesthetic <u>155</u>, <u>156</u> or narcotic <u>157</u>, <u>158</u> can provide effective analgesia, facilitating early discharge after ambulatory surgery.

Epidural analgesia with a combination of low-dose local anesthetic (e.g., 0.05% to 0.1% bupivacaine) in combination with a narcotic (e.g., 2–5 ?g/mL fentanyl) provides excellent analgesia after lower extremity surgery. These can be administered as infusions (3–10 mL/h) with patient-controlled analgesia. The rates have to be adjusted to accommodate for changing pain patterns and accumulation of drugs. Nonsteroidal anti-inflammatory drugs can be used to augment the analgesia, <u>159</u>, <u>160</u> although their use is not routinely recommended. <u>161</u> Higher doses are required after knee surgery than after hip surgery. Intrathecal infusions of bupivacaine are not recommended for postoperative pain control in orthopedic procedures. <u>162</u>

Effective analgesia with epidural infusions or peripheral blockade reduces narcotic requirements, provides better analgesia, reduces catabolism, <u>163</u> and results in improved rates of rehabilitation after total knee replacement. <u>164</u>, <u>165</u> To optimize the potential advantage of the analgesia, early rehabilitation should be encouraged. <u>166</u>

There are several limitations to the use of catheter techniques for postoperative analgesia after orthopedic surgery. First, as stated earlier, their use is contingent on an effective pain service operating with the cooperation of the nurses and orthopedic surgeons. Second, these modalities must be used in conjunction with the perioperative management of thromboembolism. The risk of epidural catheters is increased when low-molecular-weight heparin is used, <u>167</u>, <u>168</u> whereas there appears to be minimal risk with aspirin or warfarin. <u>169</u> At The Hospital for Special Surgery, almost all patients undergoing total hip or knee replacement receive postoperative epidural analgesia for 24 to 72 hours postoperatively. Patients are concurrently given aspirin or warfarin (Coumadin) after surgery in combination with foot pumps, foot exercises, and early ambulation. Low-molecular-weight heparin is not used in conjunction with epidural analgesia. No clinical epidural hematomas have been noted (>2,000 cases are performed annually at The Hospital for Special Surgery).

The final complicating factor relates to persistent lower extremity nerve injury. Patients who are at risk of developing a compartment syndrome should not have epidural infusions of local anesthetic or lower extremity nerve blocks because these may mask the early diagnostic signs (excessive pain, numbness, or muscle weakness). <u>170</u>, <u>171</u> This applies particularly to patients with fractures of the tibia and fibula. <u>172</u> A related problem concerns the use of epidural analgesia in patients who are at risk of developing nerve injuries after surgery, particularly peroneal palsy after complicated total knee replacement. <u>173</u>, <u>174</u> Patients with valgus deformities and those undergoing high tibial osteotomy are at risk of developing peroneal palsy after surgery. <u>175</u> If the palsy is diagnosed early, the knee can be flexed, bandages changed, and so on to limit injury to the nerve. An epidural infusion can delay the diagnosis, increasing the risk of permanent nerve damage. In these cases, it is preferable to avoid epidural analgesia altogether to avoid confusion and focus on early detection of potential neurologic deterioration. Epidural anesthesia and analgesia can be used to remove an infected prosthesis because the risk of epidural abscess in this setting is negligible. <u>176</u>

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COMPLICATIONS OF ORTHOPEDIC SURGERY

Tourniquet Problems

Tourniquets are applied around upper or lower extremities to eliminate intraoperative bleeding and thereby provide better operative conditions. Unfortunately, the tourniquet is unphysiologic and is associated with a number of disadvantages (Table 60–7).

TABLE 60-7. Physiologic Changes Caused by Limb Tourniquets

Neurologic effects

Abolition of somatosensory evoked potentials and nerve conduction occurs within 30 minutes.

Application for more than 60 minutes causes tourniquet pain and hypertension.

Application for more than 2 hours may result in postoperative neurapraxia.

Evidence of nerve injury may occur at a skin level underlying the edge of the tourniquet.

Muscle changes

Cellular hypoxia develops within 8 minutes.

Cellular creatine declines.

Progressive cellular acidosis occurs.

Endothelial capillary leak develops after 2 hours.

Limb becomes progressively colder.

Systemic effects of tourniquet inflation

Elevation in arterial and pulmonary artery pressures develops. This is usually slight to moderate if only one limb is occluded.

Systemic effects of tourniquet release

Transient fall in core temperature186

Transient metabolic acidosis

Transient fall in central venous oxygen tension (but systemic hypoxemia unusual)

Release of acid metabolites into central circulation (e.g., thromboxane)

Transient fall in pulmonary and systemic arterial pressures

Transient increase in end-tidal CO2

Increased oxygen consumption255

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Local Effects of Tourniquet Inflation

Mitochondrial partial pressure of oxygen decreases to zero within 8 minutes of inflating a tourniquet. Anaerobic metabolism then begins. Decrease of nicotinamide adenine dinucleotide and creatine phosphate stores in muscle occurs over the next 30 to 60 minutes. <u>177</u> Cellular acidosis (pH < 6.5) rapidly ensues. <u>177</u>, <u>178</u> Hypoxia and acidosis result in the release of myoglobin, <u>179</u> intracellular enzymes, and potassium. <u>180</u> Thromboxane is released locally with disruption of endothelial integrity. <u>181</u> Tissue edema develops if tourniquets are inflated more than 60 minutes; closure of ankle incisions may then be difficult. <u>182</u> The limb loses heat and may approach room temperature with time. Injury to muscle beneath the tourniquet may delay rehabilitation. <u>183</u>

Metabolic Response to Tourniquet Release

With deflation of the tourniquet and reperfusion of the extremity, a washout of metabolic byproducts occurs. <u>184</u> A decrease in core body temperature of 0.7°C typically occurs within 90 seconds of deflation of a lower limb tourniquet, <u>185</u>, <u>186</u> and venous oxygen saturation <u>187</u> may fall 20 percent in 30 to 60 seconds. Increases in end-tidal carbon dioxide are typically noted, <u>188</u> but decreases in arterial oxygen saturation are infrequent unless significant pulmonary shunting exists. <u>189</u>

Hemodynamic Responses

The causes of hemodynamic responses are the initial inflation of the tourniquet, subsequent prolonged tourniquet inflation, and an immediate response after tourniquet deflation (Fig. 60–7). FIGURE 60–7 The hemodynamic effect of thigh tourniquet inflation (single bar) and deflation (double bar) in an 86-year-old man with hypertension undergoing total knee replacement. Note the changes in pulmonary and systemic artery pressures with both inflation and deflation of the tourniquet. The persistent elevation in pulmonary artery pressure after reinflation of the tourniquet after deflation (after third bar) may represent transient myocardial dysfunction.

Tourniquet Inflation

Inflation of the tourniquet and exsanguination of the limb result in an expansion of central venous blood volume and a theoretical rise in peripheral vascular resistance. In ordinary practice, this results in small increases in central venous or arterial pressures. <u>190</u>, <u>191</u> However, patients with extensive varicose veins or poor ventricular compliance may experience considerable increases in pulmonary artery pressure (see <u>Fig. 60–7</u>). Bilateral simultaneous tourniquet inflation of the lower extremities may result in significant elevations in central venous pressure. <u>190</u>, <u>192</u>

Tourniquet Deflation

Deflation of the tourniquet with reperfusion of the ischemic limb is frequently associated with decreases in both central venous and arterial pressures. <u>193</u> These can be profound (see Fig. 60–7) and have resulted in cardiac arrests. <u>194</u> Contributing factors include sudden reduction in peripheral vascular resistance with pooling of blood in the extremity, acute blood loss, and the circulatory effects of the ischemic metabolites (e.g., thromboxane). <u>181</u>

Prolonged Inflation

Forty-five to 60 minutes after tourniquet inflation patients under general anesthesia may develop systemic hypertension. <u>195</u> The reason for this rather consistent timing is not entirely clear, but it may reflect a critical level of cellular ischemia in the muscle or nerve. Attempts to reduce blood pressure by deepening anesthesia are not always successful, and vasodilators such as hydralazine, nifedipine, or labetalol may be necessary. <u>195</u>

Tourniquet Pain

Patients receiving spinal or epidural anesthesia may develop a poorly defined aching or burning sensation in the distal extremity about 1 hour after tourniquet inflation. <u>196</u>, <u>197</u> Attempts to relieve "tourniquet pain" with intravenous narcotics are not always successful. Tourniquet pain may, however, be relieved by deflating the

tourniquet for 10 to 15 minutes and then reinflating it. This correlates with correction of cellular acidosis. <u>198</u> Our experience has demonstrated that a complete brachial plexus block using long-acting local anesthetics is not associated with tourniquet pain for as long as 3 to 4 hours. Neither stellate ganglion nor intercostobrachial nerve blocks are effective in relieving tourniquet pain of the upper extremity. <u>199</u> These observations taken together suggest that tourniquet pain may be related in some manner to the quality or intensity of somatic neural blockade. <u>199</u> Experience with spinal anesthesia also suggests that the intensity of blockade may be more important than the anesthetic level in preventing tourniquet pain, because isobaric spinal anesthesia has a lower incidence of tourniquet pain than hyperbaric spinal anesthesia. <u>200</u>

Neurologic Consequences

Neurologic problems may occur when tourniquets are inflated for long periods (>2 hours) or excessive inflation pressures are used. A shear force is applied to nerve trunks at the edges of the tourniquet. 201 Within 30 minutes of inflating a tourniquet, nerve conduction ceases. 202, 203 This may reflect either axonal hypoxia or the direct result of extrinsic pressure on the nerves beneath the tourniquet. 204 In clinical practice, tourniquets should be deflated every 90 to 120 minutes to minimize the risk of postoperative neurapraxias. 178, 198 Alternatively, tourniquet pressure can be lowered to 250 mm Hg while maintaining systolic pressures at 90 to 100 mm Hg. This provides a pressure gradient of 150 mm Hg between the tourniquet and systolic pressure, more than enough to maintain an exsanguinated limb. Anesthesiologists who use regional anesthesia may be implicated when postoperative neurapraxias were in fact secondary to tourniquet injury. Recognition of the adverse effects of tourniquets has led many to perform surgery without a tourniquet 205, 206, 207 or to limit the duration of its use. 208

Fat Embolism

A certain degree of lung dysfunction occurs in all patients after long bone fractures, but clinically significant fat embolism syndrome as such develops in only 10 to 15 percent of these patients. Signs include hypoxia, tachycardia, mental status changes, and petechiae on the conjunctiva, axilla, or upper thorax. Fat globules in the urine are nondiagnostic, but lung infiltrates seen on chest radiograph confirm the presence of lung injury. <u>209</u>, <u>210</u>, <u>211</u>

The pathophysiology of fat embolism represents capillary endothelial breakdown causing pericapillary hemorrhagic exudates most apparent in the lungs and brain. Pulmonary edema and hypoxemia occur as a result of pulmonary exudates. Hypoxia and areas of cerebral edema may account for the variable neurologic abnormalities seen.

The more severe cases of fat embolism involve fractures of the femur and tibia. Delays in fixation of bones and extensive reaming of the medullary canals contribute to perioperative morbidity 212 and to the severity of fat embolism syndrome. Efforts to surgically correct fractures early and minimize trauma to the bone marrow lessen the degree of fat/bone marrow embolism. Patients with coexisting lung injury are at additional risk of fat embolism. Evidence suggests that fat may pass to the systemic circulation through a patent foramen ovale 71, 72 or by transpulmonary passage. 74 The chemical composition of the fat may even contribute to this process. 213 For this reason, it is preferable to minimize pulmonary artery hypertension to reduce transpulmonary passage of fat and limit pulmonary endothelial transudation of fluid.

Treatment includes early recognition, oxygen administration, and judicious fluid management. Corticosteroids in large doses shortly after major trauma have been found to minimize the clinical presentation of fat embolism but are probably not necessary in most cases if oxygen therapy is administered. With appropriate fluid management, adequate ventilation, and the prevention of hypoxemia, outcome is usually excellent.

Deep Vein Thrombosis

Deep vein thrombosis is a common problem in orthopedics, <u>214</u> and pulmonary embolism is a major cause of postoperative mortality. <u>215</u>, <u>216</u>, <u>217</u> Rates are low after upper extremity surgery, spine surgery, <u>218</u> and knee arthroscopy (3%), <u>219</u> but more common after total hip replacement (30–50%), <u>220</u>, <u>221</u>, <u>222</u> total knee replacement (40–60%), <u>164</u>, <u>223</u>, <u>224</u> and trauma to the lower extremities (20–50%). Proximal deep vein thrombosis (popliteal femoral or iliac vessels) is more common after total hip replacement (10–20%) <u>221</u>, <u>222</u>, <u>225</u> and more likely to cause pulmonary embolism.

The emboli form during surgery during periods of venous stasis in the presence of surgical injury. During total knee replacement, there is absolute stasis with the tourniquet inflated, and on release of the tourniquet, acute increases in markers of coagulation can be detected in blood <u>226</u> (Fig. 60–8). Concurrently, thromboemboli can be detected in the right side of the heart on echocardiography. <u>227</u> During total hip replacement, obstruction of the femoral vein occurs during surgery on the femur. <u>79</u>, <u>228</u>, <u>229</u> When obstruction of the vein is relieved when the hip is relocated, an increase in markers of thrombosis is noted (Fig. 60–9) and echogenic material can be seen on echocardiography. Efforts to reduce deep vein thrombosis during surgery should be directed during these phases of venous occlusion. Maneuvers include reducing the duration of surgery (surgeon's responsibility), <u>225</u> augmenting lower extremity blood flow during surgery to reduce venous stasis, <u>230</u> and finally, administering anticoagulants during this phase of surgery. <u>79</u> If 15 to 20 units/kg of unfractionated heparin is administered before surgery on the femur, fibrin formation can be suppressed and deep vein thrombosis rates reduced to 6%. <u>231</u>

FIGURE 60–8 Fibrinopeptide A in patients receiving general or epidural anesthesia. Determinations were made before the induction of anesthesia (sample 1), 20 minutes after the start of the surgical procedure (sample 2), 45 seconds after deflation of the tourniquet at the end of surgery (sample 3) and 1 hour postoperatively (sample 4). Values are mean ± SEM. Note the increase in fibrinopeptide A after deflation of the tourniquet (sample 3) representing thrombosis in the leg while the tourniquet was inflated. (From Sharrock et al226)

FIGURE 60–9 Changes in fibrinopeptide A and D-dimer in patients receiving saline (n = 12) or intravenous heparin (1000 units) (n = 11). Determinations were made before the epidural anesthetic (1), after implantation of the acetabular component—1 to 2 minutes before operating on the femur (2), 45 seconds after relocation of hip joint after the cemented femoral component had been inserted (3), and 1 hour postoperatively in the postanesthesia care unit (4). Patients received either intravenous heparin (1000 units) or saline immediately after insertion of the acetabular component (event 2). Note a suppression of fibrinopeptide A after insertion of a cemented femoral component in patients receiving heparin compared to saline (P = .006) (event 3), reflecting inhibition of thrombosis with heparin. D-dimer was not affected by heparin administration demonstrating that heparin did not suppress fibrinolysis. Values are mean ± standard error of the mean. (From Sharrock et al<u>79</u>)

Epidural or spinal anesthesia reduces deep vein thrombosis rates after total knee replacement by 20% (from 50% to 40%) <u>164</u> and after total hip replacement by approximately 40%. <u>232</u>, <u>233</u> Deep vein thrombosis rates during total hip replacement performed with epidural anesthesia can be reduced to 10% with the concurrent use of low-dose epinephrine infusions. <u>225</u>, <u>234</u> The mechanism of action of epinephrine is unknown, <u>235</u> but it does augment lower extremity blood flow during epidural anesthesia, thereby minimizing venous stasis. <u>230</u>

Postoperative epidural analgesia does not appear to provide additional benefit in reducing deep vein thrombosis rates. <u>223</u> Epidural infusion of 0.1% bupivacaine does not increase femoral venous blood flow, whereas simple flexion/extension exercises of the foot do increase extremity blood flow. <u>236</u> The benefit of epidural analgesia may be that it facilitates early ambulation, which is beneficial in prophylaxis of deep vein thrombosis.

There is a controversy about using epidural anesthesia and heparin. <u>167</u> The effects of heparin and epidural anesthesia on deep vein thrombosis prophylaxis are, in fact, additive. <u>233</u>, <u>237</u> Deep vein thrombosis rates of 33% are noted using low-molecular-weight heparin with general anesthesia, whereas rates of 19% are noted with low-molecular-weight heparin and epidural anesthesia. <u>233</u> Perioperative anticoagulants are necessary if general anesthesia is used, but the question is whether low-molecular-weight heparin is useful (or in fact dangerous) when used in conjunction with conduction anesthesia. <u>167</u>

Postoperative modalities to reduce deep vein thrombosis that can safely be used in conjunction with epidural anesthesia include pneumatic compression boots, <u>238</u> foot pumps, <u>239</u>, <u>240</u> foot exercises, <u>236</u> early ambulation, aspirin, and low-dose warfarin started the day after surgery. Anticoagulation is not generally recommended after knee arthroscopy or spine surgery. In high-risk cases, vena cava filters may be placed preoperatively.

The role of the anesthesiologist in the prevention of deep vein thrombosis will change with a recognition of the role of anesthesia in the prevention of thrombosis and a realization that the thrombi form in the operating

room rather than after surgery. Because they form during "our watch," anesthesiologists have an opportunity and a responsibility to prevent thrombi from forming. Copyright © 2000, 1995, 1990, 1985, 1979 by Churchill Livingstone