Lesson 256: Preanesthetic Assessment of the Patient With Rheumatoid Arthritis

WRITTEN BY:
Deepa Reddy* and Amir Baluch, MD†
* Medical student, Tulane University School of Medicine, New Orleans, Louisiana
† Anesthesia resident, University of Miami Leonard M. Miller School of Medicine, Miami, Florida

REVIEWED BY:
Alan D. Kaye, MD, PhD, DABPM
Professor and Chairman, Department of Anesthesiology
Louisiana State University Health Sciences Center School of Medicine in New Orleans

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NEEDS STATEMENT
Rheumatoid arthritis (RA), a chronic and systemic inflammatory disease of unknown etiology, affects primarily the joints, usually in a symmetric pattern. Without treatment, RA may lead to destruction and deformity of the joints, after which surgical correction is often undertaken. Knowledge of RA has been identified by questionnaire as required information for anesthesiologists.

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TARGET AUDIENCE
Anesthesiologists

LEARNING OBJECTIVES
At the end of this activity, the participant should be able to:
1. Describe the epidemiology of RA.
2. Identify causative factors.
3. Explain the pathogenesis of RA.
4. Outline genetic factors that are associated with RA.
5. Describe the clinical manifestations of RA.
6. List the diagnostic criteria for RA.
7. Discuss the preoperative anesthetic evaluation of patients with RA.
8. Outline possible complications of RA.
9. Identify current therapies for RA.
10. Review the options for dealing with a difficult airway in a patient with RA.

CASE HISTORY
A 36-year-old woman, with a height of 5 ft and a weight of 47 kg, presented for left rotator cuff surgery. She had been treated for RA for 18 years and was taking medications that included celecoxib, acetaminophen, and infliximab. Her medical history included gastrointestinal bleeding 1 year previously resulting from the use of indomethacin; because of the bleeding, she had required a transfusion of 3 units of packed red blood cells. She had received several courses of steroids and had undergone a knee replacement and right shoulder surgery. On physical examination, the patient appeared emaciated and withdrawn, and radial deviation was noted at the wrists. Her hemoglobin level and hematocrit were 9.7 g/dL and 29.5%, respectively. A radiologic study of the cervical spine indicated atlanto-occipital instability. An 18-gauge catheter was inserted into the right external jugular vein after multiple unsuccessful attempts at placement of a peripheral cannula in the arms and feet.

Rheumatoid arthritis (RA) is characterized by acute and chronic systemic inflammation that involves primarily the joints but may also affect many other tissues and organs—including the blood vessels, heart, skin, lungs, and muscles. The severity and onset of the disease, which is usually insidious, are variable. The patient with RA initially presents with fatigue, musculoskeletal pain, and stiffness; only after a period of weeks to months does the condition progress to involvement of the joints.

The small joints are generally affected first—particularly the small bones of the hands. Later, the larger joints are affected, and as the disease progresses, the larger joints are also involved. The disease affects patients differently. In some, the onset is insidious; in others, the onset is more acute. The disease is usually symmetrical; however, in 10% of patients, the disease is asymmetrical.

The disease affects patients differently. In some, the onset of RA is mild, with no severe symptoms, whereas in others, disability progresses throughout life. Current treatment strategies focus on alleviating active inflammation because no curative treatment exists. The aim of treatment is to limit joint destruction. Other conditions that affect individuals with RA include infections, renal impairment, lymphomas, and cardiovascular disease. Lethal effects of therapy may also play a role in the complications that arise with RA, associated with the long-term use of nonsteroidal anti-inflammatory drugs and steroids. Although the mortality rate is low for RA alone, it can double as a result of complications. The life span of patients may be decreased by 7 to 10 years.

Epidemiology
RA affects about 1% of the world’s population. No population is immune from the disease, but women are affected 2 to 3 times as often as men. The peak age at onset is between 30 and 55 years. The annual incidence of RA is 30 cases per 100,000 people. Because women are affected more frequently than men, the prevalence of RA in women older than 65 years of age is around 5%. Risk factors for the development of RA include smoking, obesity, concurrent infections, advancing age, female gender, and certain genetic features, whereas the use of oral contraceptives and some dietary constituents may be protective.

PREANESTHETIC ASSESSMENT
Dr. Elizabeth A.M. Frost, who is editor of this continuing medical education series, is Clinical Professor of Anesthesiology at The Mount Sinai School of Medicine in New York City. She is the author of Clinical Anesthesia in Neurosurgery (Butterworth-Heinemann, Boston) and numerous articles. Dr. Frost is past president of the Anesthesia History Association and former editor of the journal of the New York State Society of Anesthesiologists, Sphere. She is also editor of the book series based on this CME program, Preanesthetic Assessment, Volumes 1 through 6 (McMahan Publishing, New York City).

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Rheumatoid arthritis is initially a clinical diagnosis after recognition of several of the following features:

- Morning stiffness ≥2 h and present for ≥6 wk
- Swelling of ≥3 joints for ≥6 wk
- Swelling of carpometacarpal, metacarpophalangeal, or proximal interphalangeal joints for ≥6 wk
- Symmetric joint swelling
- Hand X-ray changes typical of rheumatoid arthritis; include erosions and unequivocal bony decalcification
- Rheumatoid subcutaneous nodules
- Positivity for rheumatoid factor

Table 1. Clinical Features Of Rheumatoid Arthritis

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<th>Lesson 256 continued from page 47</th>
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**Etiology**

RA is a common disorder that has plagued people for centuries. Many possible causative agents have been identified, yet the etiology of the disease remains unknown. A reason that women are affected by RA more often than men may be related to the effects of estrogen on the immune system via a suppressor T-cell pathway. Genetic components to RA are believed to exist. Supporting evidence includes an increased incidence of the disease in individuals carrying an epitope in the third hypervariable region of human leukocyte antigen (HLA)-DRB1 genes. Although the etiology of RA remains uncertain, HLA-DRB1 typing may help predict relative risk, severity of disease, and response to therapy.

Serum rheumatoid factor, an immunoglobulin with anti-immunoglobulin G (IgG) Fc (crystalizable fragment) specificity, is another genetic component that can be found in patients with RA. The presence of rheumatoid factor is highly characteristic of RA but not specific, so clinicians do not rely solely on this finding for the diagnosis.

Studies of twins have also suggested that a hereditary component is involved in RA because the incidence of RA is higher in monozygotic twins than in dizygotic twins. The relationship holds true for first-degree relatives; their risk for the development of RA is 1.5 times greater than that of the general population. Also significant is the correlation between RA and other diseases believed to have an autoimmune pathogenesis. The alleles associated with RA overlap with those associated with lupus erythematosus, inflammatory bowel disease, multiple sclerosis, and ankylosing spondylitis.

Environmental factors, such as smoking, have been implicated in causation. Some studies have suggested that cigarette smoking enhances the development of RA. Cigarette smoking is also associated with more severe forms of the disease, such that smokers with a history of at least 25 pack-years are more likely to be seropositive, have nodules, or have erosions that are radiographically apparent.

Infection is another environmental factor that may explain the etiology of RA. One of the prime suspects as a microbial trigger of RA is the Epstein-Barr virus (EBV). In 1975, an antibody in the sera of patients with RA was shown to react with an EBV nuclear antigen. Since then, further findings have linked RA and EBV infection. For example, EBV is a polyclonal activator of B lymphocytes, including those that express rheumatoid factor. Other agents that have been suggested in the development of RA include Mycoplasma, Proteus mirabilis, parvoviruses, and retroviruses. Further studies may elucidate the exact role these microorganisms play in the etiology of RA.

**Pathogenesis**

Although much about the etiology of RA seems uncertain, it is believed that RA is initiated by an arthritogenic microbial antigen acting on an immunogenetically susceptible host. The origin of the arthritogenic antigen is currently unknown, yet it appears to be able to cause disease by activating an immune response. Once the host is attacked, helper CD4+ T cells are activated, which then release local inflammatory mediators and cytokines. Endothelial cells of synovial capillaries are triggered, with the expression of intercellular adhesion molecule-1. Other inflammatory cells migrate and attach to affected joints. As CD4+ T cells are activated, B cells are also triggered, which results in the production of antibodies in affected joints.

In 80% of patients with RA, autoantibodies to the Fc portion of autologous IgG (also known as rheumatoid factors) are formed. However, these factors are not diagnostic for RA because they may not be identified in all patients and can be found in other disease states. The circulating immune complexes are mostly localized within the inflamed cartilage, activating complement and enhancing the synovial inflammatory reaction. The synovium in RA organizes into invasive tissue that, if left untreated, can degrade cartilage and bone and eventually destroy the joint.

Clinical Manifestations

The clinical picture of RA varies, although most patients present initially with a slow and insidious onset of disease. The symptoms that predominate during the preliminary phase are musculoskeletal pain, stiffness, and swelling in many joints. Sites of early arthritis are typically localized in the metacarpophalangeal and proximal interphalangeal joints of the fingers, interphalangeal joints of the thumbs, the wrists, and the metatarsophalangeal joints of the toes. Other areas that may be initially affected include the joints of the upper and lower limbs, such as elbows, shoulders, ankles, and knees. Uncommonly, the upper spine may be involved, but the lumbar sacral region and hips are usually spared. Morning stiffness is typical. In about 10% of individu-als with RA, the onset of disease is acute, and severe symp-toms and polyarticular involvement develop within a few days. The typical clinical picture is one of progressive joint involvement over a period of months to years, with initially minimal limitation of motion that over time becomes more severe. The course of RA may be fast or slow and fluctuates over a period of years, with the most damage occurring during the first fifth of patients have periods of complete or partial remission.

The radiographic hallmarks of RA are joint effusions and juxta-articular osteopenia with erosions, narrowing of the joint space, and loss of articular cartilage. With the continued destruction of cartilage, ligaments, tendons, and joint capsules, characteristic deformities are observed on imaging, including radial deviation of the wrist, ulnar deviation of the fingers, and flexion–hyperextension abnormalities of the fingers. However, extension contraction occurs over time, and the severity of the erosions may reach a level beyond which further progression of the RA cannot be assessed radiographically.

**Diagnosis**

Laboratory tests for a definitive diagnosis of RA do not exist, but genetic tests for susceptibility are available. There is no guarantee that the disease will develop in patients with these genetic factors, but their risk is much greater than that of the general population. The majority of individuals who have RA are positive for both HLA-DR, and rheumatoid factor—major determinants for the disease but not solely reliable for diagnosis. Synovial fluid in the inflamed joints can also be analyzed for leukocytosis of neutrophils or lymphocytes, low levels of glucose and complement, and protein levels approaching those in plasma. Whereas the test is nonspecific for RA, it may be used to confirm the presence of inflammatory arthritis; clinical features aid in the confirmatory diagnosis.

**Current and New Therapies**

Early treatment is essential to maintain joint function. The longer that active disease persists, the less likely it is that the patient will respond to therapy. Evidence has shown that early treatment can control synovitis and may slow or even stop progression of the disease based on radiographic findings. The goals of treatment include controlling the signs and symptoms of RA, restoring physical function to joints, and preventing joint damage. If joint damage has already occurred, then the goal is to delay or halt progression of the disease. However, despite all the therapies currently available, it may not be possible to achieve complete remission.

**Pharmacologic Therapy**

A mainstay of therapy is the administration of drugs to induce remission and prevent further loss of joint tissues or function in daily activities. Physicians can manage patients by adjusting dosages, and offering different combinations of drugs. There are 5 main drug classes for the treatment of patients with RA: analgesics, nonsteroidal anti-inflammatory drugs (NSAIDs), glucocorticoids, disease-modifying antirheumatic drugs (DMARDs), and anticytokines (Table 2). Analgesics relieve the pain of mild to moderate arthritis. Included in this class are acetaminophen, tramadol, capsaicin, and opioids. Because these drugs do not exhibit any
anti-inflammatory properties, they are usually combined with agents from other classes.

NSAIDs have both analgesic and anti-inflammatory properties, but they do not change disease outcomes. The drugs in this class include ibuprofen, aspirin, naproxen, and indomethacin. They inhibit cyclooxygenase (COX–1) and COX-2, which block the synthesis of prostaglandin. NSAIDs, useful for treating the symptoms of RA, do not prevent progression of the disease. Although NSAIDs are essential to the treatment of RA, they can cause severe gastrointestinal distress and ulcers. Selective COX-2 inhibitors are just as useful in the treatment of RA, and they cause less severe adverse gastrointestinal side effects. Such drugs, including celecoxib and valdecoxib, are similar to NSAIDs except that they do not have the same corrosive effects on the gastrointestinal lining. However, before administering these drugs, the clinician must assess the patient’s medical history to ascertain that significant cardiovascular disease is not present.

COX-2 inhibitors are associated with the reduced production of prostaglandin I2 by vascular endothelium, with little or no inhibition of potentially thrombotic platelet thrombolysin A2. This sequence predisposes the patient to endotheial injury, which can increase ischemic cardiovascular events. Because there is still uncertainty about the safety of COX-2 inhibitors, the decision to administer them should be made on a case-by-case basis.

The most commonly used glucocorticoids are prednisone and prednisolone. Glucocorticoids may be administered orally, intravenously, or by intra-articular injection. Compared with NSAIDs and analgesics, glucocorticoids have much greater therapeutic effects on joint pain, but they are associated with many side effects, including adrenal suppression, ulcers, and osteoporosis. The decision of whether to initiate therapy with glucocorticoids should be based on an assessment of the patient’s other medical conditions that might increase the risk for glucocorticoid toxicity. These include established hypertension or diabetes, pre-existing cataracts or glaucoma, and risk factors for osteoporosis. Risk–benefit ratios should be clearly explained to the patient, and medical alert bracelets obtained. Finally, all patients should be counseled on smoking cessation and cholesterol reduction to curtail cardiovascular risk factors.

The DMARDs encompass a large group of antirheumatic drugs that reduce the progression of joint erosion. They have a slow onset of action and no analgesic activity. The DMARDs include gold compounds, penicillamine, hydroxychloroquine, cyclophosphamide, and methotrexate. The pharmacologic effects of these drugs are probably related to the reduction of phagocytosis and immune responses. The mechanism of action of anticytokine agents is still unknown, but it is thought that they act by decreasing the inflammatory response in affected joints. Examples are anti–tumor necrosis factor (TNF–α) agents, such as etanercept, infliximab, and adalimumab, and the interleukin-1 receptor antagonist anakinra. Case reports have noted that the anti–TNF–α agents may induce leukocytoclastic vasculitis and even neurologic manifestations, which resolve after administration is discontinued. These drugs are newer treatments, and in the future, additional biologic therapies will probably become available.

All the previously mentioned drugs useful in the treatment of RA are more effective when administered in combination. The choice of a treatment regimen is determined by the severity of disease, the adverse effects of treatment, and patient convenience and preference.

**Clinically Relevant Complications**

Systemic complications occur in patients with RA. For example, cervical joint destruction may cause atlantoaxial subluxation. Although it is rare, the anesthesiologist must be aware of this potential condition. Of all the joints in the spine, the atlantoaxial (C1-2) joint is the one most prone to subluxation. The atlas (C1 vertebra) can move anteriorly, posteriorly, vertically, or laterally, relative to the axis (C2 vertebra). Abnormal anterior movement of the atlas is the most common type of subluxation; the least common is vertical movement of the atlas in relation to the axis.27–31 (Intervertebral joints in the cervical spine become involved when the inflammatory process extends from adjacent neurocentral joints, and chronic cervical instability is initiated by apophyseal joint destruction.31 Usually, the onset and severity of cervical spinal disease are related to the progression of peripheral joint destruction—especially in individuals with hand, feet, hip, and knee erosions.32,33

The consequences of atlantoaxial subluxation include pain that radiates superiorly toward the occiput, slowly progressive spastic quadriaparesis, sensory abnormalities, and transient episodes of medullary dysfunction.34,35 These problems can cause changes in the level of consciousness, “drop” attacks, a sensation of the head falling forward on flexion of the cervical spine, loss of sphincter control, respiratory dysfunction, dysphagia, vertigo, convulsions, hemiplegia, dysarthria, nystagmus, and peripheral paresthesias without evidence of peripheral nerve disease or compression. In the presence of such neurologic signs, the origin of symptoms should be explored and appropriate treatment of the underlying condition established.

Treatment for cervical subluxation is essential and based on the degree of spinal cord compression. Usually, patients with subluxation without signs of spinal cord compression are offered medical therapy; patients with signs of spinal cord compression require surgical intervention. In patients with RA, medical therapy for cervical subluxation mostly consists of the prescription of stiff collars for stability. Benefits of this treatment are the prevention of severe injury or death associated with small falls, whiplash injuries, and intubation.36 Fiberoptic intubation with the collar in place may be required to prevent further cervical injury. Surgical therapy—usually C1-2 fusion—may prevent superior migration of the odontoid and decrease the risk for further cervical spine instability.36

Progressive RA may be complicated by cardiac involvement, including pericarditis, myocarditis, and atherosclerosis. The incidence of pericarditis associated with RA varies greatly. Most patients with RA who have pericarditis are

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**Table 2. Medications Used To Treat Rheumatoid Arthritis**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Mechanism of Action</th>
<th>Side Effects and Precautions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acetaminophen</td>
<td>Reversibly inhibits COX, mostly in the CNS</td>
<td>Hepatotoxicity at high doses</td>
</tr>
<tr>
<td>Aspirin</td>
<td>Irreversibly inhibits COX-1 and COX-2; blocks prostagland synthesis</td>
<td>Gastric upset, gastric and duodenal ulcers, hematemesis, Reye’s syndrome</td>
</tr>
<tr>
<td>Azathioprine</td>
<td>Interferes with DNA synthesis</td>
<td>Contraindicated in pregnancy: bone marrow suppression, anemia, skin rash, fever, diarrhea, nausea</td>
</tr>
<tr>
<td>Celecoxib</td>
<td>Selective reversible inhibitor of COX-2</td>
<td>Renal toxicity, causes less GI irritation than NSAIDs; possible risk for cardiovascular events</td>
</tr>
<tr>
<td>Gold salts</td>
<td>Alter the morphology and functional capabilities of macrophages; may also alter lysosomal enzyme activity</td>
<td>Dermatitis, stomatitis; may be toxic to kidneys</td>
</tr>
<tr>
<td>Ibuprofen</td>
<td>Reversible inhibitor of COX-1 and COX-2; blocks prostagland synthesis</td>
<td>Renal damage, aplastic anemia, GI distress, ulcers</td>
</tr>
<tr>
<td>Infliximab</td>
<td>Neutralizes the biologic activity of TNF-α</td>
<td>Increased incidence of lymphoma</td>
</tr>
<tr>
<td>Methotrexate</td>
<td>Unknown</td>
<td>Contraindicated in pregnancy: ulcerative stomatitis, leukopenia, nausea, abdominal distress</td>
</tr>
<tr>
<td>Prednisone</td>
<td>Decreases production of leukotrophins and prostaglandins by inhibiting phospholipase A2 and expression of COX-2</td>
<td>Iatrogenic Cushing’s syndrome</td>
</tr>
</tbody>
</table>

CNS, central nervous system; COX, cyclooxygenase; GI, gastrointestinal; NSAIDs, nonsteroidal anti-inflammatory drugs; TNF, tumor necrosis factor

Adapted and modified from reference 26.
### Table 3. Suggested Corticosteroid Regimen

Supplemental steroid administration is often used for patients who have received steroids previously. One proposed scheme is presented below.

<table>
<thead>
<tr>
<th>Day of surgery</th>
<th>Post-op days 1-3</th>
<th>Post-op day 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Give hydrocortisone 100 mg intramuscularly and every 6 h thereafter; add maintenance dose, if any.</td>
<td>Follow same regimen as on day of surgery</td>
<td>Discontinue hydrocortisone treatment; continue maintenance dose, if any.</td>
</tr>
</tbody>
</table>

### Table 4. Symptoms of Adrenal Insufficiency

There are many nonspecific symptoms of adrenal insufficiency. The presence of several of these indicators may imply—but not necessarily confirm—adrenal disease.

<table>
<thead>
<tr>
<th>Abdominal pain</th>
<th>Anaesthesia</th>
<th>Confusion</th>
<th>Fatigue</th>
<th>Headache</th>
<th>Hypertension</th>
<th>Restlessness</th>
<th>Tachycardia</th>
<th>Vomiting</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anorexia</td>
<td>Darkening skin</td>
<td>Fever/chills</td>
<td>Hyperpyrexia</td>
<td>Nausea</td>
<td>Sweating</td>
<td>Tachypnoea</td>
<td>Weakness</td>
<td></td>
</tr>
</tbody>
</table>

### Anesthetic Considerations and Management

#### Preoperative Evaluation

**Systemic Effects.** The systemic effects of RA must be evaluated. Malnutrition, poor wound healing, and anemia are often present secondary to pain and the ingestion of multiple drugs. Because NSAIDs may damage hepatic tissue and present as hypoalbuminemia, the cause of anemia, if found, should be explored. Hypovolemia and hyperproteinemia may also be present.

The leading cause of death in patients with RA is cardiovascular disease, so the cardiac system must be examined for pericardial thickening, effusions, arteritis, cardiac valve fibrosis, and rheumatoid nodules in the cardiac conduction system. Up to 45% of patients with pericardial involvement may be asymptomatic. Obtaining an echocardiogram may be helpful if clinically indicated. A suspicion of cardiac tamponade may warrant such a study, along with the possible placement of a pericardial window.

Long-standing RA can compromise the respiratory system. Pulmonary hypertension may be present secondary to an underlying pulmonary vasculitis. Pleural disease, lung nodules, interstitial pulmonary fibrosis, and obliterator bronchiolitis may coexist. Chest radiography, pulmonary function tests to indicate restrictive changes with decreased lung volumes and vital capacity, and assessment for ventilation–perfusion mismatch are tools used to diagnose underlying pulmonary pathology.

Amyloidosis or vasculitis can impair renal function. Vasculitis may damage hepatic tissue and present as hypoalbuminemia with elevated levels of hepatic transaminases. The hepatoxic effects of NSAIDs include an increased level of alkaline phosphatase. Immunosuppressant medications, such as penicillamine, methotrexate, and azathioprine may retard wound healing. The perioperative supplementation of steroids may be indicated (Table 3). General symptoms of adrenal insufficiency should be investigated, although these are nonspecific symptoms that present in a variety of diseases (Table 4).

The 14-point checklist recommended by Eisele should be used before surgery to evaluate patients with RA (Table 5). If it review of systems and the physical and laboratory findings indicate the presence of RA-induced pericarditis, myocarditis, valve abnormalities, or atherosclerosis, echocardiography may be required. A review of the patient’s recent anesthetic experience may uncover complications or reveal a successful technique.

Although a patient’s medical history may not be predictive of difficulties in administering general anesthesia, a complicated anesthetic course may influence the choice of the current strategy (ie, between regional and general anesthesia). Although a patient’s medical history may not be predictive of difficulties in administering general anesthesia, a complicated anesthetic course may influence the choice of the current strategy (ie, between regional and general anesthesia).
subluxation below C2, may also predispose to spinal cord compression on extension. Deviation, angulation, and displacement of the larynx have been recorded. The placement of airway equipment, such as supraglottic ventilatory devices, may be difficult. The recommended "sniffing" position during intubation is best avoided to decrease cervical complications; a neutral position is better tolerated. Fiber-optic intubation is often the preferred technique.

Routine perioperative radiography of the larynx is recommended by some clinicians because of the chance of cervical spine instability—even in the absence of neurologic symptoms—others believe that performing such imaging studies in all RA patients is expensive and unnecessary. The need should be determined on a case-by-case basis. The presence of symptoms such as pain radiating to the occiput, paresthesias in the shoulders or arms with head movement, or sensory loss in the hands without pain may warrant radiography. In addition, radiographs may be obtained in high-risk patients, such as the elderly, those with long-standing disease, cervical symptoms, erosive disease, or subcutaneous nodules; general anesthetic cases; and cases in which a nonsupine position is necessary.

An approach to the airway can be maintained without excessive neck extension by using a supraglottic ventilatory device or a face mask plus oral airway, but this is not always easily performed. The insertion of an airway may be difficult in some patients. Laryngeal deviation may cause difficulties when conventional methods of laryngeal mask placement are used, although the incidence of deviation tends to be low (15 among 710 fiber-optic intubations in a study by Wattenmaker and colleagues).

Intraoperative Management

General Care. Patients with RA generally undergo surgery because of joint pain or decreased functionality of joints that are deformed and unresponsive to medication: hence, many procedures are orthopedic. Current trends in anesthesia point to the use of regional anesthesia instead of general anesthesia whenever possible. Potential complications from airway manipulation, intubation, and ventilatory agitation are thus avoided. Besides the safety considerations, a regional approach provides excellent surgical conditions, successfully blunts the neurohumoral stress response during surgery, and allows good control of postoperative pain.

Positioning. Movement, positioning, and neurovascular compression are special challenges with these patients. Moderate amounts of sedation are often used to help the patient tolerate maintenance of positioning. Generous padding is used during general anesthesia to prevent neurovascular compression. The awake patient is able to attain a comfortable position unaided. Sudden movements of the neck and torso are not well tolerated and should be avoided; spines are brittle, especially in patients with ankylosing spondylitis. The use of epidors to control postoperative pain may be necessary because joint stiffness and pain increase with immobilization.

Temperature Regulation and Invasive Monitoring. There may be a subpopulation of patients with RA in whom Raynaud’s phenomena occur. Temperature regulation is always important, but in this group of patients, the prevention of hypothermia is vital because cold can precipitate vasospasm of the digital arteries, resulting in cyanosis and ischemic episodes.

When patients with RA undergo orthopedic procedures, invasive monitoring provides beat-to-beat information—which is essential if deliberate hypotension is maintained. The prompt recognition of embolism (by a sudden decrease in end-tidal CO₂, tachycardia, and hypotension), as well as cement reactions, allows early treatment of these conditions by alerting the surgeon, and with the infusion of fluids and administration of vasopressors. Methylprednisolone can embolize and cause a sudden decrease in vascular resistance due to the formation of metabolites. Hammering on joints during surgical procedures may dislodge fat emboli. Monitoring with a central venous catheter may be advised during high-risk cases, such as complicated joint revisions and hip or bilateral joint replacements in which long-stem prostheses are used.

Decreased joint mobility or distortion may create a challenge when an arterial monitor is placed. A more proximal cannulation of the wrist, or the use of a different location, such as the foot, may be necessary. Cannulation of the internal jugular vein may be complicated by limited rotation or flexion. The neck and the risk for subluxation of cervical vertebrae. The use of ultrasound techniques to locate veins can expedite the process.

Blood Conservation. Chronic anemia with a low level of erythropoietin is a common finding; patients may benefit from erythropoietin and iron supplementation. Deliberate hypotension, cell salvage techniques, and tourniquet control (if feasible) may be applied. In addition, extensive surgery may require blood replacement. Because these patients are already at risk for bleeding, the anesthesiologist should be aware of the importance of securing blood products preoperatively.

Postoperative Pain Control

Optimal outcomes with a higher level of patient satisfaction are a direct result of the active involvement of anesthesiologists in postoperative care—especially pain control. Regional anesthesia provides a smooth transition into the postoperative period and avoids long-acting blocks for painful surgeries. Continuous or demand analgesia via epidural, brachial plexus, or I.V. catheters is available. A study by DeWeese et al demonstrated more effective pain control with the use of patient-controlled epidural analgesia than with a pain control infusion pump. Patients who had an infusion pump used significantly more acetaminophen, propoxyphene naprosyate, or ketorolac after the pump was removed. Also, the infusion pump group experienced prolonged wound drainage, which is associated with a higher risk for infection.

Management of the Case Presented

The patient refused the options for regional anesthesia, such as an interscalene or supravacular block. Standard monitors (including a brain function monitor) and supplemental oxygen were placed. Premedication included metoclopramide and fentanyl. A nebulized lidocaine preparation was delivered preoperatively over 15 minutes. A total of 30 mg of dexamethasone, 50 mcg of fentanyl, and 1 mg of midazolam was administered. In addition, 100 mg of I.V. hydrocortisone was administered preoperatively. A transtracheal block was performed with 4% lidocaine. A phenylephrine-lidocaine topical preparation was administered via the nasopharynx. Fiber-optic intubation via the nasopharynx was used to insert a 6.0-mm endotracheal tube. A combination of oxygen, nitrous oxide, and desflurane was delivered to maintain anesthesia. The rotator cuff was repaired with less than 100 mL of blood loss. A total of 650 mL of Ringer’s lactate solution was administered through an 18-gauge cannula. After tracheal extubation, patient-controlled analgesia with hydromorphone was given in the postanesthesia care unit. The patient was discharged to home the following day.

Summary

RA, a devastating and debilitating systemic disease, is common and can present in many forms. The potential side effects of drug treatment should be appreciated, including the risk for bleeding from NSAIDs. Patient management and surgical intervention for complications of the disease vary dramatically. The anesthesiologist must be aware of airway pathologies, pain management techniques, and available pharmacologic therapies.

References


Table 5. Preoperative Checklist

The following 14-point list can assist with the preoperative evaluation of the patient with rheumatoid arthritis.

1. Recent general anesthesia
2. History of drug therapy
3. Jaw/neck mobility
4. Laryngoscopy (indirect)
5. Chest X-ray
6. Skeletal X-ray
7. Pulmonary function tests
8. Blood gas analysis
9. ECG
10. Hemoglobin/ESR
11. WBCs/platelets
12. Urinalysis
13. Occult blood in stool
14. Creatinine clearance

ECG, electrocardiography; ESR, erythrocyte sedimentation rate; WBCs, white blood cells

Adapted and modified from reference 52.
Lesson 256 continued from page 51

52. Eisele JH. Connective tissue disease. In: Benumof JL, ed. Anesthesia and Uncommon Dis-

The submission of post-tests by mail is no longer possible. By completing the post-test online, you obtain instant CME credit. Read the entire lesson online and take the post-test as you proceed, or read the lesson in Anesthesiology News and then take the online test.