Bone is living, growing tissue that constantly changes through a process called remodeling. This process has 2 stages: resorption and formation. During resorption, specialized cells called osteoclasts break down and remove old bone tissue. Osteoblasts then form new bone tissue to replace the old.

Bone

Osseous, or bone, tissue consists of protein fibers called collagen embedded in a matrix of intercellular liquid hardened by deposits of calcium and phosphate salts. Within and around the matrix, 3 types of bone cells build, maintain, and remodel bone. These include osteoblasts, or immature bone cells, which produce the bone matrix; osteocytes, or mature bone cells, which serve to maintain the matrix; and osteoclasts which break down and remove bone tissue (Fig.).

Bone grafts

A bone graft grows and repairs a defect according to 3 different processes. The first is osteogenesis, or the formation of new bone by living cells within the graft such as osteoblasts. The second, osteoinduction, is a chemical process in which protein molecules within the graft recruit and stimulate the patient’s undifferentiated cells to become osteoblasts. Lastly, osteoconduction is the process by which the matrix of the graft serves as a scaffold to maintain space so the recipient’s cells can generate new bone tissue. Bone grafts thus provide a structure for bone to grow, but then slowly dissolve, leaving behind only the new bone.
**Autografts**

An autograft, or bone obtained from a patient’s own body, is considered the gold standard in bone grafting procedures largely because native bone is osteogenic as well as osteoinductive and osteoconductive. It is also non-immunogenic, which means it is compatible with the patient’s own tissues and the body will not attack or reject it. As all bone requires an adequate blood supply, depending on the graft size and transplant site, a section of the periosteum (the thin layer of connective tissue that covers the bone) and its accompanying blood vessels may be included with the autograft and reattached at the site to ensure its blood supply.

There are additionally 2 types of bone that can be used for an autograft: cancellous or soft bone and cortical or hard bone. Compared to cortical bone, cancellous bone has greater surface area and is much more porous, allowing cells to infiltrate and blood vessels to form. It thus has more bone-forming potential than cortical bone, but cortical bone, being harder, can provide immediate structural support for new bone. Common sources of cancellous autograft bone are the iliac crest (upper portion of each side of the pelvis), upper tibia (shinbone), and the radius (wrist). Cortical autografts can be harvested from the diaphysis, or shaft, of the fibula (outer bone of the lower leg) and used to reinforce reconstructions, such as for spinal injuries or to replace segments where bone has been lost due to a trauma or tumor removal.

While autografts are the preferred material, harvesting bone from the patient’s own body necessitates additional surgical time and blood loss, and the amount of bone that can be harvested is always limited. The chief drawbacks to an autograft, however, are potential complications at the harvest site, such as infection and possible nerve injury.

**Allografts and synthetic materials**

A graft obtained from donor (cadaveric) bone is called an allograft. After being harvested, the bone tissue is tested for disease, cleansed, frozen, and stored in tissue banks. Allografts are not osteogenic, but they are able to stimulate cells to become osteoblasts and to provide a scaffold for bone growth. Unlike autografts, they do not require the patient to undergo an additional surgery; this reduces the risk of infection and precludes pain and loss of function at a second surgical incision site.

A variety of natural and synthetic replacement materials can also serve as substitutes for natural bone or can be mixed with either allograft or autograft tissue as bone extenders. These include ceramics like calcium phosphates, bioglass, and calcium sulphate. All of these materials are biologically active to some degree. For example, allograft bone that has been treated with a strong acid to remove the inorganic mineral deposits, known as demineralized bone matrix, possesses some osteoinductive properties. Additionally, coral, which has a biochemical and physical structure similar to bone, can act as a scaffold for new bone. Natural substances, such as bone morphogenetic proteins (BMPs), which contain growth factors, can be added to these synthetic materials to enhance their biological activity.

Allografts, either alone or mixed with extenders or enhancers, are often used in pelvic, knee, and femur (thighbone) reconstructions, but, ultimately, the type of bone graft used depends on the site and the exact nature of the injury being repaired. Since allograft tissue is not the patient’s own, the bone quality may vary. Moreover, the graft may take longer to incorporate with the patient’s native bone than an autograft. There is also a greater risk of reabsorption of the graft as well as the possibility of immune response complications, though taking anti-rejection drugs helps diminish this. Likewise, advanced testing and cleansing methods have greatly reduced the risk of transferring a disease along with an allograft.

**Bone grafting procedures**

During a bone grafting procedure, the orthopaedic surgeon will either place the graft material directly into the bone defect or lay it across to bridge the area to be fused. In some instances, the bone graft is held in place with pins, plates, or screws. When additional stability or protection is needed during the healing process, a splint, cast, or brace can be applied.

Recovery from a bone grafting procedure can take from 2 weeks to more than a year, depending on the size of the defect and the condition of the surrounding bone at the time of the surgery. More severe cases take longer and can require follow-up surgery.

**Outcomes**

Certain behaviors and conditions can affect the outcome of a graft. For instance, smoking can diminish outcomes because the carbon monoxide in cigarettes reduces local blood flow, decreasing osteoblast formation and bone metabolism at the graft site. Diabetes mellitus, which can cause peripheral nerve and vascular problems, may negatively impact fracture healing and bone grafting. Deficiencies in dietary calcium and vitamin D impair bone metabolism while metabolic conditions, such as thyroid problems and low growth hormone levels, have been associated with high rates of nonunions (fractures that fail to heal). Additionally, some drugs, such as nonsteroidal anti-inflammatory medications and corticosteroids, can interfere with bone healing. By inhibiting osteoclasts, bisphosphonates used to treat osteoporosis (low bone density) can decrease the rate of bone remodeling. Overall, bone grafting is highly successful in patients who do not smoke and follow their surgeon’s instructions when it comes to medications and activity modification.

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Staying Active with Exercise-Induced Asthma

Exercise-induced asthma (EIA), or more accurately, exercise-induced bronchoconstriction, is a narrowing of the airways of the lungs triggered by vigorous physical activity. EIA constitutes a form of respiratory difficulty in which the airways become hypersensitive and inflamed. The muscles of these airways then contract or spasm; this reaction manifests as the symptoms of asthma.

Exercise-induced asthma symptoms include:

- Wheezing
- Coughing
- Shortness of breath during or after exercise
- Chest tightness or pain
- Unusual fatigue while exercising
- Gastrointestinal discomfort

Pollutants and other airborne irritants can worsen symptoms in affected individuals. Symptoms generally occur during the first 5 to 20 minutes of exercise, or in the 5 to 10 minutes after exercise, and can last up to 30 minutes following a workout.

Who is at risk?

According to the American Academy of Allergy, Asthma and Immunology, EIA affects 12 to 15% of the population. Most people who suffer from chronic asthma also experience symptoms when exercising. EIA often begins in childhood and typically occurs in athletes who participate in sports with an aerobic component, such as running, soccer, and rowing. Athletes who play winter sports, particularly hockey or skiing, are also more susceptible to EIA due to the effects of exercising in a cold environment. Many professional and Olympic athletes, including former British footballer (soccer player) David Beckham and women’s marathon world-record holder Paula Radcliffe, have been known to suffer from EIA.

The respiratory tract

The upper respiratory tract, including the nose, nasal passages, sinuses, mouth, and pharynx (throat), is primarily involved in taking in air to breathe, then warming, moistening, and filtering it. The lower respiratory tract begins just below the vocal cords with the trachea (windpipe) which, as it descends toward the lungs, branches off into 2 main bronchi (bronchus, singular) or airway tubes. Within the lobes of the lungs, the bronchi branch into smaller airways, called bronchioles, which divide into ever-narrower branches. These terminate in the alveoli or tiny membranous air sacs rich in capillaries (small blood vessels) where the actual exchange of carbon dioxide for oxygen takes place (Fig).

Bronchial structure

The walls of the bronchi are made up of 3 layers. The outer layer consists of hyaline (bluish or transparent) cartilage rings. The lumen or inner space of the airway’s tube is covered with smooth or involuntary muscle which is, in turn, lined with respiratory epithelial or membranous tissue. Microscopic hairs on the surface of this tissue called cilia filter out dust and other inhaled particles. Moreover, the connective tissue of the smooth muscle harbors various immune cells, such as mast cells and basophils, which release histamines (chemical compounds that cause smooth muscle to contract and capillaries to dilate); it also contains goblet cells that produce mucus. A thin, protective layer of mucus covers the epithelial tissue, helping to purify the inhaled air.

During an asthma attack, mast cells and basophils release histamine and the smooth muscle of the bronchi contract or spasm, making it difficult to breathe.

What causes exercise-induced asthma?

During strenuous exercise, the body demands more oxygen and breathes faster. However, when someone with EIA exercises vigorously, a bio- and neurochemical pathway is triggered resulting in bronchospasms. While the precise mechanism governing these spasms remains unknown, 2 theories predominate; both are based on the notion that consistent and repetitive air movement can alter conditions in the bronchial tubes causing airway muscles to react. The first theory assumes that the movement of the air through the airways causes dryness that the body then combats by channeling a lot more blood into the region. This results in airway edema (swelling) and bronchospasm. The second theory assumes that the air movement decreases the temperature within the bronchi; this triggers excess blood flow in an attempt to heat the airways. As many
people breathe through their mouth when they exercise, the air they inhale is cooler than when they breathe through their nose, and this could trigger bronchospasms and an attack of EIA. Some researchers believe that a combination of these 2 theories explains an attack. There are also many other asthma triggers, including food, airborne allergens, smoke, pollution, and upper respiratory infections. Evidence, however, suggests that the cascade of events that lead to bronchospasms in people with EIA do not follow the same inflammatory pathways that lead to such spasms in people with allergic asthma.

**Preventing attacks**

The best way to treat EIA is to prevent the onset of symptoms. Patients who are prone to attacks of EIA should therefore take the time to warm up adequately before vigorous activity. If the weather is cold, a mask or scarf can be used over the mouth to reduce the effect of breathing in cold air. Alternatively, sufferers who typically breathe through their mouth while exercising should try to breathe through the nose instead so that the air is warmer. Furthermore, those with EIA should avoid outdoor activity if the pollen count or pollution level is high.

**Managing symptoms**

When an EIA attack occurs, proper management of symptoms is key. While over the counter drugs are available, these are not long-lasting and should not be used by patients who also suffer from hypertension (high blood pressure), diabetes, thyroid, or heart disease. Doctors typically prescribe medications known as beta 2 adrenergic receptor agonists or bronchodilators. These drugs cause smooth muscle to relax and the bronchial passages to dilate; some also work to stabilize the cells that release histamine. They can be short-acting or long-acting and are usually dispensed through an inhaler. Albuterol is an example of a short-acting beta agonist. It can be taken either as pretreatment 10 minutes before exercising or for quick relief during or directly after exercising. Most asthma medications, however, need to be taken up to 60 minutes before vigorous activity in order to be effective. For best results, a long-acting bronchodilator should be taken every day. Asthma medication is now also available in pill or liquid form. Some liquid medications can be used in a nebulizer or vaporizing machine that transforms the medicine into a fine mist. Strong anti-inflammatory drugs known as corticosteroids are also sometimes prescribed for EIA sufferers.

**Stay active**

Athletes and other EIA sufferers should not avoid exercise or sports. By planning ahead, doing warm up exercises, using medications as prescribed, taking extra precautions in cold weather, and avoiding allergens and pollutants, they can stay active.

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**Pelvic Fractures**

The forces required to fracture the pelvis of a person with normal bone structure are nothing short of massive. These types of forces are generally the result of high energy blunt trauma that occurs in extreme situations such as motor vehicle accidents or falls from heights. Approximately 8 to 10% of patients who sustain a blunt force and are treated at a Level 1 trauma center have a pelvic fracture. Because of the forces involved, pelvic fractures are often associated with trauma to organs and vessels inside as well as outside the bony pelvis. Due to the extensive blood supply to the region, they are also associated with hemorrhage (bleeding). For these reasons, pelvic fractures should never be considered in isolation, but rather in the context of a polytrauma or multiply injured patient. Furthermore, despite modern treatment techniques and advances in trauma care, acute pelvic fractures can be fatal. Fatality usually results from trauma to organs or surrounding blood vessels and nerves. Pelvic fractures can carry mortality rates as high as 50% if they are open (caused a break in the skin) and up to 20% if they are unstable. Fortunately, pelvic fractures represent only about 3% of the total number of skeletal injuries sustained in the US each year.

**Anatomy of the pelvis**

The pelvis supports and stabilizes both legs and the trunk of the body. The word pelvis is Latin for “basin,” and the bones of the pelvis form a basin or bowl-like structure called the pelvic ring. The enclosed space, which houses and protects the reproductive organs and rectum, is known as the pelvic cavity. This cavity is typically wider in women to accommodate pregnancy and childbirth. The large bone on either side of the pelvic ring is innominate or nameless. Each of these paired bones is formed by the fusion of 3 smaller bones—the ilium, ischium, and pubis—and connects to the sacrum (the bone in the lower back formed by 5 fused lower vertebrae) (Fig. 1) and the coccyx or tailbone (formed by a fusion of the last 4 vertebrae) (Fig. 2). The ilium is the broad upper portion of the bone where we place our hands on our hips. The ischium makes up the inferior or lower portion of the posterior pelvis; it is the bone on which we sit. At the anterior or front portion of each innominate bone is the pubis bone. Between the left and right pubis bone is a fibrocartilaginous disc that helps to make up the joint called the pubic symphysis (Fig. 1). This joint, together with the posterior sacroiliac complex (area where the pelvis joins the sacrum), stabilizes the pelvic ring. The anterior structure of the pelvis contributes approximately 40% of its overall rigidity, and the posterior approximately...
60%. The posterior portion is thus more important than the anterior to pelvic-ring stability, which makes it also more important to pelvic fracture classification.

**Diagnosis and classification**
Imaging studies are needed to accurately diagnose and classify a pelvic fracture. These can include x-rays with multiple views and stress views taken while the examiner manipulates the pelvis. A CT (computerized tomography) scan featuring multiple images of the pelvis can also be done for full assessment of the fracture pattern. A careful inspection of the entire structure must be made because following a trauma, a patient often has more than just a pelvic fracture.

Fractures of the pelvis are generally classified in 1 of 2 ways. The first is based on the stability of the pelvis and includes 3 subdivisions: 1) stable; 2) partially stable; and 3) unstable. The second, referred to as the Young-Burgess classification, is based on the mechanism of injury or the direction of the injuring force applied to the pelvis. It includes 4 subdivisions: 1) lateral compression (force is applied from the side of the pelvis); 2) anterior to posterior (force is applied from front to back); 3) vertical shear (part of the pelvis shifts upward or downward in relation to the remaining structure); and 4) a combined mechanism.

**Management**
The initial management of an unstable pelvic fracture often involves applying a sheet or pelvic binder around the patient’s pelvis to compress the area and halt any ongoing bleeding from the fracture. Once the fracture patient has been resuscitated and stabilized and the injury accurately diagnosed and classified, the goal then becomes definitive treatment. The unstable pelvis must be treated early on, not only to mobilize the patient and control pain, but also to control blood clots and chronic instability or deformity. Current pelvic fracture management employs a substantial amount of percutaneous reduction and fixation. This involves making small incisions through the skin to get the fractured bones back into the correct alignment and then fixing them in place with screws applied through these incisions. The introduction of these modern techniques and treatment options has resulted in fewer pelvic reconstructive surgeries involving large open incisions, though they are still sometimes necessary to stabilize the pelvis.

**Rehabilitation**
The type and amount of rehabilitation the patient needs largely depend on the particular fracture pattern and the fixation obtained during surgery. For stable injuries, immediate weight bearing as tolerated is often recommended, whereas for an unstable pelvic fracture, full weight bearing is often halted for at least 8 to 12 weeks after surgical treatment.

**Residual problems**
Following severe pelvic fractures, almost all patients continue to experience some degree of pelvic pain. In addition, limitations in sexual and excretory (elimination) function as well as nerve injury are common. Patients should therefore be counseled appropriately. While women who have sustained pelvic injury may still be able to give birth vaginally, the rate of cesarean, or C-section, delivery is higher than for women who have never sustained such injuries.

**Toward better outcomes**
As pelvic fractures can be fatal and often occur in tandem with other types of traumatic injuries, they should not be considered in isolation but in the context of a polytrauma patient. Consequently, although pelvic injury classification based on overall pelvic stability and the direction of the injuring force has become more standardized, it remains only a general guide to treatment. Over the past 30 years, major advances in the ability to evaluate and treat pelvic ring disruptions have led to more individualized treatment and improved outcomes.

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Reverse Total Shoulder Arthroplasty Explained

Today, there are 2 radically different kinds of total shoulder arthroplasties or replacements for individuals who suffer from severe shoulder joint pain due to arthritis or injury. While traditional shoulder arthroplasty has concentrated on replacing normal joint anatomy with plastic or metal components, the reverse total shoulder arthroplasty (RTSA) involves not only replacing joint components, but also repositioning them and altering the normal biomechanics of the shoulder. In cases where patients have sustained major muscle or tendon damage, making conventional replacement constructs more likely to fail, RTSA has become a viable solution for shoulder pain and disability. To understand how an RTSA works, a patient should first understand basic shoulder anatomy.

Shoulder anatomy

The glenohumeral, or shoulder, joint is classified as a ball-and-socket joint. It is formed by the articulation of the head (ball) of the humerus, or upper arm bone, with the glenoid cavity (socket) of the scapula (shoulder blade) (Fig. 1). Since the cavity is shallow, there is little contact between the bones, but the glenoid labrum, a ring of cartilaginous fiber that lines its circumference, deepens the cavity by about 50%, allowing for more surface contact and a better fit. Nevertheless, the joint capsule, or envelope of fibrous connective tissue that attaches to the bones of the joints to seal the joint space and to help provide stability, is very loose. This makes the glenohumeral joint the most mobile of the body, capable of flexion (bending), extension (straightening), adduction (pulling toward the body), abduction (pulling away from the body) medial and lateral rotation (turning toward or away from the midline of the body), and circumduction (moving in a circle). The capsule consists of a number of tendons (tissues that connect muscle to bone) along with bursae, or small fluid-filled sacs, strategically located to aid movement and prevent friction. Because it lacks strong ligaments (tissues that connect bone to bone) to support it, the glenohumeral joint is also known as a muscle-dependent joint. It is primarily stabilized by the biceps brachii, or muscle on the anterior (front) side of the upper arm, and the tendons of what are called the rotator cuff muscles. These include the supraspinatus, subscapularis (Fig. 2), infraspinatus, and teres minor muscles (Fig. 3). Each of these muscles originates from the scapula and has a tendon that attaches to the head of the humerus. Together they form “a cuff” around the shoulder joint, called the rotator cuff, which provides stability. This cuff is also important to shoulder movement and is frequently injured from playing sports or doing other activities involving repetitive arm and shoulder motion.

Variations on shoulder arthroplasty

In traditional shoulder replacements, a plastic cup is used for the glenoid cavity of the scapula and a metal ball for the head of the humerus (Fig. 4). Outcomes from this procedure are generally good, but when a patient with shoulder arthritis also has a massive rotator cuff tear, conventional anatomic total shoulder replacements have higher complication rates. This is because, without an intact rotator cuff to stabilize the glenohumeral joint, its centers of force shift upward, causing the arthroplasty to loosen and fail.1 The problem with patients who also have rotator cuff injuries was recognized decades ago when it led to the development of the RTSA. This new type of prosthesis or implant reversed the normal anatomic configuration of the shoulder by placing the ball component of the implant in the glenoid socket of the scapula and the socket component in the humerus. In the 1970s, early designs of this arthroplasty often failed because the implant would loosen, break, or otherwise
become unstable. Over time, the prostheses were designed to use the deltoid muscle (the rounded muscle on top of the shoulder) as a lever to compensate for the deficient rotator cuff. However, the early versions of this new design also frequently failed as the replacement constructs proved to be too constraining.

The Grammont arthroplasty
In 1985, French orthopaedist Paul Grammont advanced the design for RTSA when he came up with the idea of altering the normal biomechanics of the shoulder joint. Over the next 20 years, engineering of the Grammont RTSA evolved to provide a viable solution for patients with arthropathy (joint pain) and rotator cuff deficiency (Fig. 4). The construct functioned by converting the forces acting on the joint and, as in the previous designs, using the deltoid muscle in the absence of an intact rotator cuff as a lever so the patient could lift his or her arm. As it gave the glenohumeral articulation a larger contact surface area, the implant was more stable than earlier models and was able to prevent the humerus from shifting upward in patients with rotator cuff deficiency. Lastly, patients who were having pain from shoulder arthritis could get relief from the resurfacing provided by the stable implant. While various manufacturers have since produced prostheses that feature their own specific modifications, all of these are based on Grammont’s original design. Additionally, even though the procedure is still largely used in cases of arthropathy and rotator cuff deficiency or as revision surgery when traditional arthroplasty fails, indications for RTSA have now expanded to include some fractures of the humerus.

Outcomes and complications
RTSA offers new hope to patients suffering with shoulder pain and disability. According to doctors at Rush University Medical Center in Chicago, having this type of surgery substantially reduced shoulder pain in 9 out of 10 patients and led to a stabilized shoulder in at least 75% of cases. Despite these statistics and advancements in prosthesis design, outcomes for RTSA can vary widely and the procedure is associated with high complication rates ranging from 19 to 68% of cases. For example, scapular notching or erosion caused by the humerus or the humeral prosthesis impinging on the inferior or lower neck of the scapula, is unique to reverse total shoulder surgery. Other common problems include instability and dislocation of the prosthesis. These complications have been attributed to poor soft tissue tension, implant misplacement, an improperly-sized implant, mechanical impingement, bone loss, and neurologic dysfunction. For instance, acromial (pertaining to the protruding bone on the top of the shoulder) and scapular fractures have been reported after surgery and attributed to minor postoperative injuries. Fractures can also occur during surgery through a variety of mechanisms including bone preparation and placing instrumentation in bone that is already weak and porous. Moreover, nerve injury can result from over-lengthening the arm with the implants and overstretching nerves. Additionally, the unavoidable dead space (opening in the tissues created by the procedure itself, which allows the accumulation of blood or serum) can permit a hematoma, a solid swelling of clotted blood within the tissues, to develop. As with other joint arthroplasties, infection associated with hematomas, dead space, revision surgeries, a breach in sterile technique, or a compromised immune system, can occur. Given these possible complications, RTSA should be recommended only for select patients and performed only by experienced surgeons. Even after a successful reverse total shoulder procedure, some patients may continue to experience limitations in joint mechanics and function.

A good option for a bad shoulder
Historically, shoulder pain and arthritis have been difficult to treat in patients with a rotator cuff deficiency or following failed conventional shoulder replacement surgery. Although there can be complications, reverse total shoulder surgery addresses the problems involved directly and, to-date, offers the best treatment option available for many patients with damaged rotator cuffs.

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Reference
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