

Individual Skeletal Variation

12

12.1 Principles of skeletal variation

In Chapter 2, normal skeletal growth and development and normal anatomical features of bones and teeth were reviewed. Chapters 8–10 discussed how skeletal morphology can vary between the sexes, across geographic groups, and throughout an individual's life. Skeletal variation also exists at the individual level. As a result of different genetic and environmental influences, an individual's skeleton contains **idiosyncrasies** or individual morphological variations. These variants may be **congenital**, developmental, degenerative, or may result from disease processes or trauma. Variations in skeletal morphology can provide valuable information to forensic anthropologists, particularly for personal identification (Chapter 14). Individual variants can reveal features of an individual's health, lifestyle, or **life history**, thereby helping to further narrow the pool of potential matches. Moreover, individual variants may be rare enough to be useful for personal identification comparisons, and certain conditions (such as those requiring medical treatment) increase the chance that a record may be available that can be used for comparison. Certain skeletal variants or conditions, such as diseases or injuries that may have been fatal, may also provide information relevant to circumstances surrounding death.

It is also important for forensic anthropologists to be familiar with possible skeletal morphological variants so that they are not confused with trauma or taphonomic alterations (Chapter 13). All of the variants discussed in this chapter refer to those that result from *in vivo* processes, or those that occurred while an individual was living. Some conditions and skeletal alterations that were present during life could be mistaken for trauma, but traumas are typically associated with fractures and recently exposed internal bone surfaces, whereas individual variants reviewed in this chapter will typically have a different appearance and bone quality. A working knowledge of these individual variants combined with a conservative analytical approach should prevent or minimize any confusion of these conditions with trauma or taphonomic alterations.

Individual skeletal variations typically fall into one of four categories:

- (1) Normal anatomical variation,
- (2) Skeletal **anomalies**,
- (3) **Pathological** conditions, and
- (4) Skeletal changes related to repetitive activity.

The final conclusion as to which variant or condition is present on skeletal remains is ideally achieved through a process called **differential diagnosis**. Differential diagnosis is a deductive process of elimination used to narrow down and identify a likely condition or a small number of possibilities that cannot be excluded.

A differential diagnosis begins with a description of the variant including its location (i.e., the name of the bone or bones involved), and distribution (i.e., how much and which part or parts of the bone or bones is affected). The diagnosis then proceeds by ruling out conditions that are inconsistent with the observations, and may involve comparison with published clinical and research literature or exemplars (such as photographs, casts, or documented pathological specimens). If the condition is present on a paired bone or tooth, the suspected variant can also be compared to its normal **antimere** (the same bone or tooth from the other side) as a reference, which may help in confirming the presence or extent of the condition.

Differential diagnosis of a particular skeletal variant or condition is not always possible. In many cases, specialized methods such as radiography, microscopy, and histology may be useful. When a differential diagnosis cannot be made, a detailed description of the lesion or variant including its pattern and distribution is recommended ([Scientific Working Group for Forensic Anthropology \[SWGANTH\], 2012](#)). In any case, the analysis of skeletal variants should be amply supplemented with notes, diagrams, photos, etc., and all reasonable interpretations should be presented.

12.2 Normal skeletal variation

Normal skeletal variation refers to the range of morphological expression commonly observed in various skeletal regions. Examples of normal variation include differences in paranasal sinus shapes, cranial suture patterns, trabecular bone pattern, and external bone contours. These features and characteristics are all present in normal bone anatomy, but show small but significant differences between different people. Because these features are present in almost everyone and because they tend to show so much individual variation, they are often studied for their potential to facilitate personal identification (see Chapter 14).

Paranasal sinus shape is one example of a normal anatomical variant, with the frontal sinus being the most thoroughly studied in terms of its individual variation (e.g., [Schuller, 1921](#); [Asherson, 1965](#); [Christensen, 2005a,b](#)). To a lesser extent, the individual variation in maxillary sinuses has also been studied, particularly in relation to uses in personal identification ([Soler, 2011](#)). Because they are located within the cranial bones, the shapes of the sinuses are usually only visible radiographically. The examination of radiographs allows visualization of the dimensions, placement, and outline shape of the sinuses. Particularly in an anterior/posterior view, the variations in frontal sinus shape are quite apparent ([Figure 12.1](#)). Frontal sinuses usually appear as two irregularly shaped, asymmetric cavities that project a variable distance into the frontal bone. Although some changes in adulthood have been noted (due to, for example, trauma, disease, or bone thinning with age), they typically complete growth by around

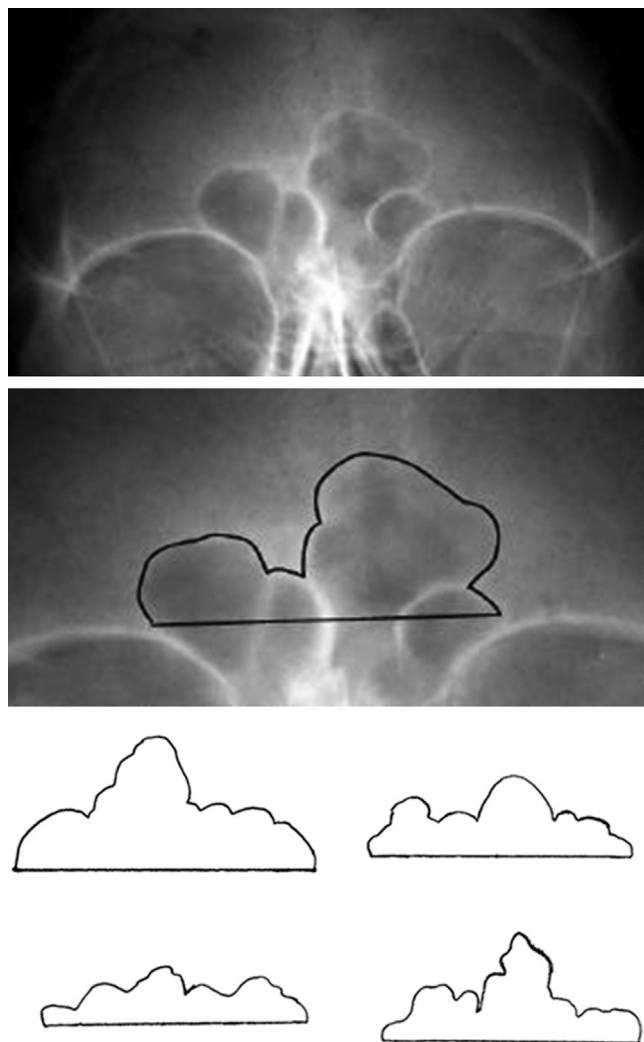


FIGURE 12.1 Examples of variation in frontal sinus shape

(From [Christensen, 2003](#))

age 20, after which their shape is stable. The variation in frontal sinus shape has been attributed to various factors including craniofacial configuration, hormonal factors, biomechanical factors, genetics, ambient air pressure, or unknown factors. It has been hypothesized that each individual's frontal sinus shape is as unique as a fingerprint.

Recall that the bones of the cranium join together along cranial sutures. During the growth process, the margins of the bones of the cranium develop projections and recesses that eventually interlock with each other, resulting in suture lines that are

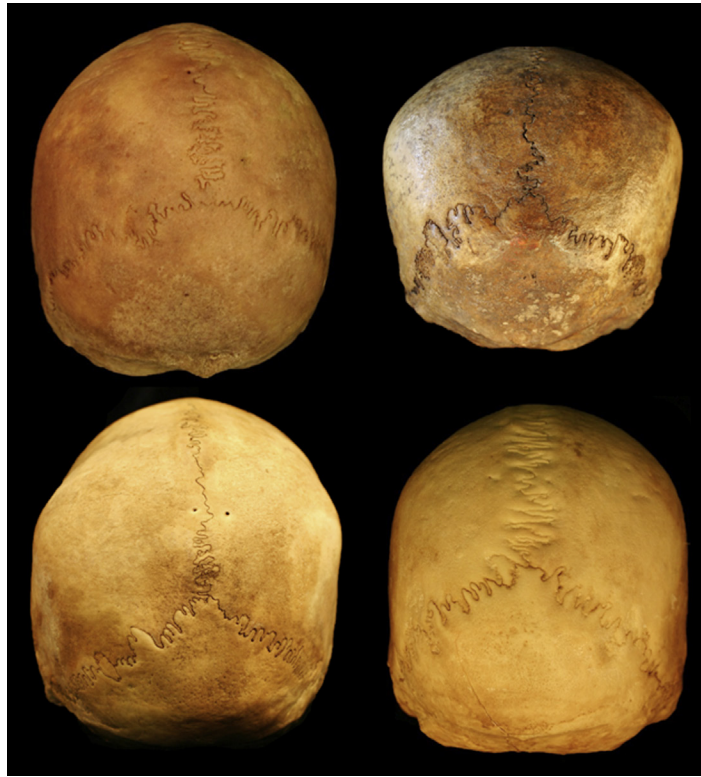


FIGURE 12.2 Differences in cranial suture patterns

jagged and seam-like on the ectocranial surface (remaining relatively straight and less remarkable on the endocranial surface). Because the growth of cranial bones is controlled largely by external stimuli (particularly neurocranial growth), cranial suture patterns are highly variable, and the inherent complexity of these projections and recesses (which are largely random) results in a nearly infinite number of possible patterns (Figure 12.2). The exact paths of the lines, therefore, vary from person to person and have been shown to be highly individualistic with no two crania having an identical pattern; in fact, bilateral suture patterns within the same individual are also different (Sekharan, 1985). It is also possible to quantify the variation in these patterns by examining the location, length, and slope of the lines of a suture's components (Rogers and Allard, 2004).

Bone's internal trabecular structure, which is visible radiographically, is also highly variable and said to be unique to each individual (Mann, 1998; Kahana et al., 1998). Normal internal bone structures have a nearly unlimited number of combinations of radiographically discernible features that show variability, including radiolucent vessel foramina and radiodense lines (Figure 12.3). Although there is a normal overall pattern to bone's external shape and contours, individual variation in these



FIGURE 12.3 Individual variants of the hand bones (white arrows) and sesamoid bone (black arrow)

surfaces has also been demonstrated. Sites studied include the clavicle and cervical spine (Stephan et al., 2011), thoracic and lumbar vertebrae (Watamaniuk and Rogers, 2010; Valenzuela, 1997), and the hyoid (Cornelison et al., 2002). Some studies have involved the examination of individual variation in multiple features such as external morphology/contour as well as trabecular pattern (e.g., Koot et al., 2005).

12.3 Anomalies

Anomalies are characteristics that are considered deviations from normal skeletal anatomy, though they may not necessarily be rare or unique. Anomalies are a product of the interaction of genetic signals and **epigenetic** interference (non-genetic factors that affect gene expression) during specific developmental events, resulting in deviations from the normal outcome (Barnes, 2012). Anomalies may be caused by disturbances from genetic mutations, maternal conditions, exposure to detrimental environmental conditions, or nutritional disorders during a particular developmental event (Barnes, 2012). These variants are also sometimes called non-metric (i.e.,

qualitative) or epigenetic variants. Examples of skeletal anomalies include **accessory** or **supernumerary** (extra, or more than the normal number) bones or teeth, accessory foramina, and non-fusion anomalies. Note that many accessory foramina and some accessory bones are technically the result of non-fusion anomalies, but certain cases will be addressed here as separate types of anomalies. An accessory facet occurs when two bones articulate in a location in addition to their normal articulation.

Accessory bones can occur in many parts of the skeleton. Examples of more commonly encountered accessory bones include having extra vertebrae, ribs, sesamoid bones, and cranial vault bones. Other “extra” bones that are technically unfused portions of parent bones will be addressed with non-fusion anomalies. Supernumerary vertebrae typically appear as transitional vertebrae at the thoracolumbar, lumbosacral, or sacrococcygeal borders, with extra vertebrae at the cervical-thoracic border being extremely rare (Barnes, 2012). When an extra vertebra is located in the thoracic region, it is typically also associated with extra ribs. In a forensic context, it may be difficult to detect the presence of a supernumerary vertebra or ribs unless the skeleton (or at least the thorax) is relatively complete. Rather than complete extra vertebrae, sometimes partial vertebrae, or **hemivertebrae**, are present, often resulting in misalignment of the spine (Figure 12.4). Sometimes a vertebra takes on characteristics of



FIGURE 12.4 Hemivertebrae in the lumbar region

(Photo by Rebecca Meeusen; specimen courtesy of the National Museum of Natural History)

another segment of the spine, a phenomenon called a **vertebral shift**. For example, a 12th thoracic vertebrae may have lumbar-like characteristics (articular facets, body shape, etc.), sometimes even resulting in the fusion of the 12th rib pair, which often appear as rudimentary structures (Barnes, 1994) (Figure 12.5).

Sesamoid bones are bones that are located within tendons where they pass over joints. The patella and the pisiform are sesamoid bones which are part of normal skeletal anatomy, but sesamoid bones can also be commonly found in other parts of the hand (particularly around the distal first metacarpal) (Figure 12.3) and the foot (often at the junction of the first metatarsal and the first proximal pedal phalanx). The number of sesamoid bones in these regions typically varies between zero and two.

Extra bones also occur in cases of accessory appendages. **Polydactyly** (or polydactylism), for example, is a condition of having extra fingers or toes. It is considered the most common congenital digital anomaly of the hand and foot, and can occur either as part of a syndrome or in isolation (Mumoli et al, 2008). Although rarely a complete and functioning digit, accessory fingers and toes often do contain bones which may occur with or without joints. They most commonly occur on the ulnar (little finger) side of the hand, but can also occur on the radial (thumb) side. Like accessory vertebrae, the identification of polydactyly in skeletonized forensic cases may be difficult unless the remains are complete, although in some cases it can be recognized by the shape of the adjacent metacarpal or metatarsal which may be broadened or bifurcated.

Another common location for accessory bones is along cranial sutures, where they are called **extrasutural bones**. These tend to occur most often along the lambdoidal suture (where they are also called *Wormian bones*), but can be found along any suture of the cranium and are typically small and irregular in appearance (Figure 12.6). Certain configurations occur regularly and have been studied more often including Inca bones, or *os incae* (so named because they were first studied in Peruvian crania), defined as a division of the squamous portion of the occipital bone (Hanihara and Ishida, 2001). Inca bones typically appear as a single, large, triangular **ossicle**, but can vary in their divisions and number of extra ossicles.

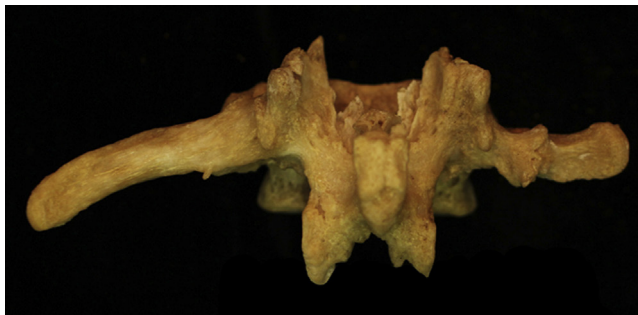


FIGURE 12.5 Vertebral shift – lumbar vertebra with fused rudimentary ribs

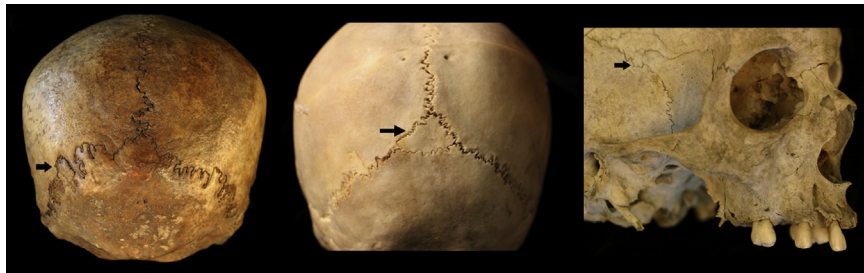


FIGURE 12.6 Extrasutural bones in the lambdoid suture, also known as a Wormian bone (left), at lambda, or Inca bone (center), and at the landmark pterion, also known as an epipteric bone (right)



FIGURE 12.7 Supernumerary teeth

(From Christensen and Anderson, 2013)

In the dentition, supernumerary teeth sometimes occur, a condition referred to as **hyperdontia** (Figure 12.7). Extra teeth can occur anywhere within the oral cavity, but are most common in the anterior maxillary dentition, and more often found in permanent than primary dentition (Garvey et al., 1999). They typically result from anomalies in dental development and although they can be asymptomatic, they often lead to clinical problems such as failure of eruption, displacement, crowding, and pathology (Garvey et al, 1999).

Accessory foramina (or extra holes in bone cortices where they typically would not be found) are seen in a number of locations throughout the skeleton including the cranium, long bones, and sternum. They are commonly the result of non-fusion or incomplete fusion. Perforations in bone can also result from trauma (such as gunshot wounds) and taphonomic agents (such as carnivore scavenging or weathering). They



FIGURE 12.8 Septal aperture in a distal humerus

(Photo by Rebecca Meeusen; specimen courtesy of the National Museum of Natural History)

should therefore always be assessed carefully to differentiate skeletal anomalies or conditions from those that result from other events or processes.

When there is incomplete ossification of the bony septum that separates the olecranon fossa from the coronoid fossa of the distal humerus, a **septal aperture** (sometimes also called a *supratrochlear foramen*) may be present (Figure 12.8). This trait tends to occur more frequently in females, more often on the left side, and may be associated with joint hypermobility (Mays, 2008; Koyun, 2011; Ndou, 2012). Although a septal aperture is expressed as an *in vivo* condition, care should be exercised when examining dry bones to ensure that a perforation in this region of typically thin bone did not result from taphonomic damage (Finnegan, 1978).

Occasionally a hole is seen in the sternum, called a **sternal foramen** (Figure 12.9). The sternum ossifies from a number of ossification centers, and a sternal foramen can result from incomplete union of any number of the sternabrae segments. It is usually seen in the inferior sternal body, but can also occur in the xiphoid process.



FIGURE 12.9 Sternal foramen

(Photo by Rebecca Meeusen; specimen courtesy of the National Museum of Natural History)

Although potentially mistaken for trauma (such as a gunshot wound) at first glance, careful inspection of a sternal aperture will reveal well-organized, mature bone and an absence of fractures around the margins.

Non-fusion anomalies result from a failure of union between two ossification centers, and can occur in virtually any part of the skeleton. The result can be a cleft or perforation in the bone, or accessory bone portions. They may simply be developmental anomalies, but may also be linked to pathological conditions including trauma. In cases where non-fusion may result in accessory bones, it is especially important that these regions are carefully assessed to differentiate them from traumas. In forensic contexts, the failure to recover these (typically small) unfused portions can make the diagnosis particularly challenging.

Cleft neural arch occurs when the two sides of the neural arch of the vertebra fail to unite ([Figure 12.10](#)). It typically affects only one or sometimes two presacral vertebrae, and can range in appearance from a slight bifurcation of the vertebral laminae to cleft laminae with or without the spinous process, and can even involve complete aplasia of one-half of the neural arch ([Barnes, 2012](#)). **Spina bifida**, a defect



FIGURE 12.10 Cleft neural arch of C1

(Image courtesy of the National Museum of Health and Medicine)



FIGURE 12.11 Spina bifida occulta

(Photo by Rebecca Meeusen; specimen courtesy of the National Museum of Natural History)

of the developing spinal cord, also disrupts neural arch development, but can be distinguished from a cleft neural arch by its widened vertebral canal, distorted pedicles, and involvement of more than two presacral vertebrae (Barnes, 2012). *Spina bifida occulta* is a less severe, often asymptomatic, form of spina bifida (Figure 12.11).

The scapula has a number of secondary ossification centers which can fail to unite, resulting in additional ossicles. The distal end of the acromion process is a



FIGURE 12.12 Metopic suture

(Image courtesy of the National Museum of Health and Medicine)

common location, resulting in a condition called **os acromiale**. A **bipartite patella**, or segmented patella, occurs when the ossification centers of the patella fail to coalesce. It usually appears as a notch in the superolateral border of the patella. The smaller, separated ossicle may eventually partially unite, may be connected only by fibrocartilage, or may fail to ossify at all.

Another type of non-fusion anomaly occurs in the cranium when parts of the cranial bones fail to coalesce prior to ossification, resulting in two ossification centers for the bone. While extra sutures can arise in the parietals or occipital, the most commonly observed location is the frontal bone. In normal growth and development, the frontal bone begins as two halves which unite along the midline. This union normally occurs by around age four (Scheuer and Black, 2000), but occasionally the two halves fail to unite, resulting in a condition called **metopism**, or the retention of a metopic suture (Figure 12.12). The suture typically has the appearance of the other normal sutures of the cranium. This feature also tends to show population differences, being more commonly seen in those of European ancestry. Facial clefts such as cleft palate are also non-fusion anomalies and tend to result in significant facial deformities.

Another type of non-union anomaly is a **pseudarthrosis**, or “false joint,” which is created when two fractured portions of a bone fail to reunite. It can result from repeated disruption of the fracture callus which impedes mineralization (Ortner, 2003). When a fracture occurs in infancy and progresses to non-union, it is referred



FIGURE 12.13 Spondylolysis of L5 with nonunion

to as a congenital or infantile pseudarthrosis. While congenital pseudarthrosis can occur in any long bone in the body, it is most frequently seen in the tibia, is typically unilateral, and will usually also involve the fibula of the same limb. It may be associated with disease, hereditary, or mechanical factors (Hefti et al., 2000).

Another example of a common non-union anomaly at a fracture site is **spondylolysis**, also known as *pars defect*. In this condition, the posterior portion of the neural arch is separated from the rest of the vertebra at the pars interarticularis (Figure 12.13). Although sometimes attributed to a congenital defect, it most often results from a fatigue fracture from low-grade stress in the lower back (Ortner, 2003). While it has been known to occur in any thoracic or lumbar vertebra, it is most common in the lower lumbar region, particularly at L5.

12.4 Pathological conditions

Pathology refers to the study of **disease**, and a **pathological condition** is the abnormal anatomy which is a manifestation of a disease process. These disease processes may be the result of infection, injury, or a disorder. Not all diseases affect the skeleton, of course, but when they do, they manifest as localized bony alterations that are

BOX 12.1 OSTEOGENESIS IMPERFECTA (OI)

Osteogenesis imperfecta (or OI) is the name for a group of pathological conditions, all of which result in osteoporosis and skeletal and dental fragility (Ortner, 2003; Waldron, 2009). The condition is uncommon, affecting approximately one in 35,000. Individuals with OI commonly suffer from skeletal fractures due to the fragile nature of their bones. Type I OI is the most common variant and predominately affects periosteal bone formation. Individuals with Type I OI often exhibit thin cancellous bone and delayed cortical remodeling. Type II OI is a more severe variant where the bones are so fragile that death often occurs during birth or childhood.

In some cases of alleged child abuse, defense attorneys have argued that the child's fractures were due to an undiagnosed form of OI rather than abuse. The simplest way to differentiate OI from trauma is through the examination of the bone histologically and evaluating the quality of the bone. The images shown below feature an infant skeleton recovered from a human rights context in Guatemala. Examination of the numerous healed bone fractures is consistent with a diagnosis of OI.



(Images courtesy of Jason Wiersema)

called **lesions**. Pathological lesions on bone may be proliferative, lytic, or deformative (Ortner, 2003). **Proliferative** (or osteoproliferative) **lesions** are those that are characterized by excess deposition of bone, while **lytic** (or osteolytic) **lesions** involve a loss of bone. **Deformative lesions** involve changes in overall bone shape.

As with other skeletal variants, pathological conditions and lesions must be examined carefully to distinguish them from other types of skeletal conditions that may be the result of traumatic or taphonomic processes (Box 12.1). Proliferative lesions



FIGURE 12.14 Proliferative lesion of the proximal tibia and fibula

(Photo by Rebecca Meeusen; specimen courtesy of the National Museum of Natural History)

are typically not confused with possible taphonomic alterations; naturally, in order to produce additional bone, the individual must have been alive. Lytic lesions, on the other hand, may require closer examination. Pathological processes and taphonomic processes can both result in destruction of bone, so caution should be exercised in distinguishing lytic lesions from taphonomic change. Likewise, bone deformation can be an *in vivo* process, but can also result from traumatic forces and taphonomic processes such as warping. **Pseudopathology** is the term used for a skeletal condition or alteration that can mimic a pathological condition. Certain pathological conditions, particularly some diseases, may provide information regarding medicolegal significance. For example, certain diseases were much more prevalent in ancient or historic contexts and are rarely seen in modern populations due to improved hygiene and healthcare.

Proliferative lesions result from increased osteoblastic activity as a reaction to a disease (Figure 12.14). They may involve a localized increase in bone density (**osteosclerosis**), or the projection of bony processes from the normal bone anatomy. Proliferative lesions that are commonly seen include those that are associated with infections of the bone, or **osteomyelitis**. Abnormal periosteal bone formation (i.e., on



FIGURE 12.15 Periostitis of the tibial shaft

(Photo by Rebecca Meeusen; specimen courtesy of the National Museum of Natural History)

the outer surface of the bone) is commonly referred to by the general term **periostitis**, meaning inflammation of the periosteum (Figure 12.15); the term *periostoses* may be more appropriate when lesions are traumatic in nature (Ortner, 2003). Inflammatory processes affecting the inner bone structures and the medullary cavity are called **osteitis**.

Some proliferative lesions are the result of ossification of other soft tissues such as cartilage and muscle. With increasing age, many cartilaginous regions have a tendency to become calcified. One such region is the costal cartilages (Figure 12.16), and as was discussed in Chapter 8, the pattern or progression of this ossification tends to vary between men and women. In some cases, especially in response to trauma, the attached connective tissue can become ossified in response to the trauma by producing bone directly in the tissue. Generally the condition is called **heterotopic**



FIGURE 12.16 Ossification of costal cartilage with fusion of first ribs to sternum

(Photo by Rebecca Meeusen; specimen courtesy of the National Museum of Natural History)

ossification (Figure 12.17), however specifically in muscle the condition is called **myositis ossificans**, and in tendons is called **ossific tendonitis**. This excess bone growth may be completely separated from the bone, or may become part of the bone, often appearing as a bony projection (Ortner, 2003).

Other proliferative lesions also take the form of bony projections. **Osteophytes** are bony projections that form at the margins of joints and signify joint damage. They are often associated with **osteoarthritis** and other degenerative joint conditions in older individuals. They are commonly found along the spine (Figure 12.18) but can be found in virtually any joint in the body (Figure 12.19). **Enthesophytes** are bony projections that form at the site of ligament or tendon attachments (Figure 12.20). Osteoarthritis can also be associated with the wearing away of joint cartilage, exposing subchondral bone and resulting in the **eburnation** of joint surfaces, a form of sclerosis that gives the bone a hard and dense quality and sometimes a shiny surface (as on the capitulum of the humerus in Figure 10.23).



FIGURE 12.17 Heterotopic ossification of the tissues surrounding the shoulder

(Photo by Christopher Rainwater; specimen courtesy of the Cleveland Museum of Natural History)

Primary **neoplasms** (or tumors) of bones are those that arise primarily in bone, and can result in prominent proliferative lesions. Osteosarcoma, for example, can form prominent bone spicules or have the appearance of cauliflower. Button osteomas are typically small benign growths found on the cranial vault ([Figure 12.21](#)). If the nerve supply to bone is damaged (often due to an injury), the lack of pain in that site can lead to continued use of the broken bone and affect healing. The resulting exuberant bony response in these cases is called a **Charcot joint** ([Ortner, 2003](#)) ([Figure 12.22](#)).

Lytic lesions involve the destruction of bone. The speed of bone destruction can affect the appearance of the lesion ([Ortner, 2003](#)). Lytic lesions that form very slowly will typically have reactive, normal density bone at their margins which have a more smooth appearance. If the pathological process destroys the bone more quickly, less



FIGURE 12.18 Osteophytosis of vertebrae

reactive bone will form and the margins of the lesion will have more sharply defined borders. These sharper borders could potentially be confused with trauma and should therefore be carefully examined.

Necrosis is a general term that refers to the death of any tissue. In bone, necrosis (also called osteonecrosis) presents as a lytic lesion often resulting from a lack of blood supply to the bone. This disrupted blood supply can result from a fracture or dislocation, especially in locations such as the shoulder, hip, and knee. The resulting condition is called **avascular necrosis** which typically appears as a collapse or destruction of the joint surface.

Secondary neoplasms (also called metastatic tumors) of bone are those that arise in other parts of the body and metastasize (spread) to the bone, and are much more common than primary bone neoplasms. In contrast with primary neoplasms of bone which tend to produce proliferative lesions, many secondary neoplasms produce lytic lesions (Ortner, 2003). Examples include osteolytic sarcoma, metastatic carcinoma (Figure 12.23), and multiple myeloma.



FIGURE 12.19 Osteoarthritis of the knee

A number of infectious diseases result in lytic bone lesions. **Brucellosis** is an infectious disease that in humans presents as a chronic infection of the lungs and associated fever. The disease also produces cavitating lytic lesions of the spine or sacroiliac joint (Figure 12.24). It is rarely seen in modern populations, and its presence may suggest that the remains are historic rather than modern in origin. Other infectious diseases including tuberculosis (Figure 12.25), leprosy, and syphilis also produce osteolytic lesions.

Osteoporosis (and its less severe manifestation, **osteopenia**) is a condition of lower than normal bone density. Although these conditions also involve a loss of bone, they are not technically considered lesions since they are not localized. Bones with osteoporosis and osteopenia retain the basic gross morphology of the bone, but simply have a lower overall bone density and typically an associated loss in bone quality. Prolonged immobility is also associated with a loss of bone density, as well as prolonged periods in low-gravity conditions of outer space, a condition referred to as **spaceflight osteopenia** (Box 12.2).

BOX 12.2 SPACEFLIGHT OSTEOPENIA

Spaceflight osteopenia is the loss of bone mass associated with prolonged exposure to the low-gravity conditions of spaceflight. The skeletal system is adapted to mechanical loading regimes that oppose gravity during normal activities such as walking, and mechanical loads play a major role in the development and maintenance of bone. Spaceflight, due to the change in gravity and weight bearing, has been shown to result in altered calcium homeostasis and bone mineral density, with the greatest losses being observed in the pelvic bones, lumbar vertebrae, and femoral neck, and the magnitude of bone loss is likely related to the duration of the flight (Turner, 2000). The precise mechanism of this loss is not well-understood, but may result from increased bone remodeling, reduced bone remodeling, or an uncoupling between bone formation and resorption. It is unclear whether these changes can have long-term consequences for bone health. Astronaut Garrett Reismann reported a 3% loss in bone density after 95 days in orbit, but states that countermeasures such as resistive exercise (pictured below) help minimize bone loss.



Astronaut Garrett Reismann engaged in resistive exercise which helps minimize bone loss in space
(Image courtesy of Garrett Reismann)



FIGURE 12.20 Enthesophyte

Some lesions involve both proliferative and lytic processes. These conditions, which normally result in proliferative periostitis, may also result in lytic necrosis if the bone in that area is deprived of blood supply and becomes necrotic. In these cases, the dead bone can become separated from the surrounding bone and is called a **sequestrum**. Hyper-developing reactive bone surrounding the sequestrum in the process of repair is called the **involucrum**. Often there is an opening in the involucrum called a **cloaca** that allows debris and pus to leave the sequestrum.

One of the more widely studied pathological conditions in skeletal remains that involves both proliferative and lytic processes is **porotic hyperostosis**, or the porous enlargement of the bone tissue, which is often associated with forms of anemia (Figure 12.26). It is commonly seen in the skull as an enlargement of the diploë, as the bone attempts to increase the available marrow space for increased red blood cell formation. Radiographically, this condition has a moth-eaten or hair-on-end appearance. When visible within the orbits, the condition is sometimes called **cribra orbitalia** (Figure 12.26).

Deformative lesions are those that involve pathologic changes in the overall bone contour or shape. Some skeletal deformations may be the result of intentional

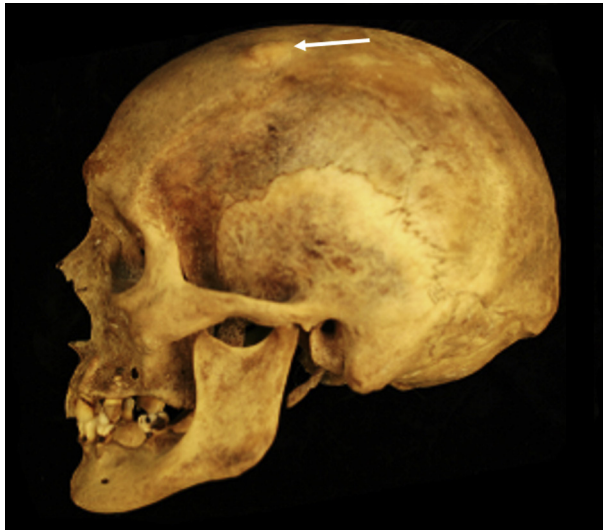


FIGURE 12.21 Button osteoma



FIGURE 12.22 Charcot joint affecting the knee

(Image courtesy of the National Museum of Health and Medicine)

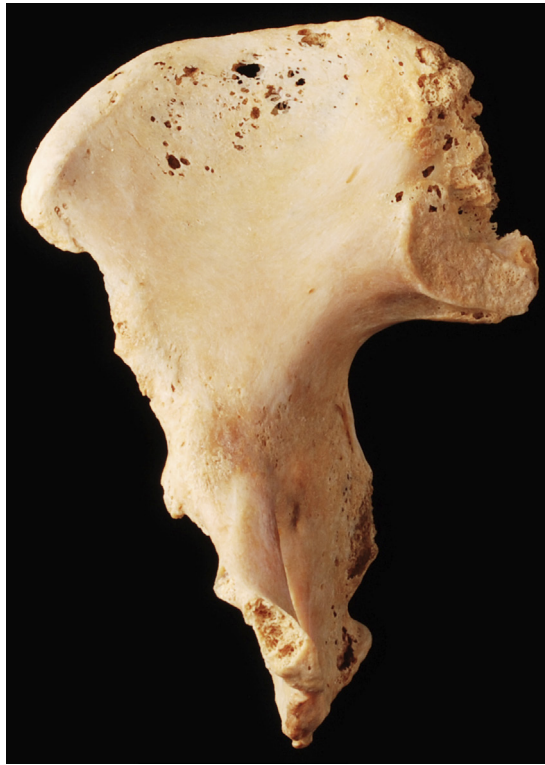


FIGURE 12.23 Metastatic carcinoma of the ilium

(Photo by Rebecca Meeusen; specimen courtesy of the National Museum of Natural History)

cultural practices and are not considered pathological. Examples include foot binding and head binding ([Box 12.3](#)). Others may be the result of trauma such as plastic deformation, or taphonomic processes such as warping.

Several skeletal deformities are recognized in the spine. **Lordosis** refers to an abnormal degree of the inward curve of the lower spine resulting in a “saddleback” appearance. **Kyphosis** is a condition of too much concave curvature of the thoracic spine resulting in a “hunchback” appearance. **Scoliosis** refers to lateral deviations of the spinal column from the midsagittal plane ([Ortner, 2003](#)). The factors leading to these deformations are varied, including compression fractures of the vertebrae, growth disturbances, or congenital defects such as hemivertebrae, a condition in which only one portion of the vertebral body develops resulting in a wedge-shaped vertebra.



FIGURE 12.24 Brucellosis lesions of the spine

(Image courtesy of Todd Fenton)



FIGURE 12.25 Tuberculosis lesions of the pubic symphysis

(Photo by Rebecca Meeusen; specimen courtesy of the National Museum of Natural History)

BOX 12.3 ARTIFICIAL CRANIAL DEFORMATION

Cranial deformation, or artificial cranial modification, is a cultural phenomenon which has been found to have been practiced on every continent except Australia ([Ortner, 2003](#)). Artificial cranial deformation is generated by applying pressure to certain areas of the cranium during the growth period, specifically the frontal, occipital, or temporoparietal. This pressure forces the bones of the vault to expand into a controlled shape. Cranial modification does not affect brain development, nor does it have any negative health consequences. Culturally, these practices were likely related to cosmetic or status ideologies.



(Photo courtesy of Diana Messer and Valerie Andrushko)



FIGURE 12.26 Porotic hyperostosis of the cranial vault (top) and cribra orbitalia (bottom)

Inadequate bone mineralization caused by insufficient calcium and phosphorus can result in a softening of the bones due to defective mineralization called **osteomalacia**. One common cause is a deficiency in vitamin D due to inadequate nutrition or any of a variety of disorders. In adults it may lead to an increase in fracture potential, deformities of the pelvis, or lordosis of the spine. In children (where the condition is called **rickets**) it can lead to significant bowing of the long bones (Figure 12.27). Poliomyelitis (polio) is a viral disease that can cause paralysis and bone deformities. Poliomyelitic paralysis affects bone growth and maintenance which can result in significant reduction in overall bone size (Figure 12.28).

Abnormalities of the development and fusion of the cranial sutures can result in the deformation of the cranium (Figure 12.29). **Craniostenosis** refers to premature fusion of the sutures of the cranium, resulting in significant cranial deformation because the normal growth of the head is altered. The alteration of cranial shape depends on which sutures are involved and the age of onset (Ortner, 2003), with the

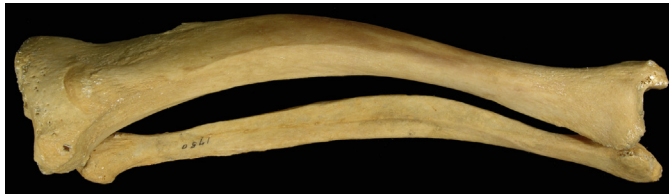


FIGURE 12.27 Rickets in a tibia and fibula

(Photo by Christopher Rainwater; specimen courtesy of the Cleveland Museum of Natural History)



FIGURE 12.28 Reduction in the size of the femur due to poliomyelitis

(Photo by Rebecca Meeusen; specimen courtesy of the National Museum of Natural History)

head typically expanding in the direction parallel to the closed suture. The condition is also often associated with altered facial features. **Hydrocephaly** is a condition involving the accumulation of fluid in the ventricles of the brain, usually causing an enlargement of the skull and a small face (Figure 12.30), and typically associated with severe neurological symptoms.



FIGURE 12.29 Craniostenosis

(Photo by Christopher Rainwater; specimen courtesy of the Cleveland Museum of Natural History)



FIGURE 12.30 Hydrocephalic skull

(Image courtesy of the National Museum of Health and Medicine)

Another form of skeletal pathology is a fracture, which may result in proliferative lesions such as calluses associated with healed fractures (Figure 12.31), lytic lesions such as necrosis from traumatic interruption of blood supply, or deformation such as misalignment from improper fracture reduction. Many of these



FIGURE 12.31 Proliferative bone and deformation associated with fracture

(Photo by Rebecca Meeusen; specimen courtesy of the National Museum of Natural History)

conditions have been addressed in previous sections of this chapter, and fracture mechanics and healing are addressed in more detail in the next chapter. In addition to lesions at a fracture site, proliferative bone often forms around the sites of surgical implants ([Figure 12.32](#)).



FIGURE 12.32 Proliferative bone growth around a surgical implant

(From Christensen and Anderson, 2013)

12.5 Repetitive activity

Repeated mechanical stresses on the skeleton can cause the bones to adapt their morphology in response to these stresses (recall Wolff's Law and the Utah Paradigm from Chapter 2). These adaptations are sometimes called "**markers of occupational stress**" or "occupational markers," in reference to their origins of often resulting from work-related physical activities. Any repetitive skeletal stresses, however, can produce changes in morphology, including those related to repetitive recreational activities or other frequent tasks or actions. Due to the many different types of activities that result in skeletal adaptations and modifications, it is not advisable to attribute any particular condition to a particular occupation in forensic anthropological casework.

One such adaptation is **hyper-development**, or the increase in size of muscle attachments or the bone's cortical area. Many studies have demonstrated an increase in the cortical area of bones that are loaded in particular ways, especially in relation to various recreational activities and sports (e.g., Bass et al., 2002; Sylvester et al., 2006). In addition, an increase in muscle size typically requires an increase in the size of the muscle attachment on the bone (larger muscles require greater surface area for attachment). For example, well-developed deltoid muscles are often associated with pronounced deltoid tuberosities of the humerus.

If an activity is performed more frequently or more intensely on one side of the body (such as with a dominant limb), asymmetry can sometimes be seen in the size or shape of paired bones, with the dominant side being larger and/or more robust. Various studies have examined the relationship between these asymmetries and handedness (for examples, see Ubelaker and Zarenko, 2012) but few have been successful



FIGURE 12.33 Central incisors worn from repeated activity

in demonstrating good predictive value for forensic casework. One reason for this is that the majority of people are right-handed, and handedness therefore has relatively little importance in forensic applications. Moreover, because of the prevalence of right-handedness, any method used to predict handedness must perform with a very high degree of accuracy and none have so far been achieved.

Teeth also show signs of repeated mechanical forces including facets, grooves, notches, and attrition. Occlusal attrition is typical with increasing age as the cusps become worn down over time from normal mastication forces. This attrition can be more pronounced if the diet contains higher grit (either from the nature of the food itself or from methods used to prepare it). Notches, grooves, and wear can occur from repeatedly holding things between the teeth. This is commonly seen in long-term pipe smokers who hold the pipe between the teeth, also known as pipe-mouth formation (Joe Hefner, personal communication, 2013), or seamstresses who hold needles between the teeth (Figure 12.33).

12.6 Case study – ankylosis

In 2012, partially mummified human remains were discovered along a river bank in northern California. The remains consisted of a complete skeleton of a White female, approximately 50–60 years of age. The skeleton exhibited a number of individual variants such as ossification of costal cartilage, and several degenerative changes of the spinal column including osteophytic lipping of vertebral centra, eburnation and osteophytic lipping of articular facets, and **ankylosis** (fusion) of two pairs of vertebrae. While the other variants of the vertebral column are



FIGURE 12.34 Ankylosis of C5 and C6 (left) and T5 and T6 (right)

relatively common among older individuals, ankylosis of vertebrae is somewhat rare. Ankylosis was identified on C5 and C6 of the cervical spine, and on T5 and T6 of the thoracic spine. Both fused pairs of vertebrae showed evidence of disc degeneration such as loss of disc height, bony bridging between vertebral bodies, and osteophyte projection beyond the normal anatomical margin (Figures 12.34). This can result from trauma such as compression fractures of the spine, which is a common finding among older females with advanced signs of osteoporosis (Ortner, 2003), or may also result from immune disorders which may be congenital. Given the decedent's age and the degenerative changes observed throughout the vertebral column, it is likely that the fused vertebrae are the result of disc collapse as opposed to a specific disease. Identification of the skeletal variants in this case was relatively straightforward, but a precise diagnosis was more complicated. In this case, the degenerative changes were an indication of relatively advanced age, and the ankylosis may be useful for identification if suitable medical records are available for comparison.

12.7 Case study – dental anomalies

In 2012, decomposed remains were recovered from along a recently flooded river bank in northern California. The remains consisted of a nearly intact skeleton of a Hispanic male, approximately 25–35 years of age. Dental anomalies were identified including “winged” incisors and a supernumerary tooth (Figure 12.35). Winged incisors are a dental feature involving a distinctive bilateral rotation of the central maxillary incisors (making the incisor junction appear V-shaped



FIGURE 12.35 “Winged” incisors and supernumerary tooth

in the occlusal view). It is most often observed in individuals of Asian and Native American ancestry. The extra tooth represented a rudimentary, non-functional tooth located distolingual to the left maxillary third molar. Supernumerary teeth that form in this location are often impacted. In this case, the tooth would likely have been considered impacted or unerupted, and would have only been visible radiographically. Dental anomalies such as these are relatively rare, and can be useful in personal identification.

12.8 Summary

- In addition to differences between sexes, across geographic groups, and throughout an individual’s lifetime, skeletal variation also exists on an individual level.
- Skeletal variation can be useful for personal identification, and it is important to be familiar with possible skeletal variants so that they are not confused with trauma or taphonomic damage.
- The main types of individual skeletal variants are those that represent variations in the shape of normal anatomy, those that are considered deviations from normal (anomalies), those that are the result of a disease process (pathological conditions), and those that result from repetitive activity.
- Variations in normal anatomy include differences in paranasal sinus shape, cranial suture pattern, trabecular pattern, and external bone contours.
- Anomalies consist of variations including supernumerary bones and teeth (such as extra vertebrae or extrasutural bones), accessory foramina (such as septal apertures or sternal apertures), and non-fusion anomalies (such as metopism or cleft neural arches).
- Pathological conditions result in localized lesions that may be proliferative (excess growth of bone), lytic (destruction of bone), or deformative (change in bone shape).

- Repetitive activities can lead to bone adaptations including increases in cortical area and density, increases in the size of muscle attachments, and the development of accessory facets.

12.9 Test yourself

- You are presented with skeletal remains that include a bone with a foramen in an unexpected (i.e., not “normal”) location. Describe the differential diagnosis process you would use to determine the likely cause of this foramen.
- Identify three sources/causes (not conditions) of variation in bone morphology.
- Explain why the detection of accessory bones can sometimes be difficult in forensic contexts.
- Describe the difference between these three types of osteoproliferative bony projections: osteophyte, enthesophyte, and myositis ossificans.
- What is the principle behind variants that result from habitual activities? Why might it be inappropriate to call these variants “occupational markers”?
- How might you be able to differentiate a traumatic fracture, a taphonomic fracture, and a rapidly formed lytic lesion?
- Why can cranial suture and paranasal sinus shape be used to facilitate identifications?
- Describe the difference between normal variation (e.g., the presence of an “Inca bone”) and pathological variation (premature cranial suture closure).

Definitions

Accessory bone An extra bone which does not occur normally

Ankylosis Fusion of two or more bones of the skeleton

Anomaly A deviation from the normal form

Antimere The opposite corresponding (left or right) part

Avascular necrosis Bone tissue death due to disruption of blood supply

Bipartite patella A condition where the ossification centers of the patella fail to coalesce; also called segmented patella

Brucellosis An infectious disease involving chronic infection of the lungs that produces cavitating lytic lesions of the spine or sacroiliac joint

Charcot joint A proliferative lesion resulting from continued use of a broken bone due to nerve supply damage and resulting lack of pain at that site

Cleft neural arch A condition where the two sides of the neural arch of the vertebra fail to unite

Cloaca An opening in an involucrum that allows debris and pus to leave a sequestrum

Congenital A defect that is present at birth, typically either arising during gestation or due to hereditary tendencies

Craniostenosis Premature fusion of the sutures of the cranium

Cribra orbitalia Porotic hyperostosis that is visible within the orbits

Deformative lesion A pathological lesion characterized by abnormal bone shape

Differential diagnosis A systematic method for narrowing down the identity of a condition

Disease An abnormal condition affecting the body including infections, injuries, and disorders

- Eburnation** A degenerative condition that results in the bone being hard and ivorylike; often associated with osteoarthritis
- Enthesophyte** A bony projection that forms at the site of ligament or tendon attachments
- Epigenetic** Non-genetic factors that cause genes to express themselves differently, resulting in different phenotypes or morphology
- Extrasutural bone** An accessory bone of a cranial suture; depending on the location and configuration, also called Wormian bone or Inca bone
- Fibrodysplasia ossificans progressiva (FOP)** A congenital disease that results in excess ossification of the connective tissues of the body
- Hemivertebrae** A condition in which only one portion of the vertebral body develops resulting in a wedge-shaped vertebra and causing spinal deformity
- Heterotopic ossification** The presence of extra, irregularly shaped bony growths typically attached to long bones
- Hydrocephaly** A condition involving the accumulation of fluid in the ventricles of the brain
- Hyper-development** Development to a high or excessive degree
- Hyperdontia** A condition of having more than the normal number of teeth
- Idiosyncrasy** A structural characteristic of a particular individual
- In vivo** A process occurring in a living organism
- Involucrum** New bone that forms around a sequestrum
- Kyphosis** Abnormal concave curvature of the thoracic spine
- Lesion** A localized alteration of a tissue, usually resulting from disease or trauma
- Life history** The history of changes that an organism undergoes throughout its lifetime
- Lordosis** Abnormal inward curve of the lower spine
- Lytic lesion** A pathological lesion characterized by abnormal loss or destruction of bone; also called osteolytic lesion
- Markers of occupational stress** Modifications to bone as a result of repetitive, work-related activity
- Metopism** Retention of a metopic suture (between the two halves of the frontal bone)
- Myositis ossificans** A condition where muscle tissue produces bone, usually in response to trauma
- Necrosis** Tissue death
- Neoplasm** An abnormal new growth of tissue or tumor; *primary* neoplasms of bone are those that arise in bone, while *secondary* neoplasms (or metastatic tumors) are those that arise in other tissues and metastasize to bone
- Os acromiale** A condition where the lateral end of the acromion process of the scapula fails to unite
- Ossicle** A small bone
- Osteitis** Inflammation involving the inner structures and medullary cavity of a bone
- Osteoarthritis** A disease of the joints characterized by degeneration of the cartilage and underlying bone, often associated with bony overgrowth; also called degenerative joint disease
- Osteogenesis imperfecta (OI)** The general name for a group of conditions which all result in pathological osteoporosis and abnormal fragility of the skeleton
- Osteomalacia** Softening of the bones due to defective mineralization, often caused by a lack of vitamin D
- Osteomyelitis** An infection of the bone or bone marrow
- Osteopenia** Low bone density that is less severe than osteoporosis

- Osteophyte** A bony projection that forms at the margins of joints
- Osteoporosis** Lower than normal bone density
- Osteosclerosis** A localized increase in bone density
- Pathological condition** Abnormal anatomy which is a manifestation of a disease process
- Pathology** The study of disease
- Periostitis** Inflammation involving the periosteal (outer) surface of a bone
- Polydactyly** A condition of having extra fingers or toes; also called polydactylism
- Porotic hyperostosis** A condition of porous enlargement of the bone tissue, often associated with anemia and commonly seen in the skull
- Proliferative lesion** A pathological lesion characterized by an abnormal excess of bone; also called osteoproliferative lesion
- Pseudarthrosis** A “false” joint that results from a failure of two fractured bone portions to reunite
- Pseudopathology** An condition or alteration that mimics a pathological condition
- Rickets** Osteomalacia in children
- Scoliosis** Lateral curvature of the spine
- Septal aperture** A foramen in the distal humerus superior to the trochlea; also called a supra-trochlear foramen
- Sequestrum** A piece of dead bone that becomes separated from the surrounding bone during the process of necrosis
- Spaceflight osteopenia** Decrease in bone density associated with prolonged exposure to low-gravity and non-weight-bearing conditions
- Spina bifida** A defect of the developing spinal cord which disrupts vertebral neural arch development, usually affecting the sacrum and numerous presacral vertebrae
- Spondylolysis** A non-union of a fracture at the pars interarticularis; also called pars defect
- Sternal foramen** A foramen occurring in either the sternal body or xyphoid process, usually a non-union anomaly
- Supernumerary** More than the normal number
- Vertebral shift** A condition where a vertebra takes on characteristics of another segment of the spine

References

- Asherson, N., 1965. *Identification by Frontal Sinus Prints: A Forensic Medical Pilot Survey*. H.K. Lewis & Co. Lts, London.
- Barnes, E., 1994. *Developmental Defects of the Axial Skeleton in Paleopathology*. University Press of Colorado, Niwot.
- Barnes, E., 2012. *Atlas of Developmental Field Anomalies of the Human Skeleton: A Paleopathology Perspective*. Wiley-Blackwell, Hoboken.
- Bass, S.L., Saxon, L., Daly, R.M., Turner, C.H., Robling, A.G., Seeman, E., Stuckert, S., 2002. The effect of mechanical loading on the size and shape of bone in per-, peri-, and postpubertal girls: A study in tennis players. *J. Bone. Miner. Res.* 17, 2274–2280.
- Christensen, A.M., 2003. *An empirical examination of frontal sinus outline variability using elliptic Fourier analysis: Implications for identification, standardization, and legal admissibility [PhD dissertation]*. The University of Tennessee, Knoxville.
- Christensen, A.M., 2005a. Testing the reliability of frontal sinus outlines in personal identification. *J. Forensic Sci.* 50 (1), 18–22.

- Christensen, A.M., 2005b. Assessing the variation in individual frontal sinus outlines. *Am. J. Phys. Anthropol.* 127 (3), 291–295.
- Christensen, A.M., Anderson, B.E., 2013. Personal identification. In: Tersigni-Tarrant, M.T., Shirley, N. (Eds.), *Forensic Anthropology: An Introduction*. CRC Press, Boca Raton, pp. 397–420.
- Cornelison, J.B., Fenton, T.W., Sauer, N.J., 2002. Comparative radiography of the lateral hyoid: A new method for human identification. *Proceedings of the 54th Annual Meeting of the American Academy of Forensic Sciences*, February 17–23, Atlanta, GA.
- Finnegan, M., 1978. Non-metric variation of the infracranial skeleton. *J. Anat.* 125 (1), 23–37.
- Garvey, M.T., Barry, H.J., Blake, M., 1999. Supernumerary teeth – an overview of classification, diagnosis and management. *J. Can. Dent. Assoc.* 65, 612–616.
- Hanihara, T., Ishida, H., 2001. Os inciae: Variation in frequency in major human population groups. *J. Anat.* 198 (2), 137–152.
- Hefti, F., Bollini, G., Dungal, P., Fixsen, J., Grill, F., Ipoolito, E., Romanus, B., Tudisco, C., Wientraub, S., 2000. Congenital pseudarthrosis of the tibia: History, etiology, classification and epidemic data. *J. Pediatr. Orthop. B.* 9 (1), 1–2.
- Kahana, T., Hiss, J., Smith, P., 1998. Quantitative assessment of trabecular bone pattern identification. *J. Forensic Sci.* 43 (6), 1144–1147.
- Koot, M.G., Sauer, N.J., Fenton, T.W., 2005. Radiographic human identification using bones of the hand: A validation study. *J. Forensic Sci.* 50 (2), 263–268.
- Koyun, N., Aydinlinglu, A., Gumrukcuoglu, F.N., 2011. Aperture in coronoid-olecranon septum: A radiologic evaluation. *Indian J. Orthopaedics* 45 (5), 392–395.
- Mann, R.W., 1998. Use of trabeculae to establish positive identification. *Forensic Sci. Int.* 98, 91–99.
- Mays, S., 2008. Septal aperture of the humerus in a mediaeval human skeletal population. *Am. J. Phys. Anthropol.* 136 (4), 432–440.
- Mumoli, N., Gandini, D., Wamala, E.K., Cei, M., 2008. Left hand polydactyly: A case report. *Cases Journal* 1, 346.
- Ndou, R., Smith, P., Gemell, R., Mohatla, O., 2012. The supratrochlear foramen of the humerus in a South African dry bone sample. *Clin. Anat.* <http://dx.doi.org/10.1002/ca.22132> (published online, not yet in print).
- Ortner, D.J., 2003. *The Identification of Pathological Conditions in Human Skeletal Remains*, second ed. Academic Press, San Diego.
- Rogers, T.L., Allard, T.T., 2004. Expert Testimony and Positive Identification of Human Remains through Cranial Suture Patterns. *J. Forensic Sci.* 49 (2), 203–207.
- Scheuer, L., Black, S., 2000. *Developmental Juvenile Osteology*. Academic Press, San Diego.
- Schuller, A., 1921. Das Röntgenogramm der Stirnhöhle: ein Hilfsmittel für die Identitätsbestimmung von Schädeln. *Monatsschrift für Ohrenheilkunde und Laryngo-Rhinologie* 5, 1617–1620.
- Scientific Working Group on Forensic Anthropology, 2012. *Identifying and Describing Pathological Conditions, Lesions and Anomalies – Revision 0*.
- Sekharan, P.C., 1985. Identification of skull from its suture pattern. *Forensic Sci. Int.* 27, 205–214.
- Soler, A., 2011. Positive identification through comparative panoramic radiography of the maxillary sinuses: A validation study. *Proceedings of the 63rd Annual Meeting of the American Academy of Forensic Sciences*, February 20–26, Chicago, IL.
- Stephan, C.N., Winburn, A.P., Christensen, A.F., Tyrrell, A.J., 2011. Skeletal identification by radiographic comparison: Blind tests of a morphoscopic method using antemortem chest radiographs. *J. Forensic Sci.* 56 (2), 320–332.

- Sylvester, A.D., Christensen, A.M., Kramer, P.A., 2006. Factors influencing osteological changes in the hands and fingers of rock climbers. *J. Anat.* 209 (5), 597–610.
- Turner, R.T., 2000. Invited review: What do we know about the effects of spaceflight on bone? *J. Appl. Phys.* 89 (2), 840–847.
- Ubelaker, D.H., Zarenko, K.M., 2012. Can handedness be determined from skeletal remains? A chronological review of the literature. *J. Forensic Sci.* 57 (6), 1421–1426.
- Valenzuela, A., 1997. Radiographic comparison of the lumbar spine for positive identification of human remains. A case report. *Am. J. Forensic Med. Pathol.* 18 (2), 215–217.
- Waldron, T., 2009. *Paleopathology*. Cambridge University Press, Cambridge.
- Watamaniuk, L., Rogers, T., 2010. Positive personal identification of human remains based on thoracic vertebral margin morphology. *J. Forensic Sci.* 55 (5), 1162–1170.