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Heterotopic Ossification about the Elbow: A Therapist's Guide to Evaluation and Management

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ABSTRACT: Heterotopic ossification (HO) is a form of pathologic bone that often occurs in the elbow after a substantial traumatic injury and can complicate the functional outcome of the affected upper extremity. This article is designed to help the treating therapist better understand the complex process of HO. The pathophysiology, causes, associated risk factors, and signs and symptoms of HO are discussed in depth. The physician's management, including a classification system, diagnostic tools, and prophylactic measures, are explained. An extensive review of the literature regarding the therapist's management of HO reveals current misconceptions about passive range of motion (PROM). Traditional thought has advocated that PROM is a contraindication when HO is present because it can lead to the development or exacerbate the formation of HO. A review of the literature only reveals a few scientific studies that concluded that forcible manipulation of stiff joints can lead to myositis ossification. Most of the articles that have concluded that PROM is contraindicated have been erroneously based on anecdotal findings. This conclusion is misleading because forcible manipulation of a joint is not synonymous with PROM exercises. This article challenges popular belief and offers some alternative thinking for the therapist treating an elbow injury with HO as well as guidelines for the rehabilitation program.

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Trauma to the elbow can be devastating and challenging to both physician and therapist as they work to restore optimum function to the patient's injured upper extremity. Severe elbow injuries can become complicated with the development of pathologic bone, often referred to as heterotopic ossification (HO), which can lead to marked stiffness and functional limitations. According to Regan and Reilly[1] there are three distinct factors that predispose the elbow to developing posttraumatic stiffness. First, an articular injury can disrupt the normal articular relationships essential for unrestrained elbow motion. Secondly, the brachialis, a large muscle covering the anterior capsule, may be torn and develop scar or pathologic bone that restricts motion. Lastly, elbow fractures or dislocations may temporarily require immobilization; this may lead to residual stiffness. The intentions of this article are to discuss the complex nature of HO, including its pathophysiology, diagnosis, associated risk factors, and medical management. In particular, an extensive literature review will serve as a foundation to reveal some current misconceptions regarding the management of HO, challenge popular belief, and offer some alternative thinking related to guidelines for the rehabilitation program.

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WHAT IS HETEROTOPIC OSSIFICATION?

Ectopic ossification refers to the formation of pathologic bone and is an umbrella term for HO, myositis ossificans (MO), and periarticular calcification. Both HO and MO represent the deposition of mature lamellar bone and share radiographic and histologic characteristics. However, the locations in which they occur are different.[2] HO develops in nonosseous tissues, while MO forms in damaged or

inflamed muscle. Periarticular calcification denotes deposits of calcium pyrophosphates that develop in soft tissues around joints, typically affecting the collateral ligaments and joint capsule. While periarticular calcification shares some histologic properties with HO and MO, radiographically it does not display the trabecular organization that is seen in both HO and MO.

There are distinct differences between normal bone and ectopic bone. In normal bone, the periosteum, covering the external surface of the bone, consists of an inner vascular cambium layer surrounded by an outer fibrous layer. Ectopic bone is not enveloped by periosteum found in normal bone.[3] Three zones have been identified microscopically in ectopic bone. The center is made up of dense cells and surrounded by a layer of osteoid. The outermost layer consists of highly organized bone. Typically, ectopic bone has a higher number of osteoblasts than normal bone and the amount of osteoclasts is nearly double that of normal bone.[4] In spite of these differences, the highly organized structure of ectopic bone is similar to that of normal bone.

The majority of pathologic bone found in the elbow after injury comprises either HO or MO (which is less common), both of which fall under the category of ectopic ossification. This topic can be confusing as many authors do not distinguish between the different types of pathologic bone and refer to all of it as HO. For the purposes of this article, the terms ectopic ossification and HO will be used synonymously (Figure 1).

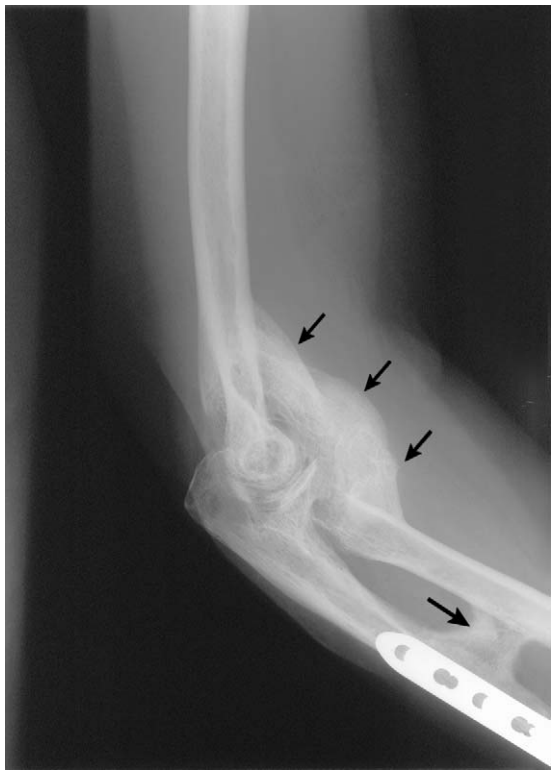

FIGURE 1. *An example of humeral–radial heterotopic ossification and radio-ulnar synostosis.*

PATHOPHYSIOLOGY

In normal bone development, pluripotential mesenchymal cells undergo migration, proliferation, and differentiation to form bone. When trauma occurs, soft tissues tear, muscle tears, and significant bleeding results. Products of the torn muscle, torn soft tissue, and bleeding have undifferentiated pluripotential mesenchymal cells that have the potential to proliferate and differentiate into bone, cartilage, muscle, or scar. It appears that there is a hormone-related mechanism that determines whether a particular cell differentiates to form bone versus scar. According to the work of Urist et al.,[5,6] bone morphogenic protein (BMP) has been identified as possibly being responsible for stimulating these mesenchymal cells to proliferate and differentiate into cartilage and bone.

This complicated physiologic process in which BMP or other similar substances interact may play a key role in the development of heterotopic bone. Major et al.[7] identified local and systemic disturbances in paraplegics, centering on the paravertebral venous plexus, that appeared to be linked to the development of heterotopic bone. Fujimori et al.[8] found BMP to play a role in the development of heterotopic bone in a mouse model and also showed the magnification of its effects with interleukin-1 treatment and collagen-induced arthritis.

Some authors working with a rat burn model have speculated on prostaglandin E_2 playing a role in the development of new bone.[9,10] Nonosteogenic cells, such as various sarcoma viruses, T-lymphocyte mitogens, and transitional epithelium from the urinary bladder, have also been associated with bone formation.[3,11]

CAUSES/ASSOCIATED RISK FACTORS

It is often difficult to determine the specific cause of HO because there are so many possible circumstances in which it can develop. The situation becomes unclear when one or more conditions are present. For example, someone with a traumatic brain injury may have a concomitant elbow dislocation. Is the development of ectopic bone a result of the neurologic insult or the elbow dislocation? It is often difficult to separate the direct cause from associated risk factors.

The most common cause of heterotopic bone formation in the elbow is direct trauma.[12] There appears to be a direct correlation between the severity of injury and the magnitude of ectopic bone that

develops. In fact, the development of heterotopic bone increases fivefold when someone has sustained an elbow dislocation along with a radial head fracture.[13]

Some associated risk factors in the development of HO include neural axis injuries and thermal injuries. The incidence of heterotopic bone appears to increase when an elbow injury is coupled with either of these associated risk factors. Garland and O'Hollaren[14] reported a notable difference in the frequency of HO at the elbow between those who sustained a neural axis injury only (5%) versus those patients who sustained both a neural axis and an elbow injury (89%). These data suggest that there is possibly a systemic cascade or hormone–related mechanism responsible for the development of HO.[15]

In the population with spinal cord injuries, the occurrence of HO, often referred to as neurogenic heterotopic ossification (NHO), appears to be related more to the degree of completeness rather than the level of injury. Several authors have noted that NHO is more frequently seen in complete transverse spinal cord injuries than in incomplete spinal cord injuries.[16–24] Additional factors seen in this population that may be associated with HO include pressure sores,[21–23,25–27] urinary tract infections or renal stones,[23,25,28–30] deep venous thrombosis,[23] and severe spasticity.[22] The exact relationship between these factors and HO remains unclear: these conditions may provoke the onset of HO, or they may occur as a result of the HO.

Heterotopic ossification in the burn population occurs at a rate of 1–3%.[31] Many agree with Hoffer et al.[32] in that the development of HO in burn patients is usually more related to the degree of thermal injury rather than the actual location of the burn. It has been documented[32] that the majority of these patients developing HO presented mostly with third-degree burns affecting more than 20% of the total body surface. This supports the opinion that the development of HO occurs as a result of the influence of systemic physiologic factors working in conjunction with local factors.[33]

The literature shows that the joint most commonly affected by HO in the burn population is the elbow.[34–37] According to Jackson,[38] the constant pressure on the elbow in a bedridden patient along with the frequent use of the elbow for leverage may in fact contribute to the development of HO. Again, it is difficult to separate the specific cause of HO in this population because there are so many factors in the burn patient that could provoke its onset. These factors include prolonged immobility, increased metabolic states, a significant elbow injury with concurrent paralysis, and an injury going undiagnosed in the midst of more life-threatening emergencies. In addition, VanLaeken et al.[33] and others[39] noted that patients with HO often showed signs of agitation and resistance to physical therapy. This agitation, seen as a forerunner in the development of HO, has been described as muscle tension, protective posturing, and a resistance to splinting or positional devices. Many believe that aggressive manipulation of these stiff joints, secondary to immobility, spasticity, or contracture, may in fact hasten the formation of heterotopic bone.[40–42]

It has been suggested that several surgery-related factors may contribute to the formation of HO. The senior author (H.H.) has observed that HO often develops in patients who have undergone repetitive surgical interventions over a short period of time. This could reflect the initial extensive soft tissue damage or the exponential deleterious effect of multiple surgical interventions. The presence of disseminated bone dust[43–50] and the formation of hematoma[47,51] have also been implicated. The length of time in surgery, along with the surgical approach and the amount of tissue dissection, has also been suggested to play a role in the predisposition of an individual to the development of HO.[48,52]

A congenital disorder that might suggest a genetic role in the development of HO is fibrodysplasia ossificans progressiva. This condition is an autosomal dominant connective tissue disorder defined by the widespread progressive ossification of soft tissues. By age 15 years, more than 90% of these patients will develop soft tissue ossifications.[53] This diagnosis is typically determined by a family history or by the occurrence of significant and widespread HO following an injury.

SIGNS AND SYMPTOMS

The onset of ectopic ossification about the elbow occurs typically two weeks after trauma, surgery, burn, or neurologic insult.[1] The development of ectopic bone may begin immediately after injury in those who are predisposed to it.[54,55] Having a medical history of developing HO, particularly after a hip surgery, predisposes one to developing HO again. In addition, a concomitant injury such as a traumatic brain injury, a spinal cord injury, or a thermal injury predisposes one to developing HO.

Early on, the elbow may display localized swelling, erythema, increased warmth, and tenderness, with patients often complaining of pain. Initially, it is impossible to differentiate normal postsurgery inflammation from the early onset of HO. However, the hallmark sign of HO is a progressive loss of range of motion (ROM) or a difficulty in regaining elbow motion at a point when the posttraumatic inflammation should be subsiding. The majority of patients affected with HO will have limited ROM, yet there are some instances when elbow ROM will be unaffected. As the ectopic ossification advances, the symptoms of

tenderness, swelling, and erythema may subside, but elbow motion continues to decrease. This loss in motion occurs even despite the intervention of dynamic and/or static progressive splinting. Radiographs at four to six weeks will usually show a subtle ''haze'' diagnostic of developing HO. Early on, the end points of flexion and extension will show a soft tissue restriction. Over time, usually three to nine months following the injury, the HO matures and the ''end feel'' can be described as rigid or abrupt, with patients usually reporting pain at the end ROM. At this time, ROM can be maintained as long as a regular exercise program is performed. A detailed discussion of therapy will be addressed later in this article.

Ectopic ossification at the elbow can also cause delayed nerve palsies, most commonly affecting the ulnar nerve, but the median and radial nerves can become compressed as well. A complete nerve lesion could result if the nerve compressions are not addressed.

COMMON SITES OF ECTOPIC OSSIFICATION

While ectopic ossification can occur anywhere in the elbow, there are several common sites in which it can develop as shown in Figure 2. Periarticular calcification is very common after elbow trauma, particularly from ligament injuries, but by itself does not lead to significant motion deficit. MO is rare but most often occurs in the brachialis muscle. Most elbow ectopic ossification leading to motion deficit occurs as HO in nonanatomic soft tissue planes.

The most frequent site in which ectopic bone develops is the posterolateral aspect of the elbow.[55] A bridge of bone typically spans from the lateral humeral condyle to the posterolateral olecranon, filling the olecranon fossa. Other common sites are the areas of the radial and ulnar collateral ligaments. Ectopic bone in the area of the radial collateral ligament can be a result of direct trauma to the lateral elbow. Ectopic bone around the ulnar collateral ligament can surround the ulnar nerve and cause a neuropathy.

Anteriorly, abundant ectopic bone can enlarge the coronoid and cause limitations in elbow flexion when it impinges on the coronoid fossa. Usually, the ectopic bone about the coronoid is in conjunction with contracture of the anterior capsule. Ectopic bone can also span from the humerus to the radius and ulna at the level of the bicipital groove, locking the elbow at 90°. As a result, the median or radial nerves could develop compression neuropathies and are at risk to injury during operative excision. In addition, a proximal radio-ulnar synostosis could occur with ectopic bone surrounding the biceps tendon. Ectopic bone can also be seen in the vicinity of the brachialis, biceps brachii, and the anterior capsule. It is important to note that ectopic bone usually does not follow anatomic planes and can present itself in a variety of locations.

CLASSIFICATION OF HO

Heterotopic ossification can be classified by its anatomic location and/or its resultant effect on ROM. The system developed by Hastings and Graham[55] classifies ectopic ossification according to how it affects functional ROM.

- Class I includes patients who have HO but display no functional ROM limitations.
- Class II includes patients having HO with limitations in functional ROM: elbow flexion/extension and/or supination/pronation. This class is subdivided into three categories depending on which plane(s) of motion is affected.
- Class III includes patients who have HO with ankylosis present, preventing elbow flexion/extension

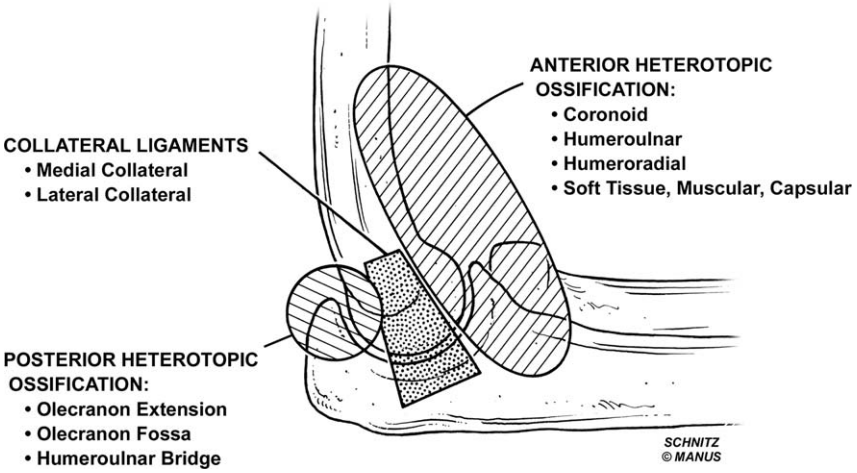

FIGURE 2. *Common sites of ectopic ossification.*

and/or supination/pronation. Again, this category is subdivided according to which plane(s) of motion is affected.

DIAGNOSTIC TOOLS

A laboratory assessment can be performed to check the levels of serum alkaline phosphatase and determine the activity of ectopic ossification. Because there are debates as to how sensitive this laboratory test is to HO, many feel that this is not the preferred diagnostic tool and that there are other more effective ways to diagnose HO. We have not found laboratory tests useful.

The diagnosis of ectopic ossification can be confirmed by a plain radiograph, which reveals both the location and maturity of pathologic bone. HO is typically seen by six weeks after injury on film but can be identified as early as two weeks after an injury.[53] HO is typically found in the area where trauma has occurred. Early on, the HO appears hazy and does not display obvious boundaries or any trabeculation. However, as it matures, the margins become more distinct and trabeculation is present.

Computed tomography or lateral trispiral tomograms can identify the definite location of ectopic ossification. This diagnostic tool is superior to a plain radiograph because it reveals the complex architecture of the articular surfaces, specifically along the ulnotrochlear and proximal radio-ulnar joints.

Ultrasound has been documented as being helpful in diagnosing HO about the hip[56] and may be beneficial in diagnosing at the elbow. Bone scans and magnetic resonance imaging (MRI) have also been mentioned in the literature as diagnostic tools but, early on, may not be specific and diagnostic of HO. Positive changes seen on an MRI or a bone scan may be due to posttraumatic inflammation that does not progress to HO. Plain radiographs are less expensive and equally diagnostic. We do not typically use a bone scan or an MRI when a diagnosis of ectopic ossification is suspected.

PROPHYLACTIC TREATMENT

Prophylactic measures can be taken to prevent the formation of ectopic bone at the elbow, in cluding pharmacologic and radiation interventions. Prophylactic treatment is recommended for patients if they have sustained a massive elbow injury or an elbow injury combined with one or more additional risk factors. These risk factors, several of which were mentioned earlier, include neurologic injury, burns, a previous history of HO, diffuse idiopathic skeletal hypertosis, hypertrophic osteoarthritis in men, ankylosis spondylitis, and Paget's disease.[53] There are two forms of prophylactic treatments currently available.

Two chemotherapeutic agents used to prevent the development of ectopic ossification are diphosphonates and nonsteroidal anti-inflammatory drugs (NSAIDs). The diphosphonates inhibit osteoid cells from calcifying and are effective in preventing heterotopic bone formation. However, once they are discontinued, the osteoid cells will begin to calcify. Diphosphonates are not the preferred choice for prophylaxis because of this phenomenon and because of the side effects of gastrointestinal disturbances and osteomalacia that can occur. It has been documented in the literature that NSAIDs have been effective in reducing the frequency and the magnitude of ectopic bone about the hip. Unfortunately, no studies have looked at the effect of NSAIDs on ectopic ossification about the elbow. The most commonly used NSAID is indomethacin. Two studies demonstrated that indomethacin inhibits precursor cells (undifferentiated cells) from differentiating into osteoblasts.[57,58] However, indomethacin has also been shown to inhibit bone formation and fracture healing. We recommend and routinely prescribe an oral dosage of 75 mg twice a day along with an oral dosage of 1 g of sucralfate four times a day after excision of heterotopic bone. This dosage is given to patients until three weeks postsurgery. We have not seen problems with fracture healing despite theoretical concerns that it could inhibit fracture repair.

Low-dose external beam radiation is the other form of prophylaxis that can be used with or without NSAIDs. The literature reports that this type of radiation has been effective in preventing the development of ectopic bone after total hip arthroplasty.[59–61] Moreover, Tonna and Cronkite[62] demonstrated that the low-dose external beam radiation prevents cell proliferation and the growth of bone in the rat femur model. Studies by Abrams et al.[45] and McAuliffe and Wolfson[63] reported the use of low-dose external beam radiation to prevent ectopic ossification from recurring after its resection at the elbow. While there was no recurrence documented in these studies, it is not apparent if these patients were indeed at risk for having ectopic ossification recur. Jupiter and Ring[64] noted that the patients in their study, who underwent resection of a proximal radio-ulnar synostosis, did not show any recurrence of ectopic bone. Interestingly, none of those patients received any postoperative prophylactic treatment. If low-dose radiation is chosen as a prophylactic measure, we recommend a dosage of 600 cGy given within 72 hours of elbow trauma or elbow surgery. For elective procedures, radiation is given preoperatively on the day of surgery. The radiation oncologists who treat our patients will simulate the radiation portals with real-time fluoroscopy to ensure that the

radiation is delivered to the proper anatomic location of the elbow.

MYTHS, FACTS, AND UNCERTAINTIES

Controversy exists in the literature as to what constitutes the most appropriate, optimal therapy program when a patient presents with HO or when it is suspected. Historically, therapists and surgeons have felt that passive range of motion (PROM) is contraindicated when HO is suspected or present because it can cause or exacerbate HO formation. After an extensive review of the literature, only three scientific studies were found.[41,65,66] All of them were performed on rabbits and analyzed the effect of daily forcible passive motion on immobilized joints. Results revealed that heterotopic bone, more specifically myositis ossification, was induced by this forcible passive motion. In two of their studies, Michelsson et al.[41,65] concluded that ''joints should be exercised very carefully during and after an immobilization period.'' Several authors have referred to these studies and concluded that PROM is contraindicated because it could lead to the development of HO. This conclusion is misleading as PROM performed by a therapist is not necessarily synonymous with ''forcible passive movements.''

A retrospective study by Thompson and Garcia[13] that is commonly cited concluded that ''passive motion during convalescence should never be used.'' Their study looked at a large group of patients and noted that those who received passive stretching to the elbow either by the therapist or from weights developed myositis ossification. Interestingly, those patients who received that kind of therapy had stiff elbows and needed an intensive passive stretch, whereas those who did not have stiff elbows did not need passive stretching. Again, it seems mistaken to conclude that the passive stretching led to the development of MO. Perhaps those elbows were stiff as a result of the traumatic injury, and the MO occurred because of the initial trauma, not the passive stretching. Several other articles[31,34,37,67–71] stating that PROM is a contraindication to HO are merely anecdotal. The statements made supporting this concept are merely assumptions made from previous authors' work, as mentioned above, or from observations made in their own practice. No well-designed scientific studies are present in these articles. Unfortunately, many physicians and therapists have based their rehabilitation guidelines on these anecdotal articles and poorly designed studies.

On the other hand, there are several articles in the literature that advocate PROM exercises as part of the therapy program even if HO is present. Stover et al.[72] reported a prospective study evaluating if the presence of an aggressive PROM program aggravated the development of HO. Their results revealed that there was no obvious difference in HO in those who received aggressive ROM exercises and those who received minimal, if any, therapy on the affected injury. A retrospective study conducted by Wharton and Morgan[73] reported that ROM did not lead to the formation, or increase the severity, of HO. Their study showed that those patients who received passive stretching did not present with a more significant amount of HO than those patients whose PROM exercises were discontinued. In fact, those patients who no longer received passive stretching quickly lost joint motion and ankylosis developed.

In their discussions of HO, several authors[74,75] recommend early and regular exercises, while Damanski advocates ''frequent passive movements,''[25] to prevent the shortening of muscles, soft tissue contractures, and joint stiffness. Linan et al.[76] documented a case study using a continuous passive machine (CPM) on a gentleman with a traumatic brain injury to restore bilateral knee motion. Despite the presence of HO, the CPM was initiated and helped recapture knee ROM. After six weeks of using it, plain radiograph revealed no change in the HO. In another study,[77] the authors induced heterotopic bone in the quadriceps muscles of rabbits and evaluated the effect of a CPM. Their study revealed that the use of a CPM did not aggravate the formation of bone in the muscle-injured rabbits.

Another case study[78] documented a gentleman with a closed head injury who presented with a significant flexion contracture of his elbow. Heterotopic bone was also present in the elbow. The use of serial casting was employed to restore elbow ROM. Functional ROM was achieved and the authors described the heterotopic ossification as ''stable.''

In summary, forced manipulation of stiff or contracted joints may lead to muscle tears and ossification within the muscle (MO), which is typically not seen about the elbow after trauma or therapy. There is essentially no scientific evidence that controlled ROM exercises or splinting causes HO about the elbow (as noted in a literature search from 1982 to the present using CINAHL and from 1966 to the present using Medline). Both active and PROM exercises, along with static progressive splinting, should be continued in the face of developing or evident HO.

THE THERAPY PROGRAM

It is important to design an effective therapy program when treating a patient who has sustained a traumatic elbow injury. The rehabilitation program can be divided into several phases with specific goals and recommended guidelines in each phase.

Acute and Edematous Phase (First Two Weeks following Injury/Surgery)

During this initial phase, it is important to apply proper edema control measures to reduce postinjury and/or postsurgery inflammation. Bleeding occurring from the initial injury and/or surgical intervention can cause significant swelling of the tissues. This edema leads to the development of scar, and minimizing edema with an effective compressive dressing will minimize scar formation. In addition, pain management is essential to allow for maximum participation in the therapy program. ROM exercises should be initiated to the elbow and forearm within the parameters of the physician as determined by the stability of the injury, with particular emphasis on regular exercise and active ROM as muscles quickly lose strength after an injury and a period of non-use.

Inflammatory Phase (Two to Six Weeks following Injury/Surgery)

Prolific unorganized scar tissue is present during this phase, which is very active yet malleable, and its formation can be influenced by therapeutic measures. The greatest potential for ROM gains exists during this phase as the scar is deformable and will respond to therapy modalities. If full PROM is permitted, self-passive stretching should be emphasized along with the use of weighted stretches and/or dynamic/static progressive splinting. It is during this phase that splinting to recapture ROM will be most effective. We stress the concept of a low-load progressive stretch[79] and oftentimes use static progressive splinting as an adjunct to restore elbow ROM. We recommend that patients wear this splint (or a dynamic splint if more of a soft end feel is present) four to six times a day for 30–45 minutes at a time. If elbow extension is limited, a nighttime static splint is fabricated to the patient's maximum extension and then serially adjusted to accommodate for gains in motion. When swelling has subsided, moist heat can be used prior to stretching exercises or the wearing of a splint to increase the elasticity of the tissues and maximize motion. If elbow ROM is not improving despite consistent splinting or if progressive losses are noted, then the treating physician should be consulted. Even if HO is revealed on plain radiographs, usually seen at four to six weeks, the patient should continue with his/her current therapy program to maximize ROM. Patients should continue to wear the dynamic or static progressive splints frequently throughout the day, as pain allows, emphasizing the most limited direction. It is important to keep in mind the goals of functional ROM. According to a study by Morrey and coworkers[80,81] an arc of 100° of elbow motion, from 30° of extension to 130° of flexion, is required for a patient to perform 90% of his or her normal daily activities. The patient's therapy program should also include a strengthening program, as advised by the treating physician. Resistive exercises will not only help the muscles regain strength, which is often quickly lost after an injury, but also maximize ROM gains. Improved muscle power can work through passive resistance to increase ROM. In addition, it is important to encourage functional use of the affected extremity to help restore elbow motion and strength.

Fibrotic Phase (Six to 12 Weeks following Injury/Surgery)

During this phase, the scar tissue is usually fully formed but is reorganizing and will continue to respond to motion and stress. If aggressive splinting has not already been initiated, it can be added to the therapy program as fractures are typically fully healed at this time. Of course, the treating physician dictates the intensity of the therapy program. It is important for the patient to wear splints regularly throughout the day to maximize the amount of prolonged stretching to the tissues. Having the patient perform resistive exercises following the wearing of a splint will help to maximize ROM gains. It is important to note that gains in ROM will become more difficult to achieve with an increasing passage of time.

Late Phase (Three to Six Months following Injury/Surgery)

During this phase, the scar is organized and fibrous tissue is present. Only mild or modest gains will be achieved at this time. Splinting to recapture ROM can continue as long as gains are noted. Patients eventually discontinue splints when either ROM goals have been achieved or ROM has reached a plateau. Typically, patients will wean from splints as long as motion is maintained instead of discontinuing them all at once. It is recommended that patients continue on a home strengthening program for a minimum of six months postinjury or postsurgery to maintain ROM and build upper extremity strength.

INDICATIONS FOR SURGICAL INTERVENTION

Surgical excision is not warranted by the mere presence of HO or a limitation in elbow motion. It is only necessary or appropriate when the limited elbow ROM prevents functional use of the affected upper extremity.

CASE STUDY

A 70-year-old male fell and sustained an intra-articular T-condylar fracture of the nondominant distal humerus. He was referred and initially seen ten days after injury. An exam revealed severe swelling and echymosis. A painful limited arc of motion was present between 50° and 70° of flexion. No heterotopic bone was present on plain radiograph. Due to his mild arthrosis and the distal nature of his fracture, along with his advanced age and low demand of activity, he was treated by replacement arthroplasty, with a triceps sparing approach, and an ulnar nerve transposition. Full ROM was achieved in the operating room: elbow extension/flexion 0°/135°, supination 80°, and pronation 80°. The patient's elbow was placed in a postoperative dressing at 40° of flexion to take tension off the skin incision. Given the patient's history of a previous olecranon fracture and the fact that he sustained a severe traumatic injury to his elbow, he was given a single dose of prophylactic radiation (600 cGy).

Therapy began at day 7 following surgery with active and passive ROM exercises to the elbow and forearm and an interval elbow extension splint. Initial ROM was as follows: elbow extension/flexion 40°/60°, supination 60°, and pronation 40°. At ten days postoperatively, early HO formation was evident on plain follow-up radiographs, as shown in Figure 3. Despite the presence of radiographic HO, therapy was continued with both active and passive ROM exercises. At three weeks postoperatively, the patient was struggling to regain flexion (100° of passive flexion present) so a dynamic elbow flexion splint, approved by the treating physician, was added to the program. Elbow flexion was emphasized over extension for functional purposes. Changes in the home program were made accordingly, with the patient wearing the dynamic elbow flexion splint twice as much as the static elbow extension splint. At six weeks postoperatively, resistive biceps and triceps exercises, starting with 2-lb weights and then progressing as tolerated, were initiated to increase elbow strength and maximize ROM.

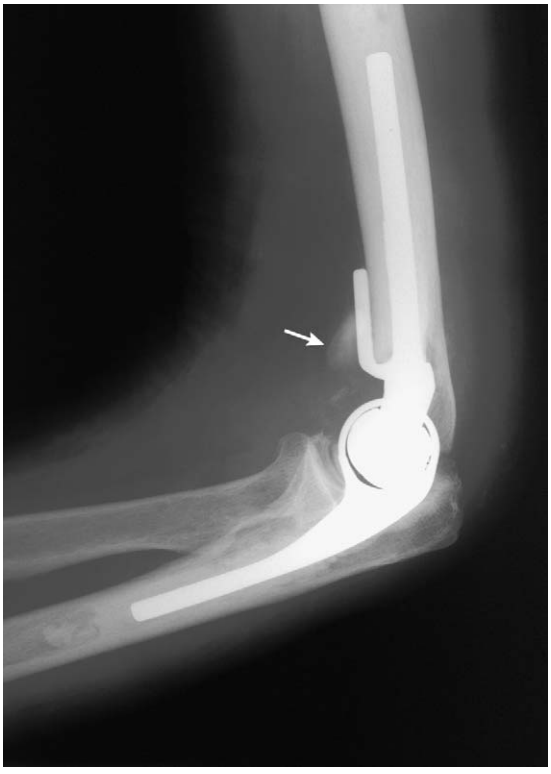

FIGURE 3. *Initial finding of anterior heterotopic ossification at ten days after total elbow replacement.*

While elbow flexion was progressing, elbow extension remained limited. Accordingly, at nine and a half weeks postoperatively, a static progressive elbow extension splint was fabricated in an attempt to restore greater extension. The patient's elbow ROM eventually reached a plateau, and at 13 weeks postoperatively, all splints were discontinued and normal use, along with a continued strengthening program, was recommended. On his final visit, the patient reported satisfaction with his ROM: elbow flexion/extension 30°/120°, supination 85°, and pronation 75° (Figures 4 and 5). The patient was able to perform almost all functional activities with ease (see Figures 6–9).

During the course of his treatment, postoperative edema decreased each successive week and the patient did not complain of significant pain. The extent of HO at six months postsurgery as compared to its initial presence on plain radiograph was essentially unchanged (Figure 10).

CONCLUSIONS

Heterotopic ossification is a complex process that can occur after a traumatic injury. The extent of its formation appears to be directly correlated to the severity of injury. Therefore, it is probable that someone who sustains a massive traumatic injury is likely to develop HO. In addition, those who have sustained neural axis injuries or thermal injuries are particularly predisposed to developing HO, especially if a concomitant elbow injury is present. Why certain cells differentiate into bone instead of scar tissue after an injury remains unclear, and more research is needed to better understand this process. As the true pathophysiology of HO becomes better understood, the means to prevent or modify its formation may ultimately be available.

The formation of HO typically begins within the first two weeks of trauma. Initially, it is barely visible on plain radiographs. The ossification process progresses over the next several months and matures anywhere from three to nine months postinjury or

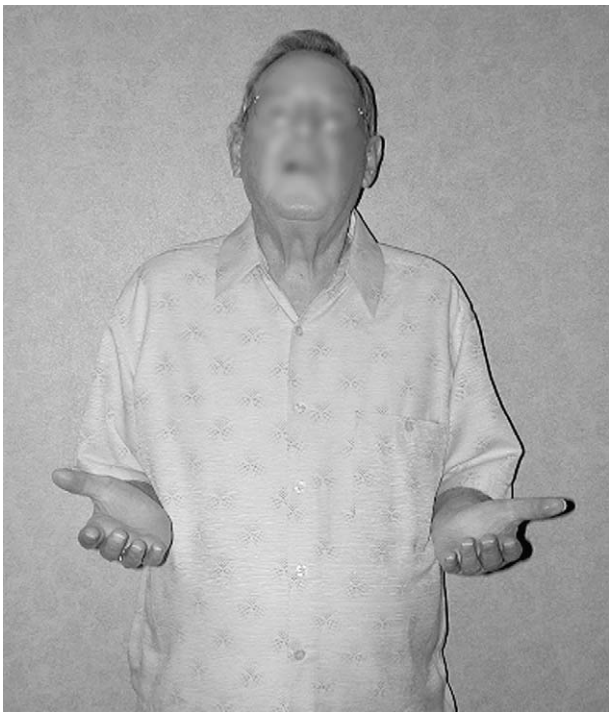

FIGURE 4. *Case study: patient's supination at six months postoperatively.*

postsurgery. Therapy is initiated during this same time frame, and the patient will usually participate in a comprehensive and intensive therapy program to

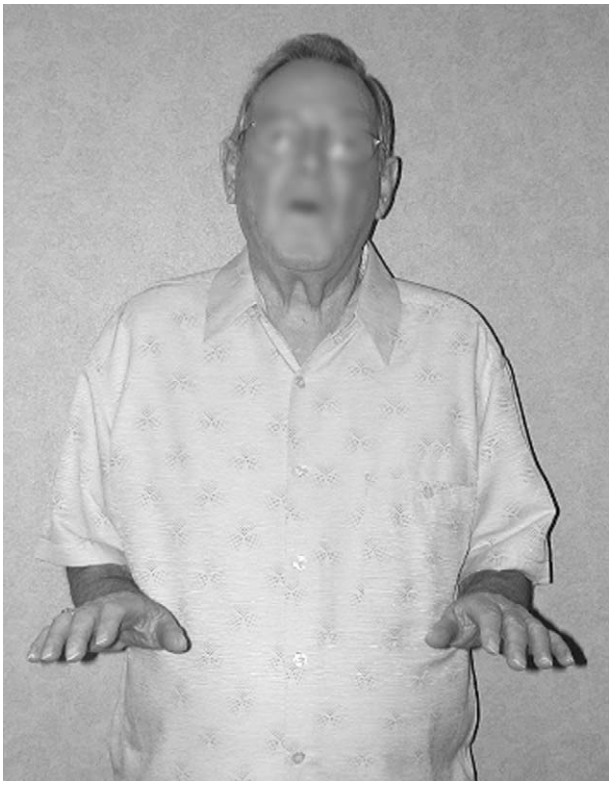

FIGURE 5. *Case study: patient's pronation at six months postoperatively.*

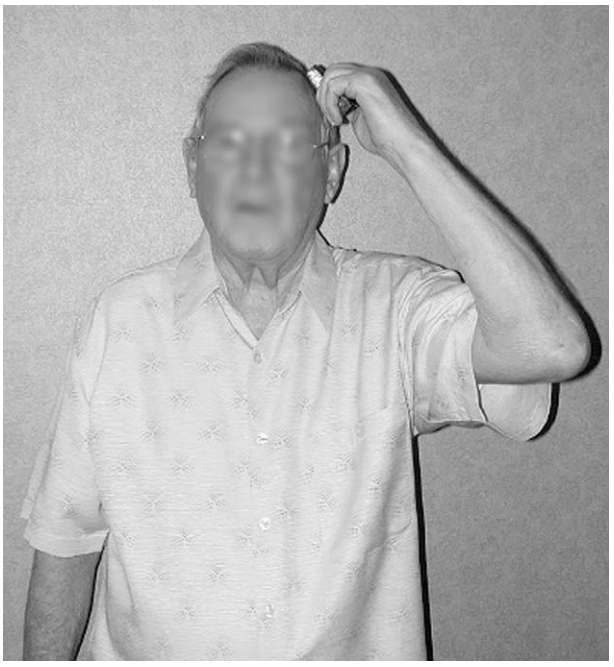

FIGURE 6. *Case study: elbow flexion demonstrating the patient's ability to comb his hair.*

recapture motion and restore function. It is inaccurate to assume that therapy is causing the ossification to occur and mature. The two just happen to occur during the same time. While manipulation of stiff joints has been associated with formation of MO, almost all posttraumatic ectopic bone about the elbow clinically is seen as HO rather than MO.

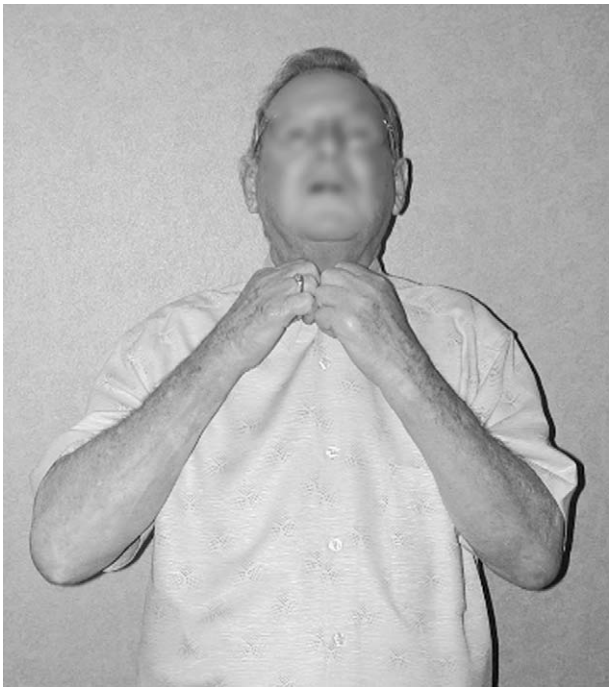

FIGURE 7. *Case study: elbow flexion demonstrating the patient's ability to button his shirt.*

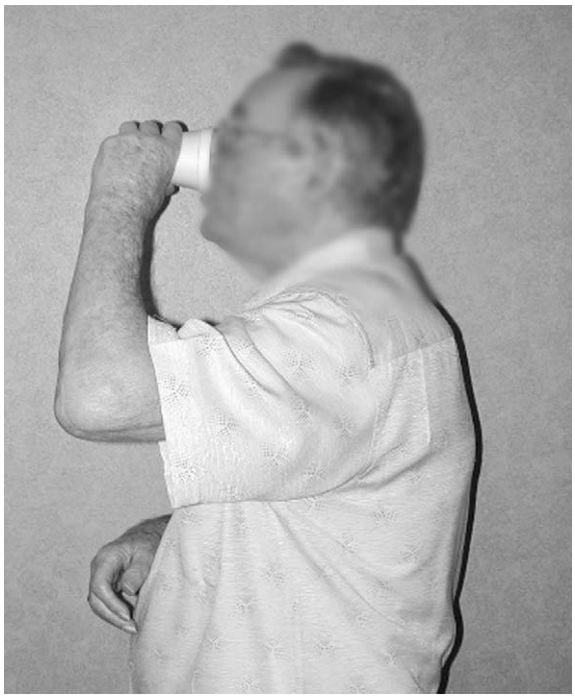

FIGURE 8. *Case study: elbow flexion demonstrating the patient's ability to drink from a cup.*

There is no solid evidence in the literature that shows that therapy, specifically PROM, causes or exacerbates HO. Until a scientific prospective study is performed that proves PROM causes HO, those patients who present with HO should participate in a comprehensive therapy program as outlined by the treating physician. Having the patient perform full active and passive ROM exercises, along with wearing splints as pain allows, will maximize ROM, restore function, and minimize the potential for ankylosis to develop.

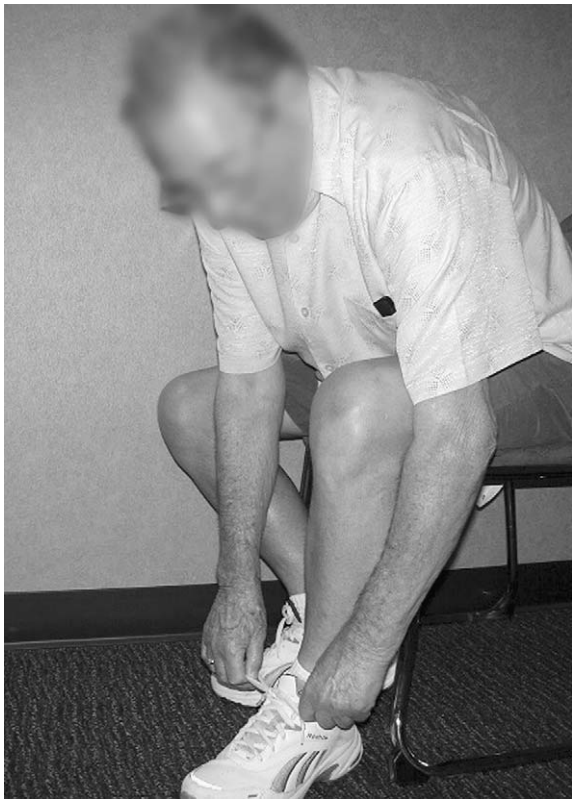

FIGURE 9. *Case study: elbow extension demonstrating the patient's ability to tie his shoe.*

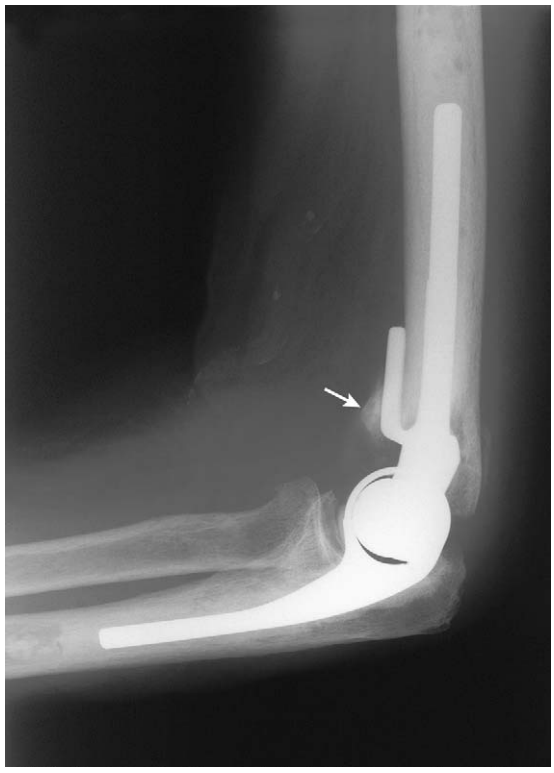

FIGURE 10. *Extent of anterior heterotopic ossification at six months postoperatively has not progressed from its initial size.*

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Quiz: Article #033

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#1. A common misconception is that HO can likely be caused by:
   a. forceful manipulation of the elbow
   b. trauma, bleeding, and pathological bone formation
   c. blunt trauma to the anterior elbow
   d. PROM of the elbow

#2. The primary pathogenesis of ectopic bone is thought to be a disturbance in:
   a. the mechanical response to motion after trauma
   b. proprioception within the anterior joint capsule
   c. the differentiation of pluripotential mesenchymal cells
   d. resorption of any hematoma in the brachialis

#3. There appears to be a correlation between the magnitude of HO formation and:
   a. the age of the patient
   b. the length of time of immobilization
   c. the patient's pre-injury blood pressure
   d. the severity of the injury

#4. The hallmark of HO is:
   a. gains in extension but loss of flexion
   b. progressive loss of ROM when the posttraumatic inflammation should be subsiding
   c. posterior joint tenderness
   d. absence of any x-ray changes

#5. The rehab program for HO is primarily focused on:
   a. strengthening
   b. ROM
   c. functional activities
   d. pain management

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